Journal Pre-proof

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PII: S1529-9430(19)31000-9
DOI: https://doi.org/10.1016/j.spinee.2019.09.017
Reference: SPINEE 58026

To appear in: *The Spine Journal*

Received date: 25 May 2019
Revised date: 14 August 2019
Accepted date: 16 September 2019

Please cite this article as: Zach Pennington BS , Daniel Lubelski MD , Erick M. Westbroek MD , Ethan Cottrill MS , Jeff Ehresman BS , Matthew L. Goodwin MD PhD , Sheng-Fu Lo MD , Timothy F. Witham MD , Nicholas Theodore MD , Ali Bydon MD , Daniel M. Sciubba MD , Spinal Cord Float Back is not an Independent Predictor of Post-operative C5 Palsy in Patients Undergoing Posterior Cervical Decompression, *The Spine Journal* (2019), doi: https://doi.org/10.1016/j.spinee.2019.09.017

C5 Palsy Float Back

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Funding: None

Devices:	None
Previous Presentations:	None
Disclosures:	
Zach Pennington	None
Daniel Lubelski	None
Erick M. Westbroek	None
Ethan Cottrill	None
Jeff Ehresman	None
Matthew L Goodwin	Consultant for Augmedics, ROM3
Sheng-Fu Lo	Unrelated grants from AO Foundation and Chordoma Foundation
Timothy F. Witham	Unrelated grants from Eli Lily and Company, Gordon and Marilyn Macklin Foundation
Nicholas Theodore	Royalties/Ownership Interest – Globus Medical; Consultant – Globus Medical, DePuy-Synthes
Ali Bydon	None
Daniel M. Sciubba	Consultant for Baxter, DePuy-Synthes, Globus Medical, K2M, Medtronic, NuVasive, Stryker
IRB Approval:	IRB approval was obtained prior to initiation of the present study (IRB00160247).

Abstract

Background

Of the more than 30,000 posterior cervical spine fusions performed annually, 7-12% will be complicated by post-operative C5 palsy, a condition characterized by new-onset deltoid weakness with or without C5 dermatomal findings and biceps weakness. Posterior translation of the cervical spinal cord has been proposed as a risk factor for this complication.

Purpose

To evaluate if C5 palsy can be predicted by spinal cord float back

Study Design/Setting

Retrospective cohort

Patient Sample

Patients ≥ 18 years of age undergoing posterior cervical decompression between 2002 and 2017 for degenerative cervical spine pathologies.

Outcome Measures

Occurrence of C5 palsy as evaluated by manual motor testing (MMT).

Methods

We recorded baseline neurological status, operative notes, details of post-operative course, and both pre- and post-operative MRI images. Float back was defined by the change in the distance between the spinal cord and posterior face of the C4/5 annulus from pre-operative to post-operative imaging. C5 palsy was defined by new-onset deltoid weakness on MMT.

Results

We identified 242 patients with a mean age of 62.4 years and mean follow-up of 27.9 months. Forty-two (17.4%) experienced post-operative C5 palsy. On univariable analysis, significant predictors of post-operative C5 palsy were mean C4/5 foraminal diameter (2.8 vs. 3.2mm; p<0.001), anterior projection of the C5 superior articular process (4.12 vs 3.70mm; p=0.04), cord float back (0.35 vs. 0.28cm; p=0.02), undergoing laminectomy of the C5 (p=0.02) or C4 and C5 levels (p=0.02), and undergoing instrumented fusion extending one level above and below the C4/5 level. Foraminotomy of the C4/5 level was not predictive of post-operative palsy. On multivariable analysis mean C4/5 foraminal diameter (OR=0.38 per mm; p<0.01) predicted C5 palsy; cord float back at the C4/5 level was not predictive of C5 palsy.

Conclusion

Spinal cord float back was not an independent predictor of C5 palsy on multivariable analysis. Only smaller foraminal diameter was independently predictive of post-operative C5 palsy. This suggests that chronic preoperative compression of the C5 roots, not post-decompression float back may be the biggest contributor to the etiology of postoperative C5 palsy.

Key Words

C5 palsy; cord float back; predictive modeling; posterior cervical decompression; cervical myelopathy; cervical radiculopathy

Abbreviations

AP	Anteroposterior
BUE	Bilateral upper extremity
CI	Confidence interval
CLA	Cord-lamina angle
CSA	Cross-sectional area
CSM	Cervical spondylotic myelopathy
CVFS	Cord-vertebral face separation
IOM	Intraoperative monitoring
LUE	Left upper extremity
MMT	Manual motor testing
MRI	Magnetic resonance imaging
OR	Odds ratio
RUE	Right upper extremity
SAP	Superior articular process

Background

Post-operative C5 palsy is defined by new onset deltoid muscle weakness following cervical spine surgery, which may also be accompanied by decreased biceps strength and/or C5 dermatomal sensory deficits.[1] Risk is greatest for patients with pre-operative foraminal stenosis[2] and in those undergoing posterior decompression, with an incidence of 7-12%.[2,3] Investigations of the impact of C5 palsy on patient quality of life have demonstrated to both negatively impact patient reported outcomes and to increase total care costs by approximately $2,000 per event.[4] Because of this, there has been much interest in investigating risk factors for C5 palsy and defining the etiology of this complication.

At present, there exist several proposed etiologies for post-operative C5 palsy, including: 1) intraoperative nerve root injury, 2) nerve root traction injury secondary to post-decompression spinal cord float back (posterior migration of the cord in the laminectomy defect), 3) spinal cord ischemia secondary to reduced blood supply via the radicular arteries, 4) reperfusion injury, and 5) segmental spinal cord injury or edema.[1,5,6] Of these, spinal cord float back is one of the most thoroughly investigated.[1,7–9] Morphological reasons offered to explain the high rates of C5 palsy relative to palsy of the other cervical roots include the shorter length,[10,11] the more horizontal orientation of the C5 roots, which makes for less redundancy or slack in the root to accommodate for the posterior migration,[11,12] and the location of the C5 segment at the apex of the cervical lordosis.[1] Consequently, migration is greatest at the C4/5 segment, placing the C5 roots at greater risk compared to the roots of adjacent levels. Clinical investigations into the relationship between spinal cord float back and palsy risk have yielded contradictory results, with some reporting a significant relationship[13–17] and others finding none.[7,18,19] To further investigate this and to ascertain whether posterior cord migration is related to risk for C5 palsy, we elected to examine a large series of patients who underwent posterior cervical decompression with pre- and post-operative MRI.

Methods

After obtaining IRB approval, we retrospectively reviewed all patients who underwent posterior cervical decompression including the C4/5 level between January 2002 and December 2017. Patients were included if the indication for surgery was a degenerative pathology of the cervical spine, if they had both pre- and post-operative MRI examinations of the cervical spine, and if they had complete medical charts, including operative notes, details of pre-surgical clinical presentation, and details of post-operative course. Patients were excluded if they were less than 18 years of age, if they had a pre-operative deficit chronic neurological condition (e.g. multiple sclerosis) that prevented consistent, accurate assessment of motor strength, if they had undergone amputation of one or both of their upper extremities, or if the indication for surgery was trauma, tumor, non-degenerative deformity (e.g. rheumatoid arthritis), infection, or hardware failure.

