

Screening for carbon monoxide exposure in selected patient groups attending rural and urban emergency departments in England: a prospective observational study

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ABSTRACT

Objectives: Carbon monoxide (CO) exposure does not produce a classical toxidrome and so it is thought that it may easily be missed, allowing patients to continue to be exposed to CO. The aim of this study was to determine the proportion of raised carboxyhaemoglobin (COHb) levels in a targeted population of patients presenting to four emergency departments (EDs) in England.

Design: A prospective observational study undertaken over a 9-month period.

Setting: Four EDs; one in a rural/suburban area and three serving urban populations.

Participants: 1758 patients presenting to the EDs with chest pain, exacerbation of chronic obstructive pulmonary disease (COPD), non-traumatic headache, seizures or flu-like symptoms.

Main outcome: Measures COHb levels measured using a pulse CO-oximeter or venous sample. Patients with COHb levels $\geq 2.5\%$ (non-smokers) or $\geq 5\%$ (smokers) completed a questionnaire assessing potential sources. Patients were defined to be positive for CO exposure if they had a positive COHb and either an identified source or no other reason for their raised level.

Results: Proportion of positive patients was: overall—4.3%; COPD—7.5%; headache—6.3%; flu-like—4.3%; chest pain—3.3%; seizures—2.1%. A variety of gas and solid (predominantly charcoal) fossil fuel sources were identified.

Conclusions: This study showed that 4.3% of patients presenting to EDs with non-specific symptoms had unexpectedly raised COHb levels 1.4% of patients had a source of CO identified. Study limitations included non-consecutive recruitment, delays in COHb measurements and a lack of ambient CO measurements, which precludes precise determination of incidence. However, this study should alert clinicians to consider CO exposure in patients presenting with non-specific symptoms, in particular headache and exacerbation of COPD, and if necessary refer patients for suitable public-health follow-up, even in the presence of low COHb readings. Further research should include standardised scene assessments.

ARTICLE SUMMARY

Article focus

- Carbon monoxide (CO) exposure is difficult to diagnose and may easily be missed.
- Previous studies from the USA and Malta have indicated that prevalence of CO exposure in an undifferentiated ED population is low but higher in patients presenting with non-traumatic headache, chest pain, exacerbation of chronic obstructive pulmonary disease, flu-like symptoms and seizures.
- Prevalence of exposure of CO among patients presenting to emergency departments (EDs) in the UK is unknown.

Key messages

- CO exposure does result in a targeted population of patients presenting to EDs: 4.3% of study patients had unexpectedly high carboxyhaemoglobin (COHb) levels.
- Diagnosing CO exposure is difficult and requires a high index of suspicion combined with a low threshold for referring patients for public health follow-up.
- Health education should include advising patients to obtain electronic CO alarms.

Strengths and limitations of this study

- This is the first study of prevalence of CO exposure in ED patients in the UK.
- COHb was difficult to interpret because of delays in measurement and confounding factors such as smoking.

INTRODUCTION

Both acute and chronic carbon monoxide (CO) exposure can produce a wide variety of non-specific clinical features, all of which mimic other pathologies.¹ In addition, it may precipitate some chronic cardiorespiratory conditions.^{2–6} Evidence suggests that CO toxicity is frequently missed by healthcare

professionals^{7–12} and those who continue to be exposed to CO are likely to experience chronic neurocognitive dysfunction.^{13–16} CO exposure has been described as a pyramid of disease,¹ the tip of which is overtly poisoning which is more likely to be recognised by clinicians. However, the base of the pyramid includes chronic, low-dose exposure, which may produce symptoms that are misdiagnosed by clinicians.

Studies have been conducted in the USA and Malta, which suggest that the level of CO poisoning is low in an undifferentiated healthcare population^{17–20} but much higher in targeted groups (those showing clinical features that may be caused by CO, such as non-traumatic headache, cardiac chest pain, exacerbation of chronic lung disease, flu-like symptoms and seizures).^{21–28} The Department of Health has recently estimated that 4000 people/year are diagnosed with CO poisoning by emergency departments (EDs) in England and Wales²⁹ but this figure does not include those whose diagnosis is missed.

