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Ventilatory support in a hyperbaric environment

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Hyperbaric oxygen, medical conditions and problems, treatment, ventilators

Abstract
As ventilatory support becomes more complex and varied, a good understanding of the changes that can occur in the hyperbaric environment to both the patient and the devices used is vital. Only with this knowledge may patients be managed safely. Unfortunately, few studies have been done so far on the physiological effects of IPPV and other respiratory support in the hyperbaric environment. What is known of these changes is summarised in this paper.

Introduction

A number of patients accepted for treatment with hyperbaric oxygen therapy (HBOT) may require some form of respiratory support. Patients with severe neurological decompression illness, arterial gas emboli, carbon monoxide poisoning and necrotising infections may all require mechanical ventilation. To manage these patients appropriately requires a good understanding of the physiological changes that occur to the respiratory system, and the effects of intermittent positive pressure ventilation (IPPV), positive end expiratory pressure (PEEP), and

continuous positive airway pressure (CPAP) under hyperbaric conditions.

Respiratory changes during hyperbaric therapy

Changes to the respiratory system during HBOT can cause problems. A number of physiologic responses may alter gas exchange and the patient's ventilatory capacity in the chamber may be inadequate. A critically ill patient may have pre-existing pulmonary pathology due to the primary disease process or even ventilator-associated barotrauma. Treatment with high-inspired oxygen can increase sensitivity to the toxic effects of oxygen on the lung under pressure. Barotrauma must be excluded, or managed, prior to compression to avoid disastrous consequences.

Aside from these possible adverse effects to the lung and risks of barotrauma, there are other effects of HBOT:

- suppression of afferent carotid and aortic chemoreceptor activity may result in initial respiratory depression in patients who are not mechanically ventilated;
- later, during treatment, hyperventilation may occur due to raised mixed venous carbon dioxide (CO_2) secondary to decreased binding of CO_2 to reduced haemoglobin;[1]
- increase in oxygen tension resulting in depression of hypoxic ventilatory drive;
- washout of nitrogen leading to absorption atelectasis.

There remains controversy over the amount of alveolar-arterial (A-a) ratio change that occurs. There is evidence that the A-a ratio remains constant, independent of inspired oxygen concentration even at increased barometric pressure.[2] However, other uncontrolled human evidence suggests that patients with lung disease seem to have arterial oxygen tensions (P_aO_2) greater then predicted, whilst patients with normal lungs have P_aO_2's that are lower than predicted when exposed to hyperbaric oxygen.[3,4]

Animal studies have demonstrated increases in pulmonary vascular resistance as well as blunting of hypoxic vasoconstriction.[5] Both may have implications in patients with significant lung pathology. Often areas of ventilation/perfusion mismatch are already present and may worsen during HBOT, resulting in marked increases in the A-a gradient. Some data suggest that, in patients requiring inspired oxygen concentrations of more than 50% for adequate oxygenation in normobaric conditions, the tissue oxygen levels achieved in the hyperbaric environment are inadequate for therapeutic effects.[5]

In patients who are not mechanically ventilated but have limited respiratory reserve, the changes above may contribute to respiratory insufficiency at increased pressure.

Physiologic effects of IPPV

The effects of IPPV have been extensively studied under normobaric conditions.[6] Unfortunately this does not appear to be the case in the hyperbaric environment. Respiratory changes induced by IPPV include increased physiological dead space, decreased functional residual capacity and increased intrathoracic pressure.[6] Cardiovascular changes include decreased cardiac output (decreased preload), increased right ventricular pressures and increased systemic and pulmonary vascular resistance. Endocrine changes include an increase in ADH release.

It is reasonable to extrapolate that the effects of IPPV and HBOT may be additive. In particular, HBOT may cause increased vascular resistance and decreased cardiac output.

Positive end expiratory pressure (PEEP)

Positive end expiratory pressure is the application of greater than ambient pressure, measured in cm of H_2O, applied during the expiratory phase of positive pressure ventilation. PEEP is often required as a method of recruiting and stabilising alveoli in the critically ill and is an important part of ventilatory strategy in the intensive care unit (ICU) setting. In the hyperbaric environment, there is evidence that significant variations in the level of PEEP may occur.

Depending on the amount of pressure and the PEEP valve used, an increase in preset PEEP by up to 4 cm of water at depth has been demonstrated.[7] Two major problems may occur as a result – cardiovascular compromise (by reducing preload), and barotrauma. Recommendations include monitoring of proximal airway pressure, use of adjustable PEEP valves and checking PEEP and readjusting after any change in pressure.[7] There may also be a problem to maintain PEEP with less sophisticated ventilators.

