Original Article

# Study of induced nystagmus in cases of motion sickness

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## ABSTRACT

**Lower Body Negative Pressure (LBNP) has been reported to induce nystagmus in previous studies. The vestibular system plays an important role in the causation of motion sickness. Nystagmus is the principle sign of any vestibular disturbance. This study analyzes the differences in LBNP induced nystagmus between those with and without history of motion sickness. Electro-oculography was used to record the nystagmus. Nystagmus was induced in only 40% of all subjected to LBNP. The mean LBNP level at which nystagmus developed in those with motion sickness was -47.5 ± 16.39 mm Hg and in controls was -50 ± 7.07 mm Hg. The frequency, slow phase velocity and fast phase velocity of the nystagmus in the test group was 0.80 ± 0.35 Hz, 4.8 ± 1.33°/s (24 ± 6.65 mV/s) and 12.6 ± 5.527s (63.0 ± 27.6 mV/s) respectively and in the control group were 0.60 ± 0.36 Hz, 4.77 + 1.237s (23.85 ± 6.15 mV/s) and 17.17 ± 9.977s (85.85 ± 46.85 mV/s) respectively. Where nystagmus developed, there was no statistical difference (p > 0.05) in its characteristics in those with and without a history of motion sickness, and there was no significant difference in the level of LBNP at which it developed between these two groups.**


**IJASM 2002; 46(2) 23 - 26**

**KEYWORDS: Nystagmus, LBNP, Motion sickness, Electro-oculography**

Motion sickness has affected man since the early ages. Motion sickness is an ever present risk whenever man relinquishes his / her intended status as a self-propelled animal and steps aboard some vehicle or device that transports him passively. This condition occurs primarily when man is exposed to real or apparent motion stimuli with which he is unfamiliar and unadapted. The vestibular apparatus plays a significant role in genesis of motion sickness [1].

In this study nystagmus induced by LBNP, in individuals presenting clinically with motion sickness is analyzed and compared with that induced in controls.

Nystagmus is the principal sign of vestibular disturbance [2]. Analysis of nystagmus, if present, helps in assessing the extent of the vestibular disturbance, in cases of motion sickness. Recent studies have used the application of lower body negative pressure (LBNP) in inducing nystagmus [3].

### Materials and Method

Human volunteers were subjected to graded LBNP up to - 60 mm Hg. Ten individuals with history of motion sickness were taken as cases and ten other

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individuals with no such history were taken as controls. LBNP was employed to induce nystagmus. Electro oculography (EOG) data was collected and analyzed to study the nystagmus induced.

The LBNP equipment used for the study was a hemi-cylindrical box made of perspex and acrylic, mounted on a tilt table and stand. The air inside this box was evacuated using a commercial vacuum pump to obtain sub atmospheric pressure inside it. One end of this box had an oval opening to permit the entry of the lower half of the subjects i.e., from waist down. The LBNP box was provided with three additional openings. These openings were for the attachment of the vacuum pump meant to create the suction, for the gate valve used to regulate the degree of suction, and for a mercury manometer used to indicate the level of suction. The subject lay on the table with the lower half of his body being enclosed by the LBNP box. Adequate sealing at the waist level was ensured using a polyester skirt.

The EOG data was recorded for both eyes on two of the 8 channels of a polygraph machine. Silver cup electrodes were used and secured in place using EC2 electrode paste. One electrode placed at the nasion served as a common electrode for both eyes. One electrode each was placed in the temporal groove at the outer canthi of both eyes to record the corneo-retinal potential difference, which changed with movements. The movements of the left eye were recorded between the G 1 on the left outer canthus and the G2 at the nasion. The movements of the right eye were recorded between the Gl at the nasion and the G 2 at the right outer canthus. The ear lobe served as the ground for recording the eye movements for both the eyes. The polygraph was first calibrated to record a deflection of 2 cm for 20 micro volts (mV). Movement of the stylus below the baseline was interpreted as movement of the eye to the left and similarly movement of the stylus above the baseline was interpreted as movement of the eye to the right. With the subject positioned and instrumented, calibration was done, so that a 20° movement of the eyes from left to right produced a deflection of 1 cm of the stylus. The direction of stylus movement in relation to movement of the eyes was confirmed before starting the experiment. The amplitude of the nystagmus was calculated by measuring the maximum deflection of the stylus from the baseline. The time period was calculated by dividing the paper travel by the paper speed of 25 mm/s. The velocity of the various components of the nystagmus beat was found by dividing the amplitude by the time period of that component. The frequency of nystagmus was calculated from the time period.

The subjects were not on any medications, for at least a week prior to the experiment. They abstained from smoking for a period of 2 hours prior to the experiment and abstained from alcohol for a period of 72 hours prior tc the experiment. They were explained the procedure. All the subjects were given a familiarization run to allay anxiety. Baseline parameters were recorded after a stabilization period of 10 minutes. The LBNP protocol followed was 10 minutes rest prior to starting, 3 minutes at -14 mm Hg, 3 minutes at -20 mm Hg, 3 minutes at -30 mm Hg, 3 minutes at -40 mm Hg, 3 minutes at -50 mm Hg and 3 minutes at -60 mm Hg.

The run was terminated after 3 minutes at -60 mm Hg of LBNP or on the occurrence of giddiness, lightheadedness, dimming or blurring of vision, nausea, sweating, chest pain, shortness of breath, or any other symptom. The hemodynamic criteria for termination was systolic BP less than 90 mm Hg, a fall of blood pressure (BP) of greater than 20 mm Hg in two successive readings, pulse pressure less than 15 mm Hg, or a difference of more than 20 beats per minute in two successive readings of head rate (HR).

