Why Does C5 Palsy Occur After Prophylactic Bilateral C4-5 Foraminotomy in Open-Door Cervical Laminoplasty? A Risk Factor Analysis

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Abstract

Study Design: Retrospective study.

Objectives: To evaluate the efficacy of bilateral C4-5 foraminotomy in preventing occurrence of postoperative C5 palsy and to identify possible risk factors for its development.

Methods: A total of 70 consecutive patients who underwent open-door laminoplasty with bilateral C4-5 foraminotomy were included. Clinical, radiographic, and operative data was reviewed. Development of postoperative C5 palsy was analyzed.

Results: A total of 54 males and 16 females were reviewed. Mean age was 56 years (range, 30-86 years). The primary pathology was spondylosis in 76% of cases and ossified posterior longitudinal ligament in 21%. Radiographic evidence of C4-5 foraminal stenosis was seen in 81% of the patients. The mean duration of preoperative symptoms was 7 ± 19 months. Four (5.7%) out of 70 patients developed C5 palsy after open-door laminoplasty with bilateral C4-5 foraminotomy. Multivariate analysis showed that a long duration of preoperative symptoms (>12 months) and the presence of preoperative C4-5 T2-MRI cord signal change were statistically significant risk factors for the development of C5 palsy even after bilateral C4-5 foraminotomy in open-door laminoplasty (P < .0001 and P = .036, respectively).

Conclusions: Prophylactic bilateral C4-5 foraminotomies do not completely eliminate the occurrence of C5 palsy. Prolonged duration of symptoms and presence of preoperative T2-MRI cord signal change increase the risk for developing postoperative C5 palsy despite foraminotomy.

Keywords
open-door laminoplasty, foraminotomy, cervical complications, C5 palsy, MRI cord signal change, cervical myelopathy

C5 palsy is a well-recognized complication known to occur after cervical laminoplasty.1-16 It is defined as the new onset of deltoid muscle paralysis after a neuro-decompression procedure.3,17-20 The incidence of C5 palsy reported after laminoplasty without concomitant nerve root decompression ranges from 5% to 17%.1,2,6,11,21-23 With 92% of the C5 palsy occurring unilaterally.3 Majority of the motor symptoms occur within the first week after the operation1,3,4,11,12,24 and are expected to resolve by 12 months.1,8,11,12,23 Permanent deficit has been reported at 10-year follow-up in up to 30% of C5 palsy in 1 case series.1 While bilateral foraminotomy performed at the time of laminoplasty may prevent the development of C5 palsy,3,12,17,20,22,25,26 little has been published on the possible clinical efficacy of bilateral foraminotomy as a prophylactic measure to prevent postlaminoplasty C5 palsy.27,28 The aim of this study was to examine whether bilateral C4-5 foraminotomy performed at the time of open-door laminoplasty will prevent the development of postoperative C5 palsy, and to identify possible risk factors for its development.

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Materials and Methods

A retrospective review of all patients who had undergone open-door cervical laminoplasty with bilateral C4-5 foraminotomy was carried out. All surgeries were performed by the senior author (K.D.R.) and were reviewed by an independent spine surgeon. Clinical, radiographic, and operative reports were examined. Patient clinical data was recorded with attention to the patients’ age and gender, duration of symptoms, neurologic deficits, pathology, and diagnosis. Patient neurological status was described according to Nurick score pre- and postoperatively. Details of the surgery, including number of laminoplasty levels, site of bilateral foraminotomy performed, laminoplasty hinge location, operative time, and blood loss were documented. Radiographs were reviewed for the presence of preoperative C4-5 foraminal stenosis, T2-weighted magnetic resonance imaging (T2-MRI) cord signal change, spinal cord flattening, and pre- and postoperative cervical lordosis. Time to resolution of the C5 palsy symptoms was also recorded. Fischer’s exact test and general linear model analyses were used to identify potential risk factors for C5 palsy development after prophylactic bilateral C4-5 foraminotomy in open-door laminoplasty.

Laminoplasty and Foraminotomy Surgical Technique

Details of the senior author’s (K.D.R.) surgical techniques have been published and a brief summary of the critical surgical steps is described below.