For included patients, we gathered details regarding presenting symptoms, baseline neurological examination, including manual motor testing (MMT) of the bilateral upper extremity, operative procedures (levels decompression, levels instrumented, use of foraminotomies at the C4/5 level), anatomy of the C4/5 level on MRI imaging, and post-operative course. Details of post-operative course comprised both in-patient and post-discharge complications, including C5 palsy, which was defined as new-onset deltoid weakness without general deterioration of strength on MMT. The occurrence of new-onset biceps weakness or sensory changes in C5 dermatome were also considered, but were not considered necessary for the diagnosis of C5 palsy. For those experiencing C5 palsy, we also gathered details regarding laterality of the deficit, times to onset and resolution, and magnitude of the deficit at the time of detection.

Anatomical data collected included C4/5 foraminal diameter on axial imaging, C4 Pavlov-Torg ratio, cord-laminar angle (CLA), anterior-posterior (AP) diameter of the spinal canal at the C4/5 level, projection of the superior articular process in front of the posterior disc face at the C4/5 level (SAP), cord and canal cross-sectional area (CSA) at the C4/5 level, facet:gutter ratio, and distance between the anterior cord and posterior disc face at the C4/5 level (CVFS). These measurements have been previously described[2] and are shown in **Figure 1**. Briefly, CLA, C4/5 AP diameter, cord CSA, and canal

CSA are as illustrated. Facet-gutter ratio was calculated as the quotient of the widths of the medial facet and bony gutter, expressed as a percent (**Figure 1D)**, and Pavlov-Torg ratio was measured as the quotient of the sagittal diameters of the spinal canal and vertebral body measured at the C4 level (ratio of dashed and solid lines, respectively in **Figure 1F)**. Posterior migration of the spinal cord was defined as the change in CVFS as the C4/5 level from pre-operative to post-operative MRI (**Figure 1G)**. Review of the radiology reports for the post-operative MRIs indicated that the majority were acquired for either persistence of pre-operative symptoms or work-up of the post-operative C5 palsy. All anatomic measurements were made by the first two authors (ZP and DL) with the average measurement value being used in the analysis.

Surgical Strategy and Use of Intraoperative Neuromonitoring

All patients in the present cohort underwent posterior cervical decompression through an open midline approach. Dissection was performed over the target segments through the midline avascular plane, extending laterally to the facet joints and exposing the lateral masses and transverse processes. Decompression was then performed using either: 1) a combination of rongeurs, 2) a high-speed drill with a matchstick, or 3) an ultrasonic cutting device. The specific technique employed was based upon surgeon preference and was not consistent across the group. The high-speed drill was the most common technique employed and ultrasonic cutting device became available within the cohort only after 2010.

When fusion was felt to be necessary by the treating surgeon, instrumented fusion was performed using lateral mas screws at the C1 and C3-7 vertebrae and pedicle screws at the C2 and T1-3 vertebrae. At our institution there exist no standard rules regarding which patients should or should not receive instrumented fusion, however, general practice is to instrument all patients receiving laminectomy of ≥ 3 contiguous levels due to the increased risk of post-laminectomy kyphosis.[20] Where fusion bridged the cervicothoracic junction, general practice amongst attending surgeons was to omit instrumentation of the C7 vertebra to better facilitate rod contouring. All cases of instrumented fusion were supported by decortication of the posterolateral elements using a high-speed drill, followed by placement of a combination of cancellous allograft, demineralized bone matrix, and morselized autograft from the laminectomy.

Statistical Analysis

Data were recorded using Microsoft Excel (Redmond, WA) and were analyzed using Statistica Version 13.3.0 (TIBCO, Palo Alto, CA). Comparisons between palsy and non-palsy patients were performed using Fisher Exact tests for dichotomous variables, Mann-Whitney U tests for continuous variables, and χ^2 tests for categorical or ordinal variables. A stepwise logistic regression was performed using all variables identified as significant at the $p \leq 0.15$ level on univariable analysis in order to develop a multivariable model predicting the occurrence of C5 palsy within the cohort. Model validation was performed using 1000 boot-strapped samples with 25% holdout and an assessment of model accuracy was performed using the C-statistic of a receiver operating curve. To assess inter-rater reliability for measurements of foraminal diameter, we used the method of Bliese and Halverson, which uses a one-way fixed-effects Analysis of Variance (ANOVA).[21,22] We also used Pearson's r and the method of Landis and Koch[23] to qualitatively categorize the interobserver reliability.

Results

Of the 870 patients treated with posterior cervical decompression for degenerative cervical spine pathologies, pre-operative MRIs were available for 611 and both pre- and postoperative MRIs were available for 242 patients. The demographics of the cohort are presented in **Table 1**. Mean age was 62.4 years, mean body mass index (BMI) was 29.13kg/m², 66% of patients were male and 58% of patients were white. The most common indications for surgery were myelopathy-alone (76.4%), radiculopathy-alone (5.0%), and myeloradiculopathy (13.2%); 5.4% of patients were operated for critical, asymptomatic stenosis of the central canal. Forty-two (17.4%) patients experienced post-operative C5 palsy with a mean onset of 3.19±2.62 days and a mean time to resolution of 34.9±36.2 weeks. Eighteen patients (43%) experienced full recovery and 20 (48%) experienced partial recovery (88% with at least partial recovery).

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Comparison of patients experiencing post-operative C5 palsy to controls demonstrated palsy patients were more likely to undergo laminectomy of C5 (97.62 vs. 80.81%; p<0.01) or both C4 and C5 (95.24vs. 78.50%; p<0.01), and were more commonly treated with fusion constructs extending 1 or more levels above and below the C4-5 levels (95.24 vs 80.00%; p=0.02). Patients experiencing post-operative C5 palsy also had smaller C4/5 foraminal diameters (2.8±0.9 vs. 3.2±0.8mm; p<0.001), more anterior projection of the superior articular process (4.12±1.53 vs 3.70±2.39; p=0.04), and greater cord float back (0.35±0.17 vs. 0.28±0.17cm; p=0.02) secondary to a larger post-operative CVFS (0.37±0.16 vs 0.31±0.16cm; p=0.02). Patients in the palsy group were also less likely to complain of gait or balance issues at the time of surgical consultation (42.86 vs 62.00%; p=0.03). None of the other variables differed significantly between the two groups, including the use of C4/5 foraminotomy (p=0.36). The presence of pre-operative C5 radicular pain trended towards being more common in the post-operative C5 group (p=0.06).

