Stress analysis of the cervical spinal cord: Impact of the morphology of spinal cord segments on stress

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Objective: Although there are several classifications for cervical myelopathy, these do not take differences between spinal cord segments into account. Moreover, there has been no report of stress analyses for individual segments to date.

Methods: By using the finite element method, we constructed 3-dimensional spinal cord models comprised of gray matter, white matter, and pia mater of the second to eighth cervical vertebrae (C2–C8). We placed compression components (disc and yellow ligament) at the front and back of these models, and applied compression to the posterior section covering 10%, 20%, 30%, or 40% of the anteroposterior diameter of each cervical spinal cord segment.

Results: Our results revealed that, under compression applied to an area covering 10%, 20%, or 30% of the anteroposterior diameter of the cervical spinal cord segment, sites of increased stress varied depending on the morphology of each cervical spinal cord segment. Under 40% compression, stress was increased in the gray matter, lateral funiculus, and posterior funiculus of all spinal cord segments, and stress differences between the segments were smaller.

Conclusion: These results indicate that, under moderate compression, sites of increased stress vary depending on the morphology of each spinal cord segment or the shape of compression components, and also that the variability of symptoms may depend on the direction of compression. However, under severe compression, the differences among the cervical spinal segments are smaller, which may facilitate diagnosis.

Key words: Crandall's classification, Cervical myelopathy, Finite element model, Hattori's classification, Spinal cord injury

Introduction

Cervical myelopathy is a disease causing dysfunction of the cervical spinal cord, and can be caused by osteophytes, or by ossified ligaments, intervertebral discs, or ligamenta flava. The onset of symptoms is associated with static^{1,2} and dynamic^{3,4} compression factors, such as encroachment by the ligamentum flavum and the pincer mechanism. The pathophysiology and symptoms of cervical myelopathy are described using Hattori's⁵ and Crandall's⁶ classifications. Hattori's classification is based on the patterns of symptom progression with reference to the cross-sectional area of lesions. According to this classification, Type I includes

instances where the gray matter in the central section is damaged, Type II includes instances where a lesion extends to the lateral and posterior funiculi, and Type III includes instances where a lesion extends to the spinothalamic tract in the ventrolateral funiculus. Crandall and Batzdorf classified patients into 5 groups based on their dominant syndromes⁶:

1. The transverse syndrome involves the corticospinal, spinothalamic, and dorsal column tracts and produces severe spasticity, frequent sphincter involvement, and Lhermitte sign.
2. The motor system syndrome involves the anterior horn cells and corticospinal tracts and produces marked spasticity but no sensory disturbances.
3. The central cord syndrome involves grey matter, central portion of corticospinal and spinothalamic long tracts and produces severe motor and sensory

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disturbances with a greater expression of weakness in the upper extremities and spasticity in the lower extremities.

4. The Brown-Séquard syndrome involves involves ipsilateral loss of motor function resulting from corticospinal tract interruption, combined with contralateral loss of pain and temperature sensation as a result of spinothalamic tract dysfunction.
5. The brachialgia and cord syndrome involves the lower motor neurons of the upper extremities and produces radicular pain.

Although these existing classifications are well established, they do not take into consideration the morphological differences between spinal cord segments. For instance, Kameyama *et al.* present histological data from spinal cord segments obtained from autopsies of patients without neurologic symptoms⁷ and show that the shape of gray and white matter varies across segments. Similarly, Ichihara *et al.* have shown that the material constants differ between gray and white matter, with gray matter being stiffer but more prone to failure at a lower mechanical strain compared to white matter.⁸ Based on these reports, under a given compression, stress distribution in spinal cord segments may differ according to the shape of gray and white matter. Despite this, no studies have yet investigated the stress distributions of different spinal cord segments. In an effort to bridge this

gap in the literature, we constructed 3-dimensional models of the second to seventh cervical spinal cord segments (C2 to C8) using the finite element method based on the organization chart presented by Kameyama *et al.*,⁷ and performed stress analyses of each segment. In this way, we were able to evaluate whether stress distributions differ between the different spinal cord segments. The results are reported here.

