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Diagnostic Role of Flexion-extension Central Motor Conduction Time in Cervical Spondylotic Myelopathy

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Study Design. Retrospective study

Objective. The purpose of this study was to assess the diagnostic usefulness of flexion-extension central motor conduction time (CMCT) for patients with cervical spondylotic myelopathy (CSM).

Summary of Background Data. Previous reports have suggested that cervical cord compression can be aggravated by neck motions. Thus, the importance of dynamic magnetic resonance imaging (MRI) has been emphasized. However, authors of this study found no reports conducted at the time of this research on whether flexion-extension CMCT was useful for detecting myelopathy.

Methods. We enrolled 227 patients with CSM for this study. We acquired CMCT recorded from the abductor pollicis brevis muscle. All patients underwent a dynamic CMCT study during neck flexion and extension as well as a static study during neutral neck. Static and dynamic MRIs were also scanned. We read all MR images using Muhle classification (MC).

Results. CMCT was significantly delayed with flexion ($P < 0.01$) and extension ($P < 0.01$) compared to neutral neck position. Patients with MC grade 1 and 2 showed significant lag in CMCT during flexion and extension. No significant lag by neck motion was observed for those in the MC grade 3. We also evaluated the amount of CMCT variation according to MC grade change (G0, G1, G2) by neck motion. Delta-CMCT of both G1 and G2 were significantly larger than those of G0 in both flexion and extension. In neutral neck, the CMCT showed significant difference between MC grades 1 and 3. They also displayed significant delay with delay with high signal intensity on T2 MRI. More than one-third of the patients whose CMCT was within normal range in neutral neck presented abnormal CMCT in neck flexion (35.3%) and extension (37.8%).

Conclusion. CMCT is significantly slower in both neck flexion and neck extension than in the neutral neck position. These findings reflect the dynamic cervical cord impingement.

Key words: central motor conduction time, cervical spinal cord, cervical spondylosis, compressive myelopathy, diagnostic technique and procedure, dynamic study, electrodiagnosis, evoked potentials, magnetic resonance imaging, transcranial magnetic stimulation.

Level of Evidence: 4

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Cervical spondylotic myelopathy (CSM) is a disease resulting from progressive compression of the spinal canal and the cervical cord due to degenerative changes of the cervical vertebrae and is usually concomitant with aging.[1,2] Representative degenerative conditions of cervical stenosis are intervertebral disc protrusion, spondylolisthesis, and ossification of posterior longitudinal ligament (OPLL).[3,4] They provoke symptoms to range from mild neck pain and radiating pain to spinal cord syndromes such as paresthesia, weakness, clumsy hand, and gait disturbance.[5]

Magnetic resonance imaging (MRI) is widely utilized as a diagnostic tool for confirming cord compression. Many patients with CSM show not only static cord compression in neutral neck position but also dynamic cord compression during neck flexion and extension.[6,7] Numerous studies exist that describe the diagnosis of compressive cervical myelopathy by using dynamic MRI as well as static MRI to identify impingement of the cervical cord.[8,9]

Electrophysiological studies have also been conducted to evaluate not only spinal cord compression but combined

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neurologic conditions such as cervical radiculopathy and peripheral neuropathies. Among those electrophysiological evaluations, motor evoked potential (MEP) recorded by transcranial magnetic stimulation is a key variable to ascertain whether there is a disturbance of central motor conduction along the corticospinal tract.[10,11] It has been widely reported that central motor conduction time (CMCT) derived from MEP and motor conduction studies is objective and sensitive values to assess central motor pathway.[12,13] Fast monosynaptic neurons in lateral corticospinal tract are disrupted in early stage of cord compression and the prolonged CMCT can discriminate myelopathy sensitively.[14] Baseline values of CMCT according to age groups have also been established.[15,16]

Although several studies have already demonstrated that dynamic MRI is necessary for the diagnosis of cord compression in CSM patients, as of the time of this study, the authors found no previous research on whether flexion-extension CMCT is useful for detecting dynamic cervical cord compression. Thus, the concept of dynamic study can be applied to electrophysiological evaluation to improve its diagnostic accuracy.

