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EFFECT OF FORWARD HEAD POSTURE ON SENSORIMOTOR INTEGRATION

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ABSTRACT

Background: Forward head posture (FHP) is a common type of postural distortion seen clinically, whereby an anterior shift of the head position occurs. Ideal posture is considered to be present when the external auditory meatus is aligned with the vertical line. There were different assessment methods of FHP and the measurement of craniovertebral (CV) angle considered as the most common assessment methods of FHP, The smaller the angle, the greater the FHP. Muscle strain considered as one of the complications of FHP, repetitive strain of the muscle lead to a change in the way of the brain processes the incoming sensory information and subsequent motor output to muscles, a process known as sensorimotor integration(SMI). This study was conducted to measure the neurophysiological effect of FHP on SMI.

Methods: Sixty volunteers participated in the study,30 normal subjects with CV angle≥49 are in group A (Normal), 30 forward head subjects with CV angle ≤49 are in group B (forward head). The amplitude of Cortical Somatosensory evoked potential (SSEPs) of N30-P22, N20-P25 and N24-P22 N30 complex (N30-P22), N20 complex (N20-P25) and N24 complex(N24-P22) of SEPS were recorded for both groups after stimulation of the right median nerve.

Results: There was a significant increase in the cortical amplitude of N30-P22, N20-P25 and N24-P22 of SEPS in the group (A) compared with a group (B) as p-value was ≤ 0.05.

Conclusion: forward head posture affects SMI through changing the response of CNS to afferent input, as demonstrated by attenuation of cortical STEPS in the group (B) compared with a group (A).

Keywords: forward head posture, sensorimotor integration, Somatosensory evoked potential, craniovertebral angle, cortical somatosensory evoked potential, central nervous system.

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INTRODUCTION

Posture can be considered as an important and frequently neglected part of overall health. Normal posture maintains the structural integrity and ideal alignment of each element of the kinetic chain. The elements of the kinetic chain are the myofascial system, articular system and the neural system [1]. When one element of this system is out of alignment, then the entire system is placed at a disadvantage. Postural malalignment is thought to lead to predictable patterns of tissue overload and dysfunction, initiating the cumulative injury cycle [2]. Forward head posture(FHP) considered as one of the most common postural disorders found clinically (66%), which occur when the head position is anterior to a vertical line passing through the centre of gravity [3]. This abnormal posture is accompanied by the development and persistence of many abnormalities including headaches [4], myofascial pain syndrome [5], abnormal scapular movement [6], and temporomandibular disorders [7], the limited cervical range of motion has also been found in patients with tension-type [4,8].

In ideal posture, the head should be positioned directly on the neck and shoulders, change of this ideal alignment place *strain* on your neck and upper back muscles, which may lead to muscle fatigue and frequently an aching neck as occurring in FHP [9]. Repetitive strain injuries and repetitive muscular activity which are common seen in FHP play a role in altering the way the brain processes incoming sensory information and subsequent motor output to muscles, a process identified as sensorimotor integration(SMI) [10].

Also, the altered afferent input may facilitate changes in the cortical representation of the area devoted to processing input from that body part due to the plastic nature of the brain. Recent work suggests that *dysfunctional joints* as occur in FHP may lead to change the afferent input to the central nervous system (CNS) and if prolonged, may result in neuroplastic changes [11].

The study of sensorimotor integration has increased dramatically in recent years, with emerging evidence of maladaptive plastic changes in the sensorimotor integration of various movement disorders such as overuse injuries [10,12-14]. It was established that the study of sensorimotor integration is important in the understanding of both normal physiological function and maladaptive plastic changes in the form of sensorimotor system malfunctions. When learning new skills and performing new tasks, it is essential to employ effective sensorimotor integration. Impaired sensorimotor integration may be useful in explaining the reoccurring or high level of injury found in workers with jobs consisting of high levels of repetitive activity [15].

There was a growing body of knowledge about the different causes which lead to altered afferent input and subsequent change SMI for example, pain, joint effusion and spinal manipulation in the previous studies but little is known about the role of asymptomatic FHP on the alternation of afferent input and subsequent changes in SMI. This study took a deeper look at how forward head posture affects the way the brain responds to altered afferent input, so it answered the question from the neurophysiological point of view, does FHP can be considered as a variant or not on SMI? In particular, the study focused on the role of FHP in sensorimotor integration.

