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Abstract: Using a percutaneous magnetic stimulator, motor evoked potentials (MEP) induced by magnetic stimulation of the cerebrum and the cervical vertebrae were recorded at the muscles to calculate the central motor conduction time after magnetic stimulation CMCT(Mg) from the motor area of the cerebral cortex to the cervical part of the spinal cord, based on latency differences in MEP. Central motor conduction time by F waves CMCT(F) was calculated by subtracting the peripheral latency from the latency of MEP produced by cranial magnetic stimulation. The subjects consisted of 20 healthy subjects, 22 patients with cervical nerve compression, 18 patients with nerve root (C6 or C7) compression, and 24 patients with peripheral nerve compression. The mean CMCT(Mg) was 6.73 ms, and the mean CMCT(F) was 5.56 ms at the abductor pollicis brevis (APB) in healthy subjects. At the abductor digiti minimi (ADM), the mean CMCT(Mg) was 7.10 ms, and the mean CMCT(F) was 6.05 ms. Both CMCT(Mg) and CMCT(F) were significantly longer in patients with spinal compression than in healthy subjects. In patients with nerve root compression, CMCT(Mg) at the APB of the impaired side was significantly longer than that at the healthy side and that in healthy subjects.

These results suggest that CMCT(Mg) represents the time required to conduct nerve impulses from the motor area of the cerebral cortex to the outlet of the intervertebral foramen of the nerve root, while CMCT(F) represents the time required to conduct nerve impulses from the motor area of the cerebral cortex to the spinal ventral horn cells.

Key words: magnetic stimulation, central motor conduction time (CMCT), motor evoked potential (MEP), spine.

Introduction

Conventional electrophysiologic diagnosis of spinal cord function relies mostly on examination of somato-sensory evoked potentials (SEP)\(^1\)\(^-\)\(^3\) and spinal cord evoked potentials (SCEP)\(^4\)\(^-\)\(^6\). However, these methods are effective primarily for evaluation of the ascending tract and posterior components of the spinal cord, and do not provide sufficient information about the motor tract, which is a descending tract of the spinal cord. Recently, therefore,
various methods to record motor evoked potentials (MEP) by stimulating the cerebrum, spinal cord, and nerve roots percutaneously to evaluate the motor tract function have been tested\textsuperscript{7,8}.

One objective of this study was to calculate the spinal central motor conduction time (CMCT) from the motor area of the cerebral cortex to the cervical spinal cord on magnetic stimulation by subtracting the latency of MEP obtained by percutaneous magnetic stimulation of the cervical region from the latency of MEP obtained by percutaneous magnetic stimulation of the cranium. A second aim was to compare this value with the peripheral latency calculated by Kimura's method using F wave, which occurs after the direct motor potential (M wave)\textsuperscript{9}, and to evaluate the characteristics of each parameter.

**Subjects**

**Healthy group**

Twenty healthy individuals were enrolled and composed the healthy group. They consisted of 11 males and 9 females aged 20–54 years (mean 31.0). Their height was 150–178 cm (mean 164.5 cm).

**Cervical spinal cord compression group**

Twenty-two patients with cervical myelopathy composed the cervical spinal cord compression group. They consisted of 18 males and 4 females aged 49–82 years (mean 62.1). Their height was 148–175 cm (mean 162.3 cm). The score according to the evaluation criteria of the Japan Orthopaedic Association for therapeutic results of cervical myelopathy (JOA score) was 6/17–13/17 (mean 8.9/17).

**Nerve root compression group**

Eighteen patients with nerve root compression at C\textsubscript{6} and/or C\textsubscript{7} formed the nerve root compression group. There were 11 with cervical herniated intervertebral disc and 7 with cervical spondylotic radiculopathy. These 12 males and 6 females were aged 23–82 years (mean 41.7). Their height was 146–176 cm (mean 160.7 cm). The level of nerve root compression according to symptoms and imaging studies was C\textsubscript{6} in 12 patients and C\textsubscript{7} in 6 patients. Patients who showed even the slightest symptom of myelopathy such as a hyperreflexia of the lower limbs were excluded.

**Peripheral nerve compression group**

Twenty-four patients were enrolled in the peripheral nerve compression group. They consisted of 14 patients with electrophysiologically diagnosed carpal tunnel syndrome, who were all females, and 10 patients with cubital tunnel syndrome, who were all males. They were aged 32–67 years (mean 50.5), and their height was 144–175 cm (mean 161.8 cm). Patients in whom differential diagnosis from symptoms due to cervical nerve root compression was needed were excluded.

