Correlation of Clinical Signs/Symptoms and Oxygen Saturation in the Hypoxic Patient

Senior Project

In partial fulfillment of the requirements for
The Esther G. Maynor Honors College
University of North Carolina at Pembroke

By

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4/27/2017

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Acknowledgements

I greatly appreciate all the guidance I received from my mentor Susan Edkins, project coordinator Dr. Teagan Decker, and Dr. Leah Fiorentino. I would also like to thank Dawn Brown, Gordon Byrd, Stefany Friel, and others that helped revise and edit my thesis. It was the support of these people that made my research possible and enjoyable.

Abstract

Hypoxia is a condition in which the body lacks oxygen, and is a problem for some populations. Hypoxia in otherwise healthy adults normally happens in situations that would prohibit the use of standard diagnostic tools, such as underwater diving and aviation. Furthermore, the loss of consciousness that follows untreated hypoxia has a high chance of being fatal in these situations. Certain programs like NASA and some military training centers induce hypoxia in their students so they can understand firsthand what signs/symptoms occur, and that they need to fix a problem. However they do not provide a concise progression to these signs/symptoms, so it is impossible to know how severely an individual has become hypoxic. This implies a population that is in need of a way of knowing when they or their partner are beginning to suffer hypoxia without the use of an electronic device or tool, and to know how severe the hypoxia is.

Keywords: Hypoxia, Hypobaric, Normobaric, Oxygen, Aviation, Symptoms

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Introduction

Hypoxia and its effect on all aerobic life has been well known for a long time. It is understood that all humans need to breathe a sufficient amount of oxygen to survive, with death occurring roughly five minutes after anoxia (Lahiri, 1974, p. 273). In response to the lethality of hypoxia, the human body has developed safeguards and warning systems to prevent oxygen levels from becoming too low. However, these safeguards only function well in normobaric air gas compositions, and are dependent upon other gases, mainly carbon dioxide, to properly alert the body to problems. Aviation and diving technology has evolved and there has been a constant concern about the recognition of hypoxia in different low oxygen environments. Military pilots and other government programs have instituted training that subjects trainees to hypoxic levels in a controlled situation. This allows trainees to become familiar with the symptoms of hypoxia and identify their *hypoxic signature*. The symptoms of hypoxia, and uniqueness of an individual's *hypoxic signature* is of interest, because despite small variation between individuals, most experience similar symptoms at specific pressures (Johnston, Iremonger, Hunt, Beattie; 2012).

Hypoxia definition and pathophysiology

Hypoxia has been defined as "a deficiency of oxygen reaching the tissues of the body" ("Hypoxia", 2017), and is generally separated into four types; hypoxemic, anemic, stagnant, and histotoxic. This paper focuses on hypoxemic hypoxia (sometimes referred to as hypoxic hypoxia) which results from exposure to a low oxygen environment. The basis of a low oxygen environment is one that provides inadequate oxygen to meet the requirements of aerobic metabolism. Due to the variety in metabolic rates between individuals with different external workloads, an exact quantitative description cannot be made for low oxygen. However, some

organizations have identified 0.16 atmospheres as the lower limit for the partial pressure of oxygen. Clinically, hypoxia is more directly assessed via oxygen saturation (Sa02) measurements and pulse oximetry. Sa02 readings at 95% or below are generally indicative of hypoxia, and below 90% require corrective treatment (AAOS, 2011, p. 334)

Hypoxic injury is more easily understood at the cellular level. Oxygen is a primary component of energy production through oxidative phosphorylation and acts as an electron acceptor. During hypoxia, oxidative phosphorylation stops, and the body has to resort to anaerobic methods of energy production that eventually are unable to keep up with cellular demand. Adenosine triphosphate (ATP) levels fall and energy dependent processes, in particular sodium-potassium pumps and calcium channel pumps, are unable to function. Because of the loss of active ion diffusion, an influx of sodium and its accompanying water cause the cell to become hypotonic and bleb. Blebbing along with overall turgor of the once cryptic cells is a hallmark sign of reversible cellular injury (Robins, Abbas, & Aster, 2015, p. 40). The efflux of potassium depolarizes the cell membrane, shutting down bioelectrical signaling. The brain, which is heavily dependent on the sodium-potassium pump for function is usually the first organ to experience deleterious effects from hypoxia (Nilsson, 2010).