METHODS

The current research was performed in the Faculty of Physical Therapy, Cairo University, in the period from November 2015 to February 2016 to measure the neurophysiological effect of forwarding head posture on sensorimotor integration, the study was prospectively registered on Australian New Zealand Clinical Trial Registry (ANZCTR) and the registration number was ACTRN12616000182493.

Design of the study: observational cross section design

Subjects

Sixty volunteers of both genders were selected and assigned into two groups after signing institutionally approved consent form before data collection. The age ranges from 18 to 25 years with a mean age (19.63± 0.964 years). Group A (Normal group): included 30 participants with CVA angle more than 49° (17 males and 13 females). Group B (Forward head): included 30 participants with CVA angle less than 49° (10 males and 20 females). The exclusion criteria were a history of neurological disease, any abnormal brain function and any history of cervical spine disc diseases. The subjects were required to be right-handed and assessed by the investigator using photographic analysis [16,17] to determine the presence of forwarding head.At the time of the experiment, all subjects were required to be asymptomatic subject (pain-free)

Instrumentation:

Two types of instrumentation were used in this study.

1. Photographic analysis
2. Computerized electromyography(EMG) device

Photographic analysis:

Photographic Posture Analysis Method [18]. This method consisted of:

1. A camera (16 megapixels).
2. A plastic pointer markers
3. Computer for downloading images, Windows8.
4. Surgimap software [19]. **http://www.surgimapspine.com**

(2) Computerized electromyography(EMG) device:

NIHON KOHDEN with serial number 02209 and, manufacturer NIHON KOHDEN country of manufacturing was Japan, and the model of the device was MEB-9400K its Volt was 220-240V and 50/60HZ.

Procedures

Assessment of forwarding head posture: Photographic analysis

To select subjects with FHP, following the same method conducted by (**Raine and Twomey.,1997**) [20], volunteers were asked to stand in their comfortable posture in front

of a plain and white wall looking forward. Volunteers were then asked to look at a point on the wall directly in front of them and focusing on it, hanging their hands at their sides. The camera height should be at the same level of the subject's shoulder. A 16.1-megapixel digital camera (NIKON WIDE 5XZoom. China) with, placed on a tripod 50 cm apart from the subject. The CVA is the angle between two lines a horizontal line passing through C7, and a line pass from the tragus of the ear to C7 was measured [17].

The C7 spinous process and tragus were palpated, for simple palpation of C7, the participants were asked to perform flexion and extension of the head and marked with adhesive skin markers (fluorescent color adhesive squares of 1 cm diameter were used). A lateral view photo of each participant was taken. A digital photo was taken and used to calculate the sagittal-C7-tragus angle. The CVA angle was measured in degrees by using surgimap software program (figure 1). According to Nemmers et al.,2009 [21] a young healthy adult was expected to exhibit an average normal head posture within a 10° range from 49° to 59° of the C7-tragus angle. Therefore, subjects encountering angles less than 49° were considered as FHP in this study.

Figure 1: Calculation of cv angle by surgimap software

Somatosensory evoked potential (SSEPs)

The position of the patient: Because of the very small size of the cortical potentials, very good relaxation of the subject is vital. Therefore the subjects were asked to sit with their eyes closed while remaining quiet and as still as possible; the lights in the room were turned off.

Skin preparation under the stimulating electrode: Skin overlying the dermatome, was carefully washed using methylated alcohol, and then dried by rubbing the skin with dry clean cotton wool. This procedure was repeated until the skin became red which aimed to reduce skin resistance. Great care was taken not to break or abrade the skin under the stimulating electrode.