Bivector skull traction via Gardner-Wells tong is used to allow easy interchange of intraoperative neck alignment. Neck flexion is achieved through a vector connecting the tong via a rope placed “in-line” along the operating table to a 15-pound single pulley system. This allows “opening up” of the interlaminar space and facet exposure to enable thorough foramen decompression. A change of vector by a separate rope placed over a crossbar on the Jackson table extends the neck. This checks for overcrowding of the laminae and prevents bony blocks from neck extension. The spine is exposed in a standard midline subperiosteal fashion and avoids unnecessary soft tissue violation at either ends of the laminoplasty.

Microscope is used for detailed interlamina-facet complex exposure, foraminotomies and laminoplasty execution. The interlaminar “V” marks the junction where the cranial lamina intersects with the caudal one at the medial facet and the start of the foraminal decompression (Figure 1). A high-speed 3-mm carbide tip cutting burr is used to resect first the overlying inferior facet to uncover the superior facet, which is the cause of the dorsal nerve root impingement. The superior facet is burred out in an “L-shaped” trough with the vertical limb of the “L” made along the lateral edge of the pedicle and the horizontal limb cranial to the pedicle. Inadequate dorsal nerve root decompression is avoided by careful removal of any residual bony hook at the cranial tip of the superior facet from an incompletely burred out “L-shaped” facet. Care to avoid more than 50% of facet removal is done to minimize postoperative facet instability and copious irrigation is used to avoid thermal trauma to the nerve.

Open-door laminoplasty is performed after foraminotomies. Bilateral bony gutters are placed near the medial border of the pedicle (may be identified through foraminotomies) at the lamina-medial facet junction. The lamina is opened on the clinically more symptomatic side. Elevation of the lamina beyond 60° is avoided to reduce nerve root traction and although suture methods have been used in the earlier cases, majority of the laminoplasty is kept open by the use of laminoplasty plates without bone graft.

All patients received 8 mg intravenous dexamethasone one hour prior to surgery. No routine steroid was given postoperatively.

Results

A total of 70 patients were retrospectively reviewed. There were 54 males and 16 females with an average age of 56 years (range, 30-86 years). Eighty-seven percent of the patients presented with myelopathic symptoms while 13% had multiple radiculopathy. The mean duration of preoperative symptoms was 7 ± 19 months. Average preoperative Nurick score was
2.3 (range, 1-5) grades. Spondylotic degeneration was the main pathology in 76% of cases and ossified posterior longitudinal ligament (OPLL) in 21%. Radiographic evidence of C4-5 foraminal stenosis was seen in up to 81% of the patients. Other radiographic findings such as C4-5 flattening of the cord and MRI cord signal change were seen in 30% and 59%, respectively.

Seventy-nine percent of the patients underwent multilevel open-door laminoplasty (5 levels). Twenty-one percent had 4-level laminoplasties done. There was near equal distribution of the side of hinge placement with right-sided hinges done in 46% and left-sided hinges in 54%. All the patients had bilateral C4-5 foraminotomies (Figure 2). Sixty-six percent of the cases had bilateral C4-5 and C5-6 foraminotomies performed. The mean operative time was 168 minutes (range, 90-270 minutes). Mean blood loss was 188 mL (range, 75-900 mL).

Postoperative C5 palsy developed in 4 out of the 70 patients reviewed (5.7%) (Figure 3). All 4 patients were males, non-smokers and nondiabetics, with a mean age of 56.3 years (range, 52-63 years) at the time of operation. The pathology was spondylotic myelopathy with C4-5 bilateral foraminal stenosis in all four cases. Three out of the 4 patients had MRI cord signal change at C4-5 as well as flattening of the cord. The mean duration of preoperative symptoms was 51 months. However, case 2 had preoperative symptoms for 120 months. If we exclude case 2 and treat it as an outlier, the mean preoperative duration of symptoms is 28 months. The average Nurick grade was 2.5 (range, 1-3) grades. Average follow up was 25.5 months (range, 16-54 months). Three patients with C5 palsy had laminoplasty performed in 4 levels, with 1 case having a 5-level laminoplasty. Cervical lordosis was preserved pre- and postoperatively. Mean operative time was 188 minutes and mean blood loss was 200 mL. Seventy-five percent of postoperative C5 palsy developed in the extremity opposite the side of the laminoplasty hinge. C5 palsy symptoms resolved at 3 months in 2 patients. The remaining 2 patients had resolution of symptoms at 12 and 54 months, respectively. A summary of the characteristics of the 4 patients with C5 palsy is seen in Table 1.