**Methods**

The method, safety, and usefulness of the test were clearly explained to the subjects, and their consent was obtained before the test. The test was performed while the subjects were seated in a chair with the shoulder joint hanging naturally, the elbow slightly flexed, and
the lower arm placed on the anterior aspect of the femoral region.

**Magnetic stimulation**

Magnetic stimulation was given using a Model 200 magnetic stimulator of Magstistm and a double cone coil. A magnetic field of 1.4T was generated in the center of the coil at the maximum stimulation intensity. Cranial magnetic stimulation was performed by stimulating the motor area of the upper limb at 20–30% higher than the threshold (minimum 80%, maximum 100%) according to the Report of the Committee for Methods of Magnetic Stimulation by the Japanese Society of Electroencephalography and Electromyography\(^1\)). Cervical spinal nerves were stimulated by adjusting the center of the coil to the level of the transverse processes of C₆–C₇ at an intensity 20–30% higher than the threshold (minimum 80%, maximum 100%) similarly to cranial stimulation. Stimulation of both the cranial and cervical regions was made at intervals of 5 seconds or longer to a maximum of 5 times per limb, and frequent stimulation was avoided.

**Records**

The recording device used was a Counterpoint from DANTEC. Motor evoked potentials (MEP) were recorded from the bilateral abductor pollicis brevis muscle (APB) and abductor digiti minimi muscle (ADM) using surface electrodes during mild voluntary contraction (facilitation). The signals were added to a maximum of 5 times until traces that allowed clear evaluation of the latency, amplitude, and duration were obtained.

**Measurements of M and F waves**

The median nerve was stimulated for APB, and the ulnar nerve was stimulated for ADM, at 7 cm proximal from the belly of each muscle in the wrist using a Counterpoint from DANTEC, and M and F waves were recorded. Records on stimulation were obtained 20 times from each muscle, and the shortest leading edge latency obtained was regarded as the latency of the F wave.

**Calculation of the central motor conduction time (CMCT)**

\(a\) **CMCT recorded by cervical magnetic stimulation:** \(CMCT(Mg)\)

\(CMCT(Mg)\) was calculated by subtracting the latency of MEP obtained by cranial magnetic stimulation from the latency of MEP obtained by cervical stimulation. Both latencies were the leading edge latency.

\[CMCT(Mg) = \text{Latency of MEP by cranial stimulation} - \text{Latency of MEP by cervical stimulation}\]

\(b\) **CMCT using F and M waves:** \(CMCT(F)\)

First, the conduction time from spinal ventral horn cells to each muscle, i.e. peripheral latency (PL), was calculated by the following formula based on Kimura's method\(^9\).

\[PL = \frac{\text{Latency of M wave} + \text{Latency of F wave} - 1}{2}\]

\(CMCT(F)\) was calculated by subtracting PL from the latency of MEP obtained by cranial magnetic stimulation.

\[CMCT(F) = \text{Latency of MEP obtained by cranial stimulation} - PL\]

Fig. 1-a and b show normal waves of a 29-year-old healthy male and the various values described above.
Results

Healthy group

In APB, the mean CMCT(Mg) was $6.73 \pm 0.37$ ms, and the mean CMCT(F) was $5.56 \pm 0.46$ ms. In ADM, the mean CMCT(Mg) was $7.10 \pm 0.55$ ms, and the mean CMCT(F)
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Fig. 2. Comparison of mean (plus standard deviation) CMCT(Mg) and CMCT(F) in healthy subjects (n=40).
Mean value of [CMCT(Mg) - CMCT(F)] at the APB in respective individuals: 1.16±0.35 ms.
Mean value of [CMCT(Mg) - CMCT(F)] at the ADM in respective individuals: 1.05±0.37 ms.
* p<0.01 Pearson's correlation coefficient test

![Fig. 2](image)

Fig. 3. Comparison of mean CMCT(Mg) and CMCT(F) between patients with cervical compression (myelopathy) and healthy subjects.
* p<0.01 student's t-test

![Fig. 3](image)

was 6.05±0.41 ms. CMCT(Mg) and CMCT(F) were significantly correlated in both APB and ADM (Pearson's test of correlation coefficient, p<0.01), and the mean difference between CMCT(Mg) and CMCT(F) in each individual was 1.16±0.35 ms in APB and 1.05±0.37 ms in ADM (Fig. 2).