Skin preparation under the recording electrode: Careful attention was paid to cleaning and scarifying the skin before the attachment of the recording electrodes in the scalp (figure 2). The hair was separated, and methylated alcohol and sandpaper thoroughly cleaned the skin in between was used to gently abrade the skin sites by removing several superficial layers of the skin and skin oils. It has been accepted that abrasion is considered sufficient when the impedance measured across two such electrode preparation

sites is between 1,000 and 5,000Ω. When the impedance is less than 1000 ohm, care must be taken to avoid a situation in which amplifier is short –circuited through aberrant conduction pathway such as excess perspiration or electrolyte paste between two electrodes. As in this instance, the impedance through the abnormal conducting pathway is less than through the electrodes and the biological signal would rather the path of least impedance, thereby bypassing the instrument [22].

Figure 2: Skin preparation under the recording electrodes

Parameters of SSEPs stimulation

The stimuli characteristics are, Electrical pulses which were square pulses,1 ms pulse duration, and the frequency was 2.47 Hz and 4.98 Hz, the stimulating electrode type was Ag/AgCl ECG skin electrodes the resistance was < 5 kO. The site of stimulation was over the median nerve on the skin 2–3 cm from the distal crease of the wrist, between the tendons of Flexor pollicis longus and Palmaris longus with the anode, placed proximally, and the cathode was distal (figure3). There were two different frequencies of SSEPs; to allow recording both the N24 and N30 SEP peak complexes. The slower frequency, 2.47 Hz optimum for N30 and didn't lead to a decrease of SEP peak, while the faster rate, 4.98 Hz, allowing N24 SEP peak to be accurately identified and measured but it leads to decrease N30 peak[23,24]. The stimulus intensity used was the motor threshold for every participant. *It was identified that* motor threshold was the lowest stimulation intensity that evoked a visible muscle contraction of the abductor pollicis brevis muscle.

Figure 3: Stimulation of median nerve

Recording parameters

The position of recording electrodes of SSEPs was placed according to the International Federation of Clinical Neurophysiologists (IFCN) guidelines [25]. The sites of recording electrodes were ipsilateral Erb's point, 2cm posterior to contralateral central C3/4 (Cc'), a frontal site (6cm anterior and 2cm

contralateral to Cz) (F) and the ground electrode was placed on the forehead of participants. These electrodes were 2 mm gold cup EEG electrodes (impedance < 5 kO). The reference electrode was placed on the earlobe of the same site of stimulation. During the recording process, the subjects sat with their eyes closed while keeping the room quiet as much as possible; the lights in the room were turned off. The amplification of SEP signal was (gain 10,000), the filtration rate was (0.2–1000 Hz) the data was saved on a laboratory computer. For the optimum recording of subcortical SEP a higher number of averaged sweeps required, however, 500 averaged sweeps can be optimum for measuring peripheral

Erb's and cortical SEP peaks [25,26]. The amplitudes of the specifically selected waveforms were measured through the averaged waveform displayed in an analysis window. All steps and parameters of SSEPs measurements were taken from to the IFCN guidelines [27].

We measure following SSEPs components: the peripheral N9, the parietal P25 (N20-P25 complex) and the frontal N24 (P22-N24 complex) and N30 (P22-N30 complex).

Statistics

All statistical analysis was done by using the statistical package for the social sciences (SPSS, version 20.0 for Windows; SPSS Inc., Chicago, Illinois, USA). Test of normality by Shapiro-Wilk test show that the data was normally. Descriptive data for participants, characteristics and dependent variables was calculated as mean ± SD. Unpaired T-test was used to compare the participant's general characteristics between both groups, and one-way ANOVA test was used to compare the values of cortical amplitude of SSEPs between both groups. The alpha level of significance was 0.05.

Sample size

The sample size estimation was based on power analysis in a pilot study with 15 subjects (mean difference 1.19 and SD 1.4) two-tailed hypothesis, alpha =0.05, power =90%

Data collection

For accurate determination of motor threshold, the participants were familiarized with the electrical stimulation required to elicit SEPs; this familiarization process was eliminating the need to discard any sweeps within the first data collection SEP trial. Peak to peak amplitudes was measured of SSEPs. Data was exported to IBM SPSS Statistics. One-way ANOVA was conducted with the alpha level of significance was set at 0.05 to compare the results between groups. For data to be included the N9 SSEPs peak

differed by no more than 10% between pre and post-trials. That's why N9 measured peripheral afferent volley, and it should be stable to be able to measure central changes in SEP peaks, there was the exclusion of three participants from the final analysis as did not fit these inclusion criteria.