Materials and methods

The ABAQUS 6.11 finite element package (Valley Street, Providence, RI, USA) was used for finite element analysis. In order to construct a 3-dimensional finite element model of the cervical spinal cord, two orthopedists plotted the gray and white matter in the left side of 7 spinal cord segments (C2 to C8), as described by Kameyama *et al.*,⁷ and the mean values of measured coordinates were determined. Each segment was then reduced or magnified in order to normalize the anteroposterior diameter of all spinal cord segments to approximately 10 mm, including the pia mater. Because stress would be affected by the presence or absence of pia mater,⁹ the pia mater was modeled around each spinal cord segment and at an equal thickness, partly due to a lack of the literature on differences in pia mater thickness among the segments, and also in order to minimize the impact of the pia mater on stress analysis (Fig. 1). In order to facilitate the analysis, our 3-dimensional finite element model did not include denticulate ligaments, radicle, dura mater, or spinal fluid. The spinal cord was assumed to be symmetrical about the mid-sagittal plane, such that only half the spinal cord required reconstruction and the whole model could be integrated by mirror image for each of the 7 spinal cord segments. Based on the length of two vertebral bodies measured on images of spinal computed tomography (CT), the vertical length of all segments was set at 40 mm. Young's modulus and Poisson's ratio of gray and white matter was set according to long-term static data obtained from stress relaxation tests of the bovine spinal cord, reported by Ichihara *et al.*,⁸ and the material constant of the pia mater was set according to data from Tuntani.¹⁰

As a model of the peripheral structures of the cervical spinal cord, intervertebral discs or a flat plate (intended osteophytes) was placed at the front. The flat plate was centrally positioned, and a posterior component (ligamentum flavum or intended calcification) was applied to the spinal cord model.

Using measurements from contrast-enhanced spinal CT images, the angle of the posterior component was set at 30° from the center of the dorsal side to the

Figure 1 Spinal cord model comprised of the second to eighth cervical spinal cord segments (C2–C8) (gray matter, white matter, and pia mater).

Figure 2 Compression components were placed in front of (anterior) and at the back of (posterior) the spinal cord model, and the angle of a ligament was set to 30°.

lateral side. Moreover, because the part encroached by the ligamentum flavum is located slightly below the center, the posterior component was placed 5 mm distal from the center (Fig. 2). These two compression components (anterior/posterior) were assigned with a material constant of a substantially hard material in order to prevent the spinal cord model from having any impact on them. The cranial and caudal sides of the spinal cord model were completely fixed. It was assumed that no friction existed among the gray matter, white matter, and pia mater, and that they were firmly adhered to one other; because there were no reports describing the anterior/posterior compression components and friction coefficients of the cervical spinal cord, to our knowledge, friction was set as “no friction.” To clarify, the positioning and material of the anterior and posterior compression components, the positioning of the spinal cord model, and the friction (i.e. everything except for the morphology of the spinal cord segments) were kept constant.

While static and dynamic compression are well-documented compression factors,^{1–4} Machino *et al.*, Miura *et al.*, and Muhle *et al.* have further reported that spinal stenosis caused by posterior compression, via the pincer mechanism in posterior flexion or via encroachment by ligamentum flavum, is a significant contributing factor to the onset of myelopathy.^{11–13} These findings suggest that an extension of the spinal cord causes ligamenta flava and/or calcified ligaments

to encroach on its posterior part. In order to model this posterior encroachment, the cranial and dorsal sides of our cervical spinal cord model were completely fixed, as was the anterior compression component. The posterior compression component was angled toward the ventral side, and compression was applied to an area covering approximately 10%, 20%, 30%, or 40% of the anteroposterior diameter of each spinal cord segment (Fig. 3).

For the cervical spinal cord, anterior compression component, and ligaments (ligamenta flava), symmetrical mesh was applied at the 15th or 20th node. The node average of the spinal cord segments was 52650 (range: 43574–58156), and the mean element count was 9291 (range: 7920–11520). Stress analyses were performed for 28 different conditions (4 compression areas $\times$ 7 spinal cord segments), and charts for stress increases at axial positions with the most severe posterior compression were compared.

