Iraqi JMS

Published by Al-Nahrain College of Medicine
ISSN 1681-6579
Email: iraqijms@colmed-alnahrain.edu.iq
http://www.colmed-alnahrain.edu.iq

# Diagnostic value of Somatosensory Evoked Potentials in Cervical Myelopathy

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## Abstract

**Background** Cervical myelopathy is a condition caused by narrowing of the spinal canal leading to cord dysfunction. The most common causes are congenital stenosis and degenerative stenosis caused by spondylosis.

**Objectives** To confirm the diagnosis of cervical myelopathies using somatosensory evoked potentials and possibly to localize the level of the lesion.

**Methods** An electrophysiological study had been carried on 61 patients with cervical myelopathy (41 female and 20 male) aged 48.66±11.72 years and 41 healthy volunteers aged 44.8±10.53 years. Sensory and motor nerve conduction study and somatosensory evoked potential for all were done to evaluate the peripheral nerves and sensory central pathways.

**Results** No significant difference was demonstrated in the sensory and motor nerve conduction studies from the healthy subjects. Somatosensory evoked potentials showed statistically highly significant changes in the N13, N20 latencies, amplitudes and N13-N9 and N20-N13 central sensory conduction times of median nerve on both sides. N13 latency has the highest specificity and sensitivity among the somatosensory evoked potentials parameters. Those patients who had prolonged central sensory conduction time between N20-N13 suggests an upper cervical lesion while those having prolonged central sensory conduction time between N13-N9 suggest lower cervical cord and/or cervical root affection.

**Conclusion** Motor and sensory conduction studies are usually normal in CM. Among SSEPs parameters, N13 latency was prolonged bilaterally, CSCT abnormal bilaterally, N13-N9 and N20-N13 latencies unilaterally (Right side). Mononeuropathies, polyneuropathies, radiculopathies and plexopathies should be excluded before diagnosis of CM was made.

**Key words** Cervical myelopathy, electroneuromyograhpy, somatosensory evoked potentials.

**List of abbreviation:** CM = Cervical myelopathy, NCS = nerve conduction studies, CMAP = compound muscle action potential, SSEP = Somatosensory evoked potentials, CNS = central nervous system, EMG = Electromyography, EPs = Evoked potentials, SL = sensory latency, DML, distal motor latency, SNCV = sensory nerve conduction velocity, MNCV = motor nerve conduction velocity, CSCT = central sensory conduction time.

## Introduction

Cervical myelopathy (CM) is a condition caused by narrowing of the spinal canal leading to cord dysfunction [(1)]. The most common causes are congenital stenosis and degenerative stenosis caused by spondylosis. [(2)]. The pathophysiology of CM involves static factors, which results in acquired or developmental stenosis of the cervical canal, and dynamic factors, which involve repetitive injury to the cervical cord [(3,4)]. The clinical findings of CM patient depending on the levels affected, involvements of the neural foramina and long tract. A variety of neurological signs and symptoms may be present, including sensory changes, reflex abnormalities, decreased dexterity, weakness, gait instability, bowel and bladder dysfunction, spasticity, presence of Hoffman's and/or Babinski's sign,

axial neck pain, radiculopathy, and even acute spinal cord injury [5-7].

**Neurophysiology assessment of CM**

Clinical neurophysiology is an area of medical practice focused primarily on measuring function in the central nervous system, peripheral nervous system and muscles as an extension of the neurologic evaluation; employs the same anatomic principles of localization as clinical examination [8,9], while nerve conduction studies (NCS) were used to evaluate the propagation of nerve action potentials along motor or sensory nerve fibers [10]. They provide means of confirming the presence and extent of peripheral nerve damage [11].