Multivariable stepwise logistic regression identified a model with four variables capable of predicting post-operative C5 palsy. Mean C4/5 foraminal diameter (OR=0.38 per mm; 95% CI [0.22-0.64]; p<0.01) was identified as the only significant independent predictor of post-operative C5 palsy. Cord-float back was not found to be independently statistically significant in multivariable analysis (p=0.27). The use of C5 laminectomy (p=0.06), extension of C4-5 fusion 1 or more levels above and below the C4-5 levels (0.06), and pre-operative complaints of C5 radiculopathy approached significance in the multivariable analysis. A receiver-operating curve of the model constructed from these variables and C4/5 foraminal diameter demonstrated a C-statistic of 0.7627 and internal validation using 1000 boot-strapped samples found an average error of 13.09±5.77%

Inter-rater reliability

To assess the accuracy of the anatomic measurements, namely the C4/5 foraminal diameter, we performed an inter-rater reliability assessment. The inter-observer correlation coefficient for these values was ICC = 0.93. Pearson’s r for the regression of the two value sets was r = 0.93±0.02 (mean ±

standard error), indicating that the measures were of "almost perfect" agreement according to the system of Landis and Koch.[23]

Discussion

Cord float back, also known as posterior spinal cord drift, is a commonly proposed potential etiology for C5 palsy.[1,24] It is believed that greater posterior drift of the spinal cord creates a tension-stretch injury of the C5 roots, creating a neurapraxia of the C5 myotome and resulting in acute/sub-acute onset paresis of the deltoid muscle of the affected arm. Here we find that in a large sample of patients undergoing posterior cervical decompression including the C4/5 level, posterior cord drift is not an independent predictor of the occurrence of post-operative C5 palsy. Rather, C4/5 foraminal diameter and pre-operative C5 radiculopathy are the best independent predictors of post-operative C5 palsy. This suggests that baseline C5 root compression may be the best predictor of post-operative C5 palsy as opposed to cord float back.

The earliest investigations of posterior spinal cord drift as a risk factor for C5 palsy were published in the Japanese literature, with several reports from the late 1990s and early 2000s hypothesizing it played a key role in the etiology of C5 palsy.[1] Authors proposed that tethering of the cord by the uncovertebral joints combined with posterior migration of the spinal cord produced a stretching of the C5 roots, creating a neurapraxic injury. This clinically correlated to the acute onset of deltoid weakness, often accompanied by biceps weakness and C5 radicular pain and dysesthesias. Compared to other cervical roots, the C5 roots are shorter[10,11] and more horizontally oriented.[11,12] Additionally, the C5 segment is located at the apex of the cervical lordosis[1] and therefore should experience the greatest posterior drift following decompression. Consequently, the C5 roots are likely subjected to the greatest change in sagittal position and have the least redundancy to accommodate for this transition. Therefore, they likely experience the highest tension during cord float back, placing them at the greatest risk for neurapraxic injury. Unlike the majority of muscles in the upper extremity, which possess substantial redundancy in their innervation, the deltoid muscle is almost exclusively innervated by the C5 root.[25,26] Consequently, injury to the C5 root is more likely to be noticed on neurological examination.

Several studies have been published supporting the role of posterior spinal cord migration as a risk factor for post-operative C5 palsy. The earliest publication in the English literature by Tsuzuki and colleagues.[27] They examined posterior cord shift and anterior protrusion of the superior articular process as risk factors for C5 palsy in patients undergoing laminectomy for cervical spondylotic myelopathy (CSM). They found that C5 palsy rates were greater for those experiencing posterior cord drift > 1.8mm who also had pronounced anterior projection (type A1)[28] of the SAP.[27] However, the mean posterior migration was not noted to be significantly different when considering all patients.

In their series of 19 patients treated with C4-6 or C4-7 laminoplasty for CSM, Shiozaki et al suggested that the time frame rather than the magnitude of the float back may be the biggest driver of C5 palsy.[29] All patients in their cohort were imaged at 24 hours and 2-weeks post-surgery. Evaluation of both post-operative images demonstrated that the float back magnitude was twice as high at 24hr in the 2 patients experiencing post-operative C5 symptoms. However, this difference did not persist at 2 weeks post-operatively, leading them to conclude that it is the acute event of spinal cord float back rather than the absolute magnitude that may mediate the C5 palsy.

Shortly after the findings of Shiozaki et al, Imagama and colleagues published a series comparing radiographic predictors of C5 palsy in patients treated with laminoplasty for CSM.[30] Comparing the 43 patients who experienced post-operative palsy to 100 matched controls, the authors noted smaller C4/5 foraminal width, greater SAP anterior protrusion, and greater posterior spinal cord shift within the C5 palsy cohort. Unlike the present study, no multivariable analysis was performed and so it is unclear if foraminal size and posterior spinal cord shift were independent predictors within the authors' cohort. Similarly, Yang and colleagues reported posterior spinal cord shift to be predictive of post-operative C5 palsy in their series of 141 patients treated with open-door laminoplasty for CSM.[31] Like Imagama et al however, they did not perform a multivariable analysis and so did not control for the potential contribution of foraminal stenosis. Therefore, it remains unclear from their results as to whether posterior spinal cord shift is an independent predictor.

By contrast, Nakashima et al performed a multivariable analysis of 84 patients treated with posterior cervical decompression and fusion for nontraumatic cervical spine disease.[17] They found that post-operative C4/5 foraminal diameter, the presence of OPLL, and posterior drift of the spinal cord at the C4/5 level were independent predictors of C5 palsy in the 10 patients that developed the complication. Notably, the authors did not find posterior drift to be significant on univariable analysis, nor did they consider either C4/5 foraminotomy or pre-operative complaints of C5 radiculopathy in their analysis.

Like Nakashima et al, Bydon et al performed a multivariable analysis of radiographic and operative predictors of C5 palsy in patients undergoing posterior laminectomy and foraminotomy for degenerative cervical spine pathologies.[9] They found that posterior cord shift and change in the size of the C4/5 foramina were independent predictors of post-operative C5 palsy. Unlike the present study, clinical variables such as presenting symptoms and baseline motor exam were not considered in the analysis. Radcliff et al examined both posterior cord drift and post-operative sagittal alignment as risk factors for C5 palsy in a 2:1 case:control design using patients treated for CSM.[13] They found that among the 17 patients with C5 palsy in their series, posterior cord migration was 213% higher at the C5 level in the C5 palsy patients, supporting the role of cord drift in the etiology of C5 palsy. Unlike the present study though, they did not consider foraminal stenosis as a risk factor.

Most recently, several additional studies have described the influence of posterior spinal cord shift on the occurrence of C5 palsy. In their cohort of 198 patients treated with open-door laminoplasty for CSM, Zhang et al found posterior spinal cord shift to be nearly 50% greater in those experiencing C5 palsy, which was significant on univariable analysis.[32] Like many prior studies however, multivariable analysis including foraminal stenosis as a risk factor was not performed. The study by Baba et al[33] examining double-door laminoplasty for CSM showed similar results, with posterior drift being greater in palsy patients, yet without a multivariable analysis controlling for other contributing variables such as foraminal stenosis. In contrast to both of these studies, Nori and colleagues considered both posterior shift and foraminal diameter in their analysis of 252 patients undergoing narrow laminectomies for CSM.[14] As in the present study, the authors found that foraminal stenosis and greater posterior spinal cord shift at the C4/5 level were significant predictors of C5 palsy on univariable analysis, but only foraminal diameter was significant on multivariable analysis.