Results

The strongest and most characteristic stress was shown from compression at the rear of the spinal cord.

Under compression applied to an area covering 10% of the anteroposterior diameter of the spinal cord, stress was increased mainly in the dorsal horn of all spinal cord segments (C2–C8), but this increase in stress was mild (Fig. 4).

Figure 3 Compression was applied from the ligamentum flavum to a posterior area covering 10%, 20%, 30%, or 40% of the anteroposterior diameter of the spinal cord.

Under compression applied to an area covering 20% of the anteroposterior diameter of the spinal cord, stress was increased mainly on the lateral side of the posterior funiculus of segments C2 and C3. At the lower spinal cord segments, stress was increased mainly in the lateral funiculus and the cuneate fasciculus (Fig. 5).

Under compression applied to an area covering 30% of the anteroposterior diameter of the spinal cord, stress was increased in the cuneate fasciculus of the posterior funiculus and in the lateral funiculus. The area of increased stress expanding towards the anterolateral funiculus and the gray matter of the anterior horn varied between spinal cord segments (Fig. 6).

Under compression applied to an area covering 40% of the anteroposterior diameter of the spinal cord, stress was increased in the entire area of gray matter/

lateral funiculus and gracile fasciculus of the posterior funiculus, as well as the anterior funiculus. Overall, the stress differences among the spinal cord segments were smaller than those seen at 10%, 20%, and 30% compression values (Fig. 7).

Discussion

Hattori's⁵ and Crandall's⁶ classifications are excellent classifications. However, compression applied to the spinal cord does not necessarily cause identical symptoms in all patients, and so symptoms do not always apply to these classifications. For example, Kokubun *et al.*, Hirabayashi *et al.* and Imajo *et al.* respectively reported that the symptoms of cervical spondylotic myelopathy at C3/4 include percentage of dysesthesia, hyperreflexia of biceps tendon reflex and shoulder muscle weakness.^{14–16} In an attempt to address this symptomatological variability, we hypothesized that the distributions of compression-induced stress could vary as a result of individual differences in the morphology of spinal cord segments or as a result of different compression patterns. We therefore performed stress analyses for each spinal cord segment.

Previous studies were conducted by Kato *et al.*^{17–19} and we have also carried out other studies using the same model.^{20–23} Employing other FEM model of the spinal cord, Carolyn *et al.* investigated the injury mechanism using cervical spine with spinal cord.²⁴ Maikos demonstrated that induced mechanical strain correlated

Figure 4 Stress charts of each spinal cord segment model under compression applied to an area covering 10% of the anteroposterior diameter (the graph is color-coded according to stress values).

Figure 5 Stress charts of each spinal cord segment model under compression applied to an area covering 20% of the anteroposterior diameter.

with the extent of the damage to spinal cord.²⁵ Czyn *et al.* reported the 3D FEM spinal cord model, based on Magnetic Resonance Image (MRI), showed good agreement with experimental results of the porcine spinal cord.^{26–27} Li *et al.* reported on the injury mechanism for acute central cord syndrome and hyperextension injury.^{28–29} They concluded that bovine and human

cranial nerves and spinal cords follow similar pathological courses.²⁸ Thus, we considered it appropriate to use the material constant for bovine spinal cord in the present study. Meyer *et al.* performed stress analysis due to the motion of the spinal cord.³⁰ Takahashi *et al.* reported on the relationship between high signal intensity on MRI and FEM model.³¹ The above

Figure 6 Stress charts of each spinal cord segment model under compression applied to an area covering 30% of the anteroposterior diameter.

Figure 7 Stress charts of each spinal cord segment model under compression applied to an area covering 40% of the anteroposterior diameter.

papers evaluated the stress distribution and thus provide valuable background information for understanding the injury mechanism of spinal cord. However, the pathology of the gray matter and white matter that caused the symptoms was not evaluated. The emphasis of the present study was on the injury mechanism relating to the pathology.