RESULTS

The participants' characteristics are illustrated in Table (1). Unpaired t-test shows no significant differences in the mean age, weight, height, sleep hours, gender and smoking.

Regarding the results of the cortical amplitude of SSEPs as shown in Table 2. One Way ANOVA revealed statistically significant difference between normal group and forward head group as shown in table (3) As the mean values of cortical amplitude of SSEPs in normal group(A) were greater than forwarding head group(B)

Table 1: Descriptive Statistics of participants age, weight, height, sleep hours, smoking and gender.

	Normal Group	Forward head Group	t-value	p-value
Age (years)	19.63 ± 0.964	19.83 ± 0.949	1.4	0.17
Weight (kg)	55.166 ± 3.85	55.133 ± 3.48	-1.13	0.97
Height (cm)	161.96 ± 5.93	160.96 ± 5.19	0.69	0.49
Sleep hours	5.36±0.718	5.133±0.776	1.208	0.232
Nonparametric				
Gender Male Female	17(13.5){0.91} 13(16.5){0.74}	10(13.5){0.91} 20(16.5){0.74}	Chi-square 3.299	0.069
Smoking Smoker Non smoker	2(1.5){0.17} 28(28.5){0.01}	1(1.5){0.17} 29(28.5){0.01}	Chi-square 0.350	0.553

*Significant at alpha level <0.05

Table 2: Descriptive statistics of Amplitude of SSEPS

	Normal group		Forward head group	
	Mean	Standard deviation	Mean	Standard deviation
Amplitude N20-P25	2.37	0.67	1.51	0.602
Amplitude N30-P22	1.33	0.747	0.894	0.579
Amplitude N24-P22	1.25	0.599	0.886	0.419

Table 3: Shows ANOVA table for amplitudes of cortical SSEPs for both group

		Sum of Squares	df	Mean Square	F	Sig.
AmplitN20-P25	Between Groups	11.239	1	11.239	27.671	.000*
	Within Groups	23.558	58	.406		
	Total	34.797	59			
AmplitN30-P22	Between Groups	2.865	1	2.865	6.411	.014*
	Within Groups	25.918	58	.447		
	Total	28.783	59			
AmplitN24-P22	Between Groups	2.049	1	2.049	7.656	.008*
	Within Groups	15.521	58	.268		
	Total	17.569	59			

*Significant at alpha level <0.05

As demonstrated in the previous table there was a significant difference in all cortical amplitudes of SSEPs (N20-P25, N30-P22, N24-P22) between normal group and the forward head group as p-value was less than 0.05. Also, it's obvious that the amplitude of cortical SSEPS is greater than forward head group

DISCUSSION

The major finding of this study was attenuation in cortical amplitude of SSEPS (parietal N20-P25, frontal N30-P22, and N24-P22). Alternation of afferent input may be the possible explanation for the current findings Bolton and Holland,1996, supported this possible mechanism; Bolton,1998; Murphy et al.,1995; Zhu et al.,1993 and Zhu et al., 2000 [28-32].

The forward head can be considered as a type of joint dysfunction as mentioned before and the presence of dysfunction in the joint lead to alter the afferent input and cause a change in the way that

CNS respond to stimulation, maladaptive neuroplastic changes in CNS may develop over time due to this alternation. These findings were in agreement with authors who suggested that altered afferent input to CNS may result from spinal dysfunction which leads to plastic changes in the way that it responds to any subsequent input [10,11,33,34].

In continuation Haavik and Murphy(2006) [35] concluded that the change in the balance of the afferent input of the CNS would result from the potential maladaptive neuroplastic change which was proposed to develop over time due to the change in the afferent input. Several studies were in agreement with the general concept of the current study which was an alternation of afferent input leads to plastic change. Haavik and Murphy(2006) [35] demonstrated a significant attenuation in the amplitude of parietal N20 and frontal N30 SEP components after a single cervical spine manipulation session compared to pre-manipulation baseline values, and the study concluded that spinal manipulation reduced excessive signaling from the involved intervertebral muscles. This alternation in the afferent input to the CNS may change the way it responded to any subsequent input. Lelic et al.,(2016) [36] performed experiments on nineteen volunteers who attended two experimental sessions, spinal manipulation, and control in random order. It was concluded that there was a reduction in the N30 amplitude from spinal manipulation by 16.9 ± 31.3% (= 0.02)

