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Original Article

Somatosensory evoked potentials in patients with obstructive sleep apnea syndrome and cerebral hypoxia

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Abstract

Aim: We aimed to examine cerebral hypoxia with Somatosensory evoked potentials (SEP) in Obstructive Sleep Apnea Syndrome (OSAS) patients, because recurring apnea attacks significantly affects cerebral perfusion.

Material and methods: SEP examination was performed by using Keypoint electromyography device (Version 2.38, Medtronic Dantec, Skovlunde, Denmark) in patients, diagnosed with severe OSAS as compared to control group in polysomnography lab of Department of Chest Diseases. Potential cortical (N20) latency and amplitudes were compared between patients and control group.

Results: Even if the average of SEP N20 latency was within the normal limits in patients group, (diagnosed with severe OSAS according to clinical and polysomnography examination), it was clearly extended and statistically significant compared to control group. (Respectively 22.31±2.13 ms, 18.35±0.80 ms P= 0.000). Moreover, when the amplitudes of obtained N20 potentials were compared between patients and control group, it was determined that the amplitudes of patients group were clearly lower and statistically significant. (Respectively 1.82±0.16 mv, 2.54±0.25 mv, P=0.000). The average of Apnea-Hypopnea index (AHI) was detected as 55.77±28.02. However, there was no correlation detected between AHI and SEP (p=0.164, r=0.261).

Conclusion: These results indicate the existence of cerebral hypoxia in OSAS and at the same time highlight the importance of SEP examinations in OSAS.

Keywords: Obstructive Sleep Apnea Syndrome; Somatosensory Evoked Potential; Cerebral Hypoxia.

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is an important health problem which is developed as a result of respiratory disorders (apnea, hypopnea) recurring due to upper airway obstructions during sleep and is systemic disease (1). OSAS has begun to be considered as an independent risk factor than risk factors known for hypertension, ischemic heart disease, Diabetes Mellitus, obesity and cerebrovascular diseases. Recurring apneas and hypoxia and later reperfusion occurred as a result of these, cause development of free oxygen radicals and ischemia-reperfusion damage in vein wall which cause vascular endothelial dysfunction. In patients who didnot have any diagnosis, it causes vascular oxidative stress with increased arterial oxygen desaturations recurring every night for several years (2).

Structures, which are responsible of potentials logged in standard somatosensory evoked potential (SEP) examination conducted by mixed nerve stimulation, are thick myelinated fibers in peripheral nerve system and cord-medial lemniscus system in central nerve system. The most commonly applied SEP examinations are stimulation of medial and ulnar nerves from wrist, tibial nerve from ankle and peroneal nerve from knee surface. The most apparent symptom which indicates an anomaly in SEP examination is the loss of potentials resulted from lesion level and its rostral, or extension of its latencies (3,4). In SEP examination, information related to pathological processes holding peripheral and central nerve system were recognized (5).

Studies presented in relation with sensory or (with more frequently used name) somatosensory evoked potentials (SEP) in OSAS patients are still limited and aetiopathogenesis of found characteristics has not been clarified yet. In this study we aimed to evaluate cerebral hypoxia in OSAS patients with SEP.

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MATERIAL and METHODS

Material

We aimed to examine the effects of cerebral hypoxia by performing SEP examination in patients, diagnosed with severe OSAS and consulting to chest diseases outpatient clinic sleep lab, and in electrophysiology lab of Neurology Department. 20 male and 10 female (total 30) patients were registered into study. Control group consisted of 12 male and 8 female (in total 20) healthy volunteers. While the average age of patients group was determined as 45.43± 11.28, the average age of control group was 42.90± 5.91. While average Body Mass Index (BMI) of patients group was 34.06±6.94, it was 25.45± 3.28 for control group (Table 1).