As the cell continues without oxygen, cytosolic calcium builds with two major consequences. The first consequence involves calcium directly inducing the activation of enzymes that attack the cell membrane, and other structural components. This process causes the cell membrane to break down and leak cellular material (proteins) into extracellular space. The second, and fairly new understanding, is calcium's role in increasing mitochondrial permeability, which release caspase enzymes into the cytoplasm, and causes apoptosis through hydrogen peroxide. (Tripathi & Chaube, 2012). Robins et al. (2015) describes both the breakdown of the

cell membrane and the self-reactive enzymatic activity to be hallmark signs of irreversible cell injury and necrosis.

Respiratory Regulation and Hypoxia Recognition

Lambertsen (1971) described respiratory regulation well when he said

> "The respiratory act is normally more driven by the requirement for elimination of metabolically produced carbon dioxide than for uptake of the oxygen to sustain life. Ordinarily, oxygen uptake occurs as an incidental consequence of the respiratory elimination of carbon dioxide" (pg. 2).

When the partial pressure of carbon dioxide begins to rise above 50 mmHg, acidosis caused by the carbon dioxide permeating the blood-brain-barrier and reacting with water stimulates the chemosensitive area of the central respiratory system located just below the medulla (Hall, 2016). The stimulated expiration then releases carbon dioxide, and subsequent inspiration draws oxygen into the lungs. The majority of respiratory regulation follows this process, and in the normobaric air gas composition setting, it does well to maintain homeostasis. However, in hypobaric or hypoxic settings, where carbon dioxide levels are maintained within normal limits and hypoxic isocapnic conditions are present, the body must resort to the peripheral chemoreceptor system to maintain adequate oxygen levels

The peripheral chemoreceptor system is composed of oxygen sensing afferent nerves at both the carotid and aortic bodies. These bodies have been frequently studied, and their denervation has been found to directly correlate with an insufficient physiological response to hypoxia (Lahiri, 1974). Current research is examining the process by which oxygen is sensed in these chemoreceptors. What is understood is that potassium channels within the chemoreceptor bodies are inhibited in some way through a transduction process brought on by hypoxia, and

their inhibition causes subsequent depolarization of the cell. Calcium channels are then stimulated to release neurotransmitter and effectively initiate afferent signaling. (Peers & Kemp, 2001). The end result is a system that is predicted to acutely recognize hypoxia and attempt to correct the oxygen insufficiency through respiratory changes, as evidenced clinically by hyperventilation in some patients (Hall, 2016, p. 542).

The central and peripheral systems are important to remember when attempting to identify symptoms of hypoxia, as hypercapnic symptoms will predominate if both are present. As stated above, hypercapnia directly affects the respiratory center, producing the symptoms one would feel during apnea in the normobaric normoxic setting. These symptoms include strong respiratory stimulation, dyspnea, restlessness, faintness, severe headache, and progressive loss of consciousness (Lambersten, 1971). More importantly, hypercapnic symptoms are noticeable to the individual, and are severely apparent in acute cases. In contrast, hypoxic symptoms are much more subtle. Stimulation of peripheral chemoreceptors only noticeably affects respiratory drive at arterial oxygen partial pressures below 60 mmHg, which can be correlated to a 92% oxygen saturation reading. Beyond this point hemoglobin's oxygen dissociation increases exponentially, giving rise to severe hypoxia in a short time frame. Studies looking at symptom recognition vary from 75.9% to 31% successful symptom identification depending on the training and mode of hypoxic exposure (Cable, 2003; Files, Webb, & Pilmanis, 2005). Overall, the body's respiratory regulation favors carbon dioxide recognition as its primary stimulus, and individuals at risk of isocapnic or hypobaric hypoxia need to be aware of the discreet symptoms that indicate inadequate oxygenation.

It is also important to understand that both of these physiological systems work in unison with (and sometimes counter) each other in the body's attempt to maintain blood gas balance.

Despite the separate presentation, both systems should be looked at as a whole. A primary example of this is the ventilation response during acute hypobaric hypoxia. As oxygen levels drop below 60 mmHg, peripheral chemoreceptors induce an increase in ventilation rate. However, carbon dioxide production remains constant, and the increase in ventilation reduces carbon dioxide tension to below normal levels. This reduction of carbon dioxide tension inhibits the ventilation response, countering the hypoxic ventilation drive. This paradox is important because it attenuates the body's attempt to correct oxygen deficit in this setting.