Multivariate logistic regression analysis showed that there was a statistically significant increase in risk for postoperative C5 palsy if the patient had a long duration of preoperative symptoms (>12 months, \(P = .0001\)). Case 2 had preoperative symptoms for 120 months. In order to determine if this skewed the results, we removed case 2 from the equation and recomputed the data. Statistical significance for preoperative duration was maintained with a \(P\) value of .003. The presence of T2 MRI cord signal change at C4-5 was another significant risk factor in the development of C5 palsy postforaminotomy with a \(P\) value of .036. Other variables such as foraminal stenosis, MRI cord compression, patient’s age and gender, diabetes, cigarette smoking, laminoplasty hinge site, preoperative Nurick score, and multilevel foraminotomies did not reach statistical significance. Neither the diagnosis of myelopathy versus radiculopathy nor the pathology of spondylosis versus OPLL have any statistical significance in the development of post foraminotomy C5 palsy (see Table 2).

**Discussion**

C5 palsy is a known complication of cervical decompression surgery. The overall incidence in literature is between 5% and 17%.\(^1\),\(^2\),\(^6\),\(^11\),\(^21\)-\(^23\),\(^36\),\(^37\) Yifei et al\(^{34}\) in a 2014 systematic review reported the incidence of C5 palsy after open-door laminoplasty, double-door laminoplasty, and laminectomy as 4.5%, 3.1%, and 11.3%, respectively. Similarly, Kaneyama et al\(^{35}\) reported a higher incidence of postoperative C5 palsy in the open door laminoplasty group when compared with a double-door laminoplasty (9.6% and 1.4%, respectively).

Several theories have been proposed to explain the occurrence of postoperative C5 palsy (Figure 4). One of the most widely accepted mechanisms is nerve root traction injury as the spinal cord migrates posteriorly after laminoplasty.\(^3\),\(^5\),\(^8\),\(^12\),\(^21\),\(^23\),\(^25\),\(^27\),\(^36\),\(^37\) Unique anatomical features such as an anteriorly located superior facet, a shorter nerve root and a larger distance of posterior shift of the cord at C5 compared to the other spinal levels, further predispose the C5 nerve root to this kind of traction injury.\(^3\),\(^22\),\(^23\) Preoperative foraminal stenosis has also been proven to be a significant risk factor to developing postoperative C5 palsy.\(^34\) Furthermore, Katsumi et al\(^{28}\) reported a significant higher incidence of postoperative C5 palsy in patients with C4-5 foraminal stenosis when compared to patients without foraminal stenosis.

Based on the hypothesis of nerve traction and foraminal stenosis as the mechanism of postlaminoplasty C5 motor palsy, the use of prophylactic C4-5 foraminotomy to decompress the C5 nerve root has been proposed as a means of eliminating this complication.\(^8\),\(^12\),\(^17\),\(^25\),\(^27\) Baba et al\(^{28}\) described laminoplasty with foraminotomy for treating cervical myelopathy in the early 1990s. They reported better neurologic improvement of myelopathy and unilateral nerve compression when doing
laminoplasty with concomitant foraminotomy. Their study, however, was limited to unilateral foraminotomy and was thus less effective for bilateral radiculopathic symptoms. Ikata et al\textsuperscript{39} advocated a sagittal splitting method of laminoplasty with bilateral foraminotomy as a solution to this problem. Sakaura et al\textsuperscript{3} reported a 0\% and 1.1\% incidence of C5 palsy after open-door laminoplasty with foraminotomy in a series of 76 and 281 patients, respectively.

Komagata et al\textsuperscript{27}, in a retrospective study, reported the incidence of C5 palsy in 230 patients with or without foraminotomies after laminoplasty. Foraminotomy was performed in 162 of 460 laminoplasty gutters. C5 palsy was observed in 1 (0.6\%) gutter with foraminotomy and in 12 (4.0\%) without foraminotomy. Only 54 patients in the series had bilateral foraminotomies and others had unilateral foraminotomy. There was no mention of whether the reported C5 palsy was from patients with bilateral or unilateral foraminotomy and what their surgical indications for unilateral foraminotomy were.