In this study, we aimed to evaluate the diagnostic usefulness of flexion-extension CMCT for confirming cervical cord compression including the concept of dynamic cord impingement. We compared the difference of CMCT according to the patient's neck positions as well as the severity of cervical MRI findings. We also hypothesized that the dynamic cervical stenosis caused by neck motions was related with the change of CMCT.

MATERIALS AND METHODS

Subjects

This study was conducted as a retrospective study using existing medical records of patients. The patients were selected from June 2017 to January 2020. To come up with the sample cohort for our study, we first selected patients suspected of CSM based on cervical MRI and clinical manifestations. We used the following clinical criteria for patient selection: upper motor neuron sign in any extremity; motor symptoms such as clumsy hand, arm or leg weakness, muscle atrophy, and gait disturbance; sensory symptoms such as sensory loss, altered proprioception, and paresthesia; autonomic dysfunctions such as overactive bladder or bowel.[17,18] If a patient presented one or more of the above symptoms, then we suspected the patient of having CSM and conducted appropriate evaluations. Simple neck pain or radiating pain was ruled out for evaluating myelopathy. For patients complaining of one or more symptoms of the aforementioned clinical criteria, we performed dynamic cervical MRI as well as conventional neutral cervical MRI. We also conducted evaluations to rule out the possibility of brain lesions. The initial selection was done by experienced neurosurgeons at our hospital. Out of the initial selection, we then picked patients who were referred to physiatrists for electrophysiologic examinations including the dynamic CMCT. After the second screening, we excluded patients with the following: previous brain lesion or brain surgery history; previous cervical spine surgery history; combined diagnosis of polyneuropathy or severe carpal tunnel syndrome; unobtainable median MEP, F-wave or compound motor action potential (CMAP); and insufficient medical records or examination results. The Institutional Review Board of our hospital approved this study (approval No. PSSH0475–202003–HR–003–01).

Electrophysiologic Studies

Electrophysiological tests were carried out using the Sirrea®wave (Cadwell, Kennewick, WA). All patients underwent routine nerve conduction study and electromyography for confirming concomitant diseases such as peripheral mononeuropathies, polyneuropathy, and cervical radiculopathy. We conducted median CMAP and F-wave by stimulating the median nerve at the wrist with supramaximal stimulation with 0.2 ms square wave pulses and by recording from the abductor pollicis brevis (APB) muscle, with a 5 to 5000 Hz filter setting. They were recorded bilaterally and repeated at least 12 times to confirm reproducibility. We performed nerve conduction studies in the supine position and set the temperature of the examination room at 23°C to 25°C to eliminate temperature-dependent effects.

We performed transcranial magnetic stimulation using the MagPro Compact with a 13–cm diameter circular coil (MagVenture, Farum, Denmark) to provoke median MEPs. We set the intensity of stimulation at 20% above the threshold during minimal isometric voluntary contraction of the APB. For cortex stimulation, we placed the center of the coil at Cz according to the international 10–20 system. We acquired CMCT data by using the calculation method that has been presented in previous literature. The method of MEP measurement and CMCT calculation formula are shown in Figure 1.[15,19] Like the Median CMAP, we also recorded Median MEPs bilaterally and repeated the recording six times to confirm reproducibility. We measured the patient's initial MEP after putting the patient in sitting and neutral neck position. Then, we measured each patient's MEP twice more in maximal neck flexion and extension within the range that the patient could perform without any symptom aggravation. Two examiners conducted the MEP study to secure the patient's safety and to fix the neck position. For analysis, we used values measured on the symptomatic side. If the patient complained of symptoms on both sides, we selected the values measured on the side with worse symptoms.

Imaging Studies

We took all cervical MRI scans with the 1.5T Philips Achieva (Philips Medical Systems, Eindhoven, Netherlands). Patients underwent static cervical MRI first; then, dynamic cervical MRI was done during flexion and extension. Same as the flexion-extension CMCT, we performed the dynamic MRI at maximal neck flexion and extension angles that the patient could achieve without any neurologic deterioration. Supporting materials were placed on the patient's head and neck to fix