In other previous studies, there was another possible explanation for this finding which was the presence of pain, the pain was considered as possible cause for neuroplastic changes (37–40). It was concluded that neuroplastic changes in both the sensory and motor cortices had been seen to take place after only brief periods of painful stimulation[37,41,42]. *While* in the current study we couldn't attribute this changes to the presence of pain as we selected asymptomatic subject. This outstanding finding regarding the presence of neuroplastic changes, even in asymptom-atic subjects. Shakespeare,1985 [43], concluded that asymptomatic (pain-free) joint dysfunction (joint effusion) had been found to inhibit surrounding muscles, and this inhibition and alteration of motor control persist even after aspiration of the joint effusion. *This finding means that the absolute cause of the neuroplastic change in the current study is attributed to forward head posture.*

All these studies revealed that any cause that can lead to change or alter afferent input to CNS could lead to a decrease or at least change in amplitude of cortical, spinal or subcortical SSEP and this explain the difference in cortical (N20-P25, N30-P22, N24-P22) between both normal and forward head group. This study was limited by Lack of investigator blinding to the group assignment. However, the investigator followed a written standardized protocol to minimize bias effect.

Limitations

Lack of investigator blinding to the group assignment, however, the investigator followed a written standardized protocol to minimize bias effect.

CONCLUSION

It was concluded that forward head posture can lead to altering afferent input, attenuate cortical amplitude of SSEPS and change the way of response of CNS which is called plastic change which later on leads to impaired sensorimotor integration may help in explaining the reoccurring or high level of injury found in workers with jobs consisting of high levels of repetitive activity and greater understanding of these changes may aid in developing appropriate treatment options for patients with movement disorders, overuse injuries such as repetitive strain injury and other musculoskeletal syndromes such as vertebral dysfunction and pain.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

[1] Clark MA. Postural Considerations, in NASM OPT Optimum Performance Training for the Performance Enhancement Specialist Course Manual. 2nd ed. Wittkop; 2001.

[2] Kendall, F. P., McCreary, E. K., Provance, P. G., Rodgers, M. M., & Romani WA. Muscles Testing and Function with Posture and Pain, 5th Ed. Lippincott Williams & Wilkins; 2005.

[3] Mustafa I, Diab A. The efficacy of forward head correction on nerve root function and pain in cervical spondylotic radiculopathy: a randomized trial. Clin Rehabil. 2011;26((4)):351–361.

[4] Fernandez-de-las-Penas C, Alonso-Blanco C, Cuadra-

do ML, Pareja JA. Forward head posture and neck mobility in chronic tension-type headache: a blinded, controlled study. Cephalalgia 2006;26(3):314–9.

[5] Cimbiz A, Beydemir F, Manisaligil U. Evaluation of trigger points in young subjects. J Musculoskelet Pain. 2006;14(4):27–35.

[6] Weon JH, Oh JS, Cynn HS, Kim YW, Kwon OY, Yi CH. Influence of forward head posture on scapular upward rotators during isometric shoulder flexion. J Bodyw Mov Ther. 2010;14(4):367–74.

[7] Evcik D, Aksoy O. Relationship between head posture and temporomandibular dysfunction syndrome. J Musculoskelet Pain. 2004;12(2):19–24.

[8] Evcik D, Aksoy O. Correlation of Temporomandibular Joint Pathologies, Neck Pain and Postural Differences. J Phys Ther Sci. 2000;12(2):97–100.

[9] Neck pain. When it's serious, when it's not. Mayo Clin Heal Lett. 2000;18(3):1–3.

[10] Byl NN, Merzenich MM, Cheung S, Bedenbaugh P, Nagarajan SS, Jenkins WM. A primate model for studying focal dystonia and repetitive strain injury: effects on the primary somatosensory cortex. Phys Ther. 1997;77(3):269–84.

