Thermal Regulation of Emergency Oxygen Supplies in Commercial Space Vehicles

James M. Pattarini; Rebecca S. Blue; David J. Alexander

INTRODUCTION: While NASA requires that commercial spaceflight vehicles provide onboard emergency oxygen supplies for crew, there are currently no requirements in place regarding thermal constraints of delivered gas. The question has been raised whether or not onboard emergency oxygen supplies must be warmed prior to administration to the crew, as inclusion of warming capabilities will increase the complexity and mass of life support systems in the vehicle. We sought to identify the risk of various inhaled oxygen temperatures and resultant pulmonary inflammatory response in potentially injured crewmembers.

METHODS: A systematic review of published literature was conducted concerning thermal regulation of inhaled gases, reactive airway response, and inflammatory reactions. In particular, we sought literature that correlated inhaled gas temperature to airway response to identify a temperature threshold that would avoid deleterious sequelae.

RESULTS: Cold air inhalation can induce acute bronchoconstriction, increased respiratory rate, and associated dyspnea and hypoxia. Physiological response to cold air varies between healthy lungs and injured tissues, and increased inflammation is associated with increasing airway reactivity. Most studies suggest that inhaled gas temperatures below 10°C may induce deleterious physiological sequelae.

DISCUSSION: Best practices would include maintenance of inhaled gas temperatures to >10°C to avoid poor physiological response, preferably as close to physiological norms as possible. Given that inhaled gas temperature may be altered by transit through an oxygen delivery system, measurement of actual delivered gas temperature should occur at the point of crewmember inhalation.

KEYWORDS: cold, bronchospasm, respiratory distress, astronaut, commercial spaceflight, environmental control and life support systems, oxygen system, space vehicle.

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Spaceflight vehicle design limitations often arise from restrictions on vehicle lift capabilities; increasing complexity and mass of onboard systems can render a vehicle impractical. NASA seeks to provide contracted commercial industry providers with minimum recommendations and requirements for the approval of a commercial vehicle for the transport of NASA astronauts and spaceflight participants; this effort must avoid overly constraining vehicular design while ensuring the health and safety of onboard occupants.[38] This struggle for balance has been apparent in the discussion of regulating onboard emergency life support systems, such as emergency oxygen (O_2) supplies. While NASA requires that commercial spaceflight vehicles provide onboard emergency O_2 supplies for crew use, there are currently no requirements in place regarding thermal control of these O_2 sources. Recently, the question has been raised whether or not onboard emergency O_2 supplies must be warmed prior to administration to the crew, as inclusion of warming capabilities will increase the complexity and mass of life support systems in the vehicle.

Pulmonary injury during spaceflight is a potential risk related to numerous etiologies, including (though not limited to) inhalation injury from noxious gases, fire and combustion

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products, or inhaled foreign body debris. Astronaut exposure to such inhalants may lead to pulmonary injury, inflammation, and resultant respiratory function deficits. Medical standard of care for such injuries commonly prompts the administration of supplemental O_2. Similarly, systemic injury or illness, such as cardiovascular events or acute infection, may warrant the administration of supplemental O_2. In general, administration of emergency O_2 resources is most likely to occur in medical or vehicular contingency scenarios for limited time periods while emergency procedures are executed.

For context, U.S. military airframes employ onboard oxygen generation systems for high-altitude and high-performance flight and emergency scenarios. These systems generally include a heat exchanger that warms oxygen supplies to >10°C at the point of hose entry.[33] Military standards documents indicate broader guidelines, with allowable breathing gas temperatures ranging from −20°C to +10°C[34] and provisions for modified masks to increase inhaled gas humidity and reduce reactive airway responses.[28] Aside from these reports, there is limited public documentation regarding use of variable temperature regulation for onboard nominal or contingency O_2 systems in current or historical aerospace vehicles and, as a result, little aerospace medical precedent for this issue. For example, documentation regarding the U.S. operating system aboard the International Space Station provides operating parameters for nominal and contingency gas supply specific to tank pressure and partial pressure of delivered gas, with some degree of passive rewarming expected between the storage tanks and point of delivery.[31] However, documentation does not directly specify normal thermal parameters of delivered gas at hardline tie-in points for medical use.[31]

Here we sought to identify the risk of providing nonwarmed supplemental O_2 to crewmembers in an emergency spaceflight scenario. In particular, we applied current terrestrial medical knowledge on pulmonary response to cold air stress, particularly in the injured crewmember, to identify the relative risk of various inhaled air temperatures and resultant pulmonary inflammatory response.

