Biomechanical study of the effect of degree of static compression of the spinal cord in ossification of the posterior longitudinal ligament

Laboratory investigation

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Objective. The authors evaluated the biomechanical effect of 3 different degrees of static compression in a model of the spinal cord in order to investigate the effect of cord compression in patients with ossification of the posterior longitudinal ligament (OPLL).

Methods. A 3D finite element spinal cord model consisting of gray matter, white matter, and pia mater was established. As a simulation of OPLL-induced compression, a rigid plate compressed the anterior surface of the cord. The degrees of compression were 10, 20, and 40% of the anteroposterior (AP) diameter of the cord. The cord was supported from behind by the rigid body along its the posterior border, simulating the lamina. Stress distributions inside of the cord were evaluated.

Results. The stresses on the cord were very low under 10% compression. At 20% compression, the stresses on the cord increased very slightly. At 40% compression, the stresses on the cord became much higher than with 20% compression, and high stress distributions were observed in gray matter and the lateral and posterior funiculus. The stresses on the compressed layers were much higher than those on the uncompressed layer.

Conclusions. The stress distributions at 10 and 20% compression of the AP diameter of the spinal cord were very low. The stress distribution at 40% compression was much higher. The authors conclude that a critical point may exist between 20 and 40% compression of the AP diameter of the cord such that when the degree of the compression exceeds this point, the stress distribution becomes much higher, and that this may contribute to myelopathy.

(DOI: 10.3171/2009.9.SPINE09314)

Key Words • myelopathy • finite element method • ossification of the posterior longitudinal ligament

OSSIFICATION of the posterior longitudinal ligament is one of the known causes of cervical myelopathy, and it is especially common in Japan. The mechanism of the myelopathy is considered to be a combination of the static compression caused by the OPLL and the dynamic effect of intervertebral motion. However, the actual location of the lesion is sometimes not in an area where there is movement—for example, it may be in an area of continuous OPLL or canal stenosis. Therefore, we considered that severe static compression may cause myelopathy without the dynamic factor of intervertebral motion. In this study, we simulated 3 different degrees of static compression and evaluated the stress distribution inside the spinal cord using a 3D finite element model.

Methods

The 3D Finite Element Model

A standard solid model with a nonlinear component was used to simulate the viscoelasticity of the spinal cord. We used a combination of linear elastic elements (Ee), a nonlinear elastic element (Ec), and a viscous element (C) to examine time-dependent material response.
Elastic parameters and viscous coefficients were determined mathematically using data obtained from tensile tests of fresh bovine spinal cord. The constitutive equation needed for the FEM calculation for this model was as follows:

\[ \sigma = E \varepsilon = f_s(E_c, \varepsilon^v) + f_d(C, \varepsilon^v, \dot{\varepsilon}^v) \quad [\text{Eq. 1}] \\
\varepsilon = \varepsilon^e + \varepsilon^v \quad [\text{Eq. 2}] 
\]

with stress being represented by \( \sigma \), strain by \( \varepsilon \), elastic strain by \( \varepsilon^e \), viscous strain by \( \varepsilon^v \), and viscous strain rate by \( \dot{\varepsilon}^v \).

The function \( f_s(E_c, \varepsilon^v) \) represented the mechanical property of nonlinear element \( E_c \) and was dependent only on viscous strain. White matter and gray matter were approximated as follows:

\[ f_s(E_c, \varepsilon^v) = E_c \cdot (\varepsilon^v)^2 \quad [\text{Eq. 3}] \]

The function \( f_d(C, \varepsilon^v, \dot{\varepsilon}^v) \) represented the mechanical property of viscous element \( C \) and was a function of viscous strain and viscous strain rate. In this study, we approximated the function to the product \( F(\varepsilon^v) \) that was a function of viscous strain only and a logarithmic function that included viscous strain rate.

\[ f_d(C, \varepsilon^v, \dot{\varepsilon}^v) = F(\varepsilon^v) \cdot \ln (1 + \beta \dot{\varepsilon}^v) \quad [\text{Eq. 4}] \]

Beta (\( \beta \)) was the material property. In short-time deformation, Eqs. 1–4 were used for FEM calculation.

On the other hand, for calculation under static compression, the time needed for deformation was infinite. Strain speed was thus assumed as follows:

\[ \dot{\varepsilon} = \dot{\varepsilon} = \dot{\varepsilon}^v \to 0 \]

Therefore, \( f_d(C, \varepsilon^v, \dot{\varepsilon}^v) \) in Eq. 4 was equal to 0. Equation 1 became:

\[ \sigma = E \varepsilon^e = f_s(E_c, \varepsilon^v) \quad [\text{Eq. 5}] \]

Under static conditions, the FEM calculation was based upon Eqs. 2 and 5.

To determine the mechanical properties of the gray and white matter, the data obtained by the tensile stress strain curve and stress relaxation under various strain rates were used. The mechanical properties of bovine spinal cord change quickly after dissection. The maximum time limit for gaining reliable data was considered to be the time taken to conduct the tensile stress strain curve and stress relaxation under various strain rates. Therefore, the bovine spinal cord itself could not be used for the experiment on stress distribution and simulation and the FEM was used instead. The mechanical properties of the pia mater were based on published data.\(^5\)

The 3D FEM spinal cord model for this study consisted of gray matter, white matter, and pia mater. The denticulate ligament was not included in the model (Fig. 1). The model simulated segmental OPLL. The spinal cord model was at the level of an upper vertebra with OPLL, an intervertebral disc without OPLL, and the upper half of the adjacent lower vertebra with OPLL. The spinal cord at the upper vertebral level comprised the first, second, and third layers; the fourth layer was the intervertebral disc level; and the fifth and sixth layers represented the cord at the level of the upper half of the adjacent lower vertebra (Fig. 2).