Katsumi et al\textsuperscript{28} prospectively compared 141 cases with cervical myelopathy who underwent open-door laminoplasty and prophylactic bilateral C4-5 foraminotomy with another 141 patients who did not have foraminotomy done at the time of laminoplasty. They reported that there was a significant decrease in incidence of postoperative C5 palsy in the foraminotomy group vs no foraminotomy group (1.4\% vs 6.4\%).\textsuperscript{28} However, the use of bilateral foraminotomy still did not totally eliminate the complication as there were 2 patients who developed C5 palsy even after bilateral prophylactic C4-5 foraminotomy. The authors suggested that there may be other spinal cord factors that can contribute to the occurrence of C5 palsy.

Table 1. Characteristics of Patients Who Developed Postoperative C5 Palsy After Open-Door Laminoplasty With Bilateral C4-5 Foraminotomy.

| Case  | Sex | Follow-up (mo) | Age at Operation (y) | Operation Level | Hinge Side | Side of C5 Palsy | Duration of Symptoms (mo) | Preoperative Nurick Grade | C4-5 Cord Compression | C2-MRI Cord Signal Change at C4-5 | C5 Palsy Resolved (mo) |
|-------|-----|----------------|----------------------|----------------|-----------|-----------------|--------------------------|--------------------------|----------------------|-----------------------------|------------------------|
| Case 1| M   | 24            | 53                   | C3-7           | L         | R               | 36                       | 1                        | N                    | Y                          | 3                      |
| Case 2| M   | 54            | 57                   | C3-7           | R         | L               | 120                      | 1                        | Y                    | Y                          | 54                     |
| Case 3| M   | 16            | 52                   | C3-7           | R         | R               | 12                       | 2                        | Y                    | N                          | 3                      |
| Case 4| M   | 16            | 63                   | C4-7           | L         | R               | 36                       | 3                        | Y                    | Y                          | 12                     |

Abbreviations: M, male; F, female; N, no; Y, yes; L, left; R, right; MRI, magnetic resonance imaging.
In our study, the incidence of postoperative C5 palsy after open-door laminoplasty with bilateral C4-5 foraminotomy is 5.7%. Spinal cord risk factor analysis including overall MRI cord signal change, MRI cord compression, diabetes, cigarette smoking, pathology (spondylosis vs OPLL), diagnosis (myelopathy vs radiculopathy), and preoperative Nurick score did not show any significant relationship in the occurrence of postoperative C5 palsy. Only 2 factors were found to be of statistical significance in the development of postforaminotomy C5 palsy. These were prolonged duration of preoperative symptoms and increase in T2-MRI cord signal change at C4-5 spinal level.

Various inherent spinal cord pathologies as a cause of C5 palsy has been proposed in literature. Mechanisms include cord ischaemia, reperfusion injury, and impairment of spinal cord gray matter. Chiba et al reported worsening of postoperative MRI spinal cord changes in patients who develop C5 palsy and suggested cord ischemia and reperfusion injury as an etiology of C5 palsy development. Sasai et al reviewed 111 patients who underwent laminoplasty and found that patients who had preoperative electromyographic changes were more likely to develop postoperative C5 palsy. This suggests that preexisting subclinical cord pathology is a risk factor for C5 palsy development. Increased cord signal intensity in MRI has been reported to suggest irreversible changes of the spinal cord. Takashima et al considered it to be reflective of edema, myelomalacia, or cord gliosis secondary to a longstanding compressive effect on the spinal cord. Ikegama et al reported that patients with preoperative MRI spinal cord signal change who had postoperative segmental motor paralysis had wider paralyzed muscle domains, weaker muscle strength, and longer recovery periods. These findings substantiate our study results, which showed that prolonged duration of symptoms and presence of T2-MRI cord signal change to be significant risk factors in postoperative C5 palsy development. The presence of preoperative MRI cord signal changes represents preoperative subclinical neural tissue damage thus making individuals prone to postoperative C5 radiculopathy from

