Hypoxia and Coriolis Illusion in Pilots During Simulated Flight

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INTRODUCTION: Pilots' vision and flight performance may be impeded by spatial disorientation and high altitude hypoxia. The Coriolis illusion affects both orientation and vision. However, the combined effect of simultaneous Coriolis illusion and hypoxia on saccadic eye movement has not been evaluated.

METHOD: A simulated flight was performed by 14 experienced pilots under 3 conditions: once under normal oxygen partial pressure and twice under reduced oxygen partial pressures, reflecting conditions at 5000 m and 6000 m (16,404 and 19,685 ft), respectively. Eye movements were evaluated with a saccadometer.

RESULTS: At normal oxygen pressure, Coriolis illusion resulted in 55% and 31% increases in mean saccade amplitude and duration, respectively, but a 32% increase in mean saccade frequency was only noted for saccades smaller than the angular distance between cockpit instruments, suggesting an increase in the number of correction saccades. At lower oxygen pressures a pronounced increase in the standard deviation of all measures was noticed; however, the pattern of changes remained unchanged.

DISCUSSION: Simple measures of saccadic movement are not affected by short-term hypoxia, most likely due to compensatory mechanisms.

KEYWORDS: Coriolis illusion, hypoxia, oculomotor activity, simulated flight.

Kowalczuk KP, Gazdzinski SP, Janewicz M, Gąsik M, Lewkowicz R, Wyleżoł M. *Hypoxia and Coriolis illusion in pilots during simulated flight*. Aerosp Med Hum Perform. 2016; 87(2):108–113.

Humans and their senses have not evolved to fly. Thus, the environmental conditions characteristic of flight (e.g., reduced partial pressure of oxygen, linear and angular acceleration) are factors contributing to the occurrence of hazards during flight. The most important of them include the phenomenon of spatial disorientation (SD) and altitude hypoxia. SD is a term used in aviation medicine to describe a situation in which the pilot incorrectly feels location, movement, or orientation of the aircraft relative to the Earth's surface and gravity.[1] Aircraft accidents caused by loss of spatial orientation are among the most dangerous consequences and often lead to the death of the crew and huge financial losses.[5,15] In practice, most pilots experience this type of phenomenon. It should be emphasized that as many as 65% of SD events go unrecognized, so the pilots are not aware of their occurrence.

Coriolis illusion is one of the most dangerous phenomena causing SD. It is associated with movements of the head in order to move the eyes to instruments (e.g., radar transponder) during the influence of acceleration on the aircraft. This illusion can produce a sensation that the aircraft is rolling, pitching, and yawing all at the same time, which could be compared with the sensation of rolling down a hillside. This illusion has been evaluated for years,[5] but until now a reliable indicator has not been specified.

The second major threat to military and civil aviation is high altitude hypoxia.[3,25] The human brain is very sensitive to hypoxia,[21] which can result in impaired vision[16] and cognitive and motor processes.[24] It may cause serious brain injury and in extreme cases lead to death.[22,25] At high altitudes (low partial pressure of oxygen), exposure of human tissue to hypoxia can lead to shortness of breath, increased heart rate, blurred vision, and mental disturbances such as delirium or euphoria.[12] Hypoxia is known to detrimentally affect cognitive functions

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This manuscript was received for review in July 2015. It was accepted for publication in November 2015.

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DOI: 10.3357/AMHP.4412.2016

such as working memory and decision making as low as 3000 m (9842.5 ft) above sea level (herein referred to as 'm').[22] It also leads to poor flight performance.[3,25]

A couple of studies have evaluated the effects of hypoxia on oculomotor activity. Merz et al.[20] demonstrated that hypoxia due to climbing up to 7500 m (24,606 ft) does not affect typical saccade parameters (latency, mean square, post-saccadic stability, error rate). However, this research expedition lasted 3 wk, giving the subjects ample time to acclimatize. Another study tested eye movement behavior in Spanish Airforce flight crews after being exposed to short-term hypobaric hypoxia in a pressure chamber.[9] They did not report changes to any eye movement parameters except for mean drift velocity (not evaluated in our study). Oculomotor activity was not tested under hypoxia, but after the pilots had rested and regained proper oxygen saturation (S_aO_2) levels. However, the effects of combined Coriolis illusion and hypoxia on flight performance and basic eye movement metrics have not yet been evaluated.[22]

Summing up, noninvasive methods for the analysis of eye movement have the potential to objectively indicate oculomotor dysfunction as a result of the loss of spatial orientation and high altitude hypoxia. Such a solution could have potential application in the automatic detection of flight safety emergencies. Therefore, we evaluated oculomotor activity during exposure to normobaric hypoxia and Coriolis illusion during a simulated flight.

