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 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 (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 hypoxia (HH) training experience by providing FIO₂ < 0.209, breathed either through a mask or within a “hypoxic tent.” Some manufacturers claim an equivalent acute hypoxic HH (HH) experience but under NH conditions. This eliminates the need for an expensive hypobaric chamber and the risk of decompression sickness associated with hypobaric hypoxia, creating a cost-effective hypoxic training regime.

The devices seem to duplicate the ambient partial pressure of O₂ (iso-pO₂) at sea level as exists at the given altitude.

The difficulty in using the upper curve in Fig. 3 is that when you provide a FIO₂ at sea level to match the pO₂ at the altitude you create a PIO₂ that is greater than the pO₂ at the altitude, a consequence of graining pH₂O.

It follows from Loeppky and our example in Fig. 4 that physiological responses would be different after peripheral and central chemoreceptor responses are integrated within the central nervous system.

Even if the Airway Gas Equation was used in reduced O₂ breathing devices one must account for the complex time-dependent role that CO₂ has in modifying FIO₂ and PIO₂ under a particular hypoxic condition.

An accurate application of the Airway Gas Equation requires that the inspired and expired FIO₂ and the expected N₅₀ value:

\[ V_{A} = V_{I} + F_{I}N_{2} = F_{I}N_{2} = 0 \]

Eq 6

Applying the Airway Gas Equation demonstrates the inability to accurately reproduce HH under NH conditions given an example of an acute hypoxic training exposure to 22.0% O₂ (Fig. 2). FIO₂ at 1.1 atm is 64.1 mmHg, equivalent to breathing air at 19.700 ft, so an iso-pO₂ machine overestimates the simulated pO₂ by 1,800 ft. This is a consequence of ignoring the contribution of pH₂O.

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