Severe carbon monoxide poisoning: outcome after hyperbaric oxygen therapy

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This paper reports the outcome after carbon monoxide poisoning in 31 consecutive patients treated with mechanical ventilation and hyperbaric oxygen therapy, compared with another study of mechanically ventilated patients treated with normobaric oxygen. We found 16.1% hospital mortality and 3.8% severe short-term memory loss, compared with 30% hospital mortality and 20% incidence of serious neurological deficit after treatment with normobaric oxygen; outcome was poor in 19.4% and 44.3% of those treated with hyperbaric and normobaric oxygen, respectively (P<0.05). Cerebral oedema caused three of five deaths despite hyperbaric therapy, occurring at 24–48 h after poisoning. Intracranial pressure monitoring and CT scan of the head before wakening should be considered in any severely poisoned patient.

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Carbon monoxide poisoning is an important cause of morbidity and mortality. There is no reliable marker of severity of poisoning, with clinicians relying on features such as level of consciousness and cardiac abnormalities. Carbon monoxide combines with haemoglobin to form carboxyhaemoglobin (Hb-CO), normal concentrations of which are approximately 2% in non-smokers and 3–8% in smokers. Acute Hb-CO measurements can confirm exposure, but Hb-CO concentrations do not correlate with severity of poisoning. Hyperbaric oxygen therapy is generally regarded as the treatment of choice for moderate to severe carbon monoxide poisoning¹ but on the UK mainland there are currently only seven hyperbaric units with facilities to treat critically ill patients. The North-West Emergency Recompression Unit (NWERU) is such a unit in the Mersey region.

In 1988, Krantz and colleagues² reviewed the outcome of 79 patients with severe carbon monoxide poisoning requiring mechanical ventilation, who did not receive hyperbaric oxygen therapy. Hospital mortality was 30%, and 20% of survivors were discharged with significant brain damage, often into long-term care after prolonged hospitalization. We report a retrospective study of the current outcome from hyperbaric oxygen therapy, that is, therapy with 100% oxygen at 2.8 atmospheres absolute pressure for 1–1.5 h,³ for severely poisoned patients receiving intensive care.

Methods

We reviewed the case records of 31 consecutive patients requiring mechanical ventilation for severe carbon monoxide poisoning treated at NWERU between November 1995 and January 1999. Information on patients’ clinical state at the time of discharge was obtained from hospitals and, where possible, more long-term information was obtained from general practitioners (GPs).

Results were analysed with a chi-squared test for binomial (good/poor) outcomes. The Mann–Whitney U-test was used to compare Hb-CO and standard bicarbonate concentrations between those with a good or poor outcome.

Results

Seventeen (54.8%) of the 31 patients were poisoned with car exhaust fumes, one unintentionally. Ten patients (32.3%) were involved in house fires and four (12.9%) were poisoned by faulty heating installations. Before hyperbaric oxygen therapy, all patients had been treated with 100% oxygen and were subsequently admitted to an intensive care unit in the Mersey area. Seven patients received only one treatment with hyperbaric oxygen; three of these died before further therapy could be given, while the other four appeared to recover well despite missing further therapy.

Five (16.1%) of the patients died in hospital; three of these had poisoned themselves and two had been involved in house fires (neither had severe burns). One patient died
from multi-system organ failure, two had severe cerebral oedema and one had cerebral oedema and extensive bowel infarction. The exact cause of death could not be determined for the fifth patient. Another patient suffered severe short-term memory loss requiring rehabilitation therapy so that, in all, six patients were considered to have a poor outcome. Of the five who died, three had received prolonged life-sustain measures following cardiorespiratory arrest before treatment with hyperbaric oxygen. The only biochemical difference that was detected between patients with a good outcome and those with a poor outcome was a lower standard bicarbonate concentration ($P<0.005$) in the latter (Table 1).

Patient characteristics were broadly similar to those reported by Krantz and colleagues and there was no difference in standard bicarbonate concentrations (Table 1). There were fewer cases with poor outcome in our survey ($P<0.05$), but the difference in hospital mortality between the two groups was not significant ($P=0.02$).

GPs provided further information about 10 patients. Four patients had some neurological symptoms (gait disturbance, short-term memory loss and fatigue) after their poisoning; all of these patients recovered spontaneously within a few weeks. One patient continued to complain of fatigue, headache and short-term memory problems up to a year later, though these symptoms had been present before the poisoning.