The main objective of this study was to determine the proportion of raised carboxyhaemoglobin (COHb) levels in a targeted population of patients presenting to four EDs in England. One ED is located in a semirural setting while the others serve urban populations, two of which are in a geographical area considered to have a high incidence of CO exposure. The secondary objective was to identify risk factors for exposure to CO in the study population.

METHODS

The study was conducted prospectively at four EDs: Frimley Park Hospital, Surrey; St Mary's Hospital, London; St James' Hospital, Leeds and Leeds General Infirmary in 2010. The former serves both suburban and rural communities, while the other three are situated in urban centres. During the course of the study, the provision of emergency services in Leeds changed with the two departments taking patients with different conditions, therefore data are presented as coming from one hospital.

Patients were considered to be eligible for enrolment if they presented to the EDs with one of the following conditions

- ▶ Chest pain characteristic of cardiac disease
- ▶ Exacerbation of chronic obstructive pulmonary disease (COPD)
- ▶ Seizures
- ▶ Flu-like symptoms
- ▶ Non-traumatic headache.

There were no age restrictions. Ethics approval was granted from Barking and Havering Research and Ethics Committee.

Patients who were recruited to the study had their COHb level measured as soon as possible after first arrival at the department, using the Masimo RAD-57 pulse CO-oximeter (Masimo Corporation, Irvine,

California, USA). Previous studies have shown that this monitor has an accuracy of $\pm 2\%$ below 15%³⁰ and 40%.³¹ A brief questionnaire was completed by a triage nurse or researcher, which included information needed to interpret COHb levels, such as time the patient left home/work, time of arrival at the ED, whether supplemental oxygen was given en route to the ED and the patient's smoking status. Blood was taken if the clinical condition required this or if the pulse CO-oximeter reading was raised, as all of the departments used point-of-care blood analysers which routinely measured COHb—blood COHb levels were not measured purely for research purposes. All sample times were recorded and a note was made of whether supplemental oxygen was administered between the different samples.

Most previous studies have used a COHb level of 10% as positive. However, because symptoms have been reported with COHb levels as low as 2.5%³² and the investigators were anxious not to miss any possible cases, the COED group decided that a more appropriate definition of a raised COHb level in this study was $\geq 2.5\%$ in non-smokers and $\geq 5\%$ in smokers. Patients who had COHb measurements above these levels were asked to complete a second questionnaire; where the patient was unable, the accompanying friend, family or staff completed this. The questionnaire captured information about possible domestic or occupational sources of exposure, use of CO and smoke alarms at home, a more detailed smoking history (type and amount of tobacco use and when the patient last smoked), and symptoms experienced by the patient or their cohabitantes/coworkers in the previous week.

In most instances, these patients were referred to the local public-health team at the Health Protection Agency (HPA) for follow-up. A standardised HPA protocol was followed,³³ this involved actions to investigate and control the hazard, such as a gas appliance check by an engineer registered with Gas Safe (<http://www.gassaferegister.co.uk>), liaison with the local authority and reporting to the Health and Safety Executive for possible occupational sources.

In a small number of cases, the patients were not referred to public health but instead Gas Safe-registered gas service engineers were contracted by the patients themselves to undertake appliance checks. The result of these checks was recorded by the research team.

All patients with raised COHb levels were treated with supplemental oxygen until their COHb levels returned to $< 2.5\%$ (or $< 5\%$ for smokers) while all other medical management was determined by clinical need.

Patients were subclassified using the following case definitions, based on Mandal *et al*³⁴

- ▶ Confirmed case—symptoms consistent with CO poisoning, raised COHb level and environmental source of CO confirmed.
- ▶ Probable case—symptoms consistent with CO poisoning, raised COHb level, no environmental source of CO identified.

- ▶ Possible case—symptoms consistent with CO poisoning, raised COHb level, no environmental source of CO identified and alternative hypothesis for COHb level likely (eg, heavy smoking/passive smoker, by expert consensus as below).
- ▶ Non-case—symptoms consistent with CO poisoning but COHb level not raised.

