APPROXIMATE SIMULATION OF ACUTE HYPOBARIC HYPOXIA WITH NORMOBARIC HYPOXIA

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INTRODUCTION. Some manufacturers of reduced oxygen (O₂) breathing devices claim a comparable hypobaric hypoxia (HH) training experience by providing F₁ O₂ < 0.209 at or near sea level pressure to match the ambient O₂ partial pressure (iso-pO₂) of the target altitude. METHODS. Literature from investigators and manufacturers indicate that these devices may not properly account for the 47 mmHg of water vapor partial pressure that reduces the inspired partial pressure of O₂ (PIO₂). Nor do they account for the complex reality of alveolar gas composition as defined by the Alveolar Gas Equation. In essence, by providing iso-pO₂ conditions for normobaric hypoxia (NH) as for HH exposures the devices ignore PₐO₂ and PₐCO₂ as more direct agents to induce signs and symptoms of hypoxia during acute training exposures. RESULTS. There is not a sufficient integrated physiological understanding of the determinants of PₐO₂ and PₐCO₂ under acute NH and HH given the same hypoxic pO₂ to claim a device that provides isohypoxia. Isohypoxia is defined as the same distribution of hypoxia signs and symptoms under any circumstances of equivalent hypoxic dose, and hypoxic pO₂ is an incomplete hypoxic dose. Some devices that claim an equivalent HH experience under NH conditions significantly overestimate the HH condition, especially when simulating altitudes above 10,000 feet (3,048 m). CONCLUSIONS. At best, the claim should be that the devices provide an approximate HH experience since they only duplicate the ambient pO₂ at sea level as at altitude (iso-pO₂ machines). An approach to reduce the overestimation is to at least provide machines that create the same PIO₂ (iso-PIO₂ machines) conditions at sea level as at the target altitude, a simple software upgrade.

Learning Objectives:

1. Applying basic principles of respiratory physiology to the design of reduced oxygen breathing devices.
2. Working toward a better understanding of hypoxia.
INTRODUCTION

Some manufacturers of reduced oxygen (O2) breathing devices claim a comparable hypoxic hyperbaric (HH) training experience by providing FIO2 < 0.209 at or near sea level pressure to match the ambient O2 partial pressure (iso-pO2) of the target altitude. METHODS: Literature from investigators and manufacturers indicate that these devices may not properly account for the 47 mmHg of water vapor partial pressure that reduces the inspired partial pressure of O2 (P02). Nor do they account for the complex reality of alveolar gas composition as defined by the Alveolar Gas Equation. In essence, by providing iso-pO2 conditions for normobaric hypoxia (NH) as for HH, the devices ignore P02 and FIO2 as more direct agents to reduce signs and symptoms of hypoxia during acute training exposures. RESULTS: There is a sufficient integrated physiological understanding of the determinants of P02, PO2, and PCO2 under acute NH and HH given the same hypoxic dose. To claim a device to induce signs and symptoms of hypoxia, ischemia is defined as the same distribution of hypoxic signs and symptoms under any circumstances of equivalent hypoxic dose, and hypoxic pO2 is an incomplete dose. Some devices that claim an equivalent NH experience under NH conditions significantly overestimate the HH condition, especially when simulating altitudes above 10,000 feet (3048 m). CONCLUSIONS: At best, the claim should be that the devices provide an approximate HH experience since they only duplicate the ambient pO2 at sea level as an iso-pO2 machine. An approach to redetermine the overestimation is to at least provide machines that create the same PO2 (iso-pO2 machines) conditions at sea level as at the target altitude, a simple software upgrade.

METHODS

Reduced O2 breathing devices create a normobaric NH (oxygen) exposure by providing an FIO2 < 0.209, breathed either through a mask or within a "hyperbaric" tent.

Some manufacturers claim an equivalent acute hypobaric hypoxic (HH) experience but under NH conditions. This eliminates the need for an expensive hypobaric chamber and the risk of decompression sickness associated with hypobaric exposure, creating a cost-effective hypoxic training niche.

Some devices deliver what they promise; however, there is not a sufficient integrated physiological understanding of the determinants of P02, PO2, and PCO2 under acute NH and HH given the same hypoxic pO2. The consequence captured in the derivation of the Alveolar Gas Equation.

The method to convert test altitude to ambient pressure was never specified, a necessary detail used to determine the operation of these devices. But through analysis, it appears that Eq. 1 is used.

\[ \text{Pbam} (\text{mmHg}) = 268.6 \times (28.8 - 0.65 \times \text{altitude (km)})^{1.166} \]  

\[ \text{Eq} \ 1 \]

The second equation used to determine the equivalent NH conditions at sea level is

\[ \text{NHH} = \text{NH} \times \text{FIO2} \times (\text{P02} \text{at} \text{altitude}) \]  

\[ \text{Eq} \ 2 \]

The difficulty in using the upper curve in Fig. 1 is that when you provide a FIO2 at sea level to match the PO2 at the target altitude you create a PO2 that is greater than the target PO2 at the altitude, a consequence of ignoring pH0.

It is best to provide a FIO2 at sea level defined by the lower curve in Fig. 3. It is at least provides the equivalent PO2 at sea level as at the target altitude, a consequence of ignoring the pH0.

Example: 9.6% FIO2 at an ATA of the display of an iso-pO2 machine would indicate that you are at about 5600 feet altitude with a pO2 of 68.5 mmHg (Eq. 3). But FIO2 at 1.154 is 64.1 mmHg, equivalent to breathing air at 19,700 feet, so an iso-pO2 machine overestimates the simulated hypoxic pO2.

The approximate simulation of acute hypobaric hypoxia with normobaric hypoxia

The transient movement of N2 changes PAN2 at constant PB, so time-dependent PAO2 and PACO2 should be considered in a calculation and possibly different time constants until a new dynamic equilibrium is achieved during a chronic NH or HH exposure.

The Alveolar Gas Equation to demonstrate the inability to accurately reproduce HH training under NH conditions given an example of an acute hyperoxic breathing exposure to 22.0% O2, a P02 of 57.3 mmHg in both conditions, the subtract for all respiratory quotient (RQ) and the use of the FIO2 at sea level as an iso-pO2 machine. There are four primary reasons for this: the arterial and mixed venous O2 are in much more closely related to the NH are solid lines and those for NH (iso-pO2) are dashed lines, all from the Alveolar Gas Equation (Eq. 5).

An accurate application of the Alveolar Gas Equation requires that the inputs to the equation be the expired N2 volume, Vn2, and the alveolar pH, PAO2, and PACO2.

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