[11] Pascual-Leone A, Torres F. Plasticity of the sensorimotor cortex representation of the reading finger in Braille readers. Brain. 1993;116(1):39–52.

[12] Classen J, Liepert J, Wise SP, Hallett M, Cohen LG. Rapid plasticity of human cortical movement representation induced by practice. J Neurophysiol. 1998;79(2):1117–23.

[13] Taylor HH, Murphy BA. Altered cortical integration of dual somatosensory input following the cessation of a 20 min period of repetitive muscle activity. Exp Brain Res. 2007;178(4):488–98.

[14] Murphy BA, Haavik Taylor H, Wilson SA, Knight JA, Mathers KM, Schug S. Changes in median nerve somatosensory transmission and motor output following transient deafferentation of the radial nerve in humans. Clin Neurophysiol. 2003;114(8):1477–88.

[15] Bosse J. Motor training and cervical spine manipulation: effects on sensorimotor integration. 2012; Masters of Health Sciences, University of Ontario Institute of Technology.

[16] Kang J-H, Park R-Y, Lee S-J, Kim J-Y, Yoon S-R, Jung K-I. The effect of the forward head posture on postural balance in long time computer-based worker. Ann Rehabil Med. 2012;36(1):98–104.

[17] Lee M-Y, Lee H-Y, Yong M-S. Characteristics of cervical position sense in subjects with forward head posture. J Phys Ther Sci. 2014;26(11):1741–3.

[18] Van Niekerk S-M, Louw Q, Vaughan C, Grimmer-Somers K, Schreve K. Photographic measurement of the upper-body sitting posture of high school students: reliability and validity study. BMC Musculoskelet Disord. 2008;9:113.

[19] Wu W, Liang J, Du Y, Tan X, Xiang X, Wang W, et al. Reliability and reproducibility analysis of the Cobb angle and assessing sagittal plane by computer-assisted and manual measurement tools. BMC Musculoskelet Disord. 2014;15:33.

[20] Raine S, Twomey LT. Head and shoulder posture variations in 160 asymptomatic women and men. Arch Phys Med Rehabil. 1997;78(11):1215–23.

[21] Nemmers TM, Miller JW, Hartman MD. Variability of the Forward Head Posture in Healthy Community-dwelling Older Women. J Geriatr Phys Ther. 2009;32(1):10–4.

[22] Dumitru D. Somatosensory evoked potential: Electrodiagnostic Medicine. 1st ed. Philadelphia; 1995.

[23] Haavik H, Murphy B. Selective changes in cerebellar-cortical processing following motor training. Exp Brain Res. 2013;231(4):397–403.

[24] Fujii M, Yamada T, Aihara M, Kokubun Y, Noguchi Y, Matsubara M. The effects of stimulus rates upon median, ulnar and radial nerve somatosensory evoked potentials. Electroencephalogr Clin Neurophysiol Potentials Sect. 1994;92(6):518–26.

[25] Nuwer MR, Aminoff M, Desmedt J, Eisen AA, Goodin D, Matsuoka S, et al. IFCN recommended standards for short latency somatosensory evoked potentials. Report of an IFCN committee. Electroencephalogr Clin Neurophysiol. 1994;91(1):6–11.

[26] Mauguiere F, Allison T, Babiloni C, Buchner H, Eisen AA, Goodin DS, et al. Somatosensory evoked potentials. The International Federation of Clinical Neurophysiology. Electroencephalogr Clin Neurophysiol Suppl. 1999; 52:79.

[27] Cruccu G, Aminoff MJ, Curio G, Guerit JM, Kakigi R, Mauguiere F, et al. Recommendations for the clinical use of somatosensory-evoked potentials. Clin Neurophysiol. 2008;119(8):1705–19.

[28] Bolton P, Holland C. Afferent Signaling of vertebral displacement in the neck of the cat. In: Society for Neuroscience, Abstracts. 1996;22:1802.

[29] Bolton PS. The somatosensory system of the neck and its effects on the central nervous system. J Manip Physiol Ther. 1998;21(8):553–63.

[30] Murphy B, Dawson N, Slack J. Sacroiliac joint manipulation decreases the H-reflex. Electromyogr Clin Neurophysiol. 1995;35(2):87–94.