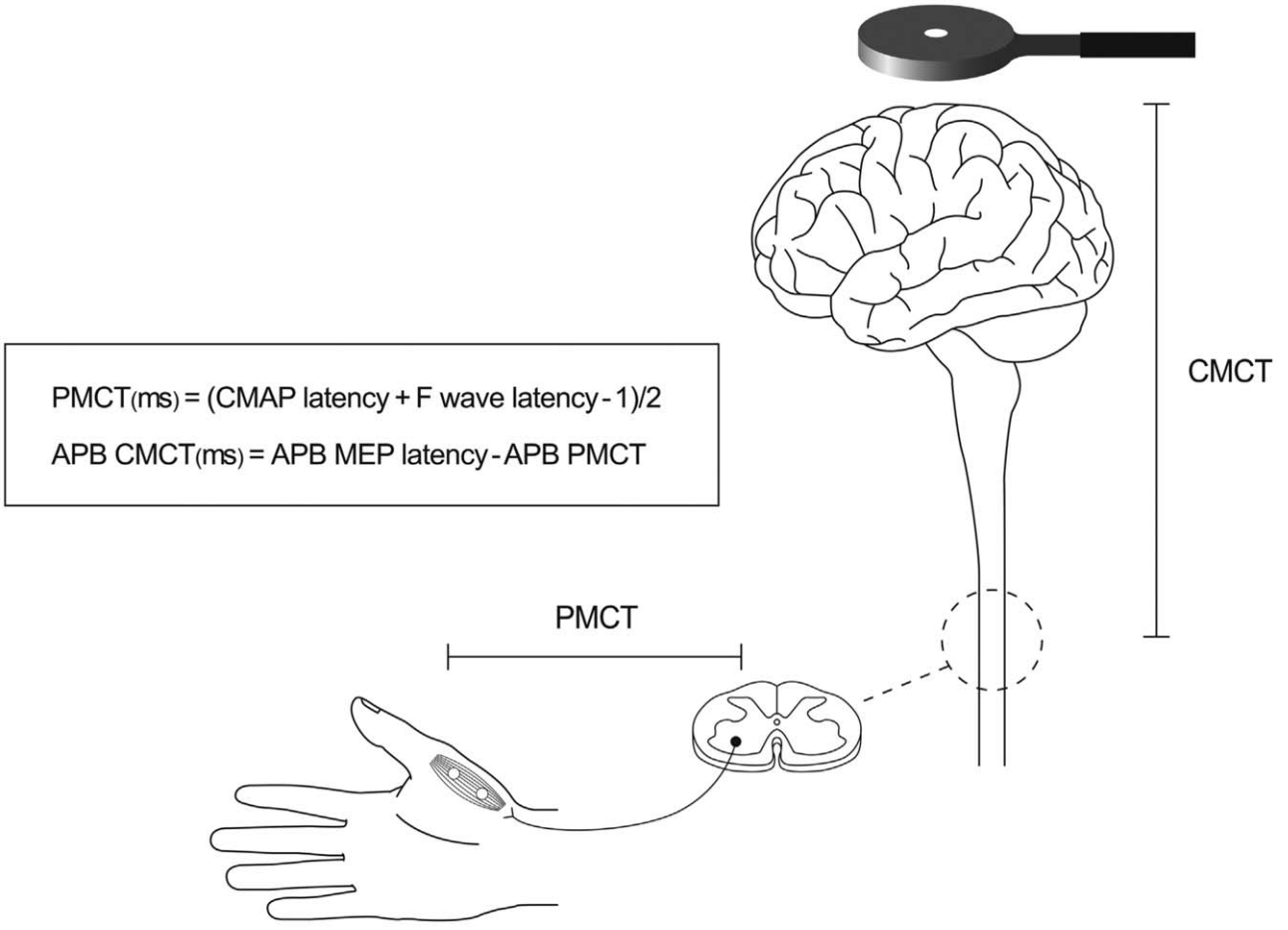

Figure 1. Median MEP measurement and calculation of APB CMCT. APB indicates abductor pollicis brevis muscle; CMCT, central motor conduction time; MEP, motor evoked potential; PMCT, peripheral motor conduction time.

the patient's position during MRI scanning. To shorten the scanning time, we only took T2 sagittal images of each neck position for the dynamic MRI. We considered the patients' discomfort and worsening of clinical symptoms because they had to be in the states of fixed neck flexion and extension for a significant duration of time to take the dynamic MRI.

The extent of cervical cord compression level ranged from C1/2 to C6/7. We measured the Muhle classification (MC) at the most stenotic intervertebral level from the midline of sagittal T2 images (Table 1).[20] We also checked for the presence of high signal intensity (HSI) on T2 images.