F-wave is a late compound muscle action potential (CMAP) [12]. Assessment of F-wave latency is very useful in clinical neurophysiology especially the proximal segment of the nerve [9,10]. While the somatosensory evoked potentials are presynaptic and postsynaptic responses recorded over the limbs, spine, and scalp following the stimulation of peripheral mixed motor and sensory nerves or cutaneous sensory nerves. The electrical potentials generated by various portions of the ascending sensory pathways can be easily elicited and recorded and can be used to examine the functional integrity of somatosensory pathways [8,11].

Median nerve responses are most commonly used [11]. It receives contributions from both the medial and lateral cords of the brachial plexus and contains fibers spanning from the C5 to T1 roots [8]. Following stimulation of the median nerve at the wrist, activity can be recorded at the Erb's point, cervical spine, and scalp. Several different peaks are identified with standard recording montages: N9, N11, N13, N14, and N20.

Abnormal somatosensory evoked potentials (SSEPs) can result from dysfunction at the level of the peripheral nerve, plexus, spinal root, spinal cord, brain stem, thalamocortical projections, or primary somatosensory cortex [13,14]. SSEP primary use is to determine compromised central nervous system (CNS) conduction. It confirms symptoms when few physical findings are noted. SSEP may confirm or reject the presence of a suspected conduction block and able to establish an anatomic region where the conduction disturbance or block occurs [15].

The intention of this study is to confirm the diagnosis of cervical myelopathies using SSEPs and conventional electromyography (EMG) study and possibly localize the level of the lesion.

## Methods

This study was conducted at the Neurophysiology Unit at Al-Imamein Al-Kadhimein Medical City for the period extended from Apr. 2013 to Apr. 2014. An ethical consent was taken from each participant to be enrolled in the study.

Sixty one patients (20 males and 41 females) aged 48.66±11.72 years with documented CM diagnosed by a senior neurosurgeon were studied. The disease duration was 1 to more than 5 years.

Forty one healthy volunteers aged 44.8±10.53 years serve as the control group; they were clinically examined by the same neurosurgeon to be included in the study. The patients and controls with history of diabetes mellitus, alcoholism, uremia and other metabolic diseases were excluded from the study.

All studied subjects underwent the following neurophysiologic tests:

- Sensory nerve conduction (SNC) of the median and ulnar nerves (both sides)
- Motor nerve conduction (MNC) and F-wave studies of the median and ulnar nerves (both sides).
- SSEPs of both median nerves.

Routine computerized EMG /EP machine (Micromed, 8-channel elecromyograph) supplemented with different types of electrodes including grounding electrode used to protect the subject against electrical hazard

and to reduce stimulus artifacts and interference, stimulating surface electrodes was used to stimulate the nerves through the skin, surface recording electrodes and disposable subdermal monopolar electrodes.

**Sensory nerve conduction study**

An antidromic method was used for SNC determination in which the nerve was proximally stimulated from the trunk and the evoked activity was distally recorded from a finger. The parameters studied were the sensory latency (SL) measured in milliseconds (msec), Sensory amplitude measured in microvolt (µV) from peak to peak and SNCV measured by dividing the conduction distance in millimeter (d) by the SL in msec.

**Motor Nerve Conduction Study and F- wave**

The motor nerve was simulated at two points along its course, by applying stimuli at the distal and the proximal sites of the nerve and recording from the muscle innervated by that nerve. The parameters studied were distal motor latency (DML). Motor nerve conduction velocity (MNCV) measured by dividing the distance between the two stimulation points over the difference between the latencies of the recorded responses ensuring both CMAP configurations must be similar in addition to F wave latency measured from the stimulus artifact to the beginning of the evoked potential.

**Somatosensory evoked potentials**

The median nerve was stimulated by bipolar stimulating electrode placed over the median nerve at the wrist. The electrical stimuli were square-wave pulses given at rate of 2-3 /sec at high pass filter 4 Hz, low pass filter 500 Hz with time base 50 ms duration and gain 5 µv/Div. The stimulus intensity was adjusted to produce a visible twitch in the APB muscle without causing any discomfort. To confirm the reproducibility of the SSEP, each measurement was carried out at least three times.