METHODS

Subjects

Participating in the study were 14 active duty male pilot instructors (age: 31.9 ± 5.8 yr old; height: 178.8 ± 3.5 cm; weight: 78.5 ± 9.2 kg; flight experience: 1060 ± 570 h; five smokers: 12.4 ± 2.5 cigarettes a day). All subjects were flying a TS-11 "Iskra" jet plane and they all had a current fitness to fly certificate given by the military and medical commission (i.e., they were healthy). All the subjects were men, reflecting the sex composition of flight instructors in the Polish Air Force in the early 21st century. All of them had normal or corrected-to-normal vision. The study protocol was approved in advance by the Bioethical Committee of the Military Institute of Aviation Medicine in Warsaw. Each subject provided written informed consent before participating.

Equipment

An Integrated Physiological Trainer (GYRO-IPT, ETC, Southampton, PA) provided all the necessary visual and motion stimulation.[17,18] It is equipped with two-way audio communication, allowing interaction with the investigator and continuous monitoring of the subject. The simulator is equipped with a data acquisition system, so the flight profile status is monitored in real time.[4] The scenario was programmed, including all voice commands. The simulated flight was based on a standard Coriolis training profile used to demonstrate SD, conducted according to the requirements of STANAG 3114 documentation (a NATO standardizing document: http://pl.scribd.com/doc/91991210/Nato-Satanag-3114-Edicion-08-2006#scribd).

The simulated flight was based on a standard training profile [ascent to a prescribed altitude of 1500 m (4921 ft) accompanied by a change in heading course and ascent over 120 s] (**Fig. 1**). The simulator rotated with subthreshold angular acceleration of $0.5° \cdot s^{-2}$ over the first 120 s of the flight, reaching an angular velocity of $60° \cdot s^{-1}$ at the moment of rolling the head to the left to change the settings on the transponder. The combined action of the head roll and simultaneous constant simulator velocity rotation induces the classic Coriolis vestibular cross-coupling effect. This particular profile was chosen because of the ability to measure angular acceleration and its high repeatability, and because it provides strong SD symptoms.

To simulate normobaric hypoxia a pilot mask (mask KM-32, oxygen regulator KP15) connected to an oxygen cylinder filled with respective mixtures was used. Two mixtures were prepared: 1) 11.1% O_2 and 88.9% N_2 at 405 mmHg $Po_2 = 85$ mm for simulating conditions at 5000 m (16,404 ft); and 2) 9.8% O_2 and 90.2% N_2 at 354 mmHg $Po_2 = 74.2$ mm to simulate conditions at 6000 m (19,685 ft).

Eye movement was measured with a JAZZ G-Plus saccadometer (Ober Consulting, Warsaw, Poland) working at 1000 Hz sampling rate, calibrated before each flight. This device has been described in detail in a number of manuscripts.[13,14,17] A saccadometer detects variations in eye movement independently of head motion; x & y eye positions, head acceleration, and pilot's voice were recorded.

Procedure

The subjects were briefed on the study and its aims and they performed one simulated flight before study onset to get acquainted with the procedure. Experiments were conducted after ensuring that all subjects had had a good night's sleep the night before. The pilot's task consisted of carrying out maneuvers with the maintenance of flight parameters according to the recorded commands. After inducing the Coriolis effect, the pilot received a command to maintain a straight and level flight at the current altitude for at least 20 s. Each pilot performed the simulated flights under three conditions of breathing mixture. The first flight was conducted in normal conditions (21.0% O_2 and 78.0% N_2 at 750 mmHg at 115 m), and the second and third flights were both conducted in hypoxic mixtures: 11.1% O_2 and 88.9% N_2 at 405 mmHg and 9.8% O_2 and 90.2% N_2 at 354 mmHg, respectively. Subjects breathed normal air in the control flight (using the mask) and the hypoxic mixtures delivered by mask to simulate high altitude hypoxia. Exposure to normobaric hypoxia began at 10 and 5 min before the flight for the second and third conditions, respectively, and terminated with the end of the study. The study was terminated in the case of at least one of these symptoms: conditions of $S_aO_2 < 70\%$, heart rate > 140 bpm, or at the pilot's request. The minimum interval between particular flights was 10 d to avoid habituation and to minimize learning effects. During the experiment subjects were constantly monitored with an ECG and pulse oximeter (Nellcor N-395 using earlobe sensor Nellcor D-YS;