TABLE 1. Muhle Classification

Grade	Description
0	Normal width of the spinal canal, no signs of anterior and posterior subarachnoid space narrowing
1	Partial obliteration of the anterior or posterior subarachnoid space or of both
2	Complete obliteration of the anterior or posterior subarachnoid space or of both
3	Anterior or posterior cord impingement or both

All imaging findings were read by experienced neurosurgeons and physiatrists at our hospital.

Statistical Analysis

We performed repeated measures analysis of variance (ANOVA) with Tukey's multiple comparisons test to compare CMCT values according to neck positions. We analyzed the comparison of mean CMCT and percentage of delta-CMCT according to the MC grade by utilizing one-way ANOVA with Bonferroni correction for multiple comparisons. The student *t* test was used to assess differences in CMCT by HSI. We performed all statistical analyses using SPSS 20.0 (IBM, Armonk, NY).

RESULTS

We examined a total of 142 men and 85 women whose average age was 58.44 ± 12.07 years. OPLL was the most common cause of CSM with 108 patients, followed by degenerative disc protrusion and spondylolisthesis. The symptom side was evenly distributed. Looking at the most severe stenosis level, C4/5 was the most common with 70 patients, followed by C3/4 with 65 patients. All patients included in the study had a MC grade 1 or above. We observed HSI on T2 images of 87

TABLE 2. Characteristics of Included Subjects

Parameter	Values (%)
Male	142 (62.6)
Average age, y	58.44 ± 12.07
Side	
Right	74 (32.6)
Left	79 (34.8)
Bilateral	74 (32.6)
Diagnosis	
Disc protrusion	85 (37.4)
Spondylolisthesis	34 (15.0)
OPLL	108 (45.6)
Location (the most stenotic level)	
C1/2	5 (2.2)
C2/3	13 (5.7)
C3/4	65 (28.6)
C4/5	70 (30.8)
C5/6	60 (26.4)
C6/7	14 (6.1)
Muhle classification (neutral neck position)	
1	162 (71.4)
2	44 (19.4)
3	21 (9.3)
HSI on T2 MRI	87 (38.3)
mJOA score	14.02 ± 2.34

HIS indicates high signal intensity; MRI, magnetic resonance imaging, mJOA, modified Japanese Orthopaedic Association scale; OPLL, ossification of posterior longitudinal ligament.

patients. The patients' baseline score on the modified Japanese Orthopaedic Association scale was 14.02 ± 2.34 (Table 2).

Flexion-extension CMCT Study

All MEP and CMCT values are provided in Table 3. In neutral neck, mean MEP onset latency was 23.80 ± 3.81 ms and mean CMCT was 8.76 ± 3.76 ms. During neck flexion and extension, CMCT was significantly delayed compared to neutral neck ($P < 0.01$ and $P < 0.01$, respectively). When we conducted subgroup analysis based on MRI findings, we discovered that CMCT was significantly prolonged during flexion and extension compared to neutral neck in the MC grade 1 ($P < 0.01$ and $P < 0.01$, respectively) and grade 2 ($P < 0.01$ and $P = 0.04$, respectively) groups. Meanwhile, the MC grade 3 group did not present a significant difference depending on the neck position. Regardless of the presence of HSI, we noted significant lagging of CMCT in flexion and extension ($P < 0.01$ and $P < 0.01$, respectively) (Figure 2).

When the CMCT cutoff value was set to 7.40, eighty-two subjects belonged to within normal range in the neutral neck position.[15] Among them, 29 patients (35.3%) with neck flexion and 31 patients (37.8%) with neck extension presented abnormal ranges of CMCT.

CMCT and Imaging findings

We categorized the subjects into different groups of G0, G1, and G2 depending on the delta-MC grade when the patient's neck positions changed. With neck flexion, 156 patients (68.7%) showed no MC grade change (G0). Sixty-three patients (27.8%) showed an MC increase of 1 grade (G1). Only eight patients (3.5%) showed an MC increase of 2 grades (G2). Meanwhile, with neck extension, 126 patients (55.5%) showed no MC grade change (G0). Eighty-three patients (36.6%) showed an MC increase of 1 grade (G1). Eighteen patients (7.9%) showed an MC increase of 2 grades (G2) (Table 4).

