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Isocapnic hyperpnea accelerates carbon monoxide elimination

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Keywords

Animal model, carbon monoxide poisoning, dogs, treatment

Comments

This paper proposes a novel and seemingly effective therapy for CO poisoning, which has numerous advantages over current best practice. However this preliminary work will require trials in spontaneously breathing and ventilated patients to prove its apparent promise. From the details given, the exact construction of their apparatus is unclear, perhaps because they wish to develop its commercial potential.

Introduction

Carbon monoxide (CO) poisoning is an all too common cause of serious or fatal poisoning. Current best therapy consists of administering high concentrations of inspired oxygen at hyperbaric pressures. However, there is often a considerable delay in administering such therapy, due to its limited availability. Whilst CO elimination is enhanced by hyperventilation, the consequent hypocapnia worsens tissue oxygen delivery by shifting the oxyhemoglobin dissociation curve to the left and induces cerebral vasoconstriction, both potentially deleterious in CO poisoning.

Aims

To demonstrate the superior efficacy, over current best therapy, of isocapnic hyperpnea, delivered by a novel ventilatory circuit, in the treatment of CO poisoning.

Methods

Five dogs were anesthetised and mechanically ventilated. Invasive monitoring was established. The dogs were administered air containing 0.28% CO for 70-90 min. Baseline ventilatory parameters were set with a tidal volume (VT) of 16 ml/kg at a frequency of 11 breaths/min and adjusted to maintain $PaCO_2$ below the apneic threshold by manipulation of VT. The subjects were then ventilated successively with room air, then 100% O_2, at baseline settings. They were then subjected to a period of isocapnic hyperpnea (VT = 50 ml/kg at a rate of 24 breaths/min). The settings were maintained for a minimum of 42 min for each section of the protocol. The isocapnic hyperpnea was achieved by adding a non-rebreathing valve to the gas inlet of the ventilator, attached to two gas sources, the first containing 100% O_2, the second containing a PCO_2 approximating that of the oxygenated mixed venous CO_2 pressure. The volume of gas delivered to the circuit from the first source equalled that delivered during baseline ventilation with the remainder coming from the second source. Arterial blood pH, PO_2, PCO_2, HCO_3^- and carboxyhemoglobin levels were measured. Rates of CO elimination were calculated and expressed as half time elimination. Mixed venous PO_2 and cardiac output were also measured.

Results

Elimination half time of CO was dramatically decreased from 212 ? 17 min during room air ventilation at baseline settings, to 42 ? 3 min during 100% O_2 ventilation, and further to 18 ? 2 min during isocapnic hyperventilation with 100% O_2. A further comparison was made by ventilating a further two dogs with hyperbaric oxygen, in whom elimination half time was reduced to 20 and 28 min, respectively.

Discussion

The authors make convincing arguments to defend their methods and conclude that isocapnic hyperventilation may offer a superior therapy to current practice. They emphasise that in human models of CO elimination, an increase in minute volume above 10-15 l/min would be sufficient to achieve the beneficial effects demonstrated in their experiment. However, to offset the deleterious effects of the resulting hypocapnia, supplementation of inspired gas with 5-10% CO_2 is intolerable to spontaneously breathing subjects and exacerbates the metabolic acidosis present in severe poisoning. Rebreathing circuits also fail as they recirculate CO along with CO_2. They argue that their method, which passively maintains isocapnia regardless of the level of hyperpnea, is simple, effective and portable. Indeed the potential benefits of being able to administer this therapy at the scene of a poisoning and/or in transport may improve long-term outcome by the earlier elimination of CO with the consequent improvement in oxygen delivery. In addition, such early therapy may reduce extravascular accumulation of CO. Finally, they argue that the addition of isocapnic hyperventilation to hyperbaric oxygen therapy would improve this intervention still further hence limiting complications of barotrauma and hyperoxia.

References

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