Ono *et al.*³² reported the pathology of compressive cervical myelopathy as follows: (1) mild compression mainly causes degeneration of the lateral funiculus, and demyelination of the posterior funiculus is not as severe as that of the lateral funiculus; (2) the anterior funiculus is relatively preserved; and (3) the severity of compression strongly correlates with that of histological damage. Ito *et al.*³³ reported that severe compression causes necrosis and defluction of the gray matter and the ventrolateral side of the posterior funiculus. Together, these findings are consistent with those of the stress charts of posterior compression obtained in the present study, which not only indicates that our spinal cord model is histologically relevant, but also that posterior compression, which is dynamic compression, is a significant contributor to spinal cord degeneration.

Kokubun *et al.*¹⁴ report that cervical myelopathy starts with numbness of the fingers, which is followed by spastic gait, and then by bladder and rectal disturbance. This corresponds to the transition from Type I to Type II and then to Type III of Hattori's

classification, and from the central cord syndrome to the transverse lesion syndrome according to Crandall's classification. Because these popular classifications do not identify specifically which spinal cord segment is the source of numbness of the fingers (the initial symptom), Imajo *et al.* applied electrical stimulation during surgery for cervical myelopathy at C3/4 and recorded evoked potentials of the spinal cord in neural pathways. They found that numbness of the fingers was attributable to the cuneate fasciculus of the posterior funiculus, which the authors identified as being prone to damage. Also the severity of symptoms subsequently advanced, the lateral funiculus and then the gracile fasciculus of the posterior funiculus were also damaged.¹⁶ Similarly, analysis of the C5 spinal cord segment revealed that stress was increased in the cuneate fasciculus and the lateral funiculus, whereas stress in the gracile fasciculus was not increased unless compression was severe. These results are also consistent with the histological findings of Ito *et al.*,³³ which demonstrate that, while the lateral funiculus and cuneate fasciculus of the posterior funiculus necrotizes in cervical spondylotic myelopathy, the gracile fasciculus does not. We found that the courses of increase in stress also followed this pattern in the other spinal cord segments.

The analyses performed in the present study have limitations: damage caused by the motion of the spinal cord itself, such as stretch-associated injury of

the spinal cord,³⁴ was not taken into account. Moreover, the model was symmetrical and only compression in the anteroposterior direction was applied. In addition, the effect of the speed of compression of components and the impact of aging were not considered.³⁵ In order to facilitate analysis, the models did not include nerve roots, denticulate ligaments, dura mater or spinal fluid. With respect to blood flow, Ono *et al.* reported ischemic changes in the white matter and gray matter at the stenosis level.³² In the present study, we did not take into account that stress distribution caused by compression may have affected blood flow to gray matter and white matter, thereby affecting the susceptibility or injury of these tissues. Finally, the absence of distance or friction between the anterior and posterior compression components may have also affected stress results. In order for future models to address these various factors, it will be necessary to design models according to specific cases or conditions. We have now emphasized the simplicity of the analysis model and that it may not fully reflect the pathology features of individual cervical myelopathy patients.

However, the stress distributions obtained by our analyses were similar to those described in previous histological and clinical reports. Under compression applied to an area covering 10% to 30% of the anteroposterior diameter of the spinal cord model, sites of increased stress were found to differ depending on differences in spinal cord segments. This indicates that stress distributions could also be changed by the shape or angle of the anterior and posterior compression components, which has formed one explanation as to why symptoms vary among patients, even when they suffer from cervical myelopathy of the same severity. However, under compression applied to an area covering 40% of the anteroposterior diameter of spinal cord, stress was increased in the entire spinal cord, in all spinal cord segments and with smaller differences between the spinal cord segments. Thus, under moderate compression, areas of increased stress vary depending on differences in the compression components and in the segments, while under severe compression, stress differences between segments are smaller, which may explain why the diagnosis of cervical myelopathy is easier to make in more severe cases.

Conclusion

In this study, we constructed models for each spinal cord segment and determined the stress distributions in each of these segments under the same conditions of posterior compression. Stress analyses yielded results consistent with previously reported clinical and histological

findings. Under moderate compression (10% to 30%), patterns of stress increase varied between spinal cord segments, presumably due to their different morphologies. Under severe compression (40%), these differences were found to be smaller.

Disclaimer statements

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