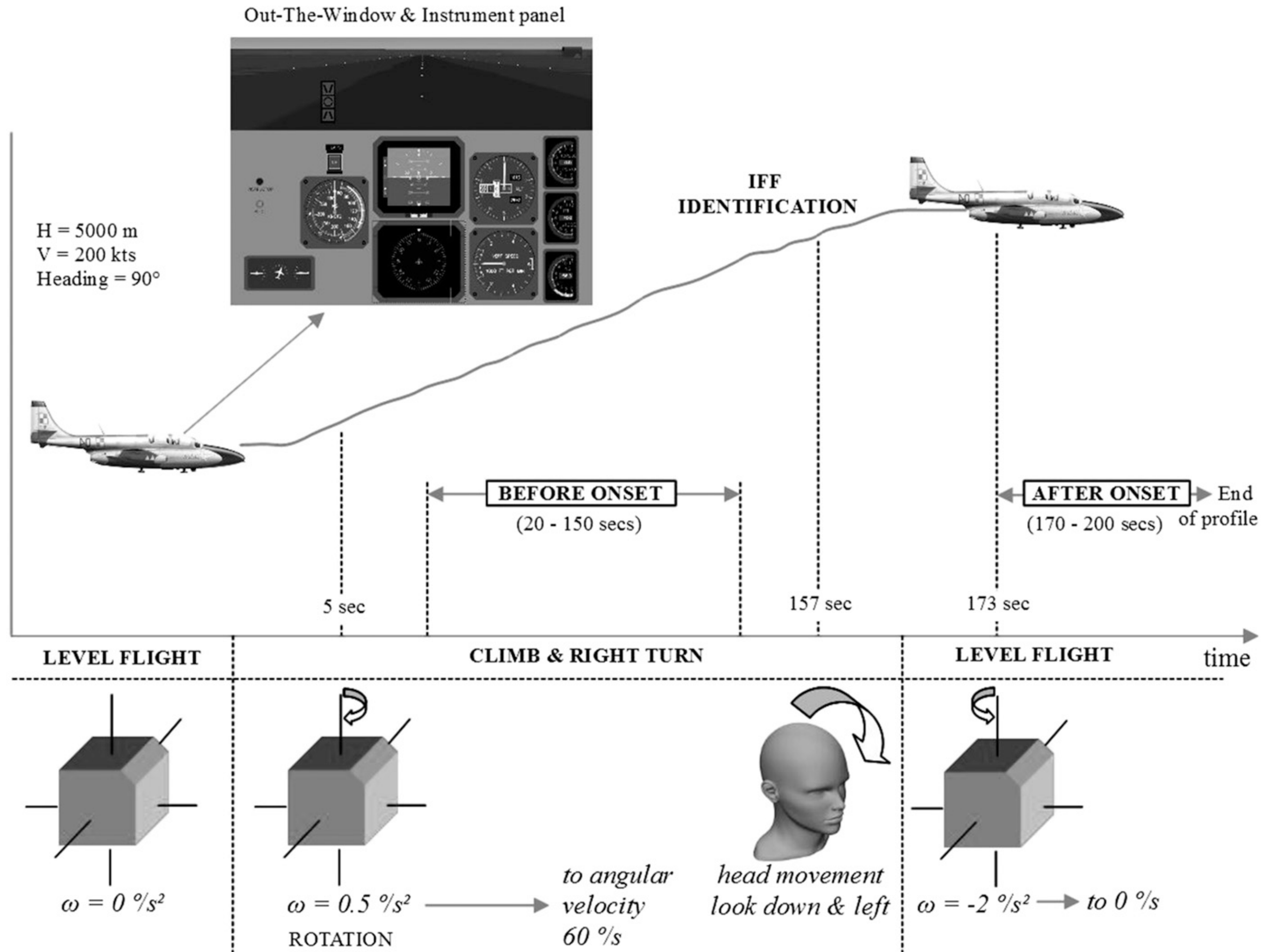

Fig. 1. Simulated flight profile. Before Onset refers to the time interval preceding onset of the Coriolis illusion; After Onset refers to the measuring time afterwards. Time count starts with the onset of the prerecorded instructions.