| Risk Factors                                      | P    |
|--------------------------------------------------|------|
| Foraminal stenosis                               | 1.000|
| Overall MRI cord signal change                   | .6369|
| MRI cord compression                             | .6086|
| Patient age                                      | .9260|
| Gender                                           | .5672|
| Diabetes                                         | 1.0000|
| Cigarette smoker                                 | .3008|
| Diagnosis (myelopathy vs radiculopathy)          | .2529|
| Pathology (spondylosis vs OPLL)                  | .3295|
| Preoperative Nurick score                        | .4796|
| Laminoplasty hinge site                          | 1.0000|
| Multilevel foraminotomies                        | .8630|
| Preoperative symptoms duration                   | <.0001*|
| Preoperative symptoms duration (excluding case 2) | .0027*|
| T2 MRI cord signal change at C4-5                | .0355*|

Abbreviations: MRI, magnetic resonance imaging; OPLL, ossification of posterior longitudinal ligament.
*Statistically significant (P < .05).

In our study, the incidence of postoperative C5 palsy after open-door laminoplasty with bilateral C4-5 foraminotomy is 5.7%. Spinal cord risk factor analysis including overall MRI cord signal change, MRI cord compression, diabetes, cigarette smoking, pathology (spondylosis vs OPLL), diagnosis (myelopathy vs radiculopathy), and preoperative Nurick score did not show any significant relationship in the occurrence of postoperative C5 palsy. Only 2 factors were found to be of statistical significance in the development of postforaminotomy C5 palsy. These were prolonged duration of preoperative symptoms and increase in T2-MRI cord signal change at C4-5 spinal level.

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![Figure 4. Theories for the development of postoperative C5 palsy.](image-url)
minimal traction on the nerve root during dorsal cord migration after laminoplasty.

A limitation of the current study is its retrospective nature and the lack of a matched control cohort of laminoplasty patients without foraminotomy. It would also have been valuable to assess the amount of posterior cord drift post foraminotomy and its relationship with the development of C5 palsy as this is one of the most commonly accepted theories of this complication. However, this was not possible in this study since routine postoperative MRI was not done for all patients.

Our study raises awareness that routine bilateral C4-5 foraminotomy in open-door laminoplasty does not prevent the development of postoperative C5 palsy. For patients with foraminal stenosis we would still do prophylactic foraminotomy to try to decrease this incidence. However, C5 palsy may still occur in patients with prolonged duration of symptoms and presence of high intensity cord signal changes at C4-5. In our series, the reason behind this complication may not be related to nerve root traction or the tether theory but due to intrinsic spinal cord pathology. No further surgical management is recommended for this subset of patients who develop C5 palsy. Medical management to reduce inflammation, for example, Riluzole, may perhaps be beneficial. Overall, patients should be counseled accordingly about the risk of developing C5 palsy despite existing measures to prevent it.

**Conclusion**

The etiology of postoperative C5 palsy remains multifactorial. Performing prophylactic bilateral C4-5 foraminotomy does not eliminate the development of postoperative C5 palsy. Inherent subclinical cord pathology contributes to the development of this complication. Prolonged duration of preoperative symptoms and C4-5 T2-MRI cord signal change appear to be statistically significant risk factors for C5 palsy development even after bilateral C4-5 foraminotomy.

