Nystagmus induced by lower-body suction

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We investigated the hypothesis that brain stem ischaemia induced by the application of lower-body suction pressure (LBSP) may disturb the function of the nystagmus generator in the brain stem and lead to the appearance of a clinically significant spontaneous nystagmus. For this, we exposed nine healthy male subjects to graded LBSP, starting at –20 mmHg, and recorded a significant nystagmus at an average pressure of 48.9 mmHg. The amplitude of the nystagmus signal was 48.2 ± 4 μV (SEM) at a mean frequency of 1.4 ± 0.06 Hz, and a slow 65.3 ± 4.4 μV/s. The pattern was divergent in nature, indicating a central nystagmus. No significant change in the nystagmus was observed even at a suction level of –60 mmHg. The central generator was affected as in five other subjects, the characteristics of the cold-stimulus-generated nystagmus were changed during exposure to a LBSP of –40 mmHg. The cardiovascular responses of all our subjects to LBSP stress were normal. It is possible that in apparently normal subjects who have had vasovagal syncopal attacks, nystagmus generated during the application of LBSP may show a distinctive pattern. This may also be true for individuals who may have motion (air) sickness. The findings suggest interesting possibilities for clinico-physiological investigations of cases of aeromedical interest.

Keywords: Brain stem ischaemia; LBSP; Nystagmus

Moderate hypovolaemia (of the order of 600–700 ml) occurs commonly even under non-clinical circumstances like heat stress and dehydration. This is usually asymptomatic and the person remains normotensive and, hence, is considered fit for flying. Asymptomatic, normotensive hypovolaemia has, however, been associated with a decrease in cerebral circulation [1–3]. Under the circumstances, some derangement of function is likely to occur. The vestibular system is one of the important systems affecting the ability to pilot an aircraft, thus making it important for us to determine the effect of moderate hypovolaemia on the vestibular system.

The laboratory method of lower-body subatmospheric pressure (LBSP) has long provided a standard model for controlled non-haemorrhagic hypovolaemia [1, 4, 5]. Application of suction at the level of the iliac crests translocates blood from the chest cavity to the periphery, reducing the central and circulating blood volumes and pertusion to various tissues [1, 5]. In the present study, we have used LBSP as a method of inducing controlled hypovolaemia without the vestibular stimulation attendant to change in posture and studied its effect on the neurovestibular system using electro-oculography.

Material and methods

Nine healthy male subjects (mean age 29.2 yr; height 170.5 cm; weight 63.5 kg) volunteered to take part in this study, which was approved by the local ethical committee. The subjects were clinically examined to rule out the presence of cardiovascular, CNS and ENT diseases. None had a spontaneous idiopathic nystagmus. Each subject was given an experience of LBSP (–40 mmHg) in order to familiarize him with the procedure and also to allay any associated anxiety. He abstained from alcohol for 24 h before the test, and from smoking and tea/coffee on the morning of the test. None of the subjects were taking any long- or short-term medication.

A semi-cylindrical box made of acrylic and a domestic vacuum cleaner (Euroclean 2000) were used to apply the LBSP. The subject was placed feet first inside the box at the level of the iliac crests after wearing a neoprene rubber skirt, which was taped firmly around his waist around the same level. The edges of the skirt were then slid over the opening of the box and taped around it in order to achieve a seal. The degree of suction applied could be varied using a perforated rotating cuff. With all ports closed, the apparatus could easily generate and maintain an LBSP of –70 mmHg, while with all ports open a pressure of 15 mmHg could be generated. LBSP was applied in a stepwise fashion, starting with a pressure of –20 mmHg and decreasing it by 10 mmHg every five minutes.

A model 7D polygraph (Grass Instruments Inc.USA) was used to record the electrocardiogram (ECG) and the electrooculogram (EOG). Arterial blood pressure (BP) was recorded using a standard anaeroid manometer.

Heart rate (HR) using ECG monitoring, EOG and blood pressure were recorded in the fourth minute at each step. After a pressure of –50 mmHg was reached, ECG and EOG monitoring were done continuously. Continuous monitoring was started earlier if development of nystagmus was suspected.

