Epidemiological study of cervical cord compression and its clinical symptoms in community-dwelling residents

Toru Hirai, Koji Otani*, Miho Sekiguchi, Shin-ichi Kikuchi, Shin-ichi Konno
Department of Orthopaedic Surgery, Fukushima Medical University School of Medicine, Fukushima, Japan
* kotani@fmu.ac.jp

Abstract

Background
Degenerative compressive myelopathy (DCM) is caused by cervical cord compression. The relationship between the magnitude and clinical findings of cervical cord compression has been described in the literature, but the details remain unclear. This study aimed to clarify the relationship between the magnitude and clinical symptoms of cervical cord compression in community-dwelling residents.

Methods
The present study included 532 subjects. The subjective symptoms and the objective findings of one board-certified spine surgeon were assessed. The subjective symptoms were upper extremity pain and numbness, clumsy hand, fall in the past 1 year, and subjective gait disturbance. The objective findings were: Hoffmann, Trömner, and Wartenberg signs; Babinski’s and Chaddock’s signs; hyperreflexia of the patellar tendon and Achilles tendon reflexes; ankle clonus; Romberg and modified Romberg tests; grip and release test; finger escape sign; and grip strength. Using midsagittal T2-weighted magnetic resonance imaging, the anterior–posterior (AP) diameters (mm) of the spinal cord at the C2 midvertebral body level ($D_{C2}$) and at each intervertebral disc level from C2/3 to C7/T1 ($D_{C2/3-C7/T1}$) were measured. The spinal cord compression ratio ($R$) for each intervertebral disc level was defined and calculated as $D_{C2/3-C7/T1}$ divided by $D_{C2}$. The lowest $R$ (LR) along C2/3 to C7/T1 of each individual was divided into 3 grades by the tertile method. The relationship between LR and clinical symptoms was investigated by trend analysis.

Results
The prevalence of subjective gait disturbance increased significantly with the severity of spinal cord compression ($p = 0.002812$), whereas the other clinical symptoms were not significantly related with the severity of spinal cord compression.
Conclusions

The magnitude of cervical cord compression had no relationship with any of the neurologic findings. However, subjective gait disturbance might be a better indicator of the possibility of early stage cervical cord compression.

Introduction

Degenerative compressive myelopathy (DCM) can be caused by mechanical [1–5] and dynamic [6–9] compression of the cervical spinal cord, and has a variety of clinical presentations, including subjective symptoms and objective findings [10–15]. Disabilities secondary to the subjective symptoms of cervical myelopathy appear either in the upper extremities, lower extremities, or both. For example, clumsiness of the hand and fingers, and inability to grip, and dysesthetic pain are well-known characteristics of myelopathy hands [12]. In the lower extremity, unsteady gait, difficulty in going down and/or up stairs, and spontaneous muscle cramping may be present. Objective findings include long tract signs [12,14], which are brought about by the failure of the white matter [12,15] of the spinal cord conduction pathway. These signs comprise symptoms pertaining to the pyramidal tract, posterior column, and spinothalamic tract, and they include: clumsy hands; spastic paralysis; gait disturbance, including motor impairment of the lower extremities; hyperreflexia of the lower extremities; and the presence of Babinski’s sign [12,14].

To date, numerous studies have described the relationship of severe cervical cord compression with the clinical symptoms of atrophy of the extrinsic and intrinsic hand muscles, clawing of the fingers, Hoffmann’s sign, clumsy hands, and so on [16–25]. On the other hand, it is known that there are asymptomatic cervical cord compressions of the image. However, the initial symptom by the cervical cord compression is not clear. In the first place, it is not clear how much cervical cord compression causes clinical symptoms. The purpose of this study was to clarify the relationship between the magnitude and the clinical symptoms (i.e., subjective symptoms and objective findings) of single-level cord compression of the cervical spine, as evaluated by magnetic resonance imaging (MRI), in community-dwelling residents.

Materials and methods

This study was approved by the ethics committee of Fukushima Medical University (No. 1880).

Study design and subjects

In May, August, and November of 2005, in the annual checkups conducted by local governments for 3236 applicants (1326 men, 1910 women; age range, 19–94 years; average age, 65.5 years) of Tadami Town, Ina Village, and Tateiwa Village in mountainous areas of Fukushima Prefecture, Japan (Table 1), 582 people provided written, informed consent to undergo MRI, medical interviews, and physical examinations as a cervical spine medical examination by one board-certified spine surgeon (KO) in each place. When they were recruited, those who underwent cervical spinal cord surgery were excluded. All participants were self-sufficient; they lived in their own houses without the need for supplemental care and walked independently with or without support with a cane or a walker [26–28]. After a medical interview, neurological
examination, and MRI, subjects with visual impairment, dementia, brain surgery, fracture of the lower extremities, and poor quality MRI were excluded. Finally, 532 subjects (163 men, 369 women; age range, 25–93 years and average age, 64.2 years) were available for analysis in this study (Table 1). More women than men participated in this study, and the most common age group was the 70s, with few in their 40s.

