Carbon monoxide poisoning treated with hyperbaric oxygen: metabolic acidosis as a predictor of treatment requirements

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Abstract
A retrospective case note analysis was made of patients who received hyperbaric oxygen for carbon monoxide poisoning and were admitted to the Royal Naval Hospital Haslar between 1991 and 1995. Males predominated (38 v 10) as did cases of deliberate self poisoning (31 v 17). The most common presenting feature was unconsciousness, which is an indication for hyperbaric oxygen and therefore reflects referral patterns.

If patients had not recovered completely after one hyperbaric oxygen treatment further treatments were given. The initial hydrogen ion concentration of those requiring more than one treatment was significantly higher than those who recovered after the first treatment. The initial carbonoxyhaemoglobin (COHb) concentration showed only a trend to being higher in the multiple treatment group. Although metabolic acidosis is well recognised, its relationship to treatment requirements has not been shown previously. Initial COHb does not always correlate well with severity of poisoning which relates to the mechanism of toxicity of carbon monoxide: binding of carbon monoxide to the intracellular oxygen carrying proteins (for example cytochromes) rather than solely to haemoglobin. These findings are consistent with this mechanism and suggests that initial acidosis is a better predictor of treatment requirements and severity than initial COHb.

Keywords: carbon monoxide poisoning; hyperbaric oxygen treatment
Carbon monoxide poisoning (CMP) is a leading cause of death from poisoning in the UK: in 1992 there were 1208 deaths in England and Wales. Treatment with hyperbaric oxygen is recommended in severe cases characterised by neurological and cardiovascular manifestations.

There is evidence, assessed by self-administered questionnaire, that patients who never lose consciousness have a good outcome. Hyperbaric oxygen may be of value in preventing late sequelae (new neurological or cognitive manifestations presenting more than three days after poisoning) and has been used to treat symptoms many days after exposure. The possible reduction in late sequelae may be important because delayed morbidity may go unrecognised after CMP.

Measurement of carboxyhaemoglobin (COHb) concentration is indicated to confirm the diagnosis, but the percentage of COHb in the blood is not always a good indicator of severity. The main impact of the poisoning is on the intracellular oxygen carrying proteins rather than on the blood, therefore it is more logical to seek a marker of tissue poisoning. Poisoning of the cytochrome system leads to cellular dysfunction and metabolic acidosis which has previously been described in carbon monoxide poisoning, but its importance as a marker of severity has never been shown.

Methods
A retrospective analysis of hospital notes was performed. All patients admitted to the Royal Naval Hospital Haslar after treatment with hyperbaric oxygen between 1991 and April 1995 were identified. Some patients given hyperbaric oxygen were returned to the referring hospital without being admitted to Haslar and are therefore not included.

Initial COHb and hydrogen ion concentration ([H⁺]) were ascertained from the casualty notes or laboratory reports from the referring hospital. Plasma salicylate concentrations were recorded when available. At the time of the study patients who had fully recovered after one hyperbaric treatment did not receive any further therapy. If the patient had made an incomplete recovery or if assessment could not be made, due to the continued need for respiratory support and sedation, a further hyperbaric treatment was undertaken. Subsequent treatments were given if the patient had responded to the previous treatment but still had not made a complete recovery. On this basis, the patients were separated into two groups: single and multiple treatments. The null hypotheses are that the multiple treatment group did not have higher COHb or [H⁺] at the time of presentation than the single treatment group. Non-parametric statistics were applied in the form of one tailed Kolmogorov-Smirnoff tests.

Results
Forty eight patients were identified: 14 received multiple treatments and 34 a single treatment. The majority (n=31) of the patients were deliberate self poisonings (caused by running a hose from the exhaust pipe to the interior of the car). The next most common cause was incomplete combustion in a malfunctioning domestic heater (n=12). The vast majority of patients had a history of unconsciousness at some time. Other symptoms included headache, nausea, and drowsiness.

Despite the severity of poisoning 36 patients made a good initial recovery, six improved, and two had serious psychiatric illness, one died, and in two cases the outcome was not clearly recorded.

COHb and initial [H⁺] are shown in figs 1 and 2. Some data were missing. Of the 14 patients requiring multiple treatments, the [H⁺] was available in 13; salicylates were undetectable in 10, minimal in one, and unrecorded in three (only two of whom were acidaemic).

The mean partial pressure of carbon dioxide was 4.62 kPa in the multiple treatment group and 4.53 kPa in the single treatment group (no significant difference). It was not always clear, from the notes from the referring hospital, whether patients were receiving assisted ventilation at the time of the initial arterial blood gas analysis. Of the 34 patients requiring one treatment the [H⁺] was available in 23. There was a trend towards COHb concentrations being higher in the multiple treatment group (p=0.1). The majority of patients in the single treatment group had a [H⁺] in the normal range compared with only 3/13 in the multiple.
The initial $[H^+]$ was significantly higher in the multiple treatment group (p<0.05), thus rejecting this null hypothesis.

**Discussion**

The cause and manifestations of CMP in this group are consistent with previously-reported series from other hyperbaric centres. The key finding is the difference in $[H^+]$ at admission between the single and multiple treatment groups. This is consistent with the mechanism of toxicity of CMP.

Initial $[H^+]$ may be an index of severity of poisoning because of its effect on cellular metabolism due to binding of carbon monoxide to intracellular oxygen carrying proteins, for example cytochromes. Despite the small numbers, a significant association with acidemia on admission and the need for multiple treatments was found and should be investigated further.

Previous studies have found that COHb does not correlate well with severity of poisoning. The reasons include: timing of measurement, rate of elimination, duration of exposure, and concentration of carbon monoxide in the inspired gas. In our patients there was a trend towards COHb being predictive of the need for multiple treatments but statistical significance was not reached.

The need for more than one treatment is only a surrogate measure of severity. The assessment of outcome of patients with CMP, particularly those due to deliberate poisoning, is difficult and may not necessarily be correlated with the severity of poisoning. Outcome may be confounded by premorbid state, cognitive reserve, and the response to treatment. It is therefore equally valid to use the amount of treatment required, so long as indications for repeat treatments are consistent and were not influenced by the initial $[H^+]$. During the period of this study the initial $[H^+]$ was not one of the indications for repeat treatment. A predictor of the amount of treatment needed in itself may be helpful.

The management of CMP is based primarily on the clinical condition of the patient, however some recommend hyperbaric oxygen on the basis of a high COHb concentration at presentation. Acidemia may give an additional measure of severity of poisoning and, in this study, correlated better with the need for multiple treatments than did COHb. More specific measures of cellular dysfunction, such as lactate, should also be considered for further investigation. These data alone do not suggest that patients with an initial metabolic acidosis require multiple treatments, however patients with such an acidosis must be considered to have significant poisoning even if the COHb is low.

**Conclusion**

Initial assessment of the patient with CMP should include a full history and clinical examination, COHb concentration (confirming exposure), and arterial $[H^+]$. Future studies of carbon monoxide poisoning should include data on initial $[H^+]$.

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**British Hyperbaric Association carbon monoxide database, 1993–96**

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