EOG recording was done simultaneously for both eyes on two separate channels of the polygraph. Silver/silver chloride electrodes were placed on the nasion (reference electrode) and at the outer canthus of each eye. Right side of the chest was used as a common ground for ECG (CM5 configuration) and EOG, so that any disruption of circuit could be promptly detected. The output was fed through a 7P4 amplifier (Grass Instruments Inc.) with a time constant of 0.45 s and a half-amplitude high frequency of 35 Hz.. The calibration 100 μV/cm was checked before and after the experiment. The pen deflections were adjusted so that the upper of the two tracings represented the right eye. The pen representing the right eye moved up when the right eye looked right. Similarly, the pen representing the left eye moved up when the left eye looked left. The subject kept his eyes closed throughout the experiment to prevent suppression of nystagmus.

The subject reported to the laboratory at about 12 noon. After wearing the neoprene skirt, he was positioned inside the LBSP box and instrumented. A rest period of 45 min was given to stabilize him. After recording baseline HR, BP and eye movements, LBSP was applied starting at –20 mmHg and increased in a stepwise manner to –60 mmHg. ECG, EOG and BP recordings were done in the fourth minute at each level of LBSP. Post-LBSP recordings were done immediately, and after 5 and 10 min of stopping suction.

The experiment was terminated in the event of subjective discomfort, or objective evidence of approaching presyncope. The predetermined end-point was 5 min at a pressure of –60 mmHg. In the absence of subjective symptoms, the experiment was extended if nystagmus was not detected by this time.

In addition to the experiment described above, five healthy male human volunteers were subjected to a caloric test before and during LBSP at –40 mmHg. The caloric stimulation was done prior to application of LBSP and 3 min after reaching –40 mmHg. A gap of 15 min was given between two stimulations to ensure the absence of residual effects. For this purpose a straight 'jump' was made to a pressure of –40 mmHg. Differences between the recordings before and during LBSP were studied.

Results

All individuals completed the experiment successfully without subjective discomfort or presyncope. The cardiovascular variables showed changes consistent with those reported by other workers. There was a continuous fall in the systolic blood pressure (SBP) from 117.9 + 2.2 mmHg (mean ± SE) to 112.2 + 2.3 mmHg (Table 1). The diastolic blood pressure (DBP) gradually increased from 71.6 ± 1.9 to

Table 1. Cardiovascular data at different levels of LBSP (mean values)

Parameter	Baseline	−20	30	40	−50	−60	Change
SBP (mmHg)	117.9	115.1	114.4	113.9	114.4	112.2	−5.7
DBP (mmHg))	71.6	74.7	77.3	77.3	80.3	80.1	+ 8.5
MAP (mmHg)	87.0	88.3	89.1	89.7	91.6	91.8	+ 4.8
PP (mmHg)	49.7	43.8	38.1	36.6	34.1	32.1	17.6
HR (bpm)	67.6	68.8	72.9	76.4	88.6	95.0	+ 27.4

Table 2. Characteristics of the spontaneous nystagmus developing with LBSP

Subject no.	LBSP at onset (mmHg)	Frequency (Hz)	Amplitude (μV)	Slow-component velocity (μV/s)
1	−20	1.5	40	66
2	−40	1.5	40	66
3	−40	1.7	40	66
4	−50	1.3	44	66
5	−50	1.0	40	40
6	−50	1.6	50	80
7	−60	1.33	70	80
8	−60	1.0	70	80
9	−70	1.7	40	50

80.1 ± 2.4 mmHg. The pulse pressure dropped from 49.7 ± 2.7 to 32.1 + 2.8 mmHg. There was an increase in the mean arterial pressure (MAP) from 87 ± 1.5 to 91.8 ± 2 mmHg. The heart rate (HR) increased from 67.6 ± 2.9 to 95 ± 5 beats/min (Table 1).

