INTRODUCTION: Acute traumatic central cervical spinal cord injury without fracture or dislocation develops classically during a hyperextension injury in patients with spondylosis. The mechanism of the injury could be a traumatic compression of the spinal cord caused by the presence of anterior osteophytes and the buckling of the ligamentum flavum, however, the precise pathophysiology of this spinal cord injury is still unknown.

In the present study, mechanical characteristics of the spinal cord were used in a computerized simulation model to predict the stress distribution within the spinal cord during a combination of acute and chronic compression, and we discuss the pathophysiology of this spinal cord injury.

METHODS/MATERIALS: The mechanical properties of the spinal cord (Ichihara 2001, 2003) were included in the Finite Element Method (FEM), allowing us to model transverse cord compression (Fig. a).

Model A: Chronic compression from the anterior direction.
Model B: Chronic compression from the anterior direction + acute moderate compression from the posterior direction.
Model C: Chronic compression from the anterior direction + acute severe compression from the posterior direction.

We hypothesized that irreversible injury in the white matter occurs at 27kPa (Bain 2001) and in the gray matter occurs at 43kPa (Ichihara 2001).

RESULTS: Model A: All gray matter and white matter were areas of low stress distribution (Fig. b).
Model B: The lateral and posterior columns were areas of high stress distribution (Fig. c).
Model C: The stresses in the white matter adjacent to the gray matter, and all gray matter were areas of high stress distribution (Fig. d).
DISCUSSION: Acute traumatic central cervical spinal cord injury without fracture or dislocation was defined as hemorrhage, necrosis or contusion within the central portion of the spinal cord by Schneider (1958). However, Bunge (1992) reported no evidence of blood or blood products within the cord parenchyma: the primary finding was diffuse disruption of axons within the lateral columns of the cord, but central gray matter was intact.

From our FEM results, rapid traumatic spinal cord compression from the posterior direction tends to selectively injure the white matter adjacent to the gray matter, especially in the lateral and posterior columns. There is no destruction in the gray matter because the gray matter is more rigid than the white matter (Ichihara 2001). This condition was reported by Bunge and matches the results of our model B (Fig. c).

In addition, greater stress during spinal cord compression from posterior direction immediately destroys all of the gray matter, which is mechanically more fragile than the white matter (Ichihara 2001). This damage produces a hemorrhage and maceration in the center of the spinal cord (Fig. d). This matches the report by Schneider.

Because the presence of hemorrhage is related to bad prognosis in spinal trauma, Bunge’s stage follows Schneider’s stage (Table), and both were very similar in spinal cord injury from posterior direction with cervical spondylosis.

CONCLUSIONS: We analyzed the stress distribution of the acute traumatic central cervical spinal cord injury without fracture or dislocation with FEM utilizing mechanical features of the spinal cord. We hypothesize that there are some stages in the acute traumatic central cervical spinal cord injury without fracture or dislocation, and Bunge’s stage follows Schneider’s stage.

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