Table 1. Demographical data of patients and control group

	Age	BMI	Gender
Patients group	45.43±11.28	34.06±6.94	20 M / 10 F
Control group	42.90±5.91	25.45±3.28	12 M / 8 F

Method

Keypoint electromyography device (Version 2.38, Medtronic Dantec, Skovlunde, Denmark) was used for recording, data saving and analysis. In patients and control group for recording of median nerve SEP examinations, the median nerve was stimulated from wrist. During SEP recording median nerve was stimulated by placing it aligned with wrist, placing cathode just proximal to palmar line and anode in a 3 cm distance to cathode, between the tendons of palmaris longus muscle. We placed the ground electrode approximately 5-6 cm above from the place that we stimulated in forearm. While active electrode was placed into a point which is suitable for parietal part which is 2 cm rear and 7 cm lateral of Cz point according to international 10/20 system at the opposite side to the hand that is stimulated for cortical potential (N20) recording, the reference electrode was placed into ear lobe. We tried to keep the tissue impedance below 5 kΩ for better recording. While 0.2 ms waves were preferred for stimulation, we kept the current intensity between 5-25 mA so that visible contractions could be obtained in thenar muscle. Lower and upper filtration rates were 30-3000 Hz and we adjusted the average analysis time as 100 ms and averaged each recording 500 times. Independent Sample T Test was used in patients and control group comparison with SPSS for windows 20.0 program for statistical analysis.

RESULTS

Even if the average of SEP N20 latency was within the normal limits in patients group, diagnosed with severe OSAS according to clinical and polysomnography examination, it was clearly extended and statistically significant compared to control group. (Respectively 22.31±2.13 ms, 18.35±0.80 ms P= 0.000). Moreover, when the amplitudes of obtained N20 potentials were compared between patients and control group, it was determined that the amplitudes of patients group were clearly lower and statistically significant. (Respectively 1.82±0.16 mv, 2.54±0.25 mv, P=0.000). The average of Apnea-Hypopnea index (AHI) was detected as 55.77±28.02. However, there was no correlation detected between AHI and SEP (p=0.164, r=0.261) (Table 2).

Table 2. N20 latency and amplitude values of patients and control group

	N20 latency	N20 Amplitude	Significance (p)
Patients group	22.31±2.13	1.82 ±0.16	0.000
Control group	18.35±0.80	2.54 ±0.25	0.000

DISCUSSION

Nowadays it is very obvious that the role of SEP examination in cerebral hypoxia is limited by highly advanced radiological imagining methods, including Magnetic Resonance Imagining techniques. Therefore, in recent years there are limited studies evaluating the SEP usage in cerebral hypoxia in OSAS.

It is known that apnea attacks occurring in OSAS lead to fluctuations in cerebral blood flow rate and blood pressure (6). Any decrease in cerebral blood flow affects cerebral perfusion in OSAS significantly. Following this, the increase in cerebral perfusion pressure results in an increase in intracranial pressure and vascular resistance increases. As a result, the decrease in cerebral blood flow can lead to cerebral ischemia (7,8). Recurring hypoxemia and reoxygenation, which occur in OSAS patients, can trigger oxidative stress mechanisms (9). Episodic hypoxic attacks during sleep, play a central role in pathophysiological mechanisms in ischemic brain lesions development by causing obvious hemodynamic changes during apneas (10,11).

In a study comprised of 20 patients, Waters et al. indicated that there is no correlation between the presence and intensity of SEP abnormalities and the presence and intensity of OSAS (12). In study we conducted, patients group was mainly consisted of patients, diagnosed with severe OSAS in polysomnographic aspect (AHI index > 30). In our study there was no correlation detected between AHI and SEP in similar way with the study of Waters et al. (p=0.164, r=0.261).

SEP examinations were used in many clinical studies, including ischemic cerebral incidences. Monitoring of SEPs in medial nerve and/or tibial nerve is a technically simple and proven method in determination of ischemic incidences in middle cerebral artery area (13,14).

In many studies related to carotid endarterectomy, same side cortical SEPs were evaluated after crosswise clamping of the same side carotid artery. In these studies, in clinical practice, important changes in SEP wave forms working as alarm criteria for insufficient cerebral blood flow, were generally identified as a decrease more than 50% of cortical N20-P25 SEP amplitude and/or 10% extension time in latency (15,16,17,18).

CONCLUSION

When the results were evaluated in our study, in parallel with alarm criteria for cerebral blood flow, mentioned in studies of De Vleeschauwer, Nwachuku, Haupt, Horsch et al. the amplitudes of N20 potentials obtained from patients group was clearly lower and their latency was obviously extended, compared to control group. In this respect, SEP examination is highly important due to the fact that it indicates the presence of cerebral hypoxia in OSAS as in ischemic stroke and carotid artery disease. However, studies related to SEP examination in OSAS are extremely limited in literature. More studies in this topic would contribute more to literature.

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