### Subjective symptoms

All subjective symptoms were determined from interviews conducted by KO and included upper extremity pain and numbness, clumsy hand, gait disturbance, and fall in the past 1 year. Clumsy hand was judged as positive when there was subjective impairment in at least 1 of 3 hand and finger actions, such as using chopsticks, writing, and fastening buttons. The number of fall episodes in the past 1 year was classified as 0, 1–2, or ≥3 times. In this study, 2 patterns based on the number of fall episodes were used for statistical analysis; these included pattern 1 (≥1 time) and pattern 2 (≥3 times) (Table 2). Gait disturbance was evaluated according to the lower extremity dysfunction score of the Japanese Orthopaedic Association (JOA) scoring system for cervical myelopathy (17–2) [29]. Gait disturbance was considered present when the lower extremity score was <3 points (Table 3).

| Table 1. Characteristics of annual checkup applicants and subjects of this study. |
|---------------------------------|-----------------|-----------------|-----------------|
| **Annual checkup applicants**   | **Total (n = 3,236)** | **Male (n = 1,326)** | **Female (n = 1,910)** |
| **Characteristics**             | **Age (y), mean (SD)** | **Age (y), mean (SD)** | **Age (y), mean (SD)** |
| **Age range (%)**              | **≤39 y** | **40–49 y** | **50–59 y** | **60–69 y** | **70–79 y** | **≥80 y** |
| **Age (y), mean (SD)**         | 65.5 (13.1) | 65.7 (13.3) | 65.3 (12.9) | 170 (5.3) | 247 (7.6) | 493 (15.2) | 849 (26.2) | 1119 (34.6) | 358 (11.1) |
| **Subjects of this study**      | **Total (n = 532)** | **Male (n = 163)** | **Female (n = 369)** |
| **Characteristics**             | **Age (y), mean (SD)** | **Age (y), mean (SD)** | **Age (y), mean (SD)** |
| **Age range (%)**              | **≤39 y** | **40–49 y** | **50–59 y** | **60–69 y** | **70–79 y** | **≥80 y** |
| **Age (y), mean (SD)**         | 64.2 (12.3) | 64.5 (12.1) | 64.1 (12.4) | 25 (4.7) | 42 (7.9) | 106 (19.9) | 143 (26.9) | 181 (34.0) | 35 (6.6) |

| Table 2. Patterns of falls, Romberg test, and modified Romberg test. |
|-----------------|-----------------|-----------------|-----------------|
| **Fall down**   | **Romberg test** | **Modified Romberg test** |
| **Pattern**     | **1** | **2** | **1** | **2** | **1** | **2** |
| **0 times**     | negative | negative | (-) | negative | negative | (-) | negative | negative |
| **1–2 times**   | positive | positive | (+) | positive | positive | (+) | positive | positive |
| **≥3 times**    | positive | positive | (+) | positive | positive | (+) | positive | positive |

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Objective findings

One experienced spine surgeon (KO) performed the neurologic examinations to evaluate the finger flexion reflexes (i.e., Hoffmann’s sign [22], Trömner’s sign [30], and Wartenberg’s sign [31]); patellar tendon reflex (PTR); Achilles tendon reflex (ATR); ankle clonus [38]; and the pathological reflexes (i.e., Babinski’s sign [32] and Chaddock’s sign [33]). The finger flexion reflex was considered positive when flexion of the thumb was observed. PTR and ATR were assessed according to the National Institute of Neurological Disorders and Stroke Scale Myotatic Reflex Scale. Scale 4 was judged as hyperreflexia and represented an enhanced and more than normal reflex; it included clonus, if present, which can be optionally noted in the additional verbal description of the reflex [34,35]. The pathological reflexes were tested by stroking the lateral border of the sole of the foot (Babinski’s sign) or the lateral malleolar area (Chaddock’s sign) with a blunt object and were considered present when dorsiflexion of the hallux in the proximal to distal direction was observed.

The Romberg test [30] and modified Romberg test [36,37] were performed with the eyes closed for more than 30 seconds while standing erect with feet together and on a straight line, respectively. The findings were classified into 3 categories, including (−) for stable, (+) for swaying but able to maintain a standing position, and (+) for impossible to maintain a standing position. In this study, 2 patterns were used for statistical analysis, as follows: pattern 1, when both (±) and (+) were positive; and pattern 2, when only (+) was positive (Table 2).

The finger escape sign (FES), which reflected motor dysfunction, was classified as grade 0–4 [15,38]. In this study, grade ≥1 was regarded as positive (Table 4). FES was assessed as positive if either the left or right hand was graded as >1. The grip and release test was conducted on

| Grade | Fingers | Deficiency | Assessment of FES |
|-------|---------|------------|-------------------|
| 0     | All     | None       | Negative          |
| 1     | Little  | Unable to hold adduction | Positive |
| 2     | Little or little and ring | Unable to assume adduction |
| 3     | Little and ring | Unable to assume adduction or full extension |
| 4     | Little, ring, and middle | Unable to assume adduction or full extension |

FES: Finger escape sign.
FES was considered positive if at least 1 side was grade >1.
the left and right hands. The subject was asked to grip and release the fingers (i.e., full finger flexion and extension) as rapidly as possible, and the number of movement cycles completed within 10 seconds was counted [15,39,40]. Grip strength of the left and right hands was assessed. Using the preliminary cutoff values reported in our previous study [41,42], the results of the grip and release test and grip strength were classified into 2 groups, including normal and impaired (positive) (Table 5). The grip and release test and grip strength were assessed as positive if one of the values in the left or right hand was less than the cutoff value.

