Delayed Drowsiness After Normobaric Hypoxia Training in an F/A-18 Hornet Simulator

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BACKGROUND: In military aviation, due to high-altitude flight operations, hypoxia training is mandatory and nowadays is mainly done as normobaric hypoxia training in flight simulators. During the last decade, scientific data has been published about delayed recovery after normobaric hypoxia, known as a "hypoxia hangover." Sopite syndrome is a symptom complex that develops as a result of exposure to real or apparent motion, and it is characterized by yawning, excessive drowsiness, lassitude, lethargy, mild depression, and a reduced ability to focus on an assigned task.

CASE REPORT: In this study, we present the case of a 49-yr-old pilot who participated in normobaric hypoxia refreshment training in an F/A-18C Hornet simulator and experienced delayed drowsiness, even 3 h after the training.

DISCUSSION: This case report demonstrates the danger of deep hypoxia. Hypoxia training instructions should include restrictions related to driving a car immediately after hypoxia training. In addition, hypoxia may lower the brain threshold for sopite syndrome.

KEYWORDS: hypoxia training, sopite syndrome, normobaric, simulator sickness.

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Motion sickness can occur when a person is exposed to a visual or vestibular mismatch. The primary symptoms are nausea, vomiting, pallor, and a cold sweat. Such sickness is usually provoked by traveling by boat, car, or airplane, but it can also result from simulators, virtual reality, or space travel.[6] It is known that motion can also cause yawning and lethargy, and these can sometimes be the sole manifestations of motion sickness. These symptoms were first described as "sopite syndrome" by Graybiel and Knepton in 1976.[8]

Sopite syndrome has been later defined as a symptom complex that develops as a result of exposure to real or apparent motion, and it is characterized by excessive drowsiness, lassitude, lethargy, mild depression, and a reduced ability to focus on an assigned task.[13] It is considered distinct from "regular" motion sickness because it has different cardinal symptoms (drowsiness vs. nausea) and a different time course. Sopite syndrome usually appears before nausea and persists longer.[10] Cognitive performance has been noticed to decline, even when motion sickness and soporific symptoms are mild.[15] In aviators, sopite syndrome may persist without being recognized and it may threaten flight safety.[10] Yawning has been shown to be a viable behavioral marker that can be used to recognize the onset of soporific effects.[14]

It is still unclear how motion sickness develops, but the most widely accepted theory is the sensory conflict theory. It proposes that when the motion detected by vestibular, visual, and proprioceptor systems conflicts with the expected or previously learned motion, the mismatch of neural signals may result in motion sickness. This is supported by experienced pilots having more simulator sickness during flight simulator training than student pilots, since the latter have not yet become accustomed to the real motion of aircraft.[6] Subjects who have lost their normal vestibular function have been noted to be free of such symptoms.[7] It is speculated that sopite syndrome is evoked by

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the inhibition of the noradrenergic neurons of the locus coeruleus.[16]

In military aviation, due to high-altitude flight operations, hypoxia training is mandatory and nowadays is often done as normobaric hypoxia training in flight simulators. The Finnish Air Force has conducted normobaric hypoxia training since 2008.[11] During the last decade, scientific data has been published about delayed recovery after normobaric hypoxia; this is called a "hypoxia hangover."[21] In this case report, we present a case of delayed drowsiness that occurred after normobaric hypoxia training.

CASE REPORT

A 49-yr-old male pilot participated in normobaric hypoxia refreshment training in an F/A-18C Hornet simulator. He had completed chamber hypobaric hypoxia training in the U.S. Navy and simulator hypoxia refreshment training in both BAE Hawk and F/A-18C Hornet simulators at 3-yr intervals. His previous hypoxia training sessions had been uneventful. The pilot had completed an annual aeromedical flight physical that noted a near-vision correction requirement and the need for medication for both hypercholesterolemia and gastroesophageal reflux disease. He had experienced motion sickness in a car as a child and had once experienced airsickness (including vomiting) during early flight training with a Hawk jet trainer. This Hawk flight also included yawning and lethargy before the vomiting. However, he had adapted to the sensory mismatch of military flying and had had no motion sickness for over 20 yr. Over the three nights before the hypoxia training, he had 7 h, 8 h, and 8 h of sleep and felt well-rested.