Several series have also been published which find no relationship between posterior spinal cord shift and the odds of experiencing C5 palsy. In their series of patients undergoing anterior or posterior decompression for degenerative cervical spine disease, Yonenobu and colleagues found no difference in the average posterior shift between the 21 patients experiencing C5 palsy and the remaining 367 patients.[18] Subsequently, Sodeyama et al investigated posterior spinal cord shift in a cohort of 65 patients with pre- and post-operative CT myelograms treated with open-door laminoplasty for degenerative cervical spine pathologies.[19] Comparison of the four patients experiencing C5 palsy to unaffected patients demonstrated no significant difference in posterior spinal cord shift. Hatta et al similarly noted no relationship between posterior migration and risk of C5 palsy in their series of 26 patients treated with selective laminoplasty for CSM.[34] Most recently, Hitchon et al reported no significant relationship between posterior migration and risk for C5 palsy in their cohort of 63 patients treated with posterior decompression and fusion for CSM.[7]

We found that the extant literature provides evidence both for and against the model of posterior spinal cord shift as a core factor in the etiology of post-operative C5 palsy. The present study provides one of the largest series of patients examined, and unlike the majority of prior series espousing posterior shift as a key contributor to C5 palsy, we provide a multivariable analysis that considers C4/5 foraminal diameter, C4/5 foraminotomy, and pre-operative C5 radiculopathy.[35] We find that in doing so, foraminal diameter and clinical signs of C5 root compression i.e. C5 dermatomal parasthesias/brachialgia are the strongest predictors of post-operative C5 palsy. Interestingly, we also note that among included patients, the performance of a C5 laminectomy was more strongly associated with the occurrence of post-operative C5 palsy than was either C4 laminectomy alone, or a multilevel decompression including both C4 and C5. Though this difference was not significant, it is a curious observation and, in our minds, further argues for C5 palsy being a product of baseline compression and intraoperative manipulation rather than a post-operative stretch type injury.

Limitations

The results of this study must be interpreted with the inherent limitations. As a retrospective study, we are unable to reach any statement regarding the causality of C5 palsy. However, the fact that posterior spinal cord shift was not associated with C5 palsy on multivariable analysis suggest that no causal link exists. Our sample size was larger than most in the literature and therefore it was likely sufficiently powered to identify a relationship, if one truly existed. Another significant limitation is that post-operative MRIs are not uniformly acquired, and therefore it is possible that the present cohort is affected by selection bias, potentially limiting the generalizability of the results. To this end though, we noted that the indication for post-operative imaging in the majority of patients was either the evaluation of C5 palsy or the evaluation of persistent pre-operative symptoms. Additionally, comparison of the anatomic parameters, including pre-operative CVFS, demonstrated no significant differences between patients who did and did not receive a post-operative MR, which suggests against an underlying anatomic confounder. In spite of this, to adequately address both the potential selection bias and the drawbacks inherent in the retrospective design, it is necessary for a large, prospective study to be conducted with acquisition of both pre-operative and post-operative MR to allow for assessment of all radiographic parameters. Our study is also limited in that we were unable to consider change in sagittal balance as a predictor of post-operative C5 palsy. It has been suggested that increased cervical lordosis may collapse the C4/5 foramina and beget a post-operative palsy. However, less than one-third of included patients had pre- and post-operative plain films. Amongst this subgroup however, change in cervical lordosis was not found to be associated with C5 palsy. We were similarly unable to consider post-operative foraminal stenosis as the impact of post-operative foraminal diameter could not be disentangled from the impact of change in lordosis due to the absence of pre- and post-operative cervical radiographs for the majority of patients. Lastly, in this study we identify pre-operative C4/5 foraminal diameter as a significant predictor of the occurrence of post-operative C5 palsy. However, we note that the standard deviations associated with these measures led to overlapping intervals. While the difference is statistically significant and validates prior findings showing a similar effect, further research with larger datasets is necessary to validate these findings prior to guiding clinical management.

Conclusion

Here we present one the largest series to date investigating posterior spinal cord drift as a risk factor of C5 palsy in patients undergoing cervical laminectomy for degenerative cervical spine disease. We find that posterior spinal cord migration is not an independent risk factor for C5 palsy. Rather, C5 palsy is best predicted by foraminal stenosis and pre-operative C5 sensory abnormalities or brachialgia. Given the outstanding heterogeneity in the literature however, future, multicenter investigations are required to better identify the etiology of post-operative C5 palsy.

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Figures

Figure 1. Figure illustrating anatomic measurements obtained from pre-operative MRI imaging. A) cord-lamina angle (°),[36] B) foraminal diameter (mm), C) C4/5 anterior-posterior canal diameter, D) facet-gutter ratio – defined as the quotient of the distances indicated by the double-arrowed lines (g/f × 100%) (star – midline; diamond – medial gutter; circle – medial facet), E) cord and canal cross-section, F) Pavlov-Torg ratio – defined by the quotient of the canal diameter (dashed) to the vertebral body diameter (solid), G) cord float back as measured by change in the distance between the ventral cord and posterior annulus of the C4/5 disk.

Figure 2. Receiver-operating curve of the multivariable model predicting the odds of post-operative C5 palsy based upon operative procedures, presenting symptoms, and local anatomy as assessed by pre-operative MRI.

Tables

Table 1: Details of included patients (n = 242)

Table 2: Univariable analysis comparing patients who did and did not experience post-operative C5 palsy

Table 3: Multivariable analysis of surgical predictors of post-operative C5 palsy