We compared the amount of CMCT variation (delta-CMCT). Delta-CMCT of both G1 and G2 were significantly larger than those of G0 during neck flexion ($P < 0.01$ and $P = 0.02$, respectively) and extension ($P < 0.01$ and $P < 0.01$, respectively). There was no meaningful difference between G1 and G2 (Figure 3A and B).

We assessed the differences in CMCT according to MC grades in the neutral neck. CMCT presented the significant difference between MC grades 1 and 3 ($P = 0.01$), but there were no significant differences between other groups (Figure 4A). Delta-CMCT did not show any significant difference according to MC grades in the neutral neck position. CMCT also presented significant delay with HSI ($P < 0.01$) (Figure 4B). Delta-CMCT was not significantly larger with HSI than those without HSI.

TABLE 3. Summary of APB MEP Onset Latency and APB CMCT Data

	Total	MC1	MC2	MC3	HSI	No HSI
MEP						
Neu, ms	23.80 ± 3.81	23.27 ± 3.41	24.77 ± 4.60	25.89 ± 4.08	25.76 ± 4.45	22.58 ± 2.73
Flex, ms	24.42 ± 3.87	23.87 ± 3.45	25.44 ± 4.67	26.51 ± 4.14	26.62 ± 4.42	23.05 ± 2.72
Ext, ms	24.46 ± 3.90	23.98 ± 3.53	25.24 ± 4.74	26.47 ± 3.83	26.65 ± 4.39	23.10 ± 2.79
CMCT						
Neu, ms	8.76 ± 3.76	8.69 ± 3.56	9.87 ± 4.14	11.20 ± 3.70	10.50 ± 4.32	8.31 ± 3.11
Flex, ms	9.77 ± 3.84	9.30 ± 3.64	10.51 ± 4.22	11.78 ± 3.76	11.34 ± 4.30	8.79 ± 3.17
Ext, ms	9.81 ± 3.82	9.42 ± 3.65	10.33 ± 4.28	11.79 ± 3.54	11.41 ± 4.30	8.82 ± 3.12
ΔFlex (%)	8.34 ± 14.36	8.62 ± 14.19	8.32 ± 15.73	6.20 ± 13.11	10.38 ± 15.86	7.07 ± 13.25
ΔExt (%)	8.91 ± 15.15	9.99 ± 15.39	6.07 ± 15.99	6.55 ± 10.11	10.92 ± 15.67	7.66 ± 14.73

Δ indicates delta; APB, abductor pollicis brevis muscle; CMCT, central motor conduction time; Ext, neck extension; Flex, neck flexion; HSI, high signal intensity; MC, Muhle classification grade; MEP, motor evoked potential; Neu, neck neutral.

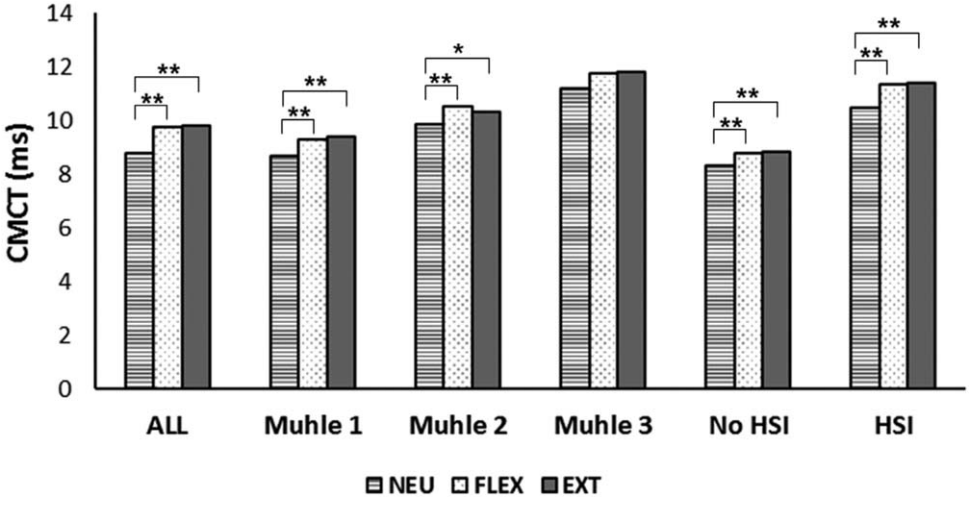

Figure 2. CMCT values according to neck motions. CMCT indicates central motor conduction time; EXT, neck extension; FLEX, neck flexion; HSI, high signal intensity; NEU, neck neutral. $^{*}P < 0.05$; $^{**}P < 0.01$.