Hypobaric hypoxia

Hypobaric hypoxia is the most common form of hypoxia, and is generally understood in two categories: acute and chronic. This research primarily focuses on acute hypoxia, in which aviators are the primary population at risk. Gradwell (2016) identified acute hypobaric hypoxia as "generally recognized to be the most serious single physiological hazard during flight" (pg. 50). With aircraft regularly flying above an altitude capable of supplying oxygen demand, the loss of cabin pressure or other supplementary oxygen device is an emergency. Moreover, as altitude increases, the more detrimental decompression becomes and the more rapidly a person experiencing decompression will become hypoxic. The speed at which a person mentally and physically deteriorates, and the loss of the ability to properly function is termed the *time of useful consciousness* (TUC). Hypoxic symptom recognition education and training is then met with the challenge of identifying hypoxia within the window of TUC, before self-correction is no longer possible.

The first problem to address is determining what signs/symptoms a person will experience when hypoxic. Appendix I contains a table outlining hypoxic signs/symptoms experienced during training exercises, and their prevalence among the different groups (Malle et

al., 2013; Smith, 2008; Johnson, Iremonger, Hunt, & Beattie 2012; Woodrow, Webb, & Wier, 2011; Khan, Adil, & Mangi 2013; Artino, Folga, & Vacchiano, 2009). Specific signs/symptoms vary greatly among individuals, however there appears to be a general group of signs/symptoms that present in most every case. Woodrow et al. (2011) claims the most common symptoms are paresthesias, lightheadedness, dizziness, decreased mental agility, and visual changes. As discussed earlier, the brain is highly sensitive to hypoxia, and so it is appropriate that cognitive impairment would be one of the first symptoms to present. Experimental data agrees, and cognitive impairment, confusion, poor concentration, and lightheaded/dizziness have been found to be the first and most widely experienced symptoms. However, the reason for cognitive impairment during hypoxia is not completely understood. A mechanism other than the cessation of oxidative phosphorylation is likely the cause, as impairment happens at an oxygen tension that would not produce the breakdown of primary aerobic pathways (Gladwell, 2016). Khan et al. (2009) identifies the "loss of judgment, self-criticism, and short term memory loss" as the first signs of mental impairment. The fact that mental impairment happens early poses a problem, as a conscious and physically able-bodied pilot may not be able to process information needed for problem solving and self-correction. At the same time, physical impairments arise, resulting in loss of fine motor skills and coordination. Some patients will exhibit these physical changes first, but remain unaware of the presentation unless a prior hypoxic signature, including these symptoms, has been identified through training.

As the duration of hypoxia progresses the number and severity of symptoms increase (Khan 2009). Increased respiratory stimulation by peripheral chemoreceptors sensing low oxygen levels and can quickly lead to hyperventilation. During hyperventilation, carbon dioxide tension decreases and if the body does not adjust for this, the decrease in carbon dioxide tension

will result in hypocapnia. Hypocapnia has its own signs/symptoms apart from hypoxia, but because of how the two are interconnected with hypobaric hypoxia, they will be treated as a continuation of progressive hypoxic symptoms. Neurologically, hypocapnia results in cerebral vasoconstriction and a subsequent drop in oxygen tension (Gladwell, 2016). This process adds to the already deteriorating mental capacity of the hypoxic patient. However, in severe hypoxia, hypoxic ventilation drive outweighs hypocapnic vasoconstriction and causes an overall increase in cerebral blood flow (Gladwell, 2016) Hypocapnia also causes paresthesia/tingling in the distal extremities, along with tremors and muscle twitching. The signs/symptoms commonly reported in hypoxic training courses, along with the data that shows the percentages of symptomatic populations can be found in Appendix I.

Severe hypoxia is normally a continuation and attenuation of moderate hypoxic symptoms. Kahn et al. (2009) concluded from hypobaric chamber testing that euphoria was the majority manifestation before closure of TUC. Gladwell (2016) describes the presents of sudden unconsciousness followed by myoclonic jerks, starting in the upper extremity and progressing to full body convulsions, as some of the final symptoms before death. The TUC window has been closed at this point, and the individual is solely relying on the actions of a third party to restore adequate oxygen supply.

