Physiological Principles of Hyperbaric Oxygenation

E. M. Camporesi, M. F. Mascia and S. R. Thom

1 Professor and Chair, Department of Anaesthesiology, Professor of Physiology, SUNY Health Science Center, Syracuse, NY 13210, USA
2 Assistant Professor of Anaesthesiology and Critical Care SUNY Health Science Center, Syracuse, NY 13210, USA
3 Assistant Professor of Emergency Medicine University of Pennsylvania Philadelphia, PA 19104, USA

Introduction

Hyperbaric oxygen therapy (HBO) entails exposure of the whole body to increased atmospheric pressure usually between 2 and 3 atmospheres (atm abs) while breathing pure oxygen or oxygen-enriched gas mixtures. The therapeutic use of 100% O₂ at pressures exceeding 3 atm abs is limited, because it is frequently accompanied by rapid onset, in minutes, of neurological symptoms, leading to seizures and loss of consciousness. Neurological toxicity sharply limits the upper limits of inspired oxygen partial pressures to 2000 mmHg. The therapeutic use of exposure pressures higher than 3 atm abs, while breathing helium-oxygen (Heliox) or nitrogen-oxygen (Nitrox) mixtures, does not appear to have significant clinical advantages over the exposure of 3 atm abs. A low limit for therapeutic oxygenation is exposure to oxygen-enriched air at 1 atm abs (ambient pressure), as is routinely used in the hospital setting. Clinical experience has shown that significant therapeutic results appear at 1.6 – 1.8 atm abs oxygen.

Several disease states have been shown to respond to either primary or adjuvant HBO therapy including intravascular gas, or bubble-mediated diseases, toxicosis (CO, CN), acute and chronic infections, as well as acute and chronic ischemic processes. The availability of low-cost monoplace chambers has made hyperbaric therapy accessible and affordable in many clinical settings. Thus, hyperbaric oxygenation will continue to be a growing area of medical practice requiring practical research. Modern HBO therapy is based on precise and evolving physiological principles. The goal of this chapter is to explain the rationale for the indications and limitations of HBO.

Alveolar Gases at Different Environmental Pressures

The physiological principles of normal oxygen transport and gas diffusion at 1 atm abs can be extended to understand the rational basis of oxygen transport.
under hyperbaric conditions. Under normal barometric conditions at sea level (1 atm abs), air is inhaled into the lungs where it mixes with the normally present water vapor and other alveolar gases. The oxygen-transport chain thus begins with a pressure gradient that continues from the lungs to the cells. If one ignores trace atmospheric gases, the alveolar partial pressure of oxygen \( (P_{A02}) \) can be calculated from the alveolar gas equation:

\[
P_{A02} = (P_b - P_{H2O}) \cdot F_{iO2} - P_{AC02} \cdot \left( F_{iCO2} \cdot \frac{(1 - F_{iO2})}{R} \right),
\]

where \( P_b \) is atmospheric barometric pressure (760 mm Hg at sea level). Alveolar partial pressure of \( CO_2 \) \( (P_{AC02}) \) is assumed to be equal to arterial \( CO_2 \) \( (P_{aC02}) \), because carbon dioxide readily diffuses through the lung parenchyma. \( R \) indicates the respiratory quotient, i.e., the ratio of \( CO_2 \) produced by the body to \( O_2 \) consumed, in moles or in volumes of gas evolved, at standard conditions (STPD). A typical healthy 70 kg adult male produces 200 ml · min\(^{-1}\) of \( CO_2 \) and requires 250 ml · min\(^{-1}\) of oxygen. \( R \) is then expressed as a dimensionless ratio, and at rest it amounts to:

\[
R = \frac{CO_2 \, \text{production}}{O_2 \, \text{consumption}} = \frac{200 \, \text{ml} \cdot \text{min}^{-1}}{250 \, \text{ml} \cdot \text{min}^{-1}} = 0.8.
\]

\( F_{iO2} \) and \( F_{iCO2} \) are the fractional concentrations of inspired (1) oxygen and carbon dioxide respectively. Assuming that our patient is not rebreathing his expired gas, \( F_{iCO2} \) can be assumed to be zero. \( P_{H2O} \) is water-vapor pressure within the lung, which is 47 mm Hg at the normal body temperature of 37°C. This value is not altered significantly when \( P_b \) increases up to 3 atm abs. \( P_{aC02} \) is usually tightly controlled by the respiratory centers around 40 mm Hg, over the range of 1–3 atm abs. The alveolar gas equation throughout this pressure range can therefore be simplified to:

\[
P_{A02} = (P_b - 47) \cdot F_{iO2} - \left( \frac{P_{AC02}}{0.8} \right).
\]

Based on the above formula, the normal alveolar partial pressure of oxygen at sea level can be calculated to be approximate by 100 mm Hg:

\[
P_{A02} = [(760 - 47) \times 0.21] - \frac{40}{0.8}
\]

\[
= (717 \times 0.21) - 50
\]

\[
= 150.57 - 50
\]

\[
P_{A02} = 100.6.
\]

The same formula can also be applied to environments where \( P_b \) is increased to values larger than 1 atm abs, or hyperbaric environments [1]. Thus, at 3 atm abs, \( P_{A02} \) will calculate exactly as 2183 mm Hg. Effects not taken into account by this