**Results**

Not all the subjects who underwent LBNP developed nystagmus. Only 40% (4 out of 10) of each of the test and control groups did so. The nystagmus, in who so ever it developed, was intermittent and had

a convergent nature. There was no difference in the characteristics of nystagmus in those with and without any history of motion sickness. The Student's 't' test was used to analyze the results which showed no significant difference in the nystagmus pattern in cases of motion sickness and the controls.

The LBNP level at which nystagmus occurred in cases of motion sickness was -47.5 ± 16.39 mm Hg and for controls was -50 ± 7.07 mm Hg. The difference was statistically insignificant ($p > 0.05$). The mean frequency of nystagmus in cases of motion sickness was 0.80 ± 0.35 Hz and the mean frequency of the nystagmus in controls was 0.60 ± 0.36 Hz. The different was not statistically significant ($p > 0.05$). The mean slow phase velocity of nystagmus in cases of motion sickness was 4.8 ± 1.337s (24 ± 6.65 mV/s) and that for controls was. 4.77 ± 1.237s (23.85 ± 6.15 mV/s). The difference was significant statistically ($p > 0.05$). The mean fast phase velocity of nystagmus in cases of motion sickness was 12.6 ± 5.527s (63.0 ± 27.6 mV/s) and that for the controls was 17.17 ± 9.377s (85.85 ± 46.85 mV/s). The difference was not statistically significant ($p > 0.05$).

**Discussion**

LBNP has been reported in some previous studies [3, 4] to induce nystagmus in normal subjects. This study employed LBNP to induce nystagmus in cases with a history of motion sickness. If nystagmus was induced, the aim was to study its pattern, with regard to its various characteristics. There has been no standardization of the protocols to be used for LBNP. The protocols used have varied from a single jump to a step-wise incremental method. The choice of a protocol has so far been dictated by the needs of the study. It has been ascertained that the changes in the various parameters are independent of the protocol employed [5]. A previous study had noted the appearance of syncope between a tolerance index of 638 to 1759 mm Hg. [6]. The lower end of this range corresponds to approximately -60 mm Hg for 3 minutes. Hence, to cater to subject safety, the present study resorted to a protocol of step wise increase in suction to the lower half of the body starting at -14 mm Hg and increasing at the rate of 10 mm Hg every 3 minutes to a maximum of -60 mm Hg. It has been documented that the reflex cardiovascular changes reach a steady state within 3 minutes of applying suction at a given level of pressure [7]. In view of this, the EOG was recorded only after a period of 2 % minutes had elapsed at any given level of suction.

Only four (40%) subjects each of the test and control groups developed nystagmus during LBNP. This occurred at a mean suction pressure of -45 mm Hg ± 15 mm Hg in the test group and at a mean pressure of -50 mm Hg ± 7.07 mm Hg in the control group. The nystagmus had the features of having a central origin. There was a breakdown in the conjugate movements of the eyes, with the slow component of the nystagmus facing away from each other and the fast components facing towards each other.

The mean frequency of the nystagmus in the test group was 0.80 ± 0.35 Hz and in the control group was 0.60 ± 0.36 Hz. The average slow phase velocity in the test group was 4.8 ± 1.33°/s (24 ± 6.65 mV/s) and that for the control group was 4.77 ± 1.23°/ s (23.85 ± 6.15 mV/s). The fast phase velocity in the test group was 12.6 ± 5.52°/s (63.0 ± 27.6 mV/s) and in the control group was 17.17 ± 9.37°/s (85.85 ± 46.85 mV/s).

There was no significant difference between the test and control groups with regard to the suction pressure at which nystagmus developed, but whenever nystagmus developed, the test and control groups did not significantly differ in their frequency, slow or fast phase velocities ($p > 0.05$). Wolthuis et al. suggested nystagmus to be due to a stimulation of the vestibular end organs following a decrease in the endolymphatic pressure as a result of LBNP producing a streaming of endolymph. In the present study, the nystagmus was convergent, indicating a central mechanism.

Lapayev et. al. [8] have proposed that the sudden return of blood to the upper regions of the body, on cessation of LBNP may be similar to

**weightlessness and that the vestibular stimulation may be the precipitating cause for space motion sickness. They however do not mention the development of nystagmus during LBNP.**

**A probable cause for the spontaneous nystagmus during LBNP appears to be the central cerebral ischaemia. Ischaemia of the vertebrobasilar system is known to produce a vestibular stimulation [9]. This is a central type of nystagmus similar to the nystagmus, which appeared with LBNP. It has been reported that a lesion in the cerebellum or the central connecting pathways could give rise to a unilateral nystagmus [10]. A sudden decrease in the thalamic blood supply before the vomiting on motion sickness is known to occur [11]. If the reduction in blood supply is the cause of vomiting then perhaps it could be used to explain the development of nystagmus as well.**

**In this study nystagmus has not developed in all the cases of motion sickness. To further compound matters, some (40%) of the controls who had no history of motion sickness also developed nystagmus.**

## Conclusion

**- This study showed that LBNP did not induce spontaneous nystagmus in all individuals. There was no statistically significant difference in the incidence of LBNP induced nystagmus between normal individuals and those with motion sickness. Hence it can be concluded that, LBNP induced nystagmus is not a suitable method to differentiate between individuals with and those without history of motion sickness.**

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