METHODS

A systematic review was conducted on currently available information and published literature of human and animal studies involving exposure to cooled air. Search terms included "cold," "hypothermic," "supplemental," "self-contained breathing apparatus (SCBA)," "self-contained underwater breathing apparatus (scuba)," "forced expiratory volume (FEV_1)," "forced vital capacity (FVC)," "pulmonary," "reactive airway," "asthma," "COPD" (chronic obstructive pulmonary disease), "bronchoconstriction," "air temperature," "dyspnea," "inflammation," "inhalation injury," "exertion," "pulmonary edema," and similar. Databases included Ovid, Medline, Web of Science, and the Defense Technical Information Center. NASA archives were searched for the same criteria. Titles obtained from these search criteria were reviewed for relevance. Studies published in a language other than English without available translation were discarded. Articles regarding pulmonary function in populations that are not directly relevant to astronaut populations (such as pediatric studies) were discarded. Studies that identified pulmonary response to cold air, reactive airway processes, or inhalational injuries were reviewed in their entirety. References of all reviewed manuscripts were also searched to identify additional applicable studies. Both animal and human studies were considered for inclusion. The most relevant studies that matched these criteria and the intent of the analysis were selected and presented below. Given the consideration of emergency O_2 provisions, it was assumed, for the purposes of this discussion, that O_2 delivery would be high flow and concentration and that provision would be limited to a short period of time during an emergency protocol, such as vehicle evacuation or atmospheric scrubbing.

RESULTS

It is well established within medical literature that cold air can induce acute bronchoconstriction, increased respiratory rate, and associated dyspnea and hypoxia. Much of this physiological response is driven by respiratory mucosal heat loss rather than the temperature of the gas itself,[9,15] though gas temperature drives convective thermal losses. In bronchial provocation testing for reactive airway diseases, patients are exposed to cold air ranging from −25°C to −10°C to induce reactive airway symptoms.[13,26,27] Elevation of inhaled air temperature from −10°C to +19°C has demonstrated a significant improvement in the pulmonary inflammatory reaction, with resolution of the clinical symptoms of the reactive airway response.[18,25] Thus, there is a direct relationship between air temperature and pulmonary function.

Tolerable temperatures for inhaled gas vary dramatically by the presence or absence of preexisting pulmonary injury, as healthy lungs respond differently to cold air inhalation than injured mucosa. For example, one study demonstrated that 10–19% of healthy, uninjured athletes developed dyspnea and mild-to-moderate reactive airway disease during exertion in cold air; in contrast, a similar study found that 78–82% of individuals with a prior history of asthma demonstrated acute dyspnea when exposed to similar conditions.[22,25] The inflammatory responses of asthma and other reactive airway diseases are similar in mechanism to acute inhalation injury,[16,23] suggesting that an inhalation injury in crewmembers would make them highly susceptible to acute dyspnea and poor respiratory response to cold air. In addition, historical studies on military divers even in a warm environment (30°C) noted a significant drop in core body temperature with the inhalation of oxygen at 5°C with associated respiratory distress and operational decline.[5,14,35] Further studies have demonstrated that thermoregulatory responses driven by peripheral thermoreceptors may not effectively compensate for thermal losses from respiratory mucosa.[29] These studies identify a risk of core body temperature drop and

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operational decline even in healthy, uninjured individuals with cold gas exposure; additional injuries would compound this risk.

The physiological stimulus of facial exposure to ambient temperatures of −5°C to −20°C has been shown in some studies to trigger an immediate bronchorestriction, regardless of preexisting reactive airway disease. Even in healthy individuals, FEV_1 can decline by as much as 3–10%.[11,19,21] In the injured or impaired lung, the cumulative effects of injury and cold air compounding pulmonary capacity decline and bronchoconstriction may be enough to cause clinical symptoms of dyspnea and hypoxia.[19,20] In addition, alteration of the physiological mechanisms of ventilation due to injury or exertion can cause an acute change of respiratory status in cold exposure. Under normal conditions, humans preferentially breathe through the nose; exposure to the nasopharynx allows for rewarming and humidification of inhaled air before it reaches the pulmonary tissues. However, after injury, during exertion, or under conditions of forced air exposure (such as positive pressure breathing), there is a shift from nasal breathing to nose-and-mouth breathing patterns, particularly when ventilation is $>30\ L \cdot min^{-1}$.[2,6,18] This increase in respiratory rate and transition to nose-and-mouth inhalation would be expected following an inhalation injury, particularly when the injured crewmember is under stress or exertion (as would be expected in most emergency scenarios).

