MEDICAL GAS RESEARCH

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Hyperbaric oxygen therapy for traumatic brain injury

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Abstract

Traumatic brain injury (TBI) is a major public health issue. The complexity of TBI has precluded the use of effective therapies. Hyperbaric oxygen therapy (HBOT) has been shown to be neuroprotective in multiple neurological disorders, but its efficacy in the management of TBI remains controversial. This review focuses on HBOT applications within the context of experimental and clinical TBI. We also discuss its potential neuroprotective mechanisms. Early or delayed multiple sessions of low atmospheric pressure HBOT can reduce intracranial pressure, improve mortality, as well as promote neurobehavioral recovery. The complimentary, synergistic actions of HBOT include improved tissue oxygenation and cellular metabolism, anti-apoptotic, and anti-inflammatory mechanisms. Thus HBOT may serve as a promising neuroprotective strategy that when combined with other therapeutic targets for TBI patients which could improve long-term outcomes.

Keywords: intracranial pressure, metabolism, apoptosis, inflammation, tissue oxygenation, cerebral blood flow

Introduction

Hyperbaric oxygen therapy (HBOT) is a treatment by which 100% oxygen is administered to a patient at a pressure greater than atmospheric pressure at sea level (i.e. one atmosphere absolute, ATA) [1]. The increased partial pressure of oxygen (pO_2) within the blood and subsequent improved mitochondrial metabolism/tissue oxygenation constitutes the net effect of HBOT [2-6]. Given that the dissolved oxygen content in the plasma increases linearly after hemoglobin is 100% saturated [7,8], plasma bound oxygen can be used more readily than that bound to hemoglobin which enables tissue oxygen delivery even in the absence of red blood cells [7,9].

Thus, HBOT induces a much larger oxygen-carrying capacity in the blood that dramatically increases the driving force of oxygen diffusion to tissues. Although HBOT-induced cerebral vasoconstriction appears to be undesirable within the context of ischemic conditions [10,11] this may not be necessarily deleterious due to increased oxygen availability to injured tissues. HBOT may also counter vasodilation of the capillaries within hypoxic tissues, thereby minimizing collection of extravascular fluids (edema) which ultimately reduces brain vasogenic edema and the ensuing decrease in intracranial pressure (ICP) [5,12-14].

Emerging evidence has shown the neuroprotective effects of HBOT in a range of multiple injuries and/or disorders (Additional file 1, Table S1) [15]. The most common clinical applications include decompression sickness, carbon monoxide poisoning, minimization of radiation therapy induced tissue damage and enhancing skin grafts [1,16], which are all covered by insurance/Medicare. There are numerous "unapproved" uses of HBOT that focus on more complex neurological disorders, including autism, multiple sclerosis and stroke, which have shown promising results in experimental settings, but clinical efficacy is still elusive. Recent efforts have applied HBOT to traumatic brain injury [5,14,17]. While significant research on HBOT therapy has been undertaken (> 10,000 citations on PubMed), very little has been reported for HBOT within the setting of TBI (< 30 citations). We now briefly review the experimental and clinical HBO research relevant to TBI.

HBOT in animal models of TBI

Early experimental research focused on the effects of HBO on brain edema, ICP and cerebral blood flow

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