Magnetic resonance imaging

Midsagittal T2-weighted images were obtained using two MRI machines. All images were measured using a workstation (ZioCube, Mita, Minato-ku, Tokyo, Japan) at Fukushima Medical University (Fukushima City, Fukushima Prefecture) by one orthopedic surgeon (TH) who was blinded to the clinical information.

Assessment of the degree of cervical cord compression

The anterior–posterior (AP) diameters (mm) of the spinal cord at the C2 midvertebral body level ($D_{C2}$) and at each intervertebral disc level from C2/3 to C7/T1 ($D_{C2/3-C7/T1}$) were measured using midsagittal T2-weighted images. There was no spinal cord compression in the C2 vertebral body level in all subjects. In the literature, there were individual differences in cervical cord size [43,44]. Because it was necessary to standardize the AP diameter of the spinal cord, the spinal cord compression ratio ($R$) was calculated, as shown in Fig 1.

Intra-observer and inter-observer reliabilities were calculated before the study results were analyzed. To evaluate intra-observer reliability, 30 MRIs of the cervical spine were randomly selected, and 180 AP diameters of the spinal cord (from C2-3 to C7-T1 of each) were measured three times by one observer (TH) every two weeks. Furthermore, to evaluate inter-observer reliability, other 30 MRIs were measured by two other orthopedic surgeons. In the measurement of AP diameter, intra-observer reliability was $\rho = 0.73$, and inter-observer reliability was $\rho = 0.82$. The intra-observer and inter-observer reliabilities were considered acceptable. Finally, all measurements were performed by TH and these measurements were adopted in this study.

In this study, the lowest R (LR) along the C2/3 to C7/T1 of each individual was classified into 3 grades by the tertile method (G1, G2, and G3) to assess single-level cord compression in the cervical spine.

Statistical analysis

The distributions of age and sex in each grade were compared by the Jonckheere–Terpstra trend test. One-way analysis of variance was used to evaluate the differences in the average age
among the 3 grades. The tendency for the prevalence of the clinical symptoms in each grade was evaluated by the Cochran–Armitage trend analysis. Data analyses were performed using IBM SPSS Statistics (ver. 24, SPSS Inc., Chicago, IL, USA) and R (version 3.4.3, Development Core Team, 2017). A p value of <0.05 was considered significant.

**Results**

The distribution of LR is shown in **Fig 2**. The LR along C2/3 to C7/T1 of each individual ranged from 0.308 to 1.11; the 1st tertile was 0.71622 and the 2nd tertile was 0.78082. Based on these results, LR was divided into 3 grades, including G1 (LR > 0.78082), G2 (0.78082 ≥ LR > 0.71622), and G3 (LR ≤ 0.71622) to reflect the increase in the severity of cervical cord compression. The results for age and sex, subjective symptoms, and objective findings in the 3 grades are shown in **Table 6**. The severity of cord compression tended to increase with older age, but this was not significant. The sex distribution was almost the same among the 3 grades. All subjective symptoms, except gait disturbance, were not significantly related to the severity of spinal cord compression. Only the prevalence of gait disturbance increased significantly with the severity of spinal cord compression (p = 0.002812) [G1 (26 subjects, 15.0%), G2 (25 subjects, 14.2%), and G3 (50 subjects, 27.3%)]. On the other hand, all objective findings were not significantly related to the severity of spinal cord compression.

The prevalence of gait disturbance increased significantly with the severity of spinal cord compression.
Discussion

Severe spinal cord compression is widely known to induce clinical symptoms [1,6]. In an autopsy study, an AP diameter of <40% of the normal cervical spinal cord diameter was reported to show severe degenerative changes in the white matter [17]. Similarly, in the clinical and hospital settings, the magnitude of cervical spinal cord compression was reported to be related to clinical symptoms, JOA score, and the postoperative recovery rate of the JOA score [22,45–52]. However, cervical cord compression is not always directly related to the presence of subjective and objective symptoms [53–59]. Boden et al reported asymptomatic cervical disc degeneration in 25% of subjects <40 years old and in almost 60% of subjects >40 years old [53]. Matsumoto et al reported that posterior protrusion and even compression of the spinal cord were not rare in asymptomatic subjects >40 years of age [60]. Moreover, Teresi et al found asymptomatic cervical disc protrusions in 20% of patients aged 45 to 54 years [55]. Based on these reports, cervical cord compression does not always induce symptoms. Therefore, the relationship between the magnitude and the onset or degree of symptoms of cervical cord compression remains unclear.