**Spinal cord compression group**

In the spinal cord compression group, the mean CMCT(Mg) was 12.90±3.37 ms in APB and 12.90±3.27 ms in ADM, and the mean CMCT(F) was 11.07±2.89 ms in APB and 10.51±3.26 ms in ADM. Both CMCT(Mg) and CMCT(F) were significantly longer...
compared with the healthy group (student's t-test: p<0.01) (Fig. 3).

**Nerve root compression group**

In the nerve root compression group, the mean CMCT(Mg) was 8.48 ± 0.78 ms in APB on the affected side and 7.08 ± 0.53 ms in APB on the normal side, and 7.47 ± 0.43 ms in ADM on the affected side and 7.01 ± 0.83 ms in ADM on the normal side. The mean CMCT(F) was 7.08 ± 1.32 ms in APB on the affected side and 6.23 ± 0.92 ms in APB on the normal side, and 6.40 ± 0.67 ms in ADM on the affected side and 6.17 ± 0.87 ms in ADM on the normal side. In this group, symptoms due to nerve root compression at C₆ or C₇ were observed, and CMCT(Mg) in APB on the affected side was longer than that on the normal side and that in the normal group (student's t-test: p<0.01), but CMCT(F) showed no significant prolongation. In ADM, no significant prolongation was observed in CMCT(Mg) or CMCT(F) (Fig. 4).

**Peripheral nerve compression group**

In the peripheral nerve compression group, the mean CMCT(Mg) was 7.65 ± 0.57 ms in APB on the affected side, 7.11 ± 0.41 ms in APB on the normal side, 7.68 ± 0.51 ms in ADM on the affected side, and 7.32 ± 0.55 ms in ADM on the normal side. The mean CMCT(F) was 5.51 ± 1.23 ms in APB on the affected side, 5.68 ± 0.24 ms in APB on the normal side, 5.62 ± 0.75 ms in ADM on the affected side, and 6.11 ± 0.60 ms in ADM on the normal side. In this group, no significant prolongation was observed in CMCT(Mg) or CMCT(F) (Fig. 5).

The amplitude was low in waves with a prolonged latency and was markedly low in patients showing severe atrophy in the muscle from which MEP was recorded.
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Fig. 5. Comparison of mean CMCT(Mg) and CMCT(F) between patients with peripheral nerve root compression and healthy subjects.

Fig. 6. [cace 1] 52 yo male. Cervical myelopathy. Plain X-ray films showed spondylotic change. MRI showed narrowing of the vertebral canal from C2 to C7 and intramedullary highintensity areas in the T2-weighted image at C2/3 and C3/4 level.

Presentation of Cases

Spinal cord compression group

[Case 1] A 52-year-old male visited with a primary complaint of impairment of dexterous movements of both upper limbs. Numbness, hypesthesia, hyperreflexia, and pathological reflexes were observed in the bilateral upper and lower limbs, and symptoms of
myelopathy were present. The condition was 10/17 according to the criteria for evaluation of the therapeutic results of cervical myelopathy of the Japan Orthopaedic Association (JOA score). Magnetic resonance imaging (MRI) showed narrowing of the vertebral canal from C2 to C7 and intramedullary high-intensity areas in the T2-weighted image at the C2/3 and C3/4 level (Fig. 6).

The latency of MEP induced by percutaneous cranial magnetic stimulation was prolonged
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Fig. 8. [case 2] 28 yo male. Cervical herniated intervertebral disc. Plain X-ray films showed disc space narrowing C5/6 level. MRI showed degeneration of the intervertebral disc and left posterolateral protrusion of the intervertebral disc at C5/6 level.

Fig. 9-a. [case 2] CMCT(Mg) : 28 yo male. Cervical herniated intervertebral disc.

on both sides and in both the APB and ADM compared with the mean of the normal group, and CMCT(Mg) was 14.5 ms in the right APB, 14.3 ms in the right ADM, 13.8 ms in the left APB, and 15.7 ms in the left ADM, all of which were markedly prolonged compared with the normal group (Fig. 7-a). The M wave was in the normal range, and the F wave was slightly prolonged compared with the normal group in all these muscles. CMCT(F) was 13.3 ms in the right APB, 13.1 ms in the right ADM, 11.0 ms in the left APB, and 14.8 ms in the left ADM (Fig. 7-b).