Medtronic, Mansfield, MA) to assure safety. Physiological parameters were recorded continuously starting 15 min prior to exposure in the simulator and ending 15 min after completion of the flight. Analysis of the results of the study consisted of:

- simulated flight time from 20 to 150 s is referred to as the first window Before Onset and after inducing Coriolis effect the flight time from 170 to 200 s is referred to as the second window After Onset for the analyzed parameters: fixation time, saccade frequency, saccade duration, saccade amplitude;
- conditions of S_aO_2 during simulated flight; and
- time to return to normal flying parameters [limits: altitude ± 100 ft (30 m), heading ± 5°, velocity ± 10 kn (± 18.5 km/h)] after the Coriolis effect (flight time from 170 to 200 s).

After the simulated flights each subject filled in a simple hypoxia symptom questionnaire to inform the researchers about any symptoms they had during the flight. In the eye movement analysis the following parameters were calculated: mean fixation time, mean saccade duration, saccade frequency, mean saccade amplitude, and mean peak saccade velocity. They were calculated over the two windows Before Onset and After Onset. The 20-s period between these windows was not used in calculations due to nystagmus elicited by the Coriolis illusion that could have biased the results of the study. Additionally, to account for the potential effects of boredom on the saccadic parameters, the Before Onset window was replaced with a shorter, 20-s window and used in follow-up analysis. To validate the calculation of means over the Before and After Onset windows, we divided each of the windows into 15-s intervals and compared them with each other to make sure that the means of saccade parameters did not change significantly within these windows.

Additionally, for eye movement analysis we introduced a coefficient relating saccade duration with duration of following fixations (DSF) defined as the total saccade duration normalized to total fixation duration over same evaluation period.[17]

$$DSF(i) = \frac{T_S(i)}{T_F(i)}$$

The following parameters were used for automatic saccade detection: saccade duration between 5 ms and 500 ms followed by fixation not shorter than 40 ms, and peak velocity and minimum amplitude greater than $30° \cdot s^{-1}$ and 2°,[10] respectively. A high limit for duration of saccades was selected to include large saccades performed during calibration. All selected saccades were visually reviewed and all artifacts were removed. In order to distinguish whether the observed effects were due to changes in saccade amplitude or fixation duration, the automatic detection was later repeated with a minimum fixation time of 120 ms and a minimum saccade amplitude of 5°, the latter corresponding to the angular distance between flight instruments in the cockpit of the simulator; i.e., smaller saccades were too small to move the gaze from one instrument to another. Only a few saccades shorter than 120 ms were detected.

Statistical Analysis

Normality of distributions was evaluated with a Kolmogorov-Smirnov test. Oculomotor parameters were compared with two-tailed *t*-tests or paired *t*-tests, where appropriate. A significance level of $P < 0.05$ (after correction for multiple comparisons) was considered statistically significant. Percentage changes in parameters due to the effects of interest were compared, as size effects underestimated the real changes in these parameters due to the increased range of these parameters at higher altitudes. All statistical tests were conducted with Excel 2010.

RESULTS

From the initial 14 subjects, only 8 sets of data corresponding to an altitude of 5000 m (16,404 ft) and 7 sets corresponding to 6000 m (19,685 ft) were completed; we obtained good quality datasets for both 5000 m and 6000 m from 5 subjects. The mean time needed to return to normal flying parameters (altitude and heading given by the investigator prior to the onset of illusion) after the onset of the Coriolis illusion was 10.63 ± 1.63 s at 5000 m and 21.9 ± 7.0 s at 6000 m, as compared to 6.58 ± 0.94 s under normoxic conditions ($P < 0.001$); these times at 6000 m were significantly longer than at 5000 m ($P < 0.001$).

The adverse effects of hypoxia obtained by self-report are summarized in **Table I**. We did not find significant correlation between subjective hypoxia symptoms and decrements in performance. Preceding the Coriolis illusion, no effects of hypoxia on mean saccade frequency, mean saccade duration, or mean saccade amplitude were recorded. No correlation was found between subjective symptoms (Table I), flight precision, or the time needed to regain correct flight parameters.