The recording disposable subdermal monopolar needle electrode was placed at the following locations: Erb's point on each side (EPi) and (Epc), over the second and fifth cervical spine process (C2S, C5S), scalp over the contralateral cortex (CPc) and cephalic Fz electrode (Reference). The parameters studied in SSEPs study of median nerve include the latency, amplitude and central sensory conduction time (CSCT).

**Statistical Analysis**

The statistical analysis was obtained using statistical package of social sciences (SPSS) version 19 software and Microsoft Office Excel 2007. All data of were expressed as mean ± SD. Data from each patient and control group were compared using independent sample t-test to calculate differences between groups. Paired t-test was used to compare the right and left side within the same group. P-value of 0.05 or less was considered significant.

Cutoff values of the prolonged latencies, CSCT, accordingly the sensitivity and specificity were evaluated by using receiver operating curve (ROC). The percentage of abnormal values in SSEP tests is calculated according to cutoff value of the normal values for the control group.

## Results

**Nerve conduction study**

The parameters of median and ulnar sensory and motor nerve conduction studies for the control subjects were presented in table 1. Paired t test was done and demonstrate no significant difference between the two sides.

**Somatosensory evoked potentials**

The latency, amplitude and CSCT of different SSEP components in the right and left upper limbs were presented in table 2. No significant difference was noticed between the two sides using paired t test.

**Table 1. Nerve conduction parameters of the right and left median and ulnar nerves in the controls**

| Parameters | Nerve | Right side N= 41 | Left side N = 41 | P value |
|---|---|---|---|---|
| SL (msec) | Median | 2.37±0.38 | 2.22±0.34 | 0.0864 |
| | Ulnar | 2.22±0.33 | 2.23±0.33 | 0.8973 |
| SNAP (µV) | Median | 26.93±5.3 | 28.95±6.56 | 0.1425 |
| | Ulnar | 26.63±5.06 | 28.5±7.17 | 0.1223 |
| SNCV (m/sec) | Median | 54.72±7.14 | 56.62±6.52 | 0.1301 |
| | Ulnar | 57.85±6.29 | 57.52±7.24 | 0.8277 |
| DML (msec) | Median | 2.79±039 | 2.91±0.32 | 0.1301 |
| | Ulnar | 2.56±0.37 | 2.59±0.41 | 0.0737 |
| Distal CMAP (mV) | Median | 7.16±1.49 | 7.85±2.21 | 0.064 |
| | Ulnar | 8.74±3.86 | 7.45±2.37 | 0.1034 |
| Proximal CMAP (mV) | Median | 7.33±1.78 | 6.59±2.75 | 0.2569 |
| | Ulnar | 7.94±2.96 | 7.03±1.92 | 0.1520 |
| MNCV (m/sec) | Median | 56.23±9.84 | 57.17±5.74 | 0.5391 |
| | Ulnar | 57.75±9.95 | 57.11±4.38 | 0.67747 |
| F-wave latency (msec) | Median | 26.52±1.69 | 26.54±1.26 | 0.9628 |
| | Ulnar | 24.92±2.06 | 24.57±2.0 | 0.5501 |

The data presented as mean±SD, SL = sensory latency, SNAP = sensory nerve action potential, SNCV = sensory nerve conduction velocity, DML, distal motor latency, CMAP = compound muscle action potential, MNCV = motor nerve conduction velocity.

