oxygen. Anecdotal reports of alternative approaches have not been followed up.[10] There is little evidence from clinical studies that normobaric oxygen is effective, and even the evidence concerning hyperbaric oxygen is difficult to interpret despite animal studies and cellular research which provide strong evidence in its favour. One important factor is that it reduces free radical concentrations, and this means that the benefit from hyperbaric oxygen may be far reaching. It provides an explanation why hyperbaric oxygen prevents the delayed manifestations of carbon monoxide poisoning.

The presentation by Hamilton-Farrell of the British Hyperbaric Association's carbon monoxide database (p98) provides an impressive panorama of the use of hyperbaric oxygen in Britain.[11] The indications for referral are clearly set out, and the reasons for delay to referral and delay to arrival in a hyperbaric facility are listed. Many of these can be reduced by making a critical examination at a local level and addressing the deficiencies identified. The rising number of referrals may be a result of increased awareness, since the number of cases of fatal poisoning is falling. It has been suggested that this is due to the introduction of catalytic converters in motor vehicles.[12]

Those working in any branch of emergency medicine would do well to study the articles

presented in this issue of the journal. One certainly wants to avoid the error of telling a patient with undiagnosed carbon monoxide poisoning to "go home and stay warm", with the result that they suffer even worse poisoning.

1 Chief Medical Officer and Chief Nursing Officer. *Carbon monoxide: the forgotten killer.* London: Department of Health, September 1998.
2 Chief Medical Officer. CO poisoning: pulse oximetry. *CMO's update 20.* London: Department of Health, November 1998: 3.
3 Carbon monoxide: health hazards and precautionary measures. *Guidance note EH43.* 2nd Ed. London: Health and Safety Executive, 1998.
4 Smith JS, Brandon S. Morbidity from acute carbon monoxide poisoning at three year follow up. *BMJ* 1973;i:318–21.
5 Chale SN. Carbon monoxide poisoning. In: Vicciello P, ed. *Handbook of medical toxicology.* Boston: Little, Brown 1993: 639–47.
6 Choi IS, Kim SK, Lee SS, *et al.* Evaluation of outcome of delayed neurologic sequelae after carbon monoxide poisoning by technetium 99m hexamethylpropylene amine oxime brain singe photon emission computed tomography. *Eur Neurol* 1995;35:137–42.
7 Kirkpatrick JN. Occult carbon monoxide poisoning. *West J Med* 1987;146:52–6.
8 Turner M, Esaw M, Clark RJ. Carbon monoxide poisoning treated with hyperbaric oxygen: metabolic acidosis as a predictor of treatment requirements. *J Accid Emerg Med* 1999;16:96–8.
9 Turner M, Hamilton-Farrell MR, Clark RJ. Carbon monoxide poisoning: an update. *J Accid Emerg Med* 1999;16: 92–6.
10 Howard RJ, Lake DR, Pall H, *et al.* Allopurinol/N-acetylcysteine for carbon monoxide poisoning. *Lancet* 1987;ii:628–9.
11 Hamilton-Farrell MR. British Hyperbaric Association carbon dioxide database, 1993–96. *J Accid Emerg Med* 1999;16:98–103.
12 Kendell RE. Catalytic converters and prevention of suicide. *Lancet* 1998;352:1525.

Carbon monoxide poisoning: an update

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Carbon monoxide is the major cause of death from poisoning in the UK. In 1996 there were 877 deaths from carbon monoxide poisoning (CMP) in England and Wales; the majority were suicides due to car exhaust fumes.[1]

Causes
The most common causes of CMP are deliberate attempted suicide using car exhaust fumes, domestic heater malfunction, open heating fires, burning buildings, and accidental exposure to car exhaust. Rarer causes include recreational boating,[2] indoor burning of charcoal briquettes,[3] obstruction by snow of vehicle exhaust systems,[4] and the hepatic metabolism of methylene chloride from paint stripper fumes.[5] The British Hyperbaric Association (BHA) CMP database suggests that, for cases managed with hyperbaric oxygen, approximately half are deliberate and half accidental.[5] This contrasts with the mortality figures, with 85% of CMP deaths being due to deliberate poisoning.

Death may be due to cardiac toxicity, neurotoxicity, systemic acidosis, or respiratory arrest.

