Hyperbaric Oxygenation

Subjects: Others
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Hyperbaric oxygenation (HBO) denotes breathing of 100% oxygen under elevated ambient pressure.It may be used in the therapy of burns.

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1. History of Hyperbaric Oxygenation

In 1662, Henshaw, a British physician first utilized hyperbaric therapy, placing patients in a steel container that was pressurized with air. Though John Priestly discovered oxygen as soon as 1775, the marginally effective compressed air therapy was only cautiously replaced by breathing of 100% oxygen under increased ambient pressure, thus initiating “hyperbaric oxygenation”. The reason for the delay was the fear of side effects based on the work of Lavoisier and Seguin who had suspected toxic effects of highly concentrated oxygen in 1789. It took almost 100 years until in 1878 Paul Bert, who is considered the “father of the hyperbaric physiology”, documented the toxic effects of hyperbaric oxygen on the central nervous system that were manifested as seizures [1]. Yet, his findings took time to settle in the hyperbaric medical community. About half a century later in 1937, Behnke and Shaw first used hyperbaric oxygen successfully for the treatment of decompression sickness. In 1955, Churchill-Davidson [2] applied HBO to potentiate the effects of radiation therapy in cancer patients, while at the same time Boerema developed HBO as an adjunct to cardiac surgery, thus prolonging the time for circulatory arrest [3]. Since that time, HBO has been applied for a variety of medical conditions, as the pathophysiological and molecular mechanisms of hyperbaric oxygen treatment were increasingly understood.

2. Principle and Mechanisms of Hyperbaric Oxygenation

HBO denotes breathing of 100% oxygen under elevated ambient pressure between 2 and 3 atmospheres absolute (ATA) in a hyperbaric chamber. In direct correlation to the pressure level, oxygen physically dissolves in the plasma increasing arterial pO_2. At a pressure of 2 ATA oxygen dissolves in the plasma resulting in an arterial pO_2 of about 1400 mmHg, which can be further raised to 2000 mmHg at a pressure of 3 ATA. At 3 ATA, the sheer amount of dissolved oxygen obviates the need for erythrocytes for oxygenation [4]. Additionally, tissue oxygen tensions rise in accordance to arterial oxygen pressure and elevated levels may persist for several hours [5]. However, the mechanism of action of HBO is not mere hyper-oxygenation counteracting tissue hypoxia but is based on the fact that hyperbaric oxygen is a highly potent drug.

HBO redistributes blood flow causing vasoconstriction in regions with increased perfusion and vasodilation in hypoxic ones. On the molecular level HBO effectuates preservation of ATP, downregulation of complex molecular cascades involving ß-2 Integrin and pro-inflammatory cytokines, upregulation of anti-inflammatory cytokines and growth factors as well as mobilization of stem cells. Since microorganisms are unable to compensate for the high levels of oxygen, HBO exerts an unspecific antibacterial action. In addition, a reduction in leukocyte chemotaxis and an increase of phagocytosis enhance the efficiency of antibiotic treatment [6][7][8]. While problems in the middle ear and the nasal sinuses may be encountered during pressurization if there is obstruction due to swelling, side effects of the hyperbaric oxygen (paraesthesia, seizures) are very uncommon, if a pressure of 3 ATA is not exceeded. Even if they occur, they are quickly reversible if hyperbaric oxygen is switched to pressurized air [9].

3. HBO in Burn Injury

The use of HBO in burns was based on a serendipitous finding. In 1965, Japan, Wada and Ikeda [10] applied HBO treatment for severe CO intoxication to a group of coal miners who had also sustained second-degree burns during an explosion. In the HBO-treated miners the burns healed remarkably better than in other victims. Since then, HBO for burns has been dealt with in experimental and clinical trials and in numerous reviews [11][12][13][14][15][16].

When delving into the history of HBO for burn injury reviewing experimental and clinical work, one finds a considerable heterogeneity of study designs, and of injury characteristics such as type, extent, and depth as well as a variety of different species used in experimental settings. Additionally, the dose of HBO deriving from the factors magnitude of pressure, duration of the individual treatment session and total number of sessions varies considerably [17][18][19], as does the interval between the burn injury and the first HBO session. Since downregulation of mediator cascades is most effective if done as early as possible, this timespan has proved to be a crucial parameter in a variety of other indications [14][20][21][22].

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