**Table 2. Somatosensory evoked potentials parameters recorded from right and left median nerves of the controls**

| SSEPs Parameters | | Right side N=41 | Left side N=41 | P value |
|---|---|---|---|---|
| Latency (msec) | N9 | 9.27±0.23 | 9.22±0.41 | 0.9254 |
| | N13 | 13.06±0.71 | 12.88±0.65 | 0.0866 |
| | N20 | 20.59±1.23 | 20.83±1.1 | 0.2239 |
| Amplitude (µV) | N9 | 4.70±1.43 | 4.46±1.32 | 0.1873 |
| | N13 | 4.04±1.23 | 4.02±1.16 | 0.4764 |
| | N20 | 4.74±1.67 | 5.64±1.71 | 0.0887 |
| CSCT (msec) | N13-N9 | 3.84±0.87 | 3.66±0.69 | 0.1629 |
| | N20-N13 | 7.53±1.43 | 7.96±1.34 | 0.0713 |
| | N20-N9 | 11.37±1.3 | 11.56±1.19 | 0.2849 |

The data presented as mean±SD, SSEPs = somatosensory evoked potentials, CSCT = central sensory conduction time

Because there was no difference between the left and right side data; thus, they were pooled together and regarded as one group for further comparison with the patient data.

**CM versus control subjects**

**Nerve conduction study**

No significant difference was observed between the CM patients and control subjects

concerning the sensory and motor data of the median and ulnar nerves (Tables 3 and 4).

**Table 3: Illustrate the data of median, ulnar sensory nerves in cervical myelopathy patient and control subjects (Unpaired t test).**

| Parameters | Nerve | CM Patients N =61 | Control subjects N =82 | P value |
|---|---|---|---|---|
| Sensory latency (msec) | Rt. Median | 2.34±0.32 | 2.31±0.35 | 0.5564 |
| | Lt. Median | 2.26±0.34 | | 0.4235 |
| | Rt. Ulnar | 2.32±0.29 | 2.23±0.33 | 0.0659 |
| | Lt. Ulnar | 2.23±0.32 | | 0.9328 |
| Sensory amplitude (µV) | Rt. Median | 28.19±6.28 | 27.94±6.01 | 0.8077 |
| | Lt. Median | 30.05±6.83 | | 0.0572 |
| | Rt. Ulnar | 28.86±7.91 | 27.57±6.24 | 0.2923 |
| | Lt. Ulnar | 26.69±8.25 | | 0.4888 |
| Sensory nerve conduction velocity (m/sec) | Rt. Median | 55.66±5.6 | 56.39±6.42 | 0.4724 |
| | Lt. Median | 57.25±5.81 | | 0.3992 |
| | Rt. Ulnar | 55.97±5.53 | 57.69±6.75 | 0.0964 |
| | Lt. Ulnar | 57.56±5.19 | | 0.8962 |

The data presented as mean±SD, CM = cervical myelopathy

**Table 4. Illustrate the data of median, ulnar nerves in cervical myelopathy patient and control subjects**

| Parameters | Nerve | CM Patients N =61 | Control subjects N =82 | P value |
|---|---|---|---|---|
| Distal latency (msec) | Rt. Median | 2.96±0.56 | 2.91±0.32 | 0.1633 |
| | Lt. Median | 3.05±0.5 | | 0.1029 |
| | Rt. Ulnar | 2.47±0.36 | 2.57±0.39 | 0.1115 |
| | Lt. Ulnar | 2.52±0.38 | | 0.4207 |
| Distal CMAP amplitude (µV) | Rt. Median | 7.83±2.42 | 7.85±2.21 | 0.3905 |
| | Lt. Median | 8.15±1.78 | | 0.4513 |
| | Rt. Ulnar | 8.07±3.2 | 8.09±3.25 | 0.9658 |
| | Lt. Ulnar | 8.25±3.21 | | 0.7785 |
| Proximal CMAP amplitude (µV) | Rt. Median | 8.16±2.62 | 7.89±2.53 | 0.2163 |
| | Lt. Median | 8.63±1.93 | | 0.0966 |
| | Rt. Ulnar | 7.49±2.59 | 7.48±2.52 | 0.998 |
| | Lt. Ulnar | 7.56±2.72 | | 0.8729 |
| Motor nerve conduction velocity (m/sec) | Rt. Median | 56.36±9.23 | 56.75±7.95 | 0.7924 |
| | Lt. Median | 58.39±4.21 | | 0.1146 |
| | Rt. Ulnar | 57.78±8.66 | 57.43±7.65 | 0.8022 |
| | Lt. Ulnar | 56.49±8.55 | | 0.497 |
| F wave latency (msec) | Rt. Median | 27.67±5.48 | 26.53±1.48 | 0.1179 |
| | Lt. Median | 27.67±5.43 | | 0.116 |
| | Rt. Ulnar | 24.94±1.94 | 24.75±2.0 | 0.5639 |
| | Lt. Ulnar | 25.25±1.77 | | 0.1137 |