TABLE 4. Proportion of Patients That Showed Changes in Dynamic MRI

		MC of the Neutral Neck		
Groups	**Total**	**MC 1**	**MC 2**	**MC 3**
Neck flexion				
G0 (Δ0)	156	107	28	21
G1 (Δ1)	63	47	16	–
G2 (Δ2)	8	8	–	–
Neck extension				
G0 (Δ0)	126	88	17	21
G1 (Δ1)	83	56	27	–
G2 (Δ2)	18	18	–	–

Δ indicates delta; MC indicates Muhle classification grade; MRI, magnetic resonance imaging.

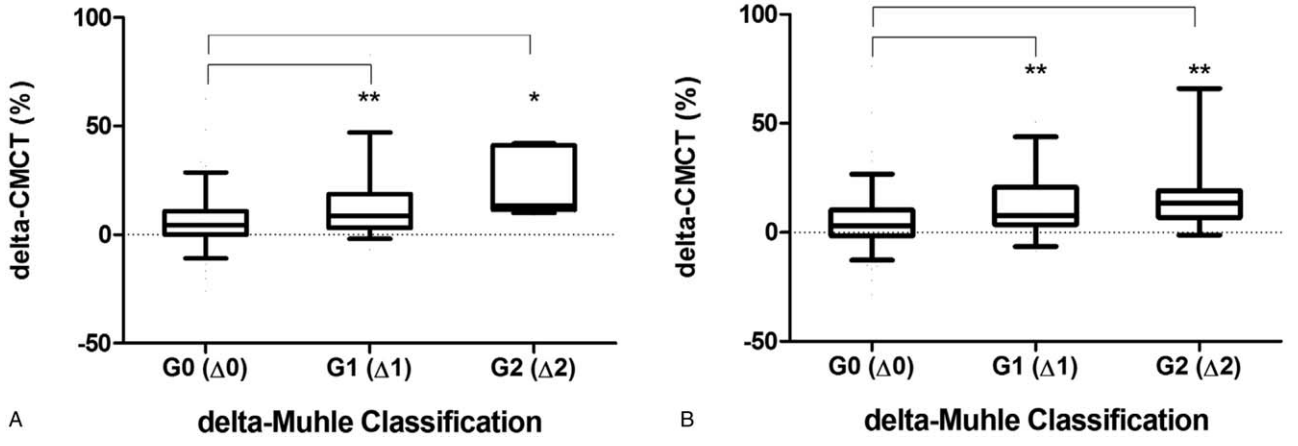

Figure 3. Comparison of delta CMCT according to the alteration of MC during neck flexion (**A**) and neck extension (**B**). Delta CMCT (%) during neck flexion = ([flexion CMCT – neutral CMCT]/neutral CMCT) × 100 (**A**). Delta CMCT (%) during neck extension = ([extension CMCT – neutral CMCT]/neutral CMCT) × 100 (**B**). CMCT indicates central motor conduction time; MC, Muhle classification grade. $^{*}P < 0.05$; $^{**}P < 0.01$.