Despite these potential problems, there are a number of physiological reasons why PEEP should be maintained during HBOT. Most particularly, PEEP recruits and stabilises alveoli, preventing areas of collapse, which are potential areas of gas trapping. PEEP also splints open airways to allow more even gas flow and again lessens the likelihood of barotrauma. It has been demonstrated that even short periods without optimal ventilation may result in deterioration that takes up to 24 hours to resolve.[8]

Continuous positive airway pressure (CPAP)

Constant positive airway pressure, which is used with spontaneously breathing patients, is easy to apply in the hyperbaric environment. The physiologic effects are similar to those seen with PEEP. A number of circuits have been tested. A simple system with a high compliance and high volume reservoir coupled with a water valve is easy to achieve.[9] The only precaution necessary is to ensure that air from the compressors is not contaminated with oil, which may cause an adult respiratory distress syndrome (ARDS).

Ventilators

A number of studies have examined the function of various ventilators under hyperbaric conditions. Blanch et al tested

19 ventilators under hyperbaric conditions to assess their function.[10] Adequate mechanical ventilation in the chamber requires a ventilator capable of functioning predictably and safely under hyperbaric conditions.[9]

Changes that occur with increasing pressure affect ventilator function. An increase in chamber pressure may result in reduction in inspiratory flow unless the supply pressure is increased. If this happens, inadequate flow to the ventilator will not maintain adequate ventilation. An increase in pressure also increases the density and viscosity of gases, which decreases the compressibility of gases and decreases the flow rate for a given pressure.

In addition to these changes, one has to consider fire prevention and maintenance of normal oxygen percentage in the chamber atmosphere. Table 1 lists the ideal requirements of a ventilator for use in a hyperbaric chamber.[9]

VOLUME CYCLED VENTILATORS

Regulation from inspiratory to expiratory phase is by tidal volume (V_t). Once the set V_t is reached, the ventilator cycles from inspiratory to expiratory mode. During HBOT the increase in viscosity and resistance results in a decrease in flow, therefore longer time is needed for the desired V_t to be reached. As a result, respiratory rate decreases.

PRESSURE CYCLED VENTILATORS

Inspiratory to expiratory timing is determined by the set pressure being reached. During HBOT at the same passage pressures, the flow is decreased due to increased resistance resulting in a longer time to reach the set pressure. This increases the inspiratory time and again decreases the respiratory rate.

TIME CYCLED VENTILATORS

Time cycling is now the most common form of inspiratory to expiratory cycling used. Unfortunately, it is also the most affected by hyperbaric conditions. After compression, the circuit senses decreased compressibility of the gases and tends to fill and empty in a shorter time, resulting in increased frequency and decreased inspiratory and expiratory time. Decreased inspiratory time and reduced flow cause a decrease in tidal volume. In addition, a decreased expiratory time may result in auto PEEP from 'breath stacking' with a significant risk of barotrauma.

Ventilators in chambers

As a general rule, ventilators used in the chamber are not as sophisticated as those used in the ICU. Ventilation modes such as pressure support, pressure-controlled volume assist, and bilevel positive airway pressure are not readily available outside the ICU. Unfortunately, chambers are usually relatively small in size and space is at a premium. The experience in our unit with the Campbell EV 500 ventilator has recently been reported.[11]

TABLE 1
DESIRABLE FEATURES OF THE HYPERBARIC VENTILATOR

- Small and compact
- No electrical requirement
- No flammable lubricants
- Powered by compressed air or chamber environment gas
- Minimum work of breathing with low gas consumption
- Low oxygen bleed into the chamber to prevent contamination of the atmosphere
- Multiple ventilatory modes
- Stable tidal volume and rate changes with pressure
- Constant PEEP
- Continuous monitoring of tidal volume, frequency, minute volume, peak and mean airway pressure, PEEP, I-E ratio

Modified from Kluger[9]

Intensive care patients who are breathing spontaneously with ventilator support may have high airway pressures and/or high levels of PEEP, and are often difficult to manage. In addition, any move from the intensive care environment and its sophisticated ventilatory strategies may result in significant changes to respiratory parameters. Unfortunately, the deterioration in respiratory function due to any disturbance may last up to 24 hours after transfer back to the intensive care unit.[9,12]

As a result of the difficulties associated with the transfer of patients from the ICU to other environments at the Prince of Wales Hospital, we have devised a transport apparatus that brings the ICU ventilator, on the patient's bed, with the patient to the chamber. This minimizes the time in which a patient's ventilation is altered. In view of these problems and the changes that may occur, an important adjunct to treatment of the ventilated patient is regular arterial blood gas sampling. The safety of the patient depends on close monitoring, especially during the compression and decompression phases of the treatment when there are large changes in pressure.

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