The data presented as mean±SD, CM = cervical myelopathy, CMAP = compound muscle action potential

**Somatosensory evoked potentials**

Apart from N9 latency and its amplitude, all other SSEPs components were significantly different between the studied groups (Table 5).

**Table 5. Somatosensory evoked potentials data of median nerves in the cervical myelopathy patients and controls**

| Parameters | | | CM Patients N =61 | Control subjects N =82 | P value |
|---|---|---|---|---|---|
| Latency (msec) | Right | N9 | 9.35±0.28 | 9.26±0.22 | 0.0501 |
| | | N13 | 14.36±1.76 | 12.97±0.68 | <0.0001 |
| | | N20 | 23.34±4.4 | 20.71±1.17 | <0.0001 |
| | Left | N9 | 9.36±0.33 | 9.26±0.22 | 0.0544 |
| | | N13 | 14.06±1.41 | 12.97±0.68 | <0.0001 |
| | | N20 | 23.75±4.37 | 20.71±1.17 | <0.0001 |
| Amplitude (µV) | Right | N9 | 4.16±1.66 | 4.58±1.37 | 0.1091 |
| | | N13 | 3.1±1.41 | 4.11±1.14 | <0.0001 |
| | | N20 | 3.63±2.02 | 5.25±1.41 | <0.0001 |
| | Left | N9 | 4.25±1.69 | 4.58±1.37 | 0.2119 |
| | | N13 | 3.49±1.79 | 4.11±1.14 | 0.0194 |
| | | N20 | 4.1±2.52 | 5.25±1.41 | 0.0019 |
| CSCT (msec) | Right | N13-N9 | 5.01±1.84 | 3.7±0.7 | <0.0001 |
| | | N20-N13 | 8.96±3.48 | 7.75±1.39 | 0.0117 |
| | | N20-N9 | 13.92±4.52 | 11.43±1.2 | 0.0001 |
| | Left | N13-N9 | 4.7±1.37 | 3.7±0.7 | <0.0001 |
| | | N20-N13 | 9.7±3.52 | 7.75±1.39 | 0.0001 |
| | | N20-N9 | 14.4±4.28 | 11.43±1.2 | <0.0001 |

The data presented as mean±SD, CM = cervical myelopathy, CSCT = central sensory conduction time

**Sensitivity and specificity of SSEP parameters**

**Median nerve**

Cutoff values of the prolonged latencies and CSCT and lower amplitudes of the median nerves were estimated and accordingly the sensitivity and specificity were evaluated. N13 latency shows the highest specificity and sensitivity (Table 6).

**Table 6. Cutoff value, sensitivity and specificity of the median somatosensory evoked potentials**

| Parameters | | Cutoff | Specificity | Sensitivity |
|---|---|---|---|---|
| Latency (msec) | N9 | 9.35 | 52.0 | 50.0 |
| | N13 | 13.25 | 70.7 | 69.7 |
| | N20 | 21.15 | 62.2 | 59.8 |
| Amplitude (µV) | N9 | 4.25 | 53.3 | 52.4 |
| | N13 | 3.85 | 67.2 | 67.1 |
| | N20 | 4.5 | 62.3 | 57.3 |
| CSCT (msec) | N13-N9 | 4.05 | 68.3 | 62.3 |
| | N20-N13 | 7.75 | 57.3 | 56.6 |
| | N20-N9 | 11.75 | 62.2 | 60.7 |

CSCT = central sensory conduction time

The percentage of abnormal median nerves SSEP data according to cutoff value were presented in table 7. The N13 latency of right and left median nerves shows the higher percentage of abnormality.