Nerve root compression group

[Case 2] A 28-year-old male visited with complaints of weakness of the left upper limb
Fig. 9-b. [case 2] CMCT(F) : 28 yo male. Cervical herniated intervertebral disc.

(a) X-P : lateral view  (b) X-P : cubital tunnel

Fig. 10. [case 3] 56 yo male. Rt. cubital tunnel syndrome. Plain X-ray films showed osteophyte formation in the olecranon fossa and the coronoid fossa of the humerus and neural groove of the ulna.

and numbness of the left lower arm to the thumb. On manual muscle testing (MMT) he was level 4, and there was decreased strength in the left triceps muscle of the left arm, extensor carpi radialis muscles and APB, and reduced tendon reflexes of the biceps muscle of the arm and brachioradial muscle were also observed. MRI showed degeneration of the intervertebral disc and left posterolateral protrusion of the intervertebral disc at the C5/6 level (Fig. 8). The latency of MEP induced by cranial magnetic stimulation was prolonged to 22.8 ms only at left C6 including the level of the affected nerve root, i.e. left APB. CMCT(Mg) was prolonged to 8.6 ms in the left APB compared with the healthy group.
The latency of F wave was markedly prolonged in the left APB, the peripheral latency (PL) was prolonged to 16.9 ms, and CMCT(F) was not prolonged at 5.9 ms (Fig. 9-b).

**Peripheral nerve compression group**

[Case 3] A 56-year-old male had noted numbness in the ulnar nerve area of the right hand for about 3 years. A sensation resembling Tinsel’s sign was observed in the right hand (Fig. 9-a).
cubital tunnel, and the right ADM was weakened and atrophied. Plain X-ray films showed osteophyte formation in the olecranon fossa and the cronoid fossa of the humerus and neural groove of the ulna (Fig. 10). The ulnar motor nerve conduction velocity was markedly reduced to 14.8 m/s in the right cubital tunnel, sensory nerve active potentials (SNAP) were not recorded, and a diagnosis of right cubital tunnel syndrome was made. The latency of MEP by cranial magnetic stimulation was markedly prolonged to 27.5 ms and was reduced in amplitude in the right ADM supplied by the affected nerve. The latency of MEP recorded by cervical stimulation was also prolonged in the right ADM, and the latency of CMCT(Mg) was not prolonged at 6.7–7.9 ms (Fig. 11-a). The amplitude of the F wave induced by wrist stimulation was reduced in the right ulnar nerve, and the latency was also markedly prolonged to 42.0 ms with a marked prolongation also of the peripheral latency (PL) to 22.4 ms. CMCT(F) was normal at 5.2–5.8 ms on all measurements (Fig. 11-b).

**Discussion**

**Evolution of changes in methods for recording of MEP by cranial stimulation**

In noninvasive stimulation of the cerebrum, effective stimulation cannot be applied to nerve tissues with conventional electric stimulators, because stimulation is blocked by the cranium which has a high electric resistance. In 1980, Merton & Morton⁸ developed a method for functional evaluation of the motor tract by stimulating the cortex by percutaneous cranial electric stimulation using a high-voltage low-output impedance stimulator and recording evoked electromyograms from limb muscles. However, this method was difficult to perform in conscious subjects, because stimulation was painful.

In 1985, Barker et al.⁹ succeeded in recording motor evoked potentials (MEP) from limb muscles by stimulating the cerebrum from the body surface using a percutaneous cranial magnetic stimulator. Magnetic stimulation is advantageous in that the cerebral cortex can be stimulated percutaneously without pain so that it can be performed readily in conscious subjects. This technique is suitable for clinical application as a method for objective and minimally invasive evaluation of the motor tract¹¹⁻¹⁴.

**Safety of cranial magnetic stimulation**

When a massive pulsed electric current is run through a coil, a fluctuating magnetic field is instantaneously generated in the body, and local eddy currents are induced. The heat caused by the eddy currents and overexcitation due to changes in the membrane potential have been reported as possible causes of nerve tissue injury¹⁵. The quantity of heat produced in tissue with this instrument is calculated to be a maximum of 2 mW and is 1/300 or less of the international standard¹⁶. Overexcitation has been suggested to induce epileptic attacks and to increase the risk of atrial fibrillation in the myocardium⁷. According to Barker et al.¹⁶, the maximum electromotive current generated by a magnetic stimulator is about 50 \( \mu \)c/pulse, which is only 1/2,000 to 1/20,000 of the capacity used for electroconvulsive therapy (100 mc-1c). “Kindling”, which has been raised as an issue in the field of psychiatry, cannot be induced with stimulation of 3 Hz or less, and the maximum stimulation of 1/3 Hz produced by this instrument is considered to be safe¹³. However, patients with a history of epilepsy or atrial fibrillation and those with metal pieces implanted in the body should be excluded for assurance. Although some of our patients
Interpretation of the central motor conduction time from the motor area of the cerebral cortex to the cervical spinal cord