Pathophysiology
The commonly held belief that CMP is mainly due to binding of haemoglobin was shown to be false over 20 years ago.[6] Goldbaum *et al* demonstrated that exchange transfusion, using a red cell concentrate with a carboxyhaemoglobin (COHb) concentration of 80%, led to indefinite survival in one group of dogs, despite equilibrium COHb concentrations of 60%. The potential to endure a diminished oxygen carrying capacity was demonstrated when other dogs were venesected isovolaemically, reducing mean packed cell volume by 68%, and survived indefinitely. In contrast, dogs in a third group inhaled 13% carbon monoxide for 15 minutes, producing a mean COHb of 65%, and all died. Goldbaum *et al* deduced that the slow rate of carbon monoxide binding by haemoglobin led to blood entering the systemic capillary beds with a

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Box 1: Clinical manifestations of carbon monoxide poisoning

Neurological
- Loss of consciousness
- Neurological and cognitive abnormality
- Gait disturbance

Cardiovascular
- Hypotension
- Cardiac ischaemia
- Arrhythmia

Biochemical
- Carboxyhaemoglobinaemia
- Metabolic acidosis

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significant burden of dissolved carbon monoxide available for diffusion. Conversely, in the exchange transfusion experiment described above, minimal dissolved carbon monoxide had been infused and the carbon monoxide was so tightly bound to haemoglobin that there was little tissue burden. The paper concluded that the toxicity of carbon monoxide is predominantly a direct action on cells, rather than mainly the denial of oxygen carriage by haemoglobin.

The mechanism for tissue damage in CMP is the poisoning of intracellular oxygen carrying haem proteins, such as cytochrome a_3 and myoglobin. This leads to cellular dysfunction and produces the clinical manifestations most rapidly in tissues with high energy requirements (brain and heart). Further injury is caused by radical oxygen species, including nitric oxide, produced locally by neutrophils.[7]

Hyperglycaemia and hypoglycaemia have an adverse effect in rats poisoned with carbon monoxide.[8] Investigation of the brain energy metabolites ATP and phosphocreatine failed to define clearly a mechanism for this finding, although lactate concentrations were lower in the normoglycaemic animals than in those with hyperglycaemia.[9] The role of plasma glucose in humans has not been investigated, however it would seem logical to maintain normoglycaemia in patients.

Ethanol has been shown to modify the severity of poisoning in mice.[10] Survival time was improved in mice pretreated with ethanol. The authors propose that the ethanol reduced blood flow and hence the rate of delivery of carbon monoxide to the tissues, resulting in a lower total body carbon monoxide burden.

In both the acute and chronic phases of CMP, cerebral perfusion has been shown to be abnormal. In the acute phase, large focal defects may occur[11] whereas diffuse abnormality is present in patients with late sequelae.[12] It remains unclear whether abnormal perfusion is a cause of brain injury or the result of other pathological mechanisms. Assessment of cerebral perfusion by isotope imaging has been suggested as a possible indicator of severity of poisoning.[13]

Clinical manifestations

Severe CMP is characterised by neurological and cardiovascular manifestations (box 1). Unconsciousness is the most common neurological manifestation although memory loss, drowsiness, poor cognitive performance, and disturbance of balance or gait (including cerebellar features) may be seen. Less severe cases may have non-specific symptoms including headache, nausea, vomiting, and lethargy. In such cases a careful history should be taken to identify potential exposure to carbon monoxide. It is possible that many mild cases are never diagnosed and there is evidence that much late morbidity from CMP goes unrecognised.[14]

The commonly quoted cherry red discoloration of skin and mucous membranes may be seen at autopsy but is extremely rare in patients presenting with acute CMP.[15] The absence of the cherry red colour does nothing to exclude the diagnosis.[16]

Clinical assessment

A careful history, specifically seeking all of the possible manifestations listed above should be taken. The type of heating in the patient's home should be established. A venous COHb concentration should be performed if there is any suspicion of the diagnosis.

Even in obvious and severe cases, clinical history and examination are most important. Specific features of the history should be sought: cause, duration of exposure, loss of consciousness (even if transient), the possibility of other poisons (cyanide or drugs), and chest pain. A history of possible chronic exposure should be sought in accidental cases, since this may have preceded the acute presentation. Neurological examination should include assessment of cognitive functions and in particular memory; patients should all be asked to stand and walk, if they are able, because balance and gait disturbance may be the only abnormality in the physical examination.