The hypoxia training was performed at 1230 h in a fixed-based tactical F/A-18C Hornet Weapons Tactics and Situational Awareness Training System simulator (Boeing Corporation, Chicago, IL, USA). The pilot's flight gear consisted of a flight helmet with a mask (Gentex Corporation, Zeeland, MI, USA) and a flight vest with a regulator (as is normally worn by pilots while flying fighter aircraft). Forehead peripheral oxygen saturation (S_pO_2), minute ventilation, and wireless electrocardiogram were monitored during the experiment by the senior flight surgeon (J.S.). Minute ventilation, S_pO_2, and subjective symptoms were manually saved to a data sheet by an experienced flight nurse. The flight instructor and the senior flight surgeon used audio-visual monitoring of the pilot. During hypoxia training, four gas mixtures were used with different concentrations of O_2: 8%, 6%, 21% (equal to sea level), and 100% (emergency oxygen). The gas change was done manually by using a gas selection box (Hypcom, Tampere, Finland). Physiologically, 8% O_2 simulates a cabin altitude of 6200 m (20,341 ft) and 6% O_2 simulates a cabin altitude of 7900 m (25,919 ft).

The training included two set-ups of the same simulated visual identification flight with a mask on and the sudden onset of different O_2 concentrations (8% and 6% O_2). At the beginning of both set-ups, the pilot was breathing air, but the flight surgeon switched on 8% or 6% O_2 during tactical maneuvering. The pilot was instructed to continue the flight mission until he recognized hypoxia symptoms or saw a system warning (a master caution and OBOGS DEGD light), and then execute hypoxia emergency procedures. The emergency procedures in hypoxia were: 1) a green ring pull, i.e., releasing emergency O_2 (100%); 2) turning the OXY FLOW KNOB off, i.e., turning the main O_2 valve off; 3) an emergency descent at a 20° nose-down attitude; and 4) sending a transponder code 7700 (an emergency squawk).

During the first set-up, when the pilot was exposed to 8% O_2, he noticed symptoms of hypoxia 74 s after the hypoxic mixture gas was induced. The symptoms were lightheadedness, deep breathing (16 L · min^{-1}), and increased heart rate (98 bpm). At this point, his S_pO_2 was 78%. The pilot consciously wanted to experience deeper hypoxia and continued the set-up mission after hypoxia recognition without emergency procedures. He cleared this intention with the senior flight surgeon via radio. After 5 min, the pilot experienced tunnel vision but was able to fly, making visual identification of unidentified aircraft and using throttle adjustments to keep a visual identification position with euphoric sensation. After 5 min 55 s (355 s), the senior flight surgeon noticed the pilot's slow speech and that his left hand on the throttle started to twitch. The master caution light, the sound warning, and the OBOGS DEGD text appeared because the flight surgeon aborted the set-up. Although S_pO_2 was 59%, the pilot was able to execute all emergency procedures. The simulated flight was frozen after an emergency descent at low altitude and level flight for 3 min in order to give feedback and instructions for the next set-up.

During the next set-up with a gas mixture of 6% O_2, the pilot noticed the same hypoxic symptoms after 43 s with S_pO_2 78%, and all emergency procedures were executed after 61 s with S_pO_2 further decreasing to 69%. Ventilation increased significantly from 14 L · min^{-1} to 21 L · min^{-1} during hypoxia. After the second set-up, a return-to-base flight was made at low altitude. The flight performance was standard level and the landing under Visual Flight Rules conditions was normal. The pilot did not experience any nausea or motion sickness during the simulator training and the simulated flight did not include intensive maneuvering.

During debriefing, 15 min after the hypoxia simulator training, the pilot felt normal and was not pale. The instruction pilot and senior flight surgeon emphasized the importance of aborting the flight mission immediately after hypoxia recognition in order to increase the time of useful consciousness for emergency procedures. The pilot was driving his car home 1 hr after the hypoxia training and, during the drive, he felt extreme lethargy and was yawning 2–3 times per min for 2 h. He considered pulling the car aside but drove all the way home. The lethargy and yawning were gone 3 h after the hypoxia training, and he felt normal the following morning.