A potent mechanism of cold-induced bronchoconstriction is the interaction between increased respiratory rate and the action of increased minute ventilation of cold and dry air on the mucosal tissue.[3] Inhalation of cold, dry air, particularly at high respiratory rate and under nose-to-mouth ventilation, increases drying of airway surfactant and the release of inflammatory mediators.[10,16,32] Dehydration of lung mucosal surfaces increases surface inflammation and subsequently leads to worsened tissue edema, tighter airways, and worsened symptoms of reactivity and dyspnea.[8,10,23] In contrast, raising air temperature to 15–19°C improves dehydration, surfactant and mucosal water loss, and resultant pulmonary function even during high respiratory demand.[18,25]

It is worth noting that injurious response to cold gas exposure is not limited to illness or injuries associated with pulmonary tissue. Studies have demonstrated that systemic illnesses, such as sepsis, without pulmonary injury patterns can be worsened with exposure to cold air during treatment with supplemental O_2.[17] Cold-induced molecular processes may lead to pulmonary edema and acute lung injury patterns independent of preexisting pulmonary injury or inflammation.[1,36,37] Further, exposure to even mildly hypothermic body conditions (30–33°C) has been associated with impaired immune response and increased susceptibility to respiratory infection.[4,12,30] As inhalation of cold air is associated with core temperature decline,[5,14,35] ill or injured crewmembers, even in the absence of lung injury, may experience injury or worsening of their clinical condition if exposed to supplemental O_2 at cold temperatures.

DISCUSSION

Depending on the initial conditions and assumptions, the tolerability of inhaled gas temperatures may vary dramatically. Previous medical literature demonstrates that physiological response to cold air varies significantly in healthy lungs compared to injured tissues, and increased inflammation is associated with increasing airway reactivity. Thus, recommendations for inhaled gas temperature will be more conservative for injured crewmembers compared to healthy individuals. Crewmembers with inhalation injuries and impaired ventilation are likely to be further injured with exposure to cold gases; however, evidence suggests that even ill crewmembers without pulmonary injury may be at higher risk of worsening condition and acute lung injury if exposed to hypothermic gas conditions. Thus, exposure to cold gas from supplemental oxygen or any other source would be contrary to best medical practice and standard of care.

In spaceflight operations, toxic inhalation has been recognized as one of the "worst-case scenarios" for pulmonary injury that may occur during spaceflight, with emergency procedures well established for crew response. The purpose of any toxic atmosphere emergency response protocol is to mitigate injury to the crew and to facilitate a safe haven for exposed crewmembers during the limited period of time the toxic agent is being scrubbed from the cabin atmosphere. In the event of a toxic inhalation, airway inflammation and bronchoconstriction or spasm should be expected to contribute to coughing, which in turn disrupts the protective seal provided by an emergency mask at high flow. If mask effectiveness is lost in the setting of a contaminated cabin atmosphere, this could easily place the affected crewmember at risk for further toxic inhalation and injury. High-flow cold breathing gases could additionally serve as a bronchoprovocative irritant, and may independently exacerbate coughing, bronchoconstriction, and bronchospasm in affected crewmembers following toxic inhalation. Thus, inappropriately cooled emergency gas supplies may exacerbate symptomatology or risk of worsened inhalation injury if cold-induced bronchospasm inadvertently leads to mask compromise.

Given that rapid functional decline has been identified at exposure to cold air at 5°C and below even during exposures of limited time, maintenance of emergency gas temperature above this level would clearly be recommended. Inhaled gas temperatures ≥10°C would further reduce dehydration and surfactant loss. Both research and clinical experience demonstrates that gas temperatures that approach physiological norms (i.e., room-temperature air) are associated with improved pulmonary response. Thus, at a minimum, best practices would include warming of emergency gases to >10°C, similar to thermal control parameters in high-performance military aircraft,[33] and preferably as close to normal body temperature (37°C) as possible.

It is worth noting that significant passive warming is expected to occur between any gas storage tank and the point of delivery in a crewmember's facemask due to exposure to tubing and mask dead-space. Previous studies on cold-temperature

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underwater diving evaluated the relative temperature of gas tanks, ambient water, and air delivered to a diver's regulator and found that, as long as ambient water temperature remained above 3°C, air delivered to the regulator would remain above freezing regardless of the temperature of the dive tank (Clarke J. How cold can scuba regulators become? Oral presentation at TekDive 2014; 17–18 May, 2014; Miami, FL; 2014),[7,24] demonstrating the passive warming of gases as they pass through water-immersed umbilical delivery systems. While temperature conduction in water varies from that of air, a warmed crew compartment may similarly allow some passive warming of O_2 delivered to the crewmember. Thus, storage supply tank temperatures may be lower and still achieve target temperatures at the point of inhalation. Preferably, gas temperature at mask-level should be ≥10°C and as close to physiological norms as reasonably achievable. Measurement of actual delivered gas temperature at the point of inhalation of current and upcoming vehicular systems may add significant value to this discussion and provide improved understanding of the actual risk posed by supplemental O_2 delivery to crewmembers in an emergency.

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