Regarding Coriolis illusion at normal S_aO_2, a 55% increase in mean saccade amplitude [$t(13) = -4.78$, $P = 0.0004$], accompanied by a 31% increase in mean saccade duration [$t(13) = -4.85$, $P = 0.0002$] was observed. Additionally, a 32% increase in saccade frequency [$t(13) = -4.15$, $P = 0.001$] was noted, reflecting the effects of SD. It was accompanied by a 24% decrease in mean fixation time [$t(13) = 3.21$, $P = 0.006$]. The above data are summarized in **Table II**. However, defining saccades as larger than 5° (angular distance between flight instruments), only a 31% increase in mean saccade amplitude [$t(13) = -3.68$, $P = 0.002$] and a 40% increase in mean saccade duration [$t(13) = -4.19$, $P = 0.0008$] were observed, accompanied by an insignificant 12% increase in frequency and an insignificant 5% decrease in mean fixation time, suggesting that the Coriolis illusion results in an increased number of correctional saccades (smaller than 5°; they are too small to move the gaze from one flight instrument to another). These changes could be alternatively described as 4.6- and 5.2-fold increases in DSF for saccades larger than 2° or 5°, respectively [$t(13) = 8.1$, $P < 0.0001$] (Table II).

At 5000 m and 6000 m (16,404 and 19,685 ft) the variability of data substantially increased. The pattern of changes remained; however, none of them remained statistically significant (see Table II). Only the DSF increased threefold for 5000 m [$t(6) = -4.46$, $P = 0.004$] and fourfold for 6000 m [$t(4) = -3.20$, $P = 0.03$].

Frequency of saccades >2° was approximately twice the frequency of saccades >5° at normal S_aO_2 and at 5000 m (16,404 ft). However, the ratio increased to 3 at 6000 m (19,685 ft), demonstrating the difficulty of maintaining proper fixations under moderate hypoxia (see Table II). The findings did not change when 120 ms was used as the lower limit for fixation

Table I. Adverse Symptoms Accompanying Normobaric Hypoxia: Number of Participants Reporting Given Symptoms and Corresponding Percentage (%).

	5000 m		6000 m	
	REPORTED SYMPTOMS		REPORTED SYMPTOMS	
SYMPTOMS	NUMBER OF SUBJECTS	% SUBJECTS	NUMBER OF SUBJECTS	% SUBJECTS
Disturbed Attention	1	6.25	4	25
Visual Disturbances	11	68.75	12	75
Sleepiness	2	12.5	4	25
Tiredness	1	6.25	2	12.5
Sweating	1	6.25	1	6.25
Breathlessness	0	0	2	12.5
Shivering	0	0	0	0
Uneasiness	1	6.25	4	25
Headache	0	0	1	6.25
Euphoria	1	6.25	4	25
Nausea	1	6.25	4	24

Table II. Oculomotor Parameters Before and After Onset of Coriolis Illusion for Particular Altitudes.

	115 m		5000 m		6000 m	
PARAMETER	BEFORE ONSET	AFTER ONSET	BEFORE ONSET	AFTER ONSET	BEFORE ONSET	AFTER ONSET
Mean saccade amplitude (degrees)	5.8 ± 1.3**	6.6 ± 1.7**	5.45 ± 0.74	6.58 ± 2.06	5.23 ± 0.68	6.38 ± 1.80
Mean saccade duration (ms)	56 ± 9*	73 ± 20*	60 ± 9	68 ± 13	62 ± 8	82 ± 13
Saccade frequency (Hz)	0.84 ± 0.30*	1.11 ± 0.42*	0.97 ± 0.16	1.20 ± 0.42	0.79 ± 0.37	0.90 ± 0.59
Mean fixation time (ms)	1269 ± 498*	964 ± 489*	988 ± 192	832 ± 292	1404 ± 626	1661 ± 1997
DSF (10^{-3})	0.44 ± 0.20**	2.30 ± 1.08**	0.55 ± 0.16*	2.08 ± 0.87*	0.45 ± 0.20*	2.59 ± 2.85*
Mean peak saccade velocity($° \cdot s^{-1}$)	443 ± 517	585 ± 356	287 ± 99	277 ± 49	244 ± 75	250 ± 68
ISF ($° \cdot s^{-1}$)	4.9 ± 2.2*	7.6 ± 3.6*	5.3 ± 1.1*	7.8 ± 3.0*	4.1 ± 1.9	5.3 ± 3.0

DSF = duration of saccade fixations; ISF = intensity of saccade fixations. Saccades were defined as larger than 2° and longer than 120 ms: *$P < 0.05$; **$P < 0.0001$.

time. Additionally, the pattern of results remained basically unchanged when the short, 20-s version of the Before Onset window was used. Furthermore, the observed trends remained when results were confined to the five subjects that completed the simulated flights at all three altitudes.