In the present study, the magnitude of cervical cord compression was evaluated as the spinal cord compression ratio on MRI. Several measurement methods for the magnitude of cervical cord compression have been reported, including the AP diameter ratio of the compressed spinal cord to the spinal canal and the AP diameter ratio of the normal (not compressed) spinal cord to the transverse area [49,60–64]. Kameyama et al reported that the transverse area of the C7 segment varied from 33.3 to 74.0 mm² in 152 cadaveric specimens [43]. This fact suggested that the individual size of the spinal cord varies widely. Therefore, the use of a relative value might be suitable for comparison of individual data and for categorization of the severity of spinal cord compression into 3 grades.

According to the results of the present study, the magnitude of cervical cord compression was related to subjective gait disturbance but not to objective findings. The Wakayama Study

Fig 2. Range of the lowest R (LR). The LR has a minimum value of 0.308 and a maximum value of 1.11. The 1st tertile is 0.71622, and the 2nd tertile is 0.78082.

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Table 6. Results for age, sex and the prevalence of subjective symptoms and objective findings in each grade.

|                          | G1          | G2          | G3          | P        |
|--------------------------|-------------|-------------|-------------|----------|
| the lowest R (LR)        | LR>0.78082  | 0.78082>LR>0.71622 | 0.71622>LR |          |
| n                        | 173         | 176         | 183         |          |
| Age (years)              |             |             |             |          |
| <40                      | 7           | 11          | 7           | 0.075    |
| 40–49                    | 20          | 11          | 11          |          |
| 50–59                    | 32          | 41          | 33          |          |
| 60–69                    | 48          | 44          | 51          |          |
| 70–79                    | 58          | 60          | 63          |          |
| ≥80                      | 8           | 9           | 18          |          |
| Average ± S.D.           | 63.4±12.4   | 63.3±12.4   | 66.0±12.0   | 0.059    |
| Sex                      |             |             |             |          |
| Male                     | 55          | 55          | 53          | 0.56     |
| Female                   | 118         | 121         | 130         |          |
| Subjective symptoms      |             |             |             |          |
| Upper extremity pain     | 23          | 20          | 34          | 0.15     |
| Upper extremity numbness | 40          | 23          | 46          | 0.6038   |
| Clumsy hand              | 11          | 12          | 16          | 0.3855   |
| ‘Gait disturbance        | 26          | 25          | 50          | 0.002812 |
| Fall down 1 (≥1)         | 40          | 32          | 47          | 0.5445   |
| Fall down 2 (≥3)         | 7           | 12          | 10          | 0.5665   |
| Objective findings       |             |             |             |          |
| Hoffmann’s reflex        | 17          | 14          | 17          | 0.8674   |
| Trömner reflex           | 9           | 5           | 11          | 0.5369   |
| Wartenberg reflex        | 33          | 26          | 33          | 0.8085   |
| Hyperreflexia of the PTR | 1           | 0           | 0           | 0.2124   |
| Hyperreflexia of the ATR | 5           | 4           | 7           | 0.5979   |
| Ankle clonus             | 6           | 5           | 11          | 0.222    |
| Babinski reflex          | 0           | 0           | 1           | 0.2298   |
| Chaddock reflex          | 0           | 0           | 0           | NA       |
| Grip and release test    | 17          | 21          | 26          | 0.2039   |
| Grip strength            | 36          | 30          | 47          | 0.25     |
| Finger escape sign       | 18          | 23          | 20          | 0.8871   |
| Romberg test 1           | 140         | 137         | 157         | 0.2266   |
| Romberg test 2           | 2           | 1           | 3           | 0.656    |
| Modified Romberg test 1 (± or +) | 158   | 164         | 170         | 0.5801   |
| Modified Romberg test 2 (+) | 104     | 101         | 120         | 0.2821   |

S.D.: Standard deviation.
PTR: Patellar tendon reflex.
ATR: Achilles tendon reflex.
NA: Not available.

[65] of community-dwelling residents reported similar results and showed that cervical cord compression was associated with physical performance (i.e., grip and release test, 6-m walking time at a maximal pace, step length at a usual and maximal pace, and chair stand time), but not with myelopathy signs (i.e., hyperreflexia of the PTR, Hoffmann’s sign, and Babinski’s sign). That study finally concluded that cervical cord compression correlated with physical performance, and that impairment of physical performance could be detected in the early stage of the disease before the appearance of objective myelopathy signs. Moreover, other
studies suggested that gait disturbance was one of the early symptoms of cervical compressive myelopathy [66–72]. In the literature, it is not clear how much cervical cord pressure results in physical symptoms. In contrast, it was clear in the present study that the prevalence of subjective gait disturbance increased if LR was less than 0.71622. In other words, the magnitude of cervical cord compression was successfully quantized. This is considered to be the most valuable point in this study.

As suggested by the results of the present study, subjective gait disturbance based on the JOA score, compared with physical performance, may be a better indicator of the possibility of cervical cord compression, which is the early stage of DCM. This implies that clinicians should keep in mind the possibility of cervical cord compression or early stage DCM in patients with subjective complaints of gait disturbance before the occurrence of any neurologic deterioration.