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DISCUSSION

Most of the symptoms caused by simulator sickness should alleviate quickly after the training is over, but around 10% of pilots experience aftereffects that persist for several hours, which may increase the risk of safety hazards.[1,6] Tiredness and fatigue are the most common adverse effects after normobaric hypoxia training.[22] Therefore, the Finnish Air Force is using a 12-h grounding from flight duty after hypoxia training. Also, car-driving problems after hypoxia training have been reported previously.[22] For safety, hypoxia training instructions should include restrictions on driving a car immediately after hypoxia training. This is supported by a recent study, which showed a delayed neurocognitive recovery after a hypoxic exposure.[2]

The pilot reported yawning and extreme tiredness 1–3 h after normobaric hypoxia training. The pilot had experienced motion sickness and sopite syndrome after Hawk IMC aerobatics during his early flight career. The symptoms in this case report matched sopite syndrome without cybersickness symptoms. However, it is not possible to determine with certainty whether symptoms were delayed sopite syndrome, hypoxia-induced drowsiness, or a combination of both. The symptoms may mirror an autonomous nervous system balance change, leading to inhibition of noradrenergic pathways, especially in the reticular formation brain area.[16]

There has been a previous report of a 23-yr-old student pilot experiencing such extreme tiredness during T-6B aerobatic training that he almost fell asleep.[5] They practiced adaptation for 7 d with a Barany chair, which resolved the nausea symptoms, but the drowsiness persisted and this ended his flight career. Sopite syndrome has also been described during a parabolic flight.[20] A 35-yr-old participant had symptoms of nausea, irritation, and drowsiness that were provoked by intermittent periods of weightlessness. At the end of the flight, the symptoms worsened to the point that she was almost unconscious, and the mood changes lasted for several hours. Interestingly, the participant had received a subcutaneous scopolamine injection prior to the parabolic flight.

On average, Finnish military pilots recognize their hypoxia symptoms at the S_pO_2 saturation level of 73% with 6% O_2 exposure.[11] S_pO_2 is known to weakly predict, for example, working memory impairment.[12] During normobaric hypoxia training, the exposure time is a more important parameter than S_pO_2, although, for example, the U.S. Navy uses 60% S_pO_2 as an abort point. It took 18 s for the case pilot to execute all the hypoxia emergency procedures. This highlights the importance of making an early decision to abort a flight mission and having the cognitive ability to change one's mental focus from an operational flight task to emergency procedures, creating a safety margin before the onset of more severe cognitive impairment when approaching the time of useful consciousness.[9] If a pilot does not execute hypoxia emergency procedures immediately, there is a risk that he or she will lose consciousness in real flights. In this case report, the pilot would not have been able to abort the flight in the first set-up without the senior flight surgeon. This highlights the reduced ability to make decisions during deep hypoxia. Our experience from over 900 normobaric hypoxia training sessions in an F/A-18 Hornet simulator is that the pilot can even fly the aircraft in deep hypoxia, but the pilot's situational awareness (SA 2 and 3 levels) and cognitive ability are decreased. Even in deep hypoxia, pilots can follow direct orders to start emergency procedures (like in our case) or follow a lead aircraft (i.e., they can perform a supported emergency descent as Dash 2 in formation).

Hyperventilation is one of the reasons for nonpressure hypoxia-like physiological episodes in flight. It is even possible that the majority of reported physiological episodes are caused by hyperventilation symptoms[3] that are recognized because of mandatory hypoxia training in military aviation. Some of what seem to be hypoxia symptoms reported in this case report are actually hyperventilation-induced symptoms.[18] This can be one explanation for why hypoxia symptoms in the same individuals can vary from one hypoxia training session to another. Pilots with a slow ventilation rate during hypoxia may lack previously learned symptoms and have difficulties in identifying hypoxia due to the lack of hyperventilation-induced hypocapnia symptoms.

Hypoxia impairs working memory, increases reaction time, and deteriorates executive functions.[4,12,19] In addition, hypoxia has a long-lasting effect on the pilot's flight performance even if hypoxia emergency procedures are executed without delay. The reaction time and regional cerebral saturation do not return to baseline levels until 24 h after hypoxia exposure.[17]

In conclusion, this case report demonstrates delayed drowsiness after normobaric hypoxia training. Yawning and extreme lethargy were even seen 3 h after the hypoxia training. A hypoxia hangover may also involve an autonomous nervous system balance change, leading to the inhibition of noradrenergic neurons. More research is needed in order to understand the complicated relationship between hypoxia and body homeostasis maintenance.

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