[31] Zhu Y, Haldeman S, Starr A, Seffinger MA, Su S-H. Paraspinal muscle evoked cerebral potentials in patients with unilateral low back pain. Spine (Phila Pa 1976). 1993;18(8):1096–102.

[32] Zhu Y, Haldeman S, Hsieh C-YJ, Wu P, Starr A. Do cerebral potentials to magnetic stimulation of paraspinal muscles reflect changes in palpable muscle spasm, low back pain, and activity scores? J Manipulative Physiol Ther. 2000;23(7):458–64.

[33] Brasil-Neto JP, Valls-Solè J, Pascual-Leone A, Cammarota A, Amassian VE, Cracco R, et al. Rapid modulation of human cortical motor outputs following ischaemic nerve block. Brain. 1993;116(3):511–25.

[34] Hallett M, Chen R, Ziemann U, Cohen L. Reorga-

nization in the motor cortex in amputees and normal volunteers after ischemic limb deafferentation. Electroencephalogr Clin Neurophysiol Supplemen. 1999;51:183-7.

[35] Haavik Taylor, H., & Murphy B. The effect of cervical spine manipulation on the ability to integrate somatosensory input following repetitive movements. In: Australian Neuroscience Society Annual Meeting. Sydney; 2006.

[36] Lelic D, Niazi IK, Holt K, Jochumsen M, Dremstrup K, Yielder P, et al. Manipulation of Dysfunctional Spinal Joints Affects Sensorimotor Integration in the Prefrontal Cortex: A Brain Source Localization Study. Neural Plast. 2016;Article ID 3704964, 9 pages.

[37] Wall JT, Xu J, Wang X. Human brain plasticity: an emerging view of the multiple substrates and mechanisms that cause cortical changes and related sensory dysfunctions after injuries of sensory inputs from the body. Brain Res Rev. 2002;39(2):181–215.

[38] Hides J, Stokes M, Saide M, Jull G, Cooper D. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine (Phila Pa 1976). 1994;19(2):165–72.

[39] McPartland JM, Brodeur RR, Hallgren RC. Chronic neck pain, standing balance, and suboccipital muscle atrophy--a pilot study. J Manip Physiol Ther. 1997;20(1):24–9.

[40] Stokes M, Young A. The contribution of reflex inhibition to arthrogenous muscle weakness. Clin Sci. 1984;67(1):7–14.

[41] Henderson LA, Bandler R, Gandevia SC, Macefield VG. Distinct forebrain activity patterns during deep versus superficial pain. Pain. 2006;120(3):286–96.

[42] Tinazzi M, Fiaschi A, Rosso T, Faccioli F, Grosslercher J, Aglioti SM. Neuroplastic changes related to pain occur at multiple levels of the human somatosensory system: A somatosensory-evoked potentials study in patients with cervical radicular pain. J Neurosci. 2000;20(24):9277–83.

[43] Shakespeare DT, Stokes M, Sherman KP, Young A. Reflex inhibition of the quadriceps after meniscectomy: lack of association with pain. Clin Physiol. 1985;5(2):137–44.