C5 Palsy Float Back

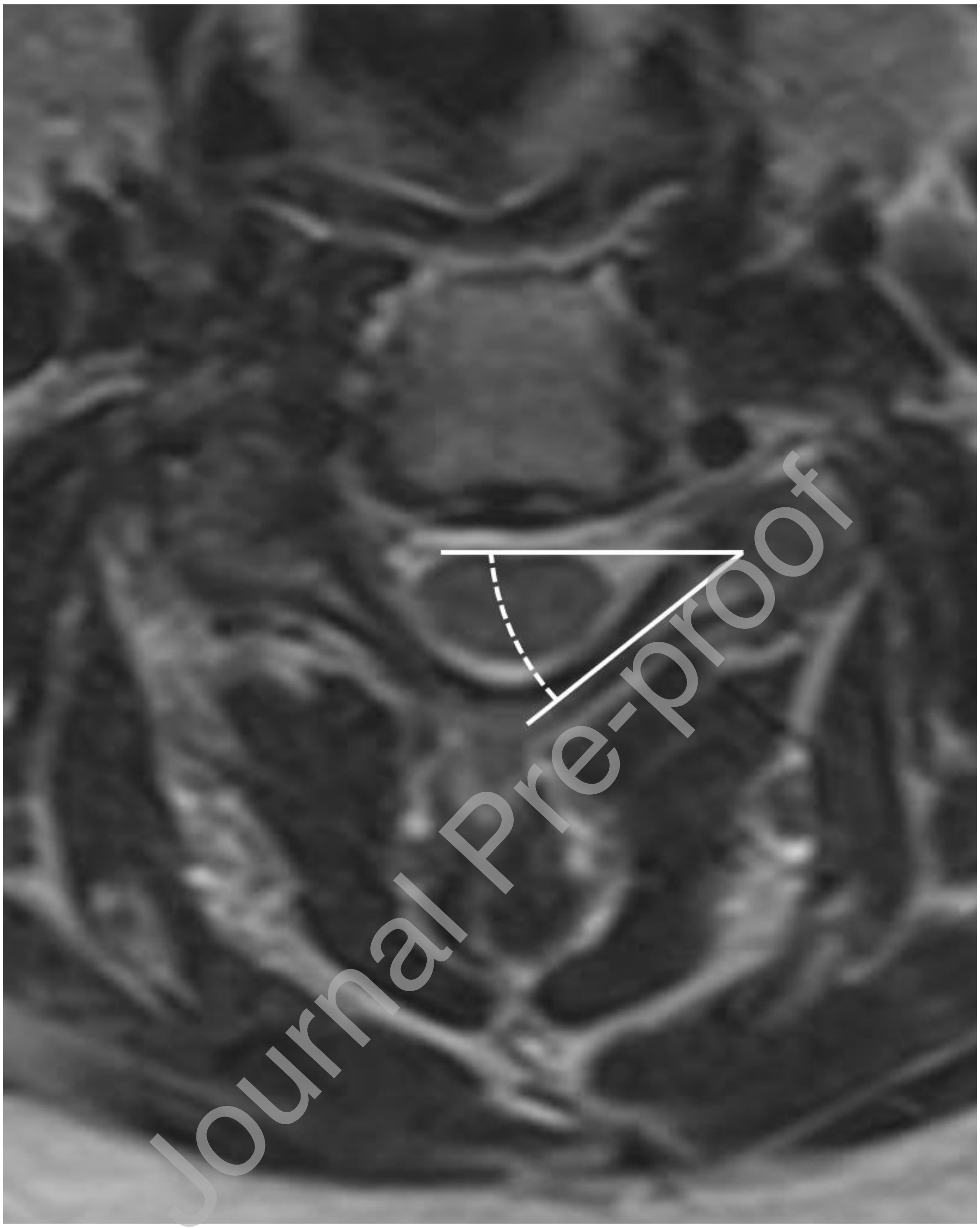

Fig 1A

C5 Palsy Float Back

Fig 1B

C5 Palsy Float Back

Fig 1C

C5 Palsy Float Back

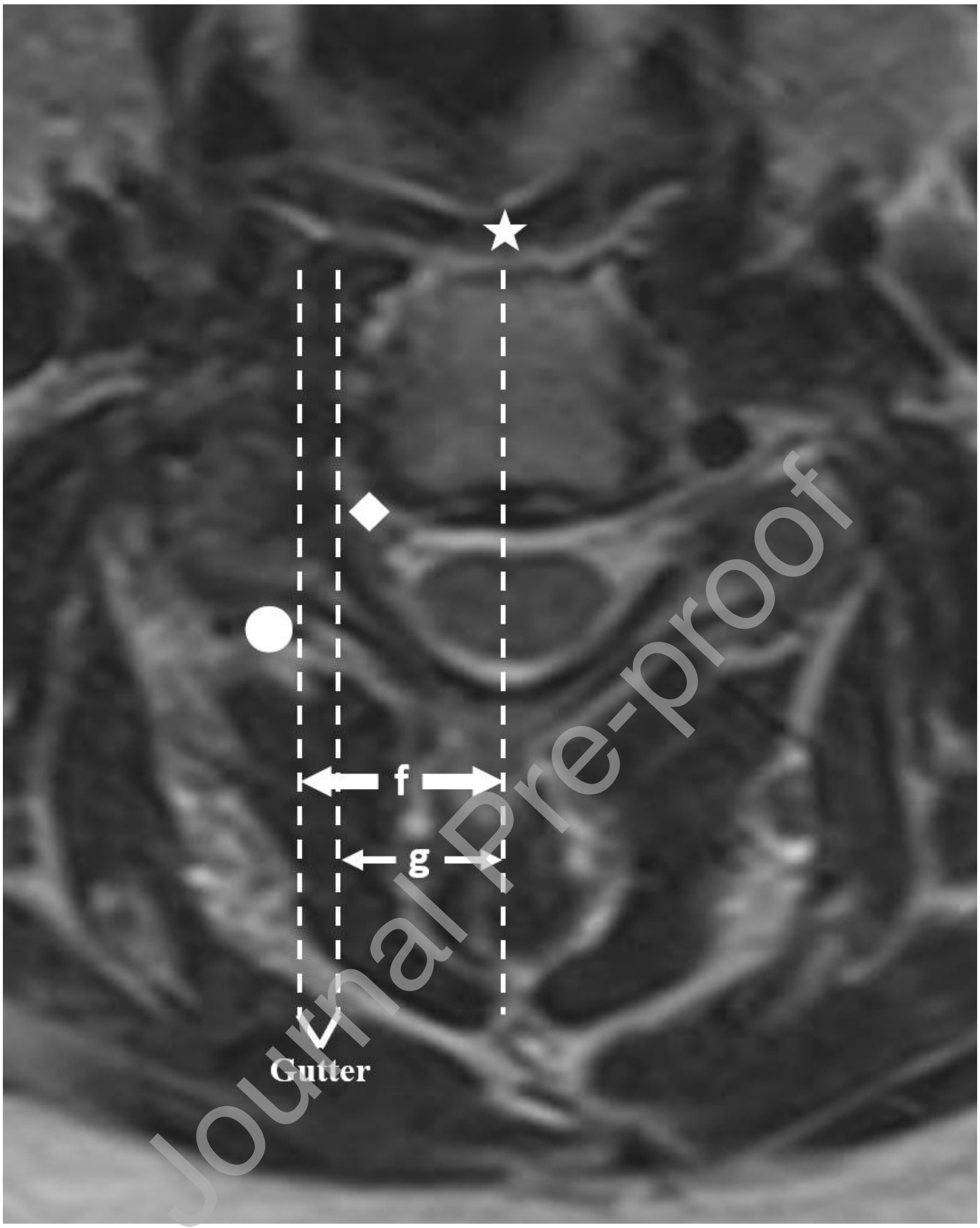

Fig 1D

C5 Palsy Float Back

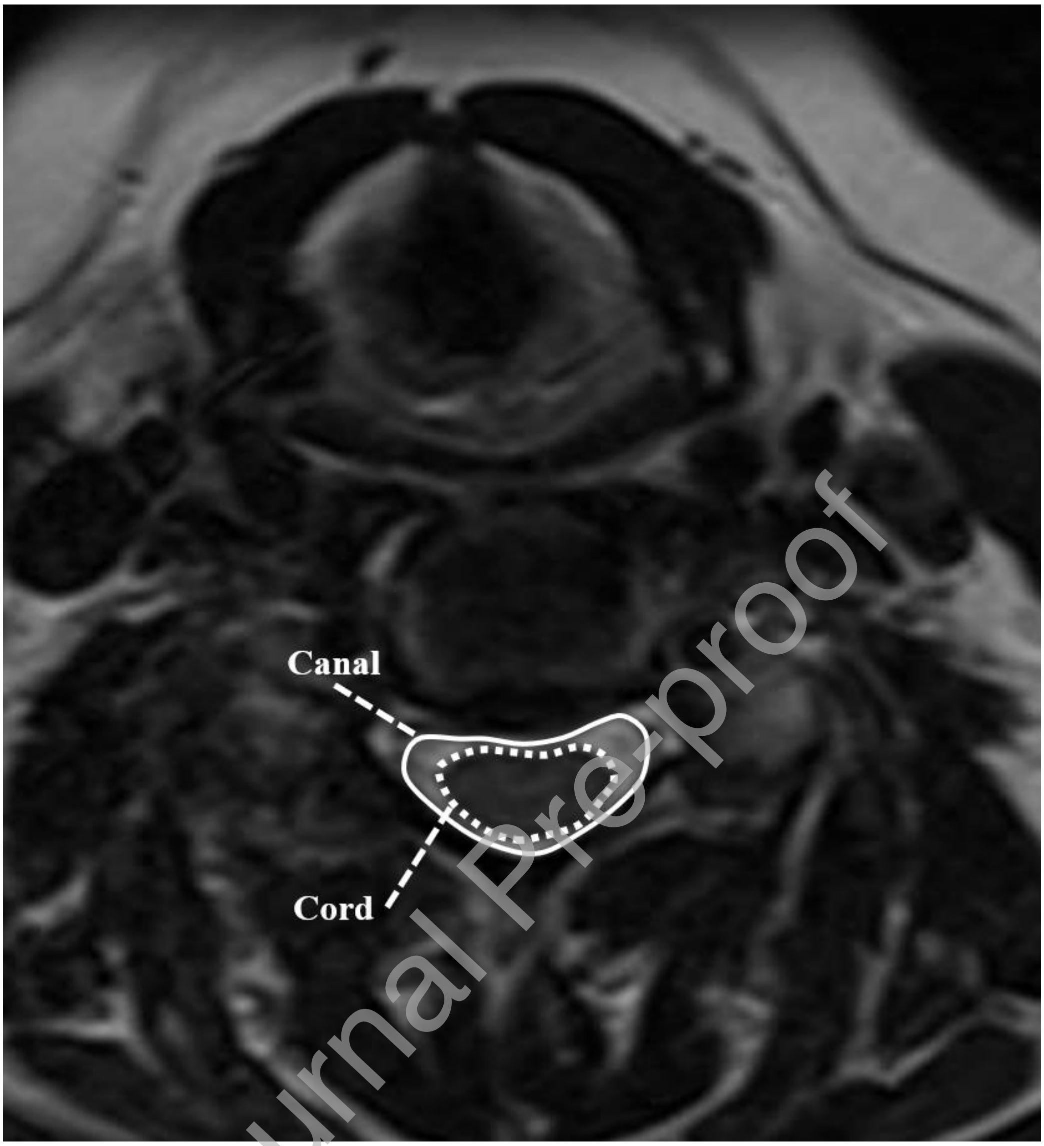

Fig 1E

C5 Palsy Float Back

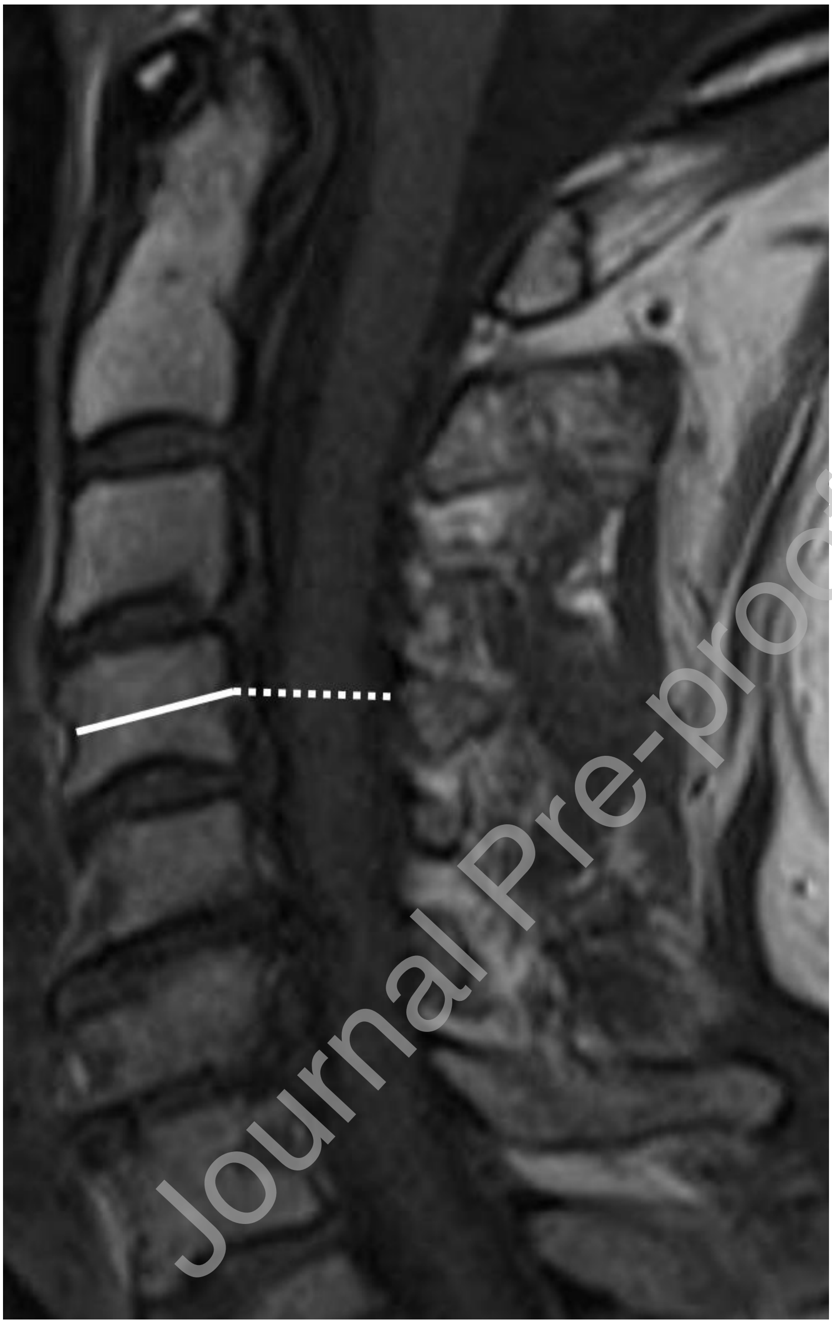

Fig 1F

C5 Palsy Float Back

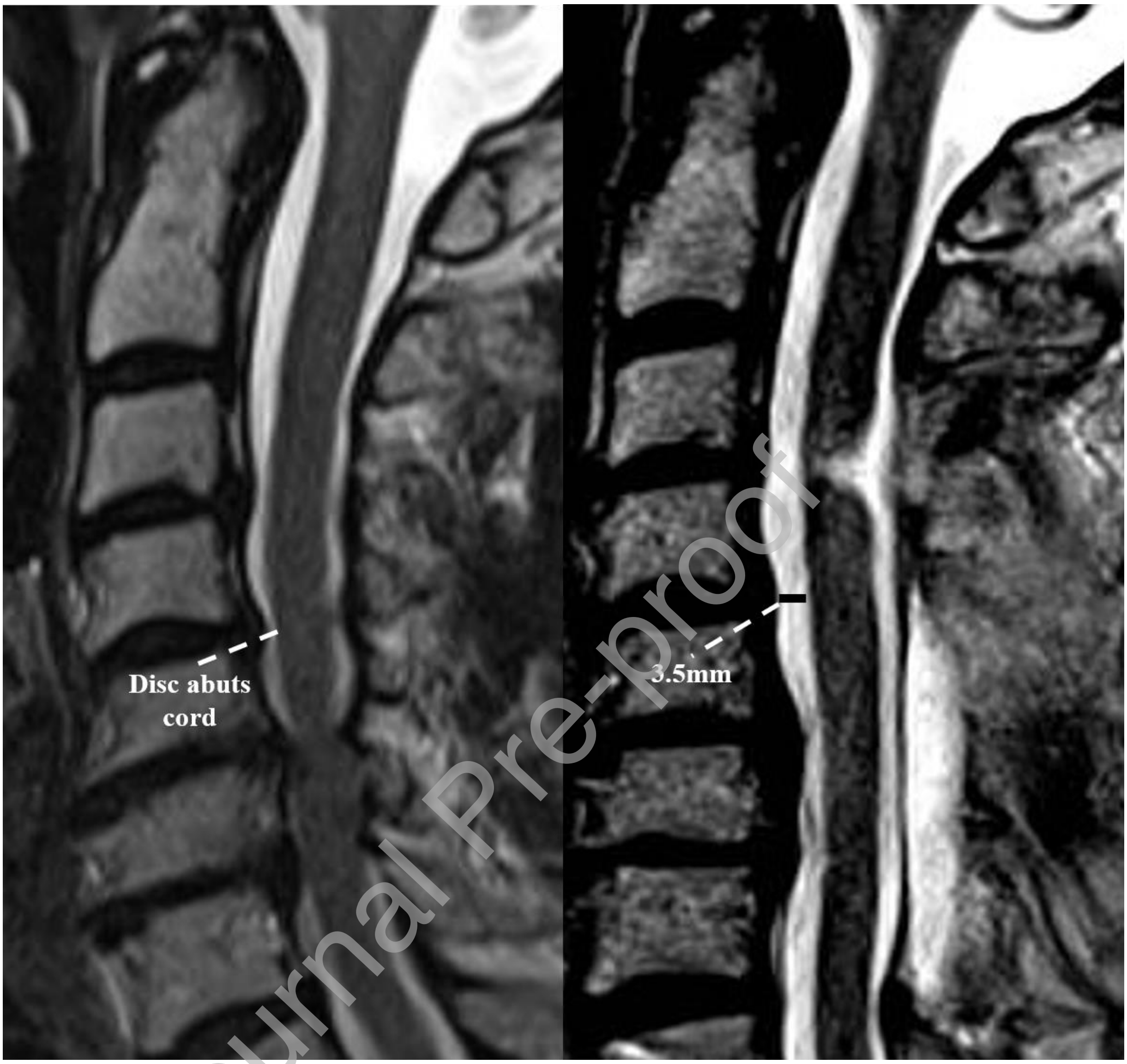

Fig 1G

C5 Palsy Float Back