Most patients with severe poisoning will present with a reduced level of consciousness, so bedside blood glucose analysis should be performed immediately to exclude hypoglycaemia, which may complicate CMP or may be the sole cause of unconsciousness. The COHb concentration should be measured in either venous or arterial blood (no significant difference in value is obtained[17]) in all patients with undiagnosed unconsciousness: greater than 10% is diagnostic of CMP in all but the heaviest of tobacco smokers.

Pulse oximetry may give falsely high readings, since COHb absorbs light at an almost identical frequency to that of oxyhaemoglobin: thus the measured value is the sum of oxyhaemoglobin and carboxyhaemoglobin.[18] Arterial blood gases should therefore be measured using a co-oximeter, to assess oxyhaemoglobin and COHb, to confirm adequacy of spontaneous or assisted ventilation, and to measure pH. Measurement of lactate may be helpful, if

available. Venous blood should also be drawn for assays of other toxins and glucose.

Electrocardiography (ECG) should be performed in all cases because ischaemic changes, ST depression, or even ST elevation (myocardial infarction pattern) can occur despite normal coronary arteries.[19] ECG monitoring is important, because the presence of arrhythmias may influence decisions about the wisdom of interhospital transfer, although haemodynamically significant arrhythmia is rare in our experience. If ST elevation is present in acute CMP, thrombolysis is probably not appropriate since the cause is generally myocardial toxicity rather than thrombotic occlusion of an epicardial coronary artery. Reversible biventricular dysfunction, leading to haemodynamic instability, is also described after CMP[20] due to the toxic effect of carbon monoxide on cardiac myocytes.

Treatment, indications for hyperbaric oxygen, and assessment of severity

Carbon monoxide binds more avidly than oxygen to haemoglobin and to tissue haem proteins, but these remain reversible processes. The premise underlying treatment is that removal of carbon monoxide will minimise further damage and reverse cellular metabolic dysfunction. The half life of COHb is variable but typically 270 minutes when breathing air, compared with 90 minutes when breathing 100% oxygen[21] and 25 minutes while breathing oxygen at a pressure of 3 bar. Cardiac output (hence circulation time) is an additional determinant of clearance rate.

Recovery depends on prompt removal from the toxic exposure and institution of effective resuscitation for life threatening cases.

NORMOBARIC OXYGEN TREATMENT

Although there is controversy surrounding hyperbaric oxygen, there is little argument that 100% normobaric oxygen should be administered. There are no studies suggesting a good outcome without any treatment, even in mild cases, therefore it should be considered mandatory to provide oxygen. As soon as the diagnosis is suspected, high concentration oxygen should be delivered via a tight fitting facemask and non-rebreathing reservoir bag, aiming to achieve as high a concentration as possible. In order to provide 100% oxygen, an air filled cushion rimmed facemask, or an endotracheal tube with the cuff inflated, is necessary. If hyperbaric oxygen is not administered, six hours of treatment with 100% oxygen at ambient pressure, as has been compared with hyperbaric oxygen in a randomised trial,[22] is recommended as a minimum. If any symptoms persist, the oxygen should be continued and hyperbaric oxygen should be considered. Before hospital discharge, all patients should be carefully re-examined to ensure that no neurological or cognitive abnormality (which would indicate the need for hyperbaric oxygen) has developed during normobaric oxygen treatment.