All the nine subjects undergoing LBSP developed a spontaneous, intermittent, divergent nystagmus. This developed at an average pressure of −48.9 ± 4.6 mmHg. The average frequency of the nystagmus was 1.4 ± 0.06 beats/s. The average slow-phase velocity was 65.3 ± 4.4°μV/s. The average amplitude of the nystagmic beat was 48.2 ± 4 μV (Table 2).

As stated above, the nystagmus was intermittent. It was not related to any subjective sensation of turning or spinning. The nystagmus was disconjugated in that the movement of one eye was not associated with the movement of the other. In addition, the eyes intermittently developed a divergent nystagmus, i.e. the slow components of the nystagmus in both the eyes were facing opposite directions, and the fast components towards each other.

Table 3. Changes (with LBSP at −40 mmHg) in the characteristics of nystagmus induced by cold caloric stimulation

Amplitude (μV)	Duration (s)	Frequency (Hz)	Slow-component velocity (μV/s)
−10	−60	−0.9	−166
−40	−104	+ 0.8	−37
0	+220	0.0	0
−60	−40	−0.3	−192
0	+ 20	+ 0.5	+ 100

The effect of LBSP on cold caloric-induced nystagmus was also studied. The amplitude as well as the frequency of the caloric-induced nystagmus before and during LBSP were different though a definite pattern could not be established (Table 3).

Discussion

The changes in the cardiovascular variables on application of LBSP were similar to those reported in normals by other authors [1, 3, 4, 5]. In a study with normal subjects, Giller *et al.* [3] have demonstrated a decrease in cerebral per-

fusion of the order of 26% under similar circumstances, using transcranial Doppler. In their study, the middle cerebral artery blood flow velocity decreased by 16 ± 4%, while there was an increase in the ratio between the systemic and transcranial Doppler pulsitalities, indicating small vessel constriction due to sympathetic stimulation under LBSP. In the present study, though brain perfusion was not measured, the response obtained is indicative of a reduction in the central blood volume, as evidenced by a fall in systolic blood pressure and an increase in heart rate. It has been assumed that a reduction in the central blood volume would have resulted in reduced cerebral perfusion.

The salient finding of the study, not heretofore reported in the literature, is that LBSP, which is known to decrease cerebral perfusion, caused a spontaneous, divergent, intermittent nystagmus in healthy subjects, with normal cardiovascular responses in the absence of any overt vestibular stimulation. This was not accompanied by any subjective feelings of discomfort, vertigo or nausea. The amplitude and frequency recorded were 'pathological' for a spontaneous nystagmus [6].

The most probable cause for the nystagmus appears to be central cerebral ischaemia. Clinically, vertebrobasilar insufficiency (VBI) is known to cause inappropriate vestibular stimulation, with a nystagmus of central origin. Unilateral nystagmus, as seen in the study, has been reported with a lesion of the cerebellum, or of the central connecting pathways. Two recently reported clinical cases of divergent nystagmus were proven to have an ischaemic pathology on MRI [7].

Our hypothesis of a disturbance in the functioning of the vestibular apparatus during the application of LBSP was also tested by comparing the results of cold caloric stimulation before and during LSBP, using experiments very similar to those used by Claussen and De Sa [6]. While a derangement was seen in all the cases, our results differed from those of Claussen and De Sa in that we could establish no consistent relationship between the amplitude, frequency or the power density spectrum of the nystagmus before and during LBSP. This, however, strengthens our argument that central cerebral ischaemia, an induced by LBSP, causes a derangement rather than stimulation (or depression) of the central nystagmus generator as hypothesized by Claussen and De Sa [6].

In view of our observations we hypothesize that if the nystagmus generator is affected by localized cerebral ischaemia, then during +Gz manoeuvres an exaggerated effect on the same areas may, in fact, contribute to in-flight disorientation. We are also of the opinion that a study of the characteristics of nystagmus induced by LBSP may be used as an investigative tool in cases of aeromedical interest, particularly those of low G tolerance, vasovagal syncope and motion (air) sickness.

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