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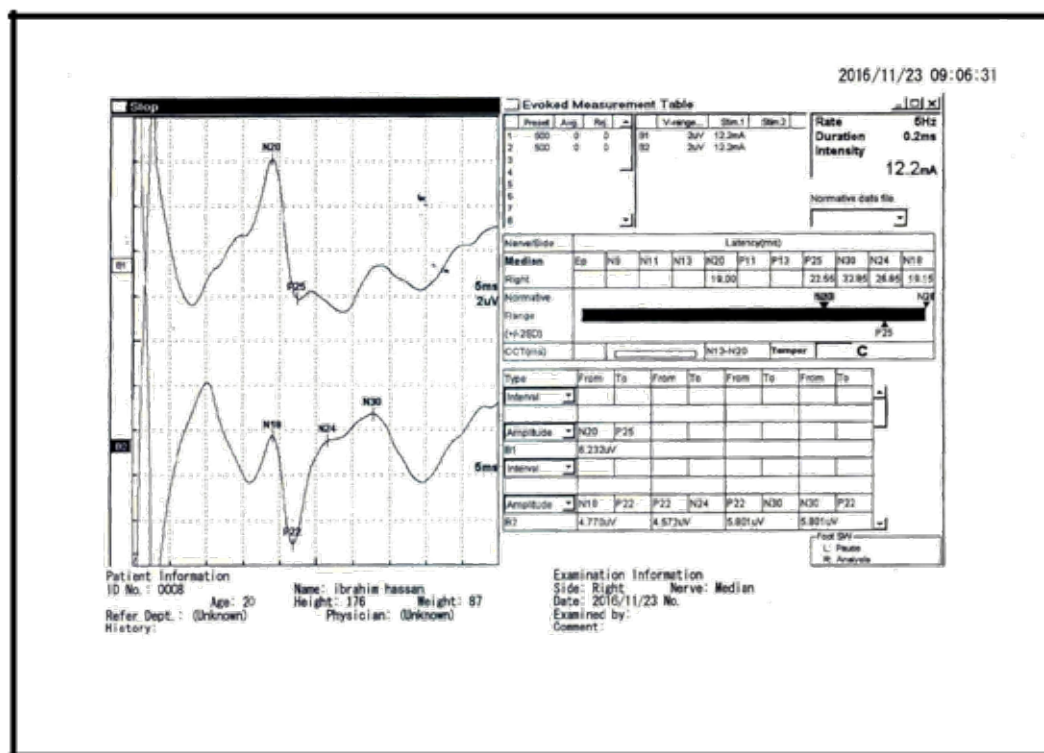
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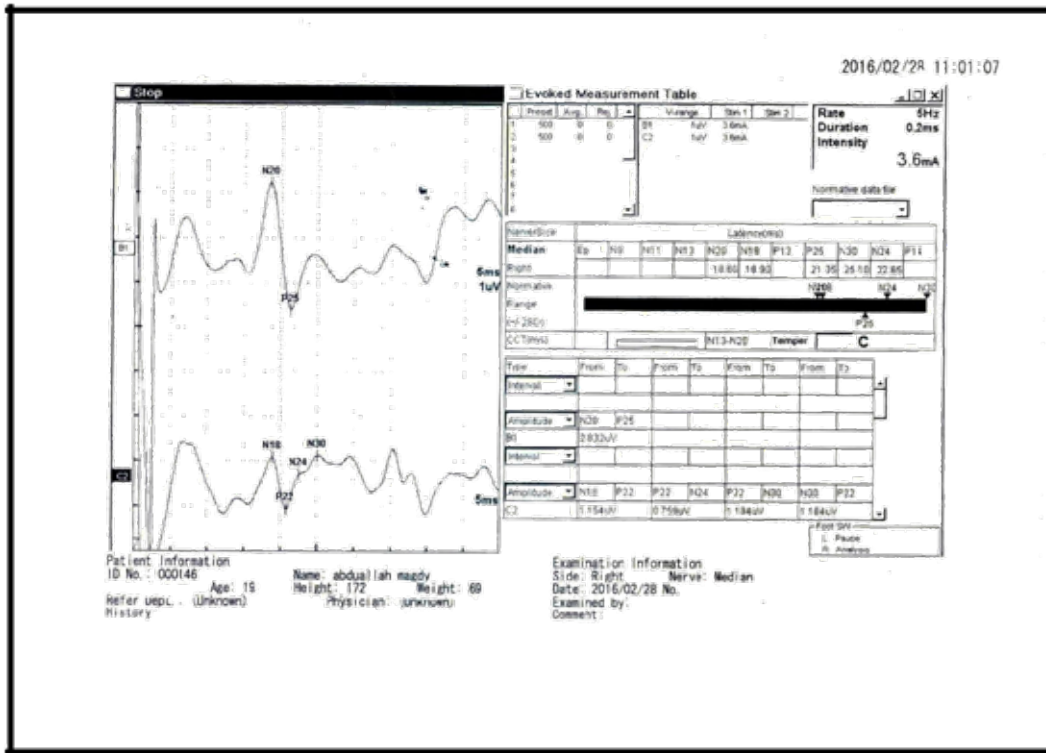
(B) Somatosensory evoked potential graph for forward head subject