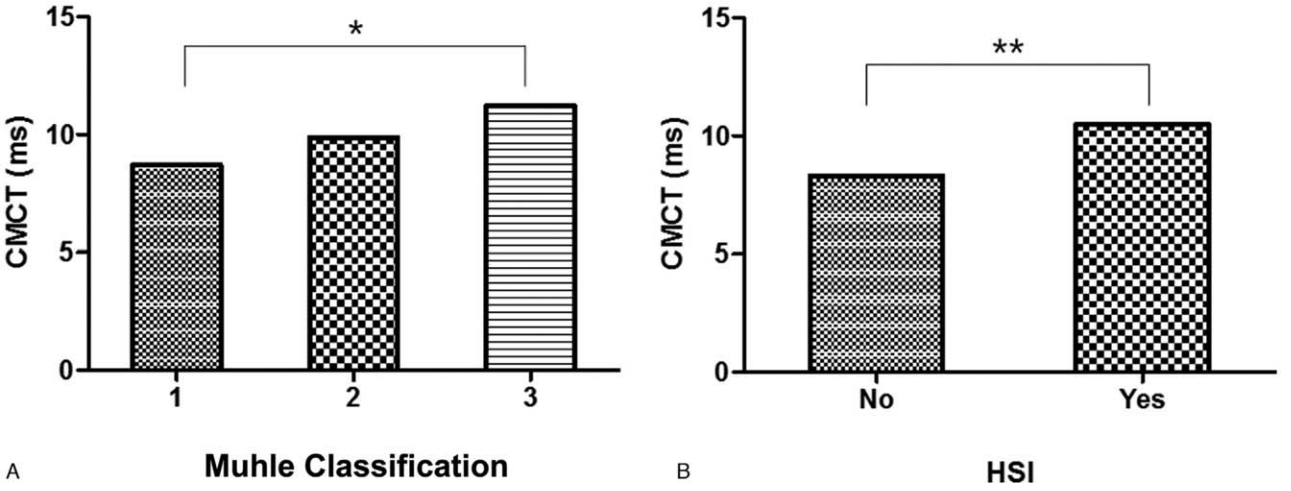

Figure 4. CMCT values by imaging findings in the neutral neck. Comparisons according to MC (**A**) and HSI (**B**). CMCT indicates central motor conduction time; HSI, high signal intensity; MC, Muhle classification grade. $^{*}P < 0.05$; $^{**}P < 0.01$.

DISCUSSION

Our results demonstrated that CMCT could reflect dynamic aspects of cervical cord compression according to neck motions already seen in studies on dynamic MRI. We saw that the CMCT was significantly slower both in neck flexion and extension than in the neutral neck position.

Also, groups with higher severity on cervical MRI–MC grade 3, HSI–showed delayed CMCT compared to groups with mild severity such as MC grade 1 and no HSI. From these results, we could confirm that there was relevance between the cervical MRI and the CMCT in the neutral neck position. Another notable observation was that delta CMCT showed significant differences according to MC grade changes. Therefore, we also identified the association between the dynamic MRI and the flexion-extension CMCT.

When we conducted subgroup analysis, we found that, for the MC grade 3 group, CMCT during flexion and extension was not significantly delayed than that in the neutral neck position. This indicates that if severe cord compression has already been discovered in a static study, then the effect of neck movement on cervical cord impingement is relatively low. This finding does not seem to have an important implication because severe CSM can be easily diagnosed clinically with static studies without performing dynamic studies. In our experience, there were patients whose MEP was already not evoked or extremely delayed in the static study. In those cases, we have not conducted the dynamic studies. Moreover, they have not been considered in view of the symptom aggravation and patient's safety.

On the contrary, our findings suggest that dynamic CMCT has considerable diagnostic usefulness in MC grade 1 and 2 groups whose disease severity is mild to moderate. Our results revealed that more than one-third of patients whose CMCT was within normal range in neutral neck turned out to show delayed CMCT during neck flexion or extension. These results imply that the static CMCT alone is limited in confirming myelopathy, especially in patients with borderline CMCT values. Thus, we can infer that the dynamic CMCT contributes significantly to diagnosing cervical compressive myelopathy and reducing the risk of surgical delay as well as misdiagnosis. In our study, we excluded patients who showed no response to MEP stimuli. However, we have experienced some cases that MEP was triggered in neutral neck, but not during flexion or extension. Consequently, including such cases can increase the importance of dynamic CMCT.