The second concern when dealing with hypoxia and signs/symptoms recognition is how long an individual has before they are no longer able to self-correct. TUC is the general measurement for determining when self-correction is no longer possible. There is no single TUC for hypoxia, as altitude, temperature, and individual physiological factors play a large role in altering that time for each individual. Beyond that, if all external and physiological features were controlled, the very determination of TUC is subjective, as cognitive or psychomotor function

may be considered within normal limits by one individual and impaired by another. Appendix I lists the available TUC for the studies reviewed, and it is evident that while the parameters for testing were near identical in some cases, TUC was almost two times longer in duration in some cases than in others. Gradwell (2016) found similar results and attempted to give a reference chart for TUC at different altitudes. The high standard deviation for the values indicate large variation among subjects.

Normobaric Hypoxia

Normobaric hypoxic situations occur when ambient oxygen tension is inadequate to support normal aerobic metabolism despite the gas pressure being within normal levels. This situation doesn't occur very often, and is more limited to the research and training fields as a substitution of traditional hypobaric chambers. However it is worth review, as hypoxic training is now being done with reduced oxygen breathing devices (ROBD) to familiarize at risk populations with personal signs/symptoms (Woodrow et al. 2011). The question then becomes, are the signs/symptoms of hypoxia the same in hypobaric and normobaric settings? Artino et al. (2009) conducted a study reviewing normobaric hypoxia through ROBD, and despite not being the main objective of the study, the data can be compared to experiments in hypobaric hypoxia. The results showed that individuals reported air hunger much more often in the normobaric setting compared to hypobaria, even with adequate flow rate. Feelings of anxiety also appear to be lower in normobaric hypoxia than compared to data from hypobaric studies. Overall, these studies seem to show a consistency between normobaric and hypobaric symptomology. There is disagreement on substituting normobaric hypoxia for hypobaric hypoxia, but the evidence to support the opposing theory focuses on chronic exposure (DiPasquale 2017). Further research is

still needed to determine the effectiveness of normobaria as a standalone replacement for hypobaric hypoxia training.

Symptom Variation, Sensitivity, and Correlation with Oxygen Saturation

The symptomology presented in the reviewed studies presents a clear picture of the current problem of hypoxia recognition in the medical field. The variation and severity of symptoms make a standard symptomology flow sheet unreasonable. Further it should be noted that most of the studies included in this review had the same basic parameters for simulated flight level and preparation protocol. In a non-training situation, the variables are rarely, if ever, going to be the same as those in the test chamber. Therefore comparing symptom progression with the prevalence of the symptoms as reported by the studies should be understood to be unspecific at best. Despite this a few generalizations can be made.

The first is cognitive impairment, memory loss, blurred vision, and dizziness/lightheadedness appear to be reliably sensitive symptoms demonstrating mental impairment from hypoxia. The second is tingling, incoordination, and tremors, appear to be reliably sensitive symptoms demonstrating psychomotor impairment from hypoxia. The third is both mental impairment and psychomotor impairment prevalence, 51% - 81%, and 29.4% - 55% respectively, indicate symptoms from both domains are likely to be seen in every case with similar variables. Lastly Normobaric hypoxia has an increased reporting of shortness of breath, and a decreased reporting of anxiety

With these conclusions, research needs to identify at what oxygen saturation these symptoms occur. By combining a table on oxygen saturation at different altitudes and symptomology at different altitudes, we can get an idea of what symptoms occur at different oxygen saturation levels (Gladwell, 2016) (Appendix II). Research shows that only a slight impairment is present

from 97% - 78%, and all symptoms may go unnoticed. This leaves a window of 78% - 66% for the recognition of reliable symptoms and self-correction.

Conclusions and Recommendations

Identifying hypoxia symptoms, and the sensitivity of those symptoms at determining hypoxia is not an easy task. Hypoxia recognition training is still key to identifying hypoxic insult. Symptoms such as paresthesia and dizziness/lightheadedness show potential in identifying the presence of hypocapnia, which can tell an individual the body has begun to hyperventilate, and oxygen tension is dropping below acceptable values. Reviews of the current understanding of hypoxic symptomology has shown promising results connecting mental/cognitive impairment to the presence of hypoxia. Data suggests a high percentage of individuals experience mental/cognitive impairments early, which could in turn be identified objectively via cognitive testing. Some research is already in the process of exploring this concept, and the King-Devick neurocognitive test has been found can "detect impairment of cognitive performance at a presymptomatic stage of hypoxia" (Stepanek et al, 2013, p. 1021). Similar to the detection of concussions on a sports field, these tests identify subtle signs of impairment otherwise unnoticeable and gives objective measures to base clinical decision making. Further research needs to be done in this area before a standard protocol can be established. As of now the best practice is to; pay close attention to symptom presentation in a hypoxic environment, assuming hypoxia when symptoms become present, and self-correcting using the available oxygen delivery devices.