**Table 7. Percentage of abnormal median somatosensory evoked potentials data according to the cutoff value**

| Parameters | | Cutoff | Right median | | Left median | |
|---|---|---|---|---|---|---|
| | | | No. | % | No. | % |
| Latency (msec) | N9 | 9.35 | 29 | 47.5 | 28 | 45.9 |
| | N13 | 13.25 | 41 | 67.21 | 42 | 68.85 |
| | N20 | 21.15 | 37 | 60.6 | 34 | 55.7 |
| Amplitude (µV) | N9 | 4.25 | 33 | 54 | 27 | 44.2 |
| | N13 | 3.85 | 40 | 65.57 | 34 | 55.7 |
| | N20 | 4.5 | 39 | 63.9 | 33 | 54.1 |
| CSCT (msec) | N13-N9 | 4.05 | 39 | 63.9 | 37 | 60.6 |
| | N20-N13 | 7.75 | 29 | 47.5 | 40 | 65.57 |
| | N20-N9 | 11.75 | 35 | 57.37 | 38 | 62.3 |

CSCT = central sensory conduction time

**Possibility of localization**

The possibility of uni- and bilateral localization of lesion level through recording CSCT between N20-N13 and N13-N9 was assumed by cutoff values of the abnormal data (Table 8).

**Table 8. Localization of lesion level by the cutoff value of abnormal central sensory conduction time of somatosensory evoked potentials**

| Level | N20-N13 | | | N13-N9 | | |
|---|---|---|---|---|---|---|
| | Right | Left | Bilateral | Right | Left | Bilateral |
| C5-C1-cortex | 29 (47.5) | 40 (65.57) | 25 (41) | - | - | - |
| C6-T1 | - | - | - | 39 (63.9) | 37 (60.6) | 37 (60.6) |
| Normal | 32 (50.8) | 21 (34.4) | 17 (27.8) | 22 (36.6) | 24 (39.3) | 19 (31.14) |

## Discussion

In the diagnosis of CM, conventional diagnostic methods such as neurologic findings, image study such as MRI and myelograms are usually performed, but conclusive diagnosis is sometimes difficult because many symptoms tend to be separate from the existing disease. The MRI demonstrates morphologic abnormalities of the cord but not the functional impairment, and not all cord compression shown by MRI is associated with cord dysfunction [16].

**Control group**

No side to side difference was observed in the control group regarding different SNC, MNC and SSEP. The current data were comparable with those reported by other researchers [9,11,17-22].

**Patient versus control group**

**Conventional sensory and motor nerve conduction study**

Conventional NCS are commonly used in lower motor neuron evaluation and they can provide an objective measure for nerve damage. They can confirm the clinical impression of nerve root compression and document or exclude other illnesses of nerves or muscles that could contribute to the patient's symptoms and signs [9,23].

Although the motor and sensory conduction studies are usually normal in CM, they still an essential part of their diagnostic evaluation. mononeuropathies, polyneuropathies, radiculopathies and plexopathies may all need to be excluded before an electrodiagnostic diagnoses of CM can be made; these all require relevant motor and sensory conduction studies.

In CM patients, motor and sensory conduction studies were within the normal limits of the control group and there was no side to side difference, a finding that was in close approximation to the data obtained by other researchers [24, 25].

The F-wave responses that provide information about the conduction rate in alpha motor fibers, especially when the pathological involvement is greater proximally or is located in anterior horn cells were normal in this study. This study documents that NCS remain complementary modalities in the evaluation of CM.