There were several limitations [26–28] in this study. First, comorbidities, such as osteoarthritis of the hip and knee, lumbar spinal stenosis, and cerebrovascular disease, including Parkinson syndrome, which can influence gait ability, were not excluded. Second, only one experienced spine surgeon performed the neurologic examinations, and the reliability of each procedure was not assessed. Third, there was no evaluation of cervical radiculopathy and peripheral neuropathy, including carpal tunnel syndrome and cubital tunnel syndrome. Fourth, the research location was in a rural and mountainous area; therefore, the data may not be extrapolated completely to the typical Japanese population. Fifth, we did not consider the sample size and do a power calculation before starting this study. Because we could not assume the number of persons who underwent MRI. As a result, there was not representative of the actual age demographics in these places, especially concerning the population of people under the age of 40 years. It is necessary to consider whether the results of this study can be applied to young people. Finally, all of the participants in this study were volunteers and, as such, there could have been an inevitable sample bias. Although this study had limitations, it clarified the relationship between the magnitude of cervical cord compression and subjective gait disturbance in community-dwelling residents.

Conclusion
In community-dwelling residents, the magnitude of cervical cord compression was related to the presence of subjective gait disturbance, but not objective findings. Therefore, subjective gait disturbance might be a good indicator of the possibility of early stage DCM.

Supporting information
S1 Table. Detail of MRI.
(DOCX)
S1 File. Doctor interview sheet in English.
(DOC)
S2 File. Doctor interview sheet in Japanese.
(DOC)

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Author Contributions

Conceptualization: Toru Hirai, Koji Otani.

Data curation: Koji Otani.

Formal analysis: Toru Hirai.

Investigation: Toru Hirai, Koji Otani.

Methodology: Koji Otani.

Project administration: Koji Otani.

Supervision: Koji Otani, Miho Sekiguchi, Shin-ichi Kikuchi, Shin-ichi Konno.

Writing – original draft: Toru Hirai.

Writing – review & editing: Koji Otani, Miho Sekiguchi, Shin-ichi Konno.

References

1. White AA III, Panjabi MM. Biomechanical considerations in the surgical management of cervical spondyloitic myelopathy. *Spine* 1988; 13:856–860. https://doi.org/10.1097/00007632-198807000-00029 PMID: 3194796

2. White AA III, Panjabi MM. *Clinical Biomechanics of the Spine*, 2nd ed. Philadelphia: J.B. Lippincott; 1990.

3. Penning L. Some aspects of plain radiography of the cervical spine in chronic myelopathy. *Neurology* 1962; 12:513–519. https://doi.org/10.1212/wnl.12.8.518 PMID: 14485123

4. Epstein NE, Hyman RA, Epstein JA, Rosenthal AD. Technical Note: “Dynamic” MRI scanning of the cervical spine. *Spine* 1988; 13:937–938. https://doi.org/10.1097/00007632-198808000-00015 PMID: 3187719

5. Adams CB, Logue V. Studies in cervical spondylotic myelopathy. I. Movement in the cervical roots, dura, and cord and their relations to the course of the extrathecal roots. *Brain* 1971; 94:557–568. https://doi.org/10.1093/brain/94.3.557 PMID: 5111717

6. Kataoka O, Kurihara A, Maruo S. Dynamic canal stenosis in cervical spondylotic myelopathy. *Clin Ortho Surg* 1975; 10;1133–1143 (in Japanese).

7. Kataoka O, Kurihara A. The role of dynamic canal stenosis in cervical spondylotic myelopathy. *J West Pacific Orthop Assoc* 1977; 14;1–22.

8. Fukui K, Kataoka O, Sho T, Sumi M. Pathomechanism, pathogenesis, and results of treatment in cervical spondylotic myelopathy caused by dynamic canal stenosis. *Spine* 1990; 15(11):1148–52. https://doi.org/10.1097/00007632-199011010-00012 PMID: 2267609

9. Matsunaga S, Kunta M, Hayashi K, Shinkura R, Chihaya K, Sakou T, et al. Pathogenesis of myelopathy in patients with ossification of the posterior longitudinal ligament. *J Neurosurg* 2002; 96:168–172. https://doi.org/10.3171/spi.2002.96.2.0168 PMID: 12450279

10. Waldman SD. *Pain Management*, 2nd ed. Philadelphia: Elsevier Saunders; 2011.

11. Curtis WS, Richard D, Frederick AS, Tom GM. *Interventional Spine: An Algorithmic Approach*. Philadelphia: Elsevier Saunders; 2007.

12. Ono K, Jilii D, Edwad D. *Cervical Spondylosis and Similar Disorders*. Singapore: World Scientific Publishing Co. Pte. Ltd; 1998.