**Declaration of Conflicting Interests**

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**References**

1. Kawaguchi Y, Kanamori M, Ishihara H, Ohmori K, Nakamura H, Kimura T. Minimum 10-year follow up after en bloc cervical laminoplasty. *Clin Orthop Relat Res.* 2003;(411):129-139.
2. Wada E, Suzuki S, Kanazawa A, Matsuoka T, Miyamoto S, Yonenobu K. Subtotal corpectomy versus laminoplasty for multilevel cervical spondylotic myelopathy: a long-term follow-up study over 10 years. *Spine (Phila Pa 1976).* 2001;26:1443-1448.
3. Sakaura H, Hosono N, Mukai Y, Ishii T, Yoshikawa H. C5 palsy after decompression surgery for cervical laminoplasty: review of literature. *Spine (Phila Pa 1976).* 2003;28:2447-2451.
4. Sakaura H, Hosono N, Mukai Y, Ishii T, Iwasaki M, Yoshikawa H. Long-term outcome of laminoplasty for cervical myelopathy due to disc herniation. A comparative study of laminoplasty and anterior spinal fusion. *Spine (Phila Pa 1976).* 2005;30:756-759.
5. Wang YM, Shah S, Green BA. Clinical outcomes following cervical laminoplasty for 204 patients with cervical spondylotic myelopathy. *Surg Neurol.* 2004;6:2487-2493.
6. Hirabayashi K, Toyama Y, Chiba K. Expansive laminoplasty for myelopathy in ossification of the longitudinal ligament. *Clin Orthop Relat Res.* 1999;(359):35-48.
7. Edwards CC 2nd, Heller JM, Murakami H. Corpectomy versus laminoplasty for multilevel cervical myelopathy. An independent matched-cohort analysis. *Spine (Phila Pa 1976).* 2002;27:1168-1175.
8. Edwards CC 2nd, Riew KD, Anderson PA, Hilibrand AS, Vaccaro AF. Cervical myelopathy: current diagnostic and treatment strategies. *Spine J.* 2003;3:68-81.
9. Hosono N, Sakaura H, Mukai Y, Fujii R, Yoshikawa H. C3-6 laminoplasty takes over C3-7 laminoplasty with significantly lower incidence of axial neck pain. *Eur Spine J.* 2006;15:1375-1379.
10. Tomita K, Kawahara N, Toribatake Y, Heller JG. Expansive midline T-saw laminoplasty (modified spinous process-splitting) for the management of cervical myelopathy. *Spine (Phila Pa 1976).* 1998;23:32-37.
11. Yonenobu K, Oda T. Posterior approach to the degenerative cervical spine. *Eur Spine J.* 2003;12(suppl 2):S195-S201.
12. Rao RD, Gourab K, Kenny SD. Operative treatment of cervical spondylotic myelopathy. *J Bone Joint Surg Am.* 2006;88:1619-1640.
13. Yoshida M, Tamaki T, Kawakami M, et al. Does reconstruction of posterior ligamentous complex with extensor muscular decrease axial symptoms after cervical laminoplasty? *Spine (Phila Pa 1976).* 2002;27:1414-1418.
14. Satomi K, Ogawa J, Ishii Y, Hirabayashi K. Short-term complications and long-term results of expansive open-door laminoplasty for cervical stenotic myelopathy. *Spine J.* 2001;1:26-30.
15. Ratliff JK, Cooper PR. Cervical laminoplasty: a critical review. *J Neurosurg.* 2003;98(3 suppl):S230-S238.
16. Rhee JM, Riew KD. Evaluation and management of neck pain, radiculopathy, and myelopathy. *Semin Spine Surg.* 2005;17:174-185.
17. Sasai K, Saito T, Akagi S, Kato I, Ohnari H, Iida H. Preventing C5 palsy after laminoplasty. *Spine (Phila Pa 1976).* 2002;174:172-1977.
18. Chang H, Park JB, Hwang JY, Song KJ. Clinical analysis of cervical radiculopathy causing deltoid paralysis. *Eur Spine J.* 2003;12:517-521.
19. Yonenobu H, Kaneko K, Taguchi T, Fugimoto H, Toyoda K, Kawai S. Nerve root distribution of deltoid and biceps brachii muscle in cervical spondylotic myelopathy: a potential risk factor for postoperative shoulder muscle weakness after posterior decompression. *J Orthop Sci.* 2004;9:540-544.
20. Kaneko K, Hashiguchi A, Kato Y, Kojima T, Imaiyo Y, Taguchi T. Investigation of motor dominant C5 paralysis after laminoplasty.
laminoplasty from the results of evoked spinal cord responses. J Spinal Disord Tech. 2006;19:358-361.

21. Uematsu Y, Tokuhashi Y, Matsuzaki H. Radiculopathy after laminoplasty of the cervical spine. Spine (Phila Pa 1976). 1998;23:2057-2062.

22. Tsuzuki N, Abe R, Saiki K, Okai K. Paralysis of the arm after posterior decompression of the cervical spinal cord: II. Analyses of the clinical findings. Eur Spine J. 1993;2:197-202.

23. Tsuzuki N, Abe R, Saiki K, Zhongshi L. Extradural tethering effects as one mechanism of radiculopathy complicating posterior decompression of the cervical spinal cord. Spine (Phila Pa 1976). 1996;21:203-211.