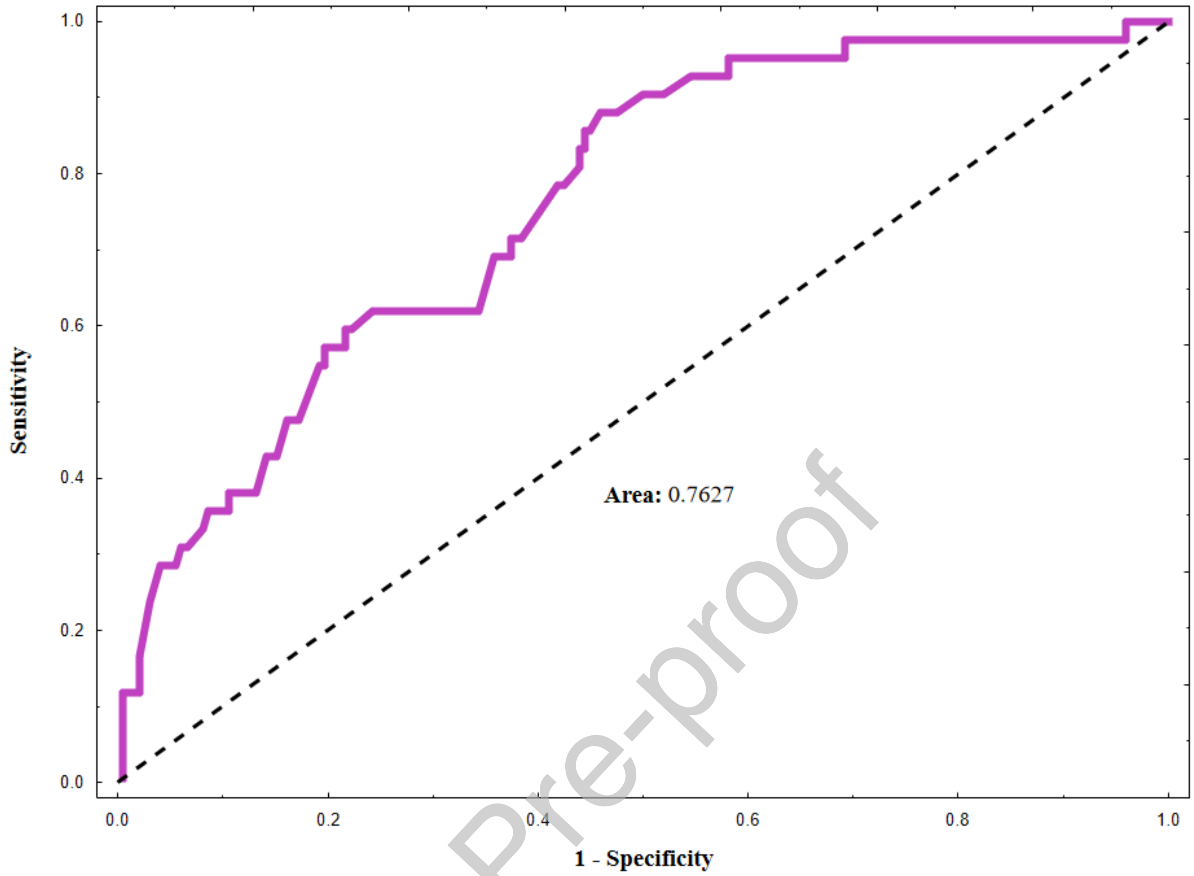

Figure 2.

Table 4: Details of included patients (n = 242)

Field	**Mean** (SD)	**Median**	**n (%)**
Demographics			
Age (yr)	62.39 (10.61)	63.5	—
Follow-up (mo)	27.90 (30.38)	16	—
Ht (m)	1.70 (0.11)	1.70	—

Wt (kg)	85.38 (21.91)	81.8	—
BMI (kg/m²)	29.13 (6.54)	27.99	—
Race			
White	—	—	140 (57.85)
Black	—	—	76 (31.40)
Asian	—	—	10 (4.13)
Other	—	—	16 (6.61)
Sex			
Male	—	—	159 (65.70)
Female	—	—	83 (34.30)
Smoking History			
Current	—	—	41 (16.94)
Former	—	—	53 (21.90)
Never	—	—	148 (1.16)
Presenting Symptoms			
Myelopathy	—	—	217 (89.3)
Abnormal Gait/Instability	—	—	142 (58.68)
Bowel/Bladder Symptoms	—	—	18 (7.44)
Dropping Objects	—	—	45 (18.60)
Hand Clumsiness	—	—	113 (46.70)
Paresthesias/Dysesthesias	—	—	136 (56.20)
Radiculopathy	—	—	44 (18.18)
C5 Radiculopathy	—	—	20 (8.26)
Nurick Grade			