13. 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. https://doi.org/10.1148/radiology.173.1.2781011 PMID: 2781011

14. Mark B, Missouri RA, Howard WB, Augustus AW. Current Concepts Review Cervical Spondylotic Myelopathy. *J Bone Joint Surg* 1993; 75(1):119–128. https://doi.org/10.2106/00004623-199301000-00016 PMID: 8419381
15. Ono K, Ebara S, Fuji T, Fuji T, Yonenobu K, Fujiwara K, et al. Myelopathy hand. New clinical signs of cervical cord damage. J bone joint surg 1987; 69(2):215–9. https://doi.org/10.1302/0301-620X.69B2.381752 PMID: 381752

16. Bruce MM, Phillip RW. Cervical spondylosis. An update. West J Med 1996; 165(1–2):43–51. PMID: 885684

17. Ogino H, Tada K, Okada K, Yamamoto T, Ono K, et al. Canal Diameter, Anteroposterior Compression Ratio, and Spondylotic Myelopathy of the Cervical Spine. Spine 1983; 8:1–15. https://doi.org/10.1097/00007632-198301000-00001 PMID: 686746

18. Jerjis JD, Gilbert RM. Early diagnosis of cervical spondylotic myelopathy. A useful clinical sign. Spine 1991; 16:1353–1355. https://doi.org/10.1097/00007632-199112000-00001 PMID: 1771463

19. Good DC, Couch JR, Wacaser L. ‘Numb, clumsy hands’ and high cervical spondylosis. Arch Neurol 1984; 22:285–291. https://doi.org/10.1001/archneur.1984.00900190047008 PMID: 6463840

20. Voskuhl RR, Hinton RC. Sensory impairment in the hands secondary to spondylotic compression of the cervical spine. Arch Neurol 1990; 47:309–311. https://doi.org/10.1001/archneur.1990.00530300850200 PMID: 2310314

21. Sung RD, Wang JC. Correlation between a positive Hoffman’s reflex and cervical pathology in asymptomatic individuals. Spine 2001; 26(1):67–70. https://doi.org/10.1097/00007632-200101010-00013 PMID: 1148648

22. Houten JK, Noce LA. Clinical Correlations of Cervical Myelopathy and the Hoffmann Sign. Journal of Neurosurgery. Spine 2008; 9(3):237–242. https://doi.org/10.3171/SPI/2008/9/9/237 PMID: 18928217

23. Omori M, Shibuya S, Nakajima T, Endoh T, Suzuki S, Irie S, et al. Hand Dexterity Impairment in Patients with Cervical Myelopathy: A New Quantitative Assessment Using a Natural Prehension Movement. Behav Neurol 2018; 2018:1–10. https://doi.org/10.1155/2018/5138234 PMID: 30073036

24. Cracknell PH, Batzdorf U. Cervical spondylotic myelopathy. J Neurosurg 1966; 25:57–66. https://doi.org/10.3171/jns.1966.25.1.0057 PMID: 5947048

25. Otani K, Ikata T, Katoh S. Implications of signal intensity on T1 weighted MR Image on the prognosis of cervical spondylotic myelopathy. Orthop Trans 1996; 20:443.

26. Otani K, Kikuchi S, Yabuki S, Igarashi T, Nikaido T, Ando R, et al. Lumbar spinal stenosis has a negative impact on quality of life compared with other comorbidities: An epidemiological cross-sectional study of 1862 community-dwelling individuals. Spine World J 2013 Dec 23: 2013:590652.

27. Otani K, Kikuchi S, Yabuki S, Onda A, Nikaido T, Watanabe K, et al. Prospective one-year follow-up of lumbar spinal stenosis in a regional community. J Pain Res 2018; 11:455–464. https://doi.org/10.2147/JPR.S148402 PMID: 29535549

28. Otani K, Kikuchi S, Nikaido T, Konno S. Magnitude of dural tube compression does not show a predictive value for symptomatic lumbar spinal stenosis for 1-year follow-up: a prospective cohort study in the community. Clin Interv Aging 2018; 13:1739–1746. https://doi.org/10.2147/CIA.S171049 PMID: 30271128

29. Hiraibayas K. Scoring system (17–2) for cervical myelopathy (Japanese Orthopaedic Association). J Jpn Orthop Assoc 1994; 68(5):498 (in Japanese).

30. Stephen MF, Arthur CC. Whiplash Injuries-The Cervical Acceleration/Deceleration Syndrome. Philadelphia: Lippincott Williams & Wilkins, 1988.

31. William WC, Russell ND. DeJong’s Neurologic Examination, 7th ed. Philadelphia: Lippincott Williams & Wilkins, 2013.

32. Rhee JM, Heflin JA, Hamasaki T, Freedman B. Prevalence of physical signs in cervical myelopathy: A prospective, controlled study, Spine 2009; 34(9):890–895. https://doi.org/10.1097/BRS.0b013e1819c444b PMID: 19352222

33. Araujo R, Firmino MJ, Correia P, Leitão-Marques M, Carvalho J, Silva M, et al. The plantar reflex: A study of observer agreement, sensitivity, and observer bias. Neurol Clin Pract 2015 Aug; 5(4):309–316. https://doi.org/10.1212/CPJ.0000000000000155 PMID: 29443235

34. Litvan I, Mangone CA, Werden W. Reliability of the NINDS myotatic reflex scale. Neurology 1996; 47 (4):969–972. https://doi.org/10.1212/wnl.47.4.969 PMID: 8857728