Previous works on dynamic MRIs have univocally reported that cervical cord compression is more provoked during neck extension.[21,22] However, studies on MRI with neck flexion have not been as uniform in their reporting.[23] Rather, some research has found that the spinal canal diameter widened during neck flexion.[6,24,25] In this study, we assessed the changes of the spinal canal diameter during neck flexion and extension by looking at the MC grades. Contrary to several existing reports on the topic, we found that MC grades were either kept at the same grade or went up a grade in both flexion and extension compared to the neutral neck position. We suggest several reasons for this result. MC is divided into different grades, and it is possible that a slight increase in the spinal canal diameter is not enough to change the MC grade. In addition, we determined the MC grade at the level with the most severe stenosis—the level where degenerative changes have progressed considerably. Because those degenerative lesions are mainly located in front of the cervical cord, the pressure on the spinal canal and the cervical cord can be increased when the patient's neck is flexed. In the same sense, most patients referred to physiatrists for the CMCT study were suspected of myelopathy. All subjects presented clinical symptoms and were read as MC grade 1 or higher in static MRI. For this reason, most patients had some degree of advanced pathological changes

and cervical canal narrowing was also aggravated during neck flexion.

Based on our results, we have identified that neck motions—flexion and extension—increase cervical cord compression in terms of neurophysiology. This finding supports previous studies on dynamic MRI which insist that the spinal canal narrowing worsens during neck flexion. Our discovery is also evidenced by the MRI findings in this study. Furthermore, our finding means that neck movement provokes either temporary or continuous cervical cord compression in CSM patients and cord impingement due to repeated neck motions causes symptoms to progress and worsen slowly.

Numerous studies have described the mechanisms causing spinal canal stenosis and cord compression due to neck motions. Most space-occupying lesions due to cervical degeneration occur in the anterior to the cervical cord.[7,26] Diseases such as disc protrusion, OPLL, vertebral osteophyte, and retrolisthesis can cause an increase in mechanical compression of the spinal canal and the cervical cord during neck flexion.[27] Meanwhile, during neck extension, spinal canal narrowing becomes prominent and the spinal cord shifts forward. Those changes increase the pressure received by the spinal canal due to aforementioned anterior cervical lesions.[28,29] Furthermore, most CSM patients experience thickening of the ligament flavum and facet joint hypertrophy which are another common causes of increasing pressure on the spinal canal and the cervical cord during neck extension.[2,30] Microvascular injury is yet another mechanism for explaining cord injury caused by repeated neck movement. Cervical cord stretching occurs during neck flexion and cervical cord shortening and thickening occur during neck extension. Such changes are known to be associated with minute ischemic events.[6,25,31]

Although the CMCT is an excellent test for diagnosing myelopathy through neurophysiological conditions, it is limited in identifying causative diseases and the level of compression. Moreover, an imaging study is essential to formulate a surgical strategy in patients with surgical decisions. Accordingly, to increase the accuracy of diagnosis and at the same time determine the correct decision for surgical treatment, it becomes critical to utilize both the dynamic CMCT and the dynamic MRI in complementary fashion.

We present the following limitations of our study. We reviewed diagnostic usefulness only with laboratory findings. Although we have presented the baseline mJOA score of patients, the score has been provided only as a demographic feature of patients and not for analytic purposes. Thus, comprehensiveness is lacking among actual clinical findings, neurophysiological studies, and imaging findings. We strongly recommend future studies that apply clinical data along with static and dynamic CMCT. Because the methods of CMCT measurement that we utilized cannot accurately reflect lesions below the C6/7 level, patients with lower cervical lesions were not included in this study.[15,32] Thus, the findings of our study are not necessarily consistent with CSM patients of all levels. Fortunately, lower cervical levels are the least mobile part of the cervical vertebrae.[33] Therefore, we view the impact of excluding patients with lower cervical levels to be minimal. Despite the fact that many CSM patients were accompanied with gait instability, our results only presented the CMCT results of upper limb. If there were results of the lower limb CMCT, our findings might be more reliable.

In conclusion, CMCT is significantly slower with neck flexion or neck extension than with neutral neck. This finding reflects the dynamic cord impingement of CSM already revealed in previous studies on dynamic MRI. Performing flexion-extension CMCT is also useful to increase the sensitivity of CSM diagnosis, especially in cases with disease severity ranging from mild to moderate. Therefore, the two studies–dynamic CMCT and dynamic MRI–can be utilized in complementary fashion for diagnosing CSM.

➢ Key Points

- ❑ Spinal cord impingement provoked by neck movement is common in CSM.
- ❑ CMCT was significantly delayed during neck flexion and extension, which demonstrated dynamic cord compression previously seen in dynamic MRI studies.
- ❑ We recommend that the flexion-extension CMCT should be considered for assessing patients with CSM, especially those with disease severity ranging from mild to moderate.

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