**SSEPs study**

In CM patients, SSEPs changed significantly from those of the control group (prolonged N13 and N20 latencies, low amplitudes and prolonged N13-N9 and N20-N13 CSCT). These findings were in harmony with the findings of other researchers [26, 27].

Patients who had abnormal Erb's point N9 latency and amplitude with normal median nerve sensory and MCS suggesting root affection and normal N9 potential in others indicates normal afferent volley reached the brachial plexus. In such condition, no slowing of impulse velocity exists in the afferent pathways.

The current study showed that N13 latency prolongation shows the highest specificity and sensitivity among other SSEPs parameters. Since studies presumed that N13 component is generated post-synaptically in the posterior horns of C2-C7 [28], more rostrally, possibly in the cuneate nucleus [29]. The timing of N13 with respect to N9 would, therefore, reflect the conduction velocity in the dorsal column fibers. Restuccia [30] found that SSEP segmental N13 medullary response was shown to be a sensitive indicator of medullary involvement in SCM and is believed to be a hallmark of potentially reversible segmental dorsal horn cervical cord dysfunction due to ischemia with a great potential for clinical improvement. Also focal demyelination of the cervical dorsal roots without blocking of the impulse transmission would obviously resulted in delayed N13 and increased N13-N9 conduction time. The N20 potential follows and is delayed in total time because of the conduction delays already demonstrated in the roots and dorsal columns.The current study presented prolonged N20-N13 interpeak latency (CSCT) suggesting an upper cervical lesion. Furthermore, N13-N9 interpeak latency was also prolonged which could suggest a lower cervical cord and/or cervical root affection. This cervical involvement may be secondary to vasculitis, degenerative disc changes or joint affection (preodentoid pannus and odentoid erosion) [25].

Moreover, the SSEPs study disclosed bilateral abnormalities in some of the patients. The increased N13-N9 conduction time may reflect a delay in impulse propagation either in the plexus, dorsal roots or the dorsal column [31]. Since lesions of brachial plexus invariably resulted in pathological N9 responses [29], SSEPs test results with normal N9 but increased N13-N9 conduction time points to a lesion proximal to the plexus, either in the cervical roots or dorsal columns.

The variability in SSEPs data of CM patients where some patients have normal results could invariably affect the sensitivity of SSEPs as a test in this group. It was observed that N13 and N20 components in some patients with CM can be entirely normal while prolonged in others.

The presence of normal SSEPs data may indicate that their symptoms either caused by an altered impulse pattern in otherwise normal large afferent fibers or by a lesion of thin, slowly conducting afferent fibers not tested by the present technique. Some patients who had evidences of radiculopathy with or without myelopathy have subjective symptoms as well as objective neurological signs of radiculopathy. The existence of N9 component and/or its normal latency indicates that a normal peripheral impulse will reach the cervical roots [32,33].

Normal SSEPs results therefore do not exclude pathological lesions outside the dorsal column/medial lemniscal system. When this system is affected, either the N13-N9 or the N20-N13 conduction times can be increased. The latter presumably reflects the conduction time between the dorsal column nuclei and the cortex, a pathway entirely located intracranially.

In conclusion the present study revealed that motor and sensory conduction studies are usually normal in CM, among SSEPs parameters, N13 latency was prolonged bilaterally, CSCT abnormal bilaterally, N13-N9 and N20-N13 latencies unilaterally (right side) and mononeuropathies, polyneuropathies, radiculopathies and plexopathies should be excluded before diagnosis of CM was made.

## Acknowledgement

We would like to thanks all members of Neurophysiology Units in Al-Imamein Al-Kadhimein Medical City for their help.

## Author contribution

Dr. Kaddori collectes and analyses the data; Dr. Hamdan interprets the data and approves the final version; and Dr. Mohammed examines the patients and referred them to the neurophysiology unit.

## Conflict of interest

The authors declare no conflict of interest.

## Funding

None.

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**Received 16th Jun. 2o15: Accepted 30th Sep. 2015**