Hyperventilation-Induced Hypocapnia in an Aviator

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BACKGROUND: Physiological episodes are a top safety concern for aviators across the United States military. While many cases and a variety of causes for physiological episodes have been described, few cases, if any, have been reported of hyperventilation-induced hypocapnia and transient loss of consciousness.

CASE REPORT: Here we describe a case of an aviator who experienced tingling extremities, confusion, and loss of consciousness during a flight. The aviator incorrectly believed he was experiencing hypoxia and continued to take multiple steps to troubleshoot the wrong underlying problem for his symptoms. Evaluation after landing suggested this was instead a stress-induced hyperventilation that resulted in symptomatic hypocapnia.

DISCUSSION: We report this case to add to the body of literature in understanding this phenomenon as well as to provide aviators, physiologists, and flight surgeons with practical suggestions for recognizing hyperventilation-induced hypocapnia and awareness of how to remedy this situation when they recognize it.

KEYWORDS: hyperventilation, hypocapnia, hypoxia, physiological episode.

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Carbon dioxide is a major product of metabolism in the body and is eliminated through respiration. Carbon dioxide in the blood directly affects blood pH and thereby respiratory drive. When respiration exceeds the amount necessary to eliminate carbon dioxide, the pH is raised and can induce symptoms such as light-headedness, headache, visual disturbances, paresthesias, and even loss of consciousness. These symptoms are very similar to hypoxia and when occurring in flight may cause an aviator to believe he or she is experiencing hypoxia due to another cause such as loss of cabin pressure, On-Board Oxygen Generation Systems (OBOGS) malfunction, or otherwise. The aviator may then take measures to address these other causes without considering hypocapnia as the cause of his or her symptoms. Fear and anxiety, among other stressors, are a prime contributor to hyperventilation.[1] The mechanism whereby hypocapnia decreases cerebral perfusion and thereby consciousness is twofold. As blood pH rises, arteries vasoconstrict, reducing cerebral perfusion. This hypoxic effect of hypocapnia may be further increased as cellular metabolism increases, raising demand for oxygen. This dangerous combination does not allow for an aviator to maintain the cerebral function required for flying an aircraft. In extreme cases such as the one presented below, it may even result in transient loss of consciousness.

CASE REPORT

A 27-yr-old male aviator was working on carrier qualifications doing his first night catapult shot. He was feeling anxious about the flight and about 8 min into the flight started feeling off. He continued to feel increasingly confused and noticed a sensation of tingling in his arms and legs. Approximately 10 min into the flight he pulled his emergency oxygen release and declared an emergency. His symptoms did not improve with backup oxygen or cabin decompression. He was redirected to land at a nearby base and briefly lost consciousness during the landing. He was given oxygen and transported via ambulance to a local emergency room. EMS reported an approximately 1-min loss of consciousness. Physical examination by the flight surgeon upon arrival in the emergency room revealed normal vital signs, normal heart, lung, and abdominal exams, as well as a normal

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complete neurological exam. Laboratory results of note included an initial carboxy-hemoglobin of 3.6 which later went down to 2.5. His venous blood gas on arrival at the emergency department was pH 7.58, carbon dioxide 25, and bicarbonate 24. After being in the emergency department for 3 h, it changed to a pH 7.43, carbon dioxide 23, and bicarbonate 22. All other laboratory tests and chest X-ray were within normal limits.

DISCUSSION

For this young male aviator who presented with progressive confusion, tingling of extremities, and brief loss of consciousness (LOC) during flight, differential diagnosis includes hypoxia, carbon monoxide poisoning, and respiratory alkalosis. The fact that the patient's symptoms did not improve with backup oxygen or cabin decompression makes hypoxia due to malfunction of the OBOGS unlikely. The venous blood gas obtained in the emergency room gives an important insight into what the cause of his physiological event was, even though it was drawn several minutes after he regained consciousness. The elevated pH of 7.58 indicates that he was in alkalosis and the carbon dioxide level of 25 indicates that his alkalosis was respiratory in nature. This would be caused by hyperventilating and blowing off carbon dioxide, which lowered the pH of his blood. The initial cause of his hyperventilation was likely anxiety combined with donning of flight gear, which decreases respiratory ability,[5] which progressively increased as he started feeling symptoms and believed he was not getting oxygen. These venous blood gas values would be improved as he continued to recover during transport and in the emergency room. His blood pH was likely even higher as he ended his flight. He continued to hyperventilate to the point of transient LOC. Hypocapnic hyperventilation has been shown to affect psychomotor performance at levels of P_ACO_2 of 15 torr, though intellectual performance deficits are still not demonstrated at that level.[2] Prior to reaching levels of 25 torr, performance of both intellectual and motor activities are largely unaffected.[2,4] Some literature suggests that hyperventilation alone is not enough to induce transient LOC and that other factors must be present to decrease cerebral perfusion to this level.[3] In this case, an increased cerebral blood flow demand from psychological stress may have been the additional factor.

It is important for both flight surgeons and aviators to be able to differentiate hypoxia from hyperventilation-induced hypocapnia. When this aviator performed multiple troubleshooting steps for hypoxia and his symptoms continued to worsen, he should have suspected that there was some other cause. Had he been aware of hyperventilation-induced hypocapnia as a cause for these types of symptoms he may have been able to recognize it and slow his breathing to help ameliorate the situation.

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