**Discussion**

Controversy persists over the most appropriate treatment for carbon monoxide poisoning. Many patients with mild poisoning make a full recovery whatever the treatment, but approximately 1000 patients die each year in the UK as a result of carbon monoxide poisoning. Even after initial recovery, there is a risk of delayed neurological sequelae. Current criteria for the treatment of carbon monoxide poisoning with hyperbaric oxygen are as follows: carboxyhaemoglobin >20%; loss of consciousness at any stage; neurological signs or symptoms other than headache; myocardial ischaemia or arrhythmia diagnosed by ECG; pregnancy.

All patients in this report required tracheal intubation before transfer for therapy with hyperbaric oxygen; in three cases the incident had been severe enough to cause the death of other casualties at the scene.

Our study supports the findings of Krantz and colleagues, in that the level of Hb-CO was of no prognostic value, though metabolic acidosis on arrival to hospital was greater in those who did badly. Overall, the severity of metabolic acidosis was comparable between the two studies, but outcome was significantly better in patients treated with hyperbaric oxygen. Many of the patients in the report by Krantz and colleagues subsequently needed institutional care owing to the severity of their neurological deficit. This did not happen for any of the patients treated with hyperbaric oxygen.

The difference in hospital mortality between the two groups was not significant, maybe because of differences in the size of groups. If the hyperbaric group size had been twice as large, a 16.1% mortality would have been significantly less (at the 5% level). Even with hyperbaric therapy, some patients developed delayed neurological sequelae. Four of the survivors who could be traced complained to their GP with minor neurological sequelae, namely headache, fatigue, gait disturbance or mild short-term memory loss.

The two studies were conducted at different times, so intensive care treatment was probably not similar. Krantz and colleagues reported a high incidence of hypotension from the treatment used to prevent cerebral oedema, and moderate hypothermia was used for cerebral protection. The patients treated hyperbarically received cardiovascular support, and were warmed to achieve and maintain normal temperature. A more recent study, on animals, reported that hypothermia was associated with a worse outcome in carbon monoxide poisoning. Krantz and colleagues did not report the exact duration of treatment with 100% oxygen, saying only that it was at least 1 h. Current guidelines would recommend 6 h.

The results of this study have led to a change in our practice. Severe cerebral oedema (responsible for three of the five deaths in this study) was an unpredictable problem at 24–48 h, so we now consider monitoring intracranial pressure and/or a doing a CT scan of the head before wakening.

The risks associated with hyperbaric oxygen therapy are well documented. It is common practice to inflate the tracheal tube cuff with saline to avoid compression and expansion of an air-filled cuff with changes in ambient pressure. In one patient, the cuff was overinflated at the referring hospital, resulting in a tracheal mucosal tear; this healed without intervention, but prolonged the patient’s ICU stay.

To obtain maximal benefit, hyperbaric oxygen should be administered within 6 h of a patient’s removal from carbon

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**Table 1.** Summary of patient characteristics and outcome. The results for Hb-CO and standard bicarbonate are shown as range (median). The proportion of patients with poor outcome in the two groups was significantly different ($P<0.05$).

<table>
<thead>
<tr>
<th></th>
<th>Normobaric treatment</th>
<th>Hyperbaric treatment</th>
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</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>79</td>
<td>31</td>
</tr>
<tr>
<td>Age (yr), range (median)</td>
<td>5–91 (49)</td>
<td>21–78 (41)</td>
</tr>
<tr>
<td>Gender, number (%)</td>
<td>Male 33 (42%) Female 46 (58%)</td>
<td>Male 20 (64.5%) Female 11 (35.4%)</td>
</tr>
<tr>
<td>Hb-CO %</td>
<td>‘normal’–63 (32)</td>
<td>12.6–52.1 (40)</td>
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<tr>
<td>Standard HCO3 (mmol litre$^{-1}$)</td>
<td>Good outcome 9.4–25.8 (20.7) Poor outcome 6.8–25.2 (17.7) Poor outcome 35/79 (44.3%)</td>
<td>Good outcome 10.4–23 (19) Poor outcome 6.2–16.4 (11.7) Poor outcome 6/31 (19.4%)</td>
</tr>
</tbody>
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monoxide.\(^{10}\) Although patients were transported over long distances, most (25/31) received treatment within 6 h (maximum interval 8 h 12 min). There was no evidence that the long journeys caused complications.

In conclusion, hyperbaric oxygen treatment of severely poisoned patients appeared to reduce the risk of serious neurological deficit and the need for prolonged care.

References