HYPERBARIC OXYGEN TREATMENT

Currently accepted indications for hyperbaric oxygen (Guy's Hospital Poisons Unit) are: loss of consciousness (even if transient), neurological or cognitive abnormality, ECG evidence of cardiac ischaemia (ST abnormality), COHb >20%, and pregnancy (as the fetus cannot be assessed and is more susceptible to poisoning than an adult). Although blood COHb measured at the time of admission helps to confirm the diagnosis, it does not always correlate well with severity of poisoning.[23] Both the type of exposure and the timing of measurement, relative to the removal from exposure, contribute to this. A prolonged exposure to relatively low concentrations of carbon monoxide may produce a large tissue load of carbon monoxide with low COHb, whereas a short exposure to a high concentration can produce a low tissue load (less severe) and a high COHb. Despite this it is considered by some an indication for hyperbaric oxygen. Interestingly, of patients in the BHA database, 298 (51.8%) had recorded COHb concentrations exceeding 25%; of these cases all but seven satisfied the criteria for hyperbaric oxygen other than the COHb concentration alone. This included patients in whom neurological abnormalities were not reported at the time of referral for hyperbaric oxygen; abnormalities were either not recognised initially or developed later, in transit. Therefore, despite the lack of specific evidence that arbitrary concentrations of COHb alone are an indication for hyperbaric oxygen, in practice high concentrations of COHb may be an indication that referral is necessary but is rarely the only pretext for treatment.

Hyperbaric oxygen has been shown to reduce the free radical concentration where 100% normobaric oxygen did not.[24] The paradox that higher partial pressures of oxygen reduce oxygen free radical production is explained by the observation that neutrophil adherence to the vascular endothelium mediated by β_2 integrins is reduced by hyperbaric oxygen. The mechanism for this appears to be an effect on the membrane guanylate cyclase of the neutrophil.[25] These data suggest that any benefit from hyperbaric oxygen may not be solely an effect on the half life of carbon monoxide in the blood and tissues, but also a local action in the tissues preventing further injury.

In the absence of a controlled clinical trial directly comparing hyperbaric oxygen with normobaric oxygen this area remains controversial, as discussed in an article published in 1994.[26] Since then, further important data have emerged suggesting that hyperbaric oxygen reduces the incidence of late sequelae[27]: new neurological or cognitive deficits presenting between three and 21 days after poisoning that contribute to the unrecognised chronic morbidity associated with CMP.[14] It had been thought that the outcome, in patients who never lose consciousness at the time of original poisoning, is not changed by hyperbaric oxygen[22]; however, this study compared hyperbaric oxygen and

Box 2: Indications for hyperbaric oxygen
● Loss of consciousness
● Neurological abnormality
● Cardiac arrhythmia/ischaemia
● COHb >20%
● Pregnancy

normobaric oxygen and used a self administered questionnaire as its outcome measure, which may not have been a sensitive enough tool to identify subtle cognitive problems. Recent data suggest that patients who never lose consciousness derive early benefit from hyperbaric oxygen, since they recover more quickly.[28] Until more data become available, we recommend using the guidelines from the National Poisons Unit at Guy's Hospital (box 2).

Unconsciousness is the most common reason for referral for hyperbaric oxygen; 65% of the patients in the BHA database (374 of 575 cases) had a definite history of loss of consciousness.[5] Patients who never lose consciousness and have no other indication for hyperbaric oxygen, despite a thorough clinical assessment, may be treated with normobaric oxygen.[22] General practitioners should be contacted and advised to assess regularly patients in the first three weeks after poisoning, as late sequelae may develop in this period. Late sequelae have been shown to respond to hyperbaric oxygen[29] and therefore patients should be referred for this treatment in this circumstance.

Follow up of 50 accidentally poisoned patients presenting to Whipps Cross Hospital has been undertaken from 1993 to 1996, three months after initial presentation (personal communication, M Hamilton-Farrell). In this group all patients with a history of carbon monoxide exposure for longer than 24 hours had residual symptoms, whereas 75% of those with shorter exposures were asymptomatic. More data will be collected to investigate this observation.

Metabolic acidosis may provide a closer reflection of tissue poisoning and has been shown to be associated with more severe poisoning as judged by treatment requirements and was certainly a better indicator of severity than COHb.[30] More specific markers of cellular dysfunction, such as lactate, should be investigated and may prove helpful, but clinical evaluation remains the most valuable tool for initial assessment of severity.

Brain imaging
The place of imaging investigations has not been established in the acute phase. Brain computed tomography is not indicated before hyperbaric oxygen unless there is doubt about the cause of unconsciousness.