35. Mark H. NINDS Myotatic Reflex Scale. Neurology 1993; 43(12):2733.

36. Shumway-Cook Anne, Horak Fay Bahling. Assessing the influence of sensory interaction of balance. Physical therapy 1986; 66(10):1548–1550. https://doi.org/10.1093/ptj/66.10.1548 PMID: 3763708

37. Agrawal Yuri, Carey John P, Sklare Daniel A, Schubert Michael C. The Modified Romberg Balance Test: Normative Data in U.S. Adults. Otology & Neurology 2011; 32:1309–1311. https://doi.org/10.1097/MAO.0b013e31822e5bee PMID: 21892121
38. Ono K, Fuji T, Okada K. Myelopathy hand and reversibility of cervical myelopathy. *Bessatsu Seikei Geka* 1982; 2:10–7 (in Japanese).

39. Yukawa Y, Nakashima H, Ito K, Machino M, Kanbara S, Kato F. Quantifiable tests for cervical myelopathy; 10-s grip and release test and 10-s step test: standard values and aging variation from 1230 healthy volunteers. *J Orthop Sci* 2013; 18(4):509–513. https://doi.org/10.1007/s00776-013-0381-6 PMID: 23564077

40. Nakashima H, Yokawa Y, Ito K, Machino M, Kanbara S, Morita D, et al. Validity of the 10-s step test: prospective study comparing it with the 10-s grip and release test and the 30-m walking test. *Eur Spine J* 2011; 20:1318–22. https://doi.org/10.1007/s00586-011-1733-6 PMID: 21380747

41. Kobayashi H, Otani K, Kato K, Watanabe K, Nkaido T, Yabuki S, et al. Diagnostic characteristics of 10-second test on diagnosis for cervical myelopathy-An investigation of sensitivity and specificity by age group with ROC curve-. *J Spine Res* 2017; 8(3):221.

42. Kobayashi H, Otani K, Nkaido T, Watanabe K, Kato K, Tominaga R, et al. Grip strength as screening tool for cervical myelopathy- Minami Aizu study: Investigation of cut-off value for age and sex groups. *J Jpn. Orthop. Assoc* 2018; 92(3):S723.

43. Kameyama T, Hashizume Y, Ando T, Takahashi Y. Morphometry of the normal cadaveric cervical spinal cord. *Spine* 1994; 19:2077–2081. https://doi.org/10.1097/00007632-199409150-00013 PMID: 7825049

44. Kato F, Yukawa Y, Suda K, Suda K, Yamagata M, Ueta T. Normal morphology, age-related changes and abnormal findings of the cervical spine. Part II: Magnetic resonance imaging of over 1,200 asymptomatic subjects. *Eur Spine J* 2012; 21(8):1499–1507. https://doi.org/10.1007/s00586-012-1716-4 PMID: 22302162

45. Morio Y, Yamamoto K, Teshima R, Nagashima H, Higino H. Clinicoradiologic Study of Cervical Laminoplasty With Posterolateral Fusion or Bone Graft. *Spine* 2000; 25(2):190–196. https://doi.org/10.1097/00007632-200001190-00006 PMID: 10685482

46. Morio Y, Teshima R, Nagashima H, Nawata K, Yamazaki D, Nanjo Y. Correlation between operative outcomes of cervical compression myelopathy and MRI of the spinal cord. *Spine* 2001; 26(11):1238–45. https://doi.org/10.1097/00007632-200106010-00012 PMID: 11389390

47. Hosono N, Makino T, Sakaura H, Mukai Y, Fuji T, Yoshikawa H. Myelopathy hand: New evidence of the classical sign. *Spine* 2010; 35(8):273–277. https://doi.org/10.1097/BRS.0b013e3181c6afeb PMID: 20354474

48. Hachiku T, Taguchi T, Kaneko K, Fujigami Y, Yonemura H, Kawai S. A correlation between magnetic resonance imaging and electrophysiological findings in cervical spondylotic myelopathy. *Spine* 2001; 26(13):1–6. https://doi.org/10.1097/00007632-200107010-00014 PMID: 11458169

49. Fujiwara K, Yonenobu K. the Prognosis of Surgery for Cervical Compression Myelopathy an Analysis of the Factors Involved. *J Bone Joint Surg* 1989; 71-B(3):393–398. https://doi.org/10.1302/0301-620X.71B3.2722928 PMID: 2722928

50. Mastronard L, Elsawaf A, Roperto R, Bozzao A, Carolu M, Ferrante M, et al. Prognostic relevance of the postoperative evolution of intramedullary spinal cord changes in signal intensity on magnetic resonance imaging after anterior decompression for cervical spondylotic myelopathy. Journal of neurosurgery. *Spine* 2007; 30(6):615–622. https://doi.org/10.1097/01.SNJ.0000271371.SF013 PMID: 18074686

51. Yonenobu K, Hosono N, Iwasaki M, Asano M, Ono K. Laminoplasty versus subtotal corpectomy. A comparative study of results in multisegmental cervical spondylotic myelopathy. *Spine* 1992; 17:1281–4. PMID: 1462201