24. Chiba K, Toyama Y, Matsumoto M, Maruiwa H, Watanabe M, Hirabayashi K. Segmental motor paralysis after expansive open-door laminoplasty. Spine (Phila Pa 1976). 2002;27:2108-2115.

25. Fan D, Schwartz D, Vaccaro A, Hilibrand AS, Albert TJ. Intraoperative neurophysiologic detection of iatrogenic C5 nerve root injury during laminectomy for cervical compression myelopathy. Spine (Phila Pa 1976). 2002;27:2499-2502.

26. Riew KD, Hurford RK, Taylor B. Microsurgery of the cervical spine. Minerva Orthop Traumatol. 2004;55:1-16.

27. Komagata M, Nishiyama M, Endo K, Ikegami H, Tanaka S, Ima-kiire A. Prophylaxis of C5 palsy after cervical expansive laminoplasty by bilateral partial foraminotomy. Spine J. 2004;4:650-655.

28. Katsumi K, Yamazaki A, Watanabe K, Ohashi M, Shoji H. Can prophylactic bilateral C4/C5 foraminotomy prevent postoperative C5 palsy after open-door laminoplasty? A prospective study. Spine (Phila Pa 1976). 2012;37:748-754.

29. Nurick S. The natural history and the results of surgical treatment of the spinal cord disorder associated with the cervical spondylosis. Brain. 1972;95:101-108.

30. Kuklo TR, Lehman RA Jr, Taylor BA, Riew KD. Surgical approaches to the subaxial cervical spine. In: Frymoyer JW, Wiesel SW, eds. The Adult & Pediatric Spine. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2004:761-769.

31. Lehman RA Jr, Taylor BA, Rhee JM, Riew KD. Surgical techniques: cervical laminoplasty. J Am Acad Orthop Surg. 2008;16:47-56.

32. O’Brien MF, Peterson D, Casey AT, Crockard HA. A novel technique for laminoplasty augmentation of spinal canal area using titanium miniplate stabilization: a computerized morphometric analysis. Spine (Phila Pa 1976). 1996;21:474-483.

33. Deutsch H, Mummaneni PV, Rodts GE, Haid RW. Posterior cervical laminoplasty using a new plating system. Technical note. J Spine Disord Tech. 2004;17:317-320.

34. Gu Y, Cao P, Gao R, et al. Incidence and risk factors of C5 palsy following posterior cervical decompression: a systematic review. PLoS One. 2014;9:e101933. doi:10.1371/journal.pone.0101933

35. Kaneyama S, Sumi M, Kanatani T, et al. Prospective study and multivariate analysis of the incidence of C5 palsy after cervical laminoplasty. Spine (Phila Pa 1976). 2010;35:E1553-E1558.

36. Shiraishi T, Fukuda K, Yato Y, Nakamura M, Ikegami T. Results of skip laminectomy- minimum 2-year follow-up study compared with open-door laminoplasty. Spine (Phila Pa 1976). 2003;28:2667-2672.

37. Seichi A, Takeshita K, Kawaguchi H, Nakajima S, Akune T, Nakamura K. Postoperative expansion of intramedullary high-intensity areas on T2-weighted magnetic resonance imaging after cervical laminoplasty. Spine (Phila Pa 1976). 2004;29:1478-1482.

38. Baba H, Chen Q, Uchida K, Imura S, Morikawa S, Tomita K. Laminoplasty with foraminotomy for coexisting myelopathy and unilateral radiculopathy. A preliminary report. Spine (Phila Pa 1976). 1996;21:196-202.

39. Ikata T, Yamada H. A recommended method of laminoplasty for cervical myelopathy. Serikeigeka MOOK 2C (Tokyo). 1993:30-34.

40. Takahashi M, Yamashita Y, Sakamoto Y, Kojima R. Chronic cervical cord compression: clinical significance of increased signal intensity on MR images. Radiology. 1989;173:219-224.

41. Ikegami S, Tsutsumimoto T, Ohta H, et al. Preoperative spinal cord damage affects the characteristics and prognosis of segmental motor paralysis after cervical decompression surgery. Spine (Phila Pa 1976). 2014;39:463-468.