Limitations

There is extensive data on hypobaric hypoxia symptomology and the prevalence of symptom appearance. This review combined the two to consolidate hypoxia sensitive symptom

data with the current understanding of symptom progression, so more accurate generalizations of hypoxic presentation can be made. The gap in research that still remains is a combination of hypoxic symptom sensitivity directly compared to objective measurements. The limitations of this review is that direct symptom to objective measurement was not able to be done, thus data acquired is only a retrospective approximation. Further experimentation needs to be done in order to have a strong sample size of individual symptoms correlated to objective measure, thus helping to identify patterns of sensitivity.

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Appendix I

	Malle et al. 2013	Smith 2008	Johnson et al. 2012	Woodrow et al. 2011	Khan et al. 2009	Artino et al. 2009	Artino et al. 2009
type of hypoxia	hypobaric hypoxia	hypobaric hypoxia	hypobaric hypoxia	hypobaric hypoxia	hypobaric hypoxia	normobaric hypoxia 30 L/min	normobaric hypoxia 50 L/min
age (years)	23.9 ± 1.7	33.7 ± 7.5	average 31.6 (21-51 range)	31-40			
n participants	57	49	26	1123	104	121	156
altitude (meters)	10,000	7620	7620	7620	7620	7620 (simulated)	7620 (simulated)
Duration	156s ± 7s	3 mins	max of 4 mins	5-10 mins	5 mins		
Oxygen saturation (Sp02 %)	79.2 ± 1.0	60-70		60-70			
heart rate (bpm)	115 ± 4			increased HR/palpitation 14.9%	palpitations 3.8%		
Time of Useful Consciousness	156s ± 7s			259s ± 43s	5.182 mean		
working memory	Significant decrease ($p < 0.001$)	57%					
cognitive function		58%	81%				
poor concentration		73%				52%	55%
confusion		67%		38.7%	4.8%		
making mistakes		57%					
drowsiness		35%	37%		21.1%		
psychomotor impairment		55%		29.4%			

slow response		67%					
incoordination		55%	46%		2.9%	25%	24%
tremors		43%	58%	11.4% (tetany)			
visual changes		54%	88%	40.4% (visual impairment)			
reduced color intensity		59%			1%		
reduced light intensity		57%				16%	20%
blurred vision		45%			10.6%	26%	35%
psychological disturbance		40%		27.3%			
anxiety		51%		21.1%		6%	5%
depression		35%					
euphoria		33%		24.4%	2.9%	14%	19%
nonspecific		50%	12%	52% (hypoxic symptoms)			
dizziness			81%	58.7%		42%	47%
lightheaded			81%	72.3%			
tingling			38%	45.2%	12.5%	36%	35%
air hunger				32.6%	2.9%	59.4%	44.2%
cyanosis				17%	4.8%		
fatigue				19.8%	15.4%	13%	11%
headache			15%	15.9%	11.5%	9%	13%
hot/cold flashes			65%	35.7%	8.7%	22%	20%
muscle weakness				13%	18.3%		
nausea				17.5%	11.5%	7%	9%
numbness				23%	5.8%		
increased respirations				17.3%			

Appendix II

Altitude (feet)	Oxygen Saturation (%)	Symptoms
Up to 10,000	97 - 90	No signs/symptoms. Novel tasks may be impaired
10,000 – 15, 000	90 - 78	Few signs/symptoms. skilled task impaired, but unnoticed. Prolonged exposure can cause headache
15,000 – 20,000	78 - 66	Signs/symptoms present. Mental impairment, loss of critical judgement, psychomotor grossly impaired, emotional changes, lightheaded, visual changes, paresthesia's, tetany, cyanosis
Above 20,000	66 -	Attenuation of above signs/symptoms. cognitive function rapidly declines, LOC without warning, myoclonic jerks, convulsions, death