In the healthy group, CMCT(Mg) was about 1 ms longer than CMCT(F) in both the APB and ADM. When the spine at the cervical region is magnetically stimulated, the nerve roots are considered to be stimulated at their emergence through the intervertebral foramen\(^{19,20}\). This was confirmed experimentally by Peter et al. in 1994\(^{21}\). The latency of MEP induced by cervical magnetic stimulation means the conduction time from the opening of the intervertebral foramen to the muscle from which the potentials were recorded. The F wave, on the other hand, is a potential generated as the stimulation applied to peripheral nerves ascends retrograde in the motor tract, excites spinal ventral horn cells, which takes about 1 ms, and induces reflex muscle contraction\(^{22}\). The peripheral latency calculated using the F wave (PL = (F + M - 1)/2) means the time from the spinal ventral horn cells to the muscle from which the potentials were recorded\(^{9,22}\). Therefore, CMCT(Mg) and CMCT(F) are considered to represent the conduction time from the motor area of the cerebral cortex to the nerve root at its emergence through the intervertebral foramen and the conduction time from the motor area of the cerebral cortex to spinal ventral horn cells, respectively. This difference is considered to have caused the difference of about 1 ms from the healthy group.

Both CMCT(Mg) and CMCT(F) were significantly prolonged in the spinal cord compression group compared with the healthy group. This was probably a result of conduction disturbance due to compression from the more cranial part of the spinal cord itself, i.e. the motor tract, in the vertebral canal, and compression of spinal ventral horn...
cells (Fig. 12).

In the nerve root compression group, which included patients with symptoms due to compression of 1 or 2 nerve roots, and excluded those who showed symptoms of myelopathy, only CMCT (Mg) of the APB on the affected side including the level of the compressed nerve roots was significantly prolonged. This was considered to be the result of conduction disturbance due to compression of a proximal part of the nerve roots caused by thickening of the intervertebral articulation, osteophyte formation, or protrusion of the intervertebral disc (Fig. 12).

In the peripheral nerve compression group, which had entrapment neuropathy, neither CMCT (Mg) nor CMCT (F) was significantly prolonged although the latency of MEP induced by cranial magnetic stimulation was prolonged in the muscle supplied by the impaired nerve, probably because the latency of MEP induced by cervical magnetic simulation and peripheral latency using the F wave were also prolonged.

Factors that affect the central conduction time

The nerve conduction time slightly increases with age, starting after 30 to 40 years of age, but this change is reportedly small even at 60 or 80 years of age\(^{22}\). Differences in the age distribution among various groups should therefore be considered. However, as the results of our measurement of the conduction time were similar between elderly and younger healthy subjects, we could not ascribe the prolongation of the conduction time to slowing of conduction due to aging alone. Further evaluation of the conduction time in elderly healthy individuals is needed to clarify this point.

Clinical application of this method

The present study has established that CMCT (Mg) and CMCT (F) represent the conduction time from the motor area of the cerebral cortex to the nerve root at its emergence through the intervertebral foramen and the conduction time from the motor area of the cerebral cortex to spinal ventral horn cells. Detailed comparison of the two conduction times may provide a means of differentiating whether upper motor neurons or lower motor neurons are impaired.

Problems to be approached in the future

Comparison of the MEP predominantly supplied by each nerve root and the central conduction time may provide an important method for determination of the level of impairment. However, the precision of this examination is presently insufficient for determination of the level of impairment, because of the lack of reproducibility due to a low MEP detection rate by magnetic stimulation from muscles other than intrinsic muscles of the hand or foot, and because of artifacts due to the close proximity of the point of magnetic stimulation and the muscle from which potentials are recorded. Improvements in the shape of the stimulation coil and modification of the recording electrodes are needed to improve the precision of this method so that it may be applied in clinical practice.

References

2) Yiannikas MB, Shahani BT and Young PR: Short-latency somatosensory-evoked potential from radial, median,


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