0	—	—	51 (21.07)
1	—	—	66 (27.27)
2	—	—	64 (26.45)
3	—	—	40 (16.53)
4	—	—	15 (6.20)
5	—	—	6 (2.48)
Neurological Exam			
Clonus	—	—	39 (16.12)
Pathological Reflexes			
Babinski	—	—	39 (16.12)
Hoffman	—	—	71 (29.34)
Hyperreflexia	—	—	113 (46.69)
Weakness	—	—	161 (66.53)
BUE Motor Exam			
RUE C5	4.66 (0.67)	5	—
RUE C6	4.70 (0.62)	5	—
RUE C7	4.69 (0.60)	5	—
RUE C8	4.60 (0.68)	5	—
RUE T1	4.46 (0.83)	5	—
LUE C5	4.63 (0.70)	5	—
LUE C6	4.65 (0.64)	5	—
LUE C7	4.62 (0.63)	5	—
LUE C8	4.59 (0.65)	5	—
LUE T1	4.42 (0.82)	5	—

C5 Palsy Float Back

Prior Surgery	—	—	61 (25.21)
Post-operative C5 Palsy	—	—	44 (18.18)

Key: BUE – bilateral upper extremity; LUE – left upper extremity; RUE – right upper extremity

C5 Palsy Float Back

Table 5: Univariable analysis comparing patients who did and did not experience post-operative C5 palsy

Field	Non-Palsy	Palsy	p-value
Demographics	200 (82.64)	42 (17.36)	
Age (yr)	62.09 (10.96)	63.86 (8.73)	0.39
Follow-up (mo)	29.94 (31.55)	18.21 (21.90)	0.05
Ht (m)	1.70 (0.12)	1.72 (0.09)	0.22
Wt (kg)	84.51 (21.81)	89.49 (22.21)	0.13
BMI (kg/m²)	28.94 (6.56)	30.08 (6.47)	0.27
Race			0.44
White	113 (56.50)	27 (64.29)	—
Black	66 (33.00)	10 (23.81)	—
Asian	7 (3.50)	3 (7.14)	—
Other	14 (7.00)	2 (4.76)	—
Sex			0.28
Male	128 (64.00)	31 (73.81)	—
Female	72 (36.00)	11 (26.19)	—
Smoking History			0.62
Current	34 (17.00)	7 (16.67)	—
Former	46 (23.00)	7 (16.67)	—
Never	120 (60.00)	28 (66.67)	—
Presenting Symptoms			
Myelopathy	181 (90.50)	35 (83.33)	0.18
Abnormal Gait/Instability	124 (62.00)	18 (42.86)	0.03
Bowel/Bladder Symptoms	16 (8.00)	2 (4.76)	0.75

Dropping Objects	36 (18.00)	9 (21.43)	0.66
Hand Clumsiness	91 (45.50)	22 (52.38)	0.50
Paresthesias/Dysesthesias	115 (57.50)	22 (52.38)	0.61
Radiculopathy	33 (16.50)	11 (26.19)	0.18
C5 Radiculopathy	14 (7.00)	6 (14.29)	0.13
Nurick Grade	1.74 (1.29)	1.33 (1.22)	0.32
0	38 (19.00)	13 (30.95)	—
1	53 (26.50)	13 (30.95)	—
2	57 (28.50)	7 (16.67)	—
3	33 (16.50)	7 (16.67)	—
4	13 (6.50)	2 (4.76)	—
5	6 (3.00)	0 (0)	—
Neurological Exam			
Clonus	34 (17.00)	5 (11.90)	0.50
Pathological Reflexes			
Babinski	29 (14.50)	10 (23.81)	0.16
Hoffman	58 (29.00)	13 (30.95)	0.85
Hyperreflexia	92 (46.00)	21 (50.00)	0.73
Weakness	135 (67.50)	26 (61.90)	0.48
Imaging Details			
# Levels Compressed†	3.05 (1.31)	3.23 (1.14)	0.62
# Levels with T2 Hyperintensity	1.09 (1.17)	1.00 (1.01)	0.82
Preoperative C4/5 Anatomy			
Kang Score	1.74 (1.04)	2.07 (0.93)	0.06

CVFS Distance (cm)	0.03 (0.07)	0.02 (0.04)	0.52
Foraminal Diameter (mm)	3.2 (0.8)	2.8 (0.9)	<0.001
Facet-Gutter Ratio	82.36 (5.30)	82.59 (5.18)	0.81
SAP (mm)	3.70 (2.39)	4.12 (1.53)	0.04
Pavlov-Torg	0.74 (0.12)	0.73 (0.12)	0.48
AP Diameter (cm)	0.75 (0.23)	0.72 (0.23)	0.17
CSA – cord (cm^2)	0.75 (0.17)	0.71 (0.21)	0.15
CSA – canal (cm^2)	1.25 (0.46)	1.11 (0.40)	0.13
CSA (cord):CSA (canal)	0.65 (0.20)	0.67 (0.18)	0.65
CLA (°)	35.10 (3.68)	34.31 (3.61)	0.27
Postoperative C4/5 Anatomy			
CVFS Distance (cm)	0.31 (0.16)	0.37 (0.16)	0.02
Cord Float back (cm)	0.28 (0.17)	0.35 (0.17)	0.02
Surgical Details			
Prior Surgery	53 (26.50)	8 (19.05)	0.43
Decompression			
Laminectomy Length	3.59 (1.48)	3.96 (1.02)	0.12
C4 laminectomy	174 (87.00)	40 (95.24)	0.18
C5 laminectomy	160 (80.81)	41 (97.62)	0.004
C4+5 laminectomy	157 (78.50)	40 (95.24)	0.009
C4-5 laminectomy ± 1lvl	92 (46.00)	23 (54.76)	0.31
C4-5 laminectomy ± 2lvl	7 (3.50)	1 (2.38)	0.99
C4/5 Foraminotomy	58 (29.00)	15 (35.71)	0.46
Fusion			

Construct Length	5.65 (1.99)	5.97 (1.37)	0.37
C4 instrumentation	194 (97.00)	41 (97.62)	0.99
C5 instrumentation	194 (97.00)	41 (97.62)	0.99
C4-5 instrumentation	194 (97.00)	41 (97.62)	0.99
C4-5 instrumentation±1lvl	162 (81.00)	40 (95.24)	0.02
C4-5 instrumentation±2lvl	99 (49.50)	21 (50.00)	0.99
C4-5 instrumentation±3lvl	4 (2.00)	0 (0)	0.99
Fusion and Laminectomy – C4-5	151 (75.50)	40 (95.24)	0.003

Key: AP – anterior-posterior; BUE – bilateral upper extremity; CLA – cord-laminar angle; CSA – cross-sectional area; CVFS – cord-vertebral face separation; LUE – left upper extremity; lvl – level; RUE – right upper extremity; SAP, projection of superior articular process in front of the posterior disc face

†Compression defined as AP diameter < 0.8cm

C5 Palsy Float Back

Table 6: Multivariable analysis of surgical predictors of post-operative C5 palsy

Variable	OR	95% CI		p-value
		LL	UL	
Signs/Symptoms				
C5 Radiculopathy	1.70	0.94	3.05	0.08
Surgery Details				
C5 laminectomy	2.74	0.97	7.78	0.06
C4-5 instrumentation±1lvl	2.01	0.95	4.28	0.06
Local Anatomy				
Foraminal Diameter – C4/5 (per mm)	0.38	0.22	0.64	<0.01