The most common abnormalities noted on computed tomography of the brain are low density lesions in the globus pallidus and deep white matter changes, although these changes do not correlate with outcome or COHb concentrations.[31] A normal tomogram has been correlated with a good outcome,[32] although this does not exclude clinical problems. In the same series, abnormalities on the computed tomography also correlated with a poorer outcome despite hyperbaric oxygen. It remains unclear if early computed tomography has any role in risk stratification. Magnetic resonance imaging (MRI) has also been performed in a small number of patients and has a greater sensitivity for white matter abnormalities.[33] Again insufficient data are available to judge if there is a place for MRI in the acute situation. There is certainly a place for brain imaging if patients fail to make a complete recovery after hyperbaric oxygen, to exclude coexistent pathology.[34]

Prevention
Regular servicing of gas appliances, adequate ventilation, and cleaning of chimneys should prevent domestic exposures. Carbon monoxide sensors are commercially available, but may not detect the low levels that could cause clinical manifestations if repeated or prolonged exposure occurs. They do, however, appear to alert victims to heavy exposures and encourage early presentation.[35] Catalytic converters reduce the carbon monoxide content of exhaust gases and may help to reduce the overall severity of exposures to car exhaust fumes.

Summary
CMP is an important toxicological emergency. The majority of cases present to accident and emergency departments, a few to general practitioners. In some cases the diagnosis is obvious, but in other cases manifestations may be non-specific when a high index of suspicion, a careful history, and venous or arterial COHb are necessary to establish the diagnosis. A thorough clinical assessment, including arterial blood gases and ECG, are necessary to estimate severity of poisoning. The role of imaging investigations is unclear in the acute phase. Computed tomography or MRI of the brain should be considered if patients do not make a complete neurological recovery after hyperbaric oxygen to exclude other pathology.

1 Office for National Statistics. *Mortality statistics cause: England and Wales.* London: HMSO, 1996. Series DH2 No 23.
2 Silvers SM, Hampson NB. Carbon monoxide poisoning among recreational boaters. *JAMA* 1995;274:1614–6.
3 Hampson NB, Kramer CC, Dunford RG, *et al.* Carbon monoxide poisoning from indoor burning of charcoal briquets. *JAMA* 1994;271:52–3.
4 Rao R, Touger M, Gennis P, *et al.* Epidemic of accidental carbon monoxide poisonings caused by snow-obstructed exhaust systems. *Ann Emerg Med* 1997;29:290–2.
5 Hamilton-Farrell MR. British Hyperbaric Association carbon monoxide database, 1993–96. *J Accid Emerg Med* 1999;16:98–103.

6 Goldbaum LR, Orellano T, Dergal E. Mechanism of the toxic action of carbon monoxide. *Ann Clin Lab Sci* 1976;6: 372–6.

7 Thom SR. Carbon monoxide mediated brain lipid peroxidation in the rat. *J Appl Physiol* 1990;68:997–1003.

8 Penney DG, Helfman CC, Dunbar JC Jr, *et al*. Acute severe carbon monoxide exposure in the rat: effects of hyperglycaemia and hypoglycaemia on mortality, recovery and neurologic deficit. *Can J Physiol Pharmacol* 1991;69:1168–77.

9 Matsuoka M, Igisu H, Tanaka I, *et al*. Effects of hypo- and hyperglycaemia on brain energy metabolites in mice exposed to carbon monoxide. *Toxicol Lett* 1994;73:135–43.

10 Tomita M, Okuyama T, Shimosato K, *et al*. Effect of ethanol on fatal carbon monoxide poisoning in awake mice. *Toxicol Lett* 1990;50:151–7.

11 Denays R, Makhoul E, Dachy B, *et al*. Electroencephalographic mapping and ^{99m}Tc-HMPAO single-photon emission computed tomography in carbon monoxide poisoning. *Ann Emerg Med* 1994;24:947–52.

12 Choi IS, Kim SK, Lee SS, *et al*. Evaluation of outcome of delayed neurological sequelae after carbon monoxide poisoning by technetium-99m hexamethylpropylene amine oxime brain single photon emission computed tomography. *Eur Neurol* 1995;35:137–42.

13 Turner M, Kemp PM. Isotope brain scanning with Tc-HMPAO: a predictor of outcome in carbon monoxide poisoning? *J Accid Emerg Med* 1997;14:139–41.