52. Einoamany H. Sensitivity of pyramidal signs in patients with cervical spondylotic myelopathy. *Asian Spine J* 2016; 10(1):65–69. PMID: 26949460

53. Boden SD, McCowin PR, Davis DO, Dina TS, Mark AS, Wiesel S. Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg* 1990; 72(8):1178–1184. PMID: 2398088

54. Lehto IJ, Tertti MO, Komu ME, Paajanen HEK, Tuominen J, Kormano MJ. Age-related MRI changes at 0.1 T in cervical discs in asymptomatic subjects. *Neuroradiology* 1994; 36(1):49–53. https://doi.org/10.1007/BF00399196 PMID: 8107998

55. Teresi LM, Lufkin RB, Reicher MA, Moffit BJ, Vinuela FV, Wilson GM, et al. Asymptomatic degenerative disk disease and spondylosis of the cervical spine: MR imaging. *Radiology* 1987; 164:83–88. https://doi.org/10.1148/radiology.164.1.5589931 PMID: 5589931

56. Matsumoto M, Fujimura Y, Suzuki N, Nishi Y, Nakamura M, Yabe Y, et al. MRI of cervical intervertebral discs in asymptomatic subjects. *J Bone Joint Surg* 1998; 80(1):19–24. https://doi.org/10.1302/0301-620x.80b1.7929 PMID: 9460946
57. Ishikawa M, Matsumoto M, Fujimura Y, Chiba K, Toyama Y. Changes of cervical spinal cord and cervical spinal canal with age in asymptomatic subjects. *Spinal Cord* 2003; 41(3):159–163. https://doi.org/10.1038/sj.sc.3101375 PMID: 12612618

58. Okada E, Matsumoto M, Ichihara D, Chiba K, Toyama Y, Fujiwara H, et al. Does the sagittal alignment of the cervical spine have an impact on disk degeneration? Minimum 10-year follow-up of asymptomatic volunteers. *Eur Spine J* 2009; 18(11):1644–1651. https://doi.org/10.1007/s00586-009-1095-5 PMID: 19609784

59. Okada E, Matsumoto M, Fujihara H, Toyama Y. Disc degeneration of cervical spine on MRI in patients with lumbar disc herniation: Comparison study with asymptomatic volunteers. *Eur Spine J* 2009; 18(11):1644–1651. https://doi.org/10.1007/s00586-009-1095-5 PMID: 19609784

60. Arvin B, Kalsi-Ryan S, Karpova A, Mercier D, Furian JC, Massicotte EM, et al. Postoperative magnetic resonance imaging can predict neurological recovery after surgery for cervical spondylotic myelopathy: a prospective study with blinded assessments. *Neurosurgery* 2011; 69:362–368. https://doi.org/10.1227/NEU.0b013e318259a65b PMID: 22511231

61. Nagata K, Yoshimura N, Muraki S, Hashizume H, Ishimoto Y, Yamada H, et al. Prevalence of cervical cord compression and its association with physical performance in a population-based cohort in Japan, *Spine* 2012; 37:1892–1898. https://doi.org/10.1097/BRS.0b013e31825a2619 PMID: 22565382

62. Nishimura H, Endo K, Suzuki H, Tanaka H, Shishido T, Yamamoto Y. Gait Analysis in Cervical Spondylotic Myelopathy. *Asian Spine J* 2015; 9:321–326. PMID: 26097646

63. Ailish M, Dara M, Ciaran B. Gait impairment in cervical spondylotic myelopathy: comparison with age- and gender-matched healthy controls. *Eur Spine J* 2012; 21:2456–2466. https://doi.org/10.1007/10.1097/BRS.0b013e318259a65b PMID: 22511231

64. Kuhtz-Buschbeck JP, Jöhnk K, Mäder S, Stolze H, Mehdorn M. Analysis of gait in cervical myelopathy. *Gait Posture* 1999; 9(3):184–189. https://doi.org/10.1016/s0966-6362(99)00015-6 PMID: 10575079

65. Lee JH, Lee SH, See IS. The characteristics of gait disturbance and its relationship with posterior tibial somatosensory evoked potentials in patients with cervical myelopathy. *Spine* 2011; 36(8):E524–E530. https://doi.org/10.1097/BRS.0b013e3181f1412d PMID: 21224774

66. Suzuki E, Nakamura H, Konishi S, Yamamoto Y, Kato T. Analysis of the spastic gait caused by cervical compression myelopathy, *J Spinal Disord Tech* 2002; 15(6):519–522. https://doi.org/10.1097/00024720-200212000-00015 PMID: 12489881

67. Kim CR, Yoo JY, Lee SH, Lee DH, Rhim SC. Gait analysis for evaluating the relationship between increased signal intensity on T2-weighted magnetic resonance imaging and gait function in cervical spondylotic myelopathy. *Arch Phys Med Rehabil* 2010; 91(10):1587–1592. https://doi.org/10.1016/j.apmr.2010.07.008 PMID: 20875519