DISCUSSION

We observed deterioration of pilot performance due to combined Coriolis illusion and hypoxia. It was reflected as longer times needed to reach required flight parameters and was not related to the pilot's blood oxygenation. Hypoxia per se did not affect basic eye movement parameters. Coriolis illusion, though, was related to increases in both mean saccade amplitude and duration, but an increase in mean saccade frequency was only noted for saccades smaller than the angular distance between cockpit instruments, suggesting an increase in the number of correction saccades. This pattern remained at lower S_aO_2 levels, although statistical significance decreased due to the small number of subjects and larger variability of results reflected as increased standard deviations. A higher number of correctional saccades was noticed for moderate hypoxia [6000 m (19,685 ft) altitude]. Even mild hypoxia significantly increases the possibility of SD in pilots during a Coriolis illusion flight profile. The increase (in some cases very high) in time needed to return to correct flight parameters poses a serious threat to flight safety. Pilots may not be aware of their decreased performance. Implications of altitude in acrobatic flying should also be considered (cumulative effect of hypoxia and $+G_z$: excessive and sustained gravitational forces draining blood away from the brain causing cerebral hypoxia).

The observed effects of hypoxia on basic eye movement are consistent with earlier research.[9,11,20] Our results also confirm and extend previous studies suggesting that observed effects were not due to acclimatization[20] or performing testing under normoxic conditions.[9]

Variability of eye movement parameters increased with hypoxia. Without the Coriolis effect, simple oculomotor activity was not affected, likely due to compensatory mechanisms. During the stimulation of the vestibular organs due to angular acceleration, the times of stable fixation were shortened and the number of correctional saccades increased. Taken together, SD induced by the Coriolis illusion exceeds the pilot's capacity to compensate. This is particularly evidenced by the increased number of correctional saccades at 6000 m (19,685 ft). Studies have demonstrated that the cerebellum plays an important role in gaze holding.[2] Because hypoxia induces alterations in the metabolic system of the cerebellum, it may increase eye instability by disrupting cerebellar inputs to the neural integrator.[9]

Our work provides information on the mechanisms responsible for developing SD under an additional adverse environmental factor: hypoxia. Altitude hypoxia induced by staying at 4000–6000 m (13,123–19,685 ft) corresponds, by adaptability, with incomplete compensation. Lack of oxygen is not fully compensated for by physiological adaptation mechanisms. The time for which one can perform successfully in such conditions depends on individual factors. The time of exposure set in our study allows the pilot to initiate physiological responses for average [5000 m (16,404 ft)] and high [6000 m (19,685 ft)] hypoxia intensity.

Recent research has found that training in hypoxic environments can improve the performance of some motor and cognitive tasks in mice,[12,19] suggesting that refresher hypoxia training might similarly improve pilot sensorimotor performance. The question of whether refresher hypoxic training may help to improve responses to emergency situations in aviation is currently open.[9,24,26] In our research oculomotor activity was recorded independently of the pilot's head movements; however, Daye et al.[8] concluded that even when no head movement is planned the brain uses feedback control of the gaze and not the eye alone. At the same time, this gaze command acts to suppress the vestibulo-ocular reflex if the head should be perturbed by an imposed movement during the initial saccade. Several studies have shown that neck muscles are activated during stimulation of the superior colliculus[6,23] or frontal eye fields,[7] even when the subject does not make a head movement.

Finally, some limitations of the present study could be identified. The study did not include fatigue associated with the performance of flight maneuvers and the impact of hypoxia. However, the pilots did perform the flights after a presumably good night's sleep, thus minimizing the effects of fatigue. Thus, future studies should also investigate the additional effects of fatigue on the oculomotor activity of pilots. Moreover, it is not clear whether similar variations in eye movement would occur if different hypoxia conditions or a different illusion was used. Comparison of our study with previous studies suggests that hypoxia conditions, or rather their duration, may play a role in evoking eye movement variations. Thus, future studies should also manipulate the duration of hypoxia.

In conclusion, onset of the Coriolis illusion does not modify the pattern of change of oculomotor metrics. Moreover, the level of significance of these changes decreased due to an increase in variability of the measurements. Simple measurements of saccadic movements remain unchanged while under hypoxia, most probably due to compensatory mechanisms (blood redistribution).

ACKNOWLEDGMENTS

We thank Jacek Dylak of Ober Consulting, Warsaw, Poland, for technical assistance along the way and Andrzej Gaździński for assistance in the computer programming needed for data analysis. The study was supported by a Military Institute of Aviation Medicine internal grant (88/2003 to K. P. Kowalczuk), by the Inspectorate of Military Health Service (01/WNiL/2007 to M. Wyleżoł), and by the Polish National Science Centre (2011/03/B/NZ4/03771 to S. P. Gaździński).

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