14 Smith JS, Brandon S. Morbidity from acute carbon monoxide poisoning at three-year follow-up. *BMJ* 1973;i:318–21.

15 Gorman DF, Clayton D, Gilligan JE, *et al*. A longitudinal study of 100 consecutive admissions for carbon monoxide poisoning to the Royal Adelaide Hospital. *Anaesth Intensive Care* 1992;20:311–16.

16 Turner M. The dangers of carbon monoxide poisoning. *N Engl J Med* 1995;332:894.

17 Touger M, Gallagher EJ, Tyrell J. Relationship between venous and arterial carboxy-haemoglobin levels in patients with suspected carbon monoxide poisoning. *Ann Emerg Med* 1995;25:481–3.

18 Buckley RG, Aks SE, Eschom JL, *et al*. The pulse oximetry gap in carbon monoxide intoxication. *Ann Emerg Med* 1994;24:252–5.

19 Marius-Nunez AL. Myocardial infarction with normal coronary arteries after acute exposure to carbon monoxide. *Chest* 1990;97:491–4.

20 McMeekin JD, Finegan BA. Reversible myocardial dysfunction following carbon monoxide poisoning. *Can J Cardiol* 1987;3:118–21.

21 Levassuer L, Galliot-Guilley M, Richter F, *et al*. Effects of mode of inhalation of carbon monoxide and of normobaric oxygen administration on carbon monoxide elimination from the blood. *Hum Exp Toxicol* 1996;15:898–903.

22 Raphael JC, Elkharrat D, Jars-Guincestre MC, *et al*. Trial of normobaric and hyperbaric oxygen for acute carbon monoxide intoxication. *Lancet* 1989;ii:414–19.

23 Norkool DM, Kirkpatrick JN. Treatment of acute carbon monoxide poisoning with hyperbaric oxygen: a review of 115 cases. *Ann Emerg Med.* 1985;14:1168–71.

24 Thom SR. Antagonism of carbon monoxide-mediated brain lipid peroxidation by hyperbaric oxygen. *Toxicol Appl Pharmacol* 1993;123:234–4.

25 Thom SR. Functional inhibition of luekocyte β_2 integrins by hyperbaric oxygen in carbon monoxide-mediated brain injury in rats. *Toxicol Appl Pharmacol* 1993;123:248–56.

26 Tibbles PM, Perrotta PL. Treatment of carbon monoxide poisoning: a critical review of human outcome studies comparing normobaric oxygen with hyperbaric oxygen. *Ann Emerg Med* 1994;24:269–76.

27 Thom SR, Taber RL, Mendiguren II, *et al*. Delayed neuropsychologic sequelae after carbon monoxide poisoning: prevention by treatment with hyperbaric oxygen. *Ann Emerg Med* 1995;25:474–80.

28 Ducasse JL, Celsis P, Marc-Vergnes JP. Non-comatose patients with acute carbon monoxide poisoning: hyperbaric or normobaric oxygen? *Undersea Hyperb Med* 1995;22:9–15.

29 Myers RAM, Snyder SK, Emhoff TA. Sub-acute sequelae of carbon monoxide poisoning. *Ann Emerg Med* 1985;14: 1163–7.

30 Turner M, Esaw M, Clark RJ. Carbon monoxide poisoning treated with hyperbaric oxygen: metabolic acidosis as a predictor of treatment requirements. *J Accid Emerg Med* 1999;16:96–8.

31 Tom T, Abedon S, Clark RI, *et al*. Neuroimaging characteristics in carbon monoxide toxicity. *J Neuroimaging* 1996;6: 161–6.

32 Pracyk JB, Stolp BW, Fife CE, *et al*. Brain computerised tomography after hyperbaric oxygen therapy for carbon monoxide poisoning. *Undersea Hyperb Med* 1995;22:1–7.

33 Horowitz AL, Kaplan R, Sarpel G. Carbon monoxide toxicity: MR imaging in the brain. *Radiology* 1987;162: 787–8.

34 Turner M, Clark RJ. Glioblastoma presenting as carbon monoxide poisoning. *J Accid Emerg Med* 1999;16:155–6.

35 Krenzelok EP, Roth R, Full R. Carbon monoxide .... the silent killer with an audible solution. *Am J Emerg Med* 1996;14:484–6.

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 exposure 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 carboxyhaemoglobin (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.

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Keywords: carbon monoxide poisoning; hyperbaric oxygen treatment