Patients in the ‘Confirmed’ and ‘Probable’ categories were considered to be positive for the purposes of data analysis, while those in the ‘Possible’ and ‘Non-Case’ groups were considered to be negative. Positive cases were classified as ‘Confirmed’ after an environmental source was established during the public-health follow-up. In patients where an alternative hypothesis was likely, an expert group from the research group consisting of an emergency physician, two toxicologists and an epidemiologist met to decide whether a case should be defined as ‘Probable’ or ‘Possible’. All of the questionnaire data (where available) were considered, including smoking status and when tobacco was last smoked, further symptoms suggestive of CO poisoning (such as confusion or feeling faint), whether cohabittees/coworkers were affected, whether symptoms got better away from home/work and information about use and state of appliances in the home.

Statistical tests were carried out using Stata V.8.0.

RESULTS

A total of 1777 patients were recruited into the study between January and October 2010; 19 patients were recruited incorrectly so analysis was undertaken on 1758 patients. Patient demographics and results at each study site are shown in table 1 while table 2 shows demographic details and results by disease group. Table 3 shows a more detailed breakdown of the positive and negative results.

Sex was recorded in 1738 cases; 911 (52%) were men. The median age was 50 years (range 10 weeks–97 years). 483 (28%) patients reported themselves as smokers. The majority of patients complained of only one symptom; however, 41 patients were recorded with two symptoms (including four positive cases) and three patients reported three symptoms (all were negative); patients with multiple symptoms were included in more than one disease group.

Only 4.3% of patients were classified as positive (95%CI 3.4% to 5.4%). By hospital, this varied from 1.8% (95%CI 1.0% to 3.0%) at St Mary’s, 4.6% (95%CI 3.2% to 6.4%) at Frimley and 9.5% (95%CI 6.5% to 13.2%) at Leeds. The proportion of raised COHb was highest in patients with COPD (7.5%; 95% CI 4.7% to 11.4%). More smokers (9.2%) were diagnosed with raised COHb than non-smokers (2.5%) and the difference was significant ($p \leq 0.05$). There was no significant difference in proportion of raised COHb between age groups (table 4).

The median COHb level was 2.6% in smokers (range 0–33.3%) and 1% for non-smokers (range 0–22.2%).

Table 1 Demographics

	Frimley park	St Mary’s	Leeds	All hospitals
Total number recruited	712	729	317	1758
Male (%)	56	52	47	52
Median age (range)	53 years (10 weeks–97 years)	47 years (7 months–96 years)	54 years (16–90 years)	50 years (10 weeks–97 years)
<18 years (%)	2.1	3.7	0.9	2.2
Smokers (%)	188 (26)	188 (26)	103 (32)	479 (27)
Median COHb (range) in smokers	3.0% (0–33.3%)	2.0% (0–21%)	3.8% (0–11%)	2.6% (0–33.3%)
Median COHb (range) in non-smokers	1.3% (0–22.2%)	1.0% (0–18.1%)	1.5% (0–8%)	1.0% (0–22.2%)
Percentage of positive cases (95% CI)	4.6% (3.2% to 6.4%)	1.8% (1.0% to 3.0%)	9.5% (6.5% to 13.2%)	4.3% (3.4% to 5.4%)
Median COHb (range) for positive cases	5.9% (2.0–33.3%)	11.4% (4.9–21%)	6.3% (2.5–11%)	6.1% (2.0–33.3%)
	n=33	n=13	n=30	n=76
Median COHb (range) for negative cases	1.4% (0–8.3%)	1% (0–7.9%)	1.7% (0–7.7%)	1.2% (0–8.3%)
	n=679	n=716	n=287	n=1682

COHb, carboxyhaemoglobin.