The spinal cord was assumed to be symmetrical, and the left-half model was established to simplify calculations. The total number of isoparametric 20-node elements was 780 and the total number of nodes was 3643.\(^1\)

**Static Compression Model**

For simulation of OPLL, anterior static compression was applied to Layers 1–3, 5, and 6, and stress distributions in each layer of the cord were computed. Layer 4 was assumed to be the intervertebral level of the spinal cord and was not compressed (simulating segmental OPLL). The degrees of compression were 10, 20, and 40% of the AP diameter of the spinal cord. First, 10% compression was applied to the spinal cord. This was followed by 20% compression and finally, 40% compression. Simulating OPLL compression, the rigid plate (representing the ossified ligament) compressed the anterior surface of the spinal cord. Degenerative disc disease also compresses the anterior aspect of the cord but the disc lesion is not as rigid as OPLL. Therefore, we considered this simulation to represent a compression model of the effect of OPLL. The spinal cord was supported from behind by the rigid body along its posterior border, simulating the lamina (Fig. 3).
Influence of static compression

**Results**

The stresses of the spinal cord were very low under 10% compression. The stresses on the compressed layers were a little higher than those on the uncompressed layer (Fig. 4). After 20% compression was applied, the stresses on the spinal cord increased very slightly (Fig. 5). After 40% compression, the stresses on the spinal cord became much higher than with 20% compression, and high stress distributions were observed in gray matter and the lateral and posterior funiculus. The stresses on the compressed layers were much higher than those on the uncompressed layer (Fig. 6).

**Discussion**

Myelopathy in patients with OPLL is considered to be caused by the combination of a static factor, which is compression of OPLL, and a dynamic factor, which is intervertebral motion. Matsunaga et al. reported the overall range of motion in the group with myelopathy was significantly greater than that in the group without myelopathy. Nishiura et al. reported that minor trauma–induced myelopathy in OPLL patients occurred at the edge or noncontinuous portion of the OPLL. These reports indicate that a dynamic factor is an essential part of the pathogenetic mechanism of myelopathy in OPLL patients. On the other hand, Matsunaga et al. also reported that the patients in whom the minimum diameter of the space available for the spinal cord was less than 6 mm all suffered from myelopathy. From this fact, we hypothesized that canal stenosis due to OPLL may cause myelopathy without a dynamic factor and that there may be a critical threshold of stenosis, beyond which myelopathy may be caused by static compression alone. To prove the hypothesis, we investigated 3 different degrees of static compression and calculated the stress distributions inside the spinal cord, simulating OPLL using a 3D finite element model. Because we used static compression in this simulation, the computed stress distribution was equivalent to that under chronic compression. Although the model used the immobile segment of the spinal column, the spinal cord inside the column at this segment still has some mobility in OPLL patients.

Bovine spinal cord was used for our model since it was impossible to obtain fresh human spinal cord. It is not known whether the mechanical properties of human and bovine spinal cords differ, and hence the model we used may not accurately simulate the human condition. However, for the purpose of this study we assumed the mechanical properties of spinal cord from the 2 species were similar.

Under the compression of 10 and 20% of the AP diameter of the spinal cord, the stress distributions were very low. This may correspond to cases of OPLL in which there is little or no myelopathy. Under 40% compression,
the stress distribution became much higher. Continuously high stress distribution may result in microvascular changes that lead to ischemia in the cord. Moore et al. have described the relationship between duration of ischemia and severity of neurological deterioration and degree of recovery. This high stress distribution can cause myelopathy without a dynamic factor in OPLL patients. This may correspond to cases of OPLL with myelopathy without a dynamic factor. In these patients the portions of the lesion causing myelopathy are the areas of canal stenosis at the level of the vertebral body affected by the OPLL, not the areas associated with intervertebral mo-

**Fig. 4.** Cross-sectional views of stress distributions at 10% compression of the AP diameter of the cord. A: The third layer of the model, representing the upper vertebral level (compressed). B: The fourth layer of the model, representing the disc level (not compressed). C: The fifth layer, representing the lower vertebral level (compressed).

**Fig. 5.** Cross-sectional views of stress distributions at 20% compression. A: Third layer, representing the upper vertebral level. B: Fourth layer, representing the disc level (not compressed). C: Fifth layer, representing the lower vertebral level.
Influence of static compression

Judging from the results of our simulation, when the degree of the compression by OPLL is less than some critical point and there is no dynamic factor at play, the stress distribution is very low. When the degree of the compression by OPLL exceeds some critical point, the stress distribution becomes very high. The high stress distribution may cause myelopathy without the presence of a dynamic factor. In our simulation, the degree of compression was limited to only 3 different degrees—10, 20, and 40%. Therefore, we considered that to decide the critical point at which myelopathy occurred was impossible. However, the difference of stress distribution between 20 and 40% was very large. Hence, we concluded the critical point may be between 20 and 40% of the AP diameter of the spinal cord.

**Conclusions**

We studied the effect that different degrees of static compression of the spinal cord might produce in OPLL patients using 3D FEM simulation. The stress distribution in the spinal cord at 10 and 20% compression of its AP diameter was very low. The stress distribution at 40% compression was much higher. We conclude that some critical point may exist between 20 and 40% compression, and when the degree of cord compression exceeds the critical point, the stress distribution becomes much higher, potentially contributing to myelopathy.

**Disclosure**

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

**References**


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Fig. 6. Cross-sectional views of stress distributions at 40% compression. **A:** Third layer (upper vertebral level). **B:** Fourth layer (disc level, not compressed). **C:** Fifth layer (lower vertebral level).