Table 2 Results by condition

	Chest pain	COPD	Headache	Seizures	Flu	Total
Total number recruited	915	265	350	146	117	1758*
Male (%)	58	47	44	58	38	52
Median age (range)	50 years (1197 years)	72 years (41–95 years)	40 years (690 years)	42 years (166 years)	37 years (10 weeks–96 years)	50 years (10 weeks–97 years)
Smokers (%)	24	33	32	34	21	27
Median COHb (range) smokers	2.6% (0–11%)	3.0% (0–13.4%)	3.0% (0–33.3%)	2.4% (0–8%)	2.0% (0–7.7%)	2.6% (0–33.3%)
Median COHb (range) non-smokers	1.0% (0–6.1%)	1.2% (0–8%)	1.0% (0–18.1%)	1.1% (0–5%)	1.0% (0–22.2%)	1.0% (0–22.2%)
Percentage of positive cases (95% CI)	3.3% (2.2% to 4.6%)	7.5% (4.7% to 11.4%)	6.3% (4.0% to 9.4%)	2.1% (0.4% to 5.9%)	4.3% (1.4% to 9.7%)	4.3% (3.4% to 5.4%)
Median COHb (range) for positive cases	5.9% (2.5–11%)	6.2% (2.5–13.4%)	8.7% (4–33.3%)	5.6% (5–8%)	6.0% (5–22.2%)	6.1% (2.0–33.3%)
Median COHb (range) for negative cases	1.2% 0–8.3%	1.4% (0–8.3%)	1.0% (0–7.8%)	1.3% (0–8%)	1.0% (0–7.7%)	1.2% (0–8.3%)

*Some patients presented with more than one complaint. COHb, carboxyhaemoglobin; COPD, chronic obstructive pulmonary disease.

Patients with headaches had the highest median COHb levels at 8.7% (range: 4–33.3%). Sixty-two of the 76 positive cases (82%) had a COHb level below 10%.

In total, 22 (29%) of the positive cases were subclassified as confirmed; the source of CO exposure was found to be:

- ▶ Shisha pipe smoking in an enclosed environment (10)
- ▶ Faulty or incorrectly installed gas boiler (6)
- ▶ Ventilation outlet blocked by snow or wallpaper (3)
- ▶ Fire smoke inhalation (1)
- ▶ Leaving a gas cooker burning (1)
- ▶ Lighting a barbecue indoors (1)

Interestingly, one case was due to a faulty boiler in a neighbouring flat which was identified by detailed investigation by the public health team undertaking the follow-up. The patient who inhaled fire smoke did not disclose that their headache had started after they had put out a chip pan fire (they were in an unventilated smoke-filled room for a number of minutes) until they were completing the questionnaire!

Ambulance crew or patients already suspected CO poisoning as the cause of symptoms at triage in 30 (2%) of patients, half of whom were subsequently categorised in the study as positive. Of all the positive patients, CO poisoning was only previously suspected in 20% of cases. The vast majority (23/30) of patients where CO was suspected had headaches. The incidence of ‘unsuspected’ positive cases was 3.5%; it is uncertain whether this reflects reality because of the Hawthorne effect; staff, including many ambulance crews, were aware that the study was taking place so CO was more likely to be suspected.

Positive cases were identified throughout the recruitment period, not just in the winter months (table 5).

DISCUSSION

Background

CO is a colourless, odourless gas that is not detectable by the human senses. It produces non-specific symptoms that mimic other conditions, and therefore may be missed by healthcare professionals, particularly following chronic and/or subacute exposure.^{7–12} This often results in continued exposure to CO which may lead to chronic neurocognitive dysfunction,^{13–16} exacerbation of chronic cardiorespiratory conditions^{2–6} or more serious acute effects if exposure to higher concentrations occurs. Wright¹ described CO exposure as a pyramid of disease with acute, overt poisoning representing the tip; case reports and press articles show that even these cases may be misdiagnosed. The base of the pyramid includes chronic, low-level exposure which is more likely to be missed yet may cause significant morbidity or even mortality particularly in susceptible individuals with chronic cardiorespiratory disease.

There is a paucity of evidence of both the prevalence of CO exposure and the resulting burden of health effects in the UK. A recent study of ambient CO levels in

Table 3 Total cases identified by presenting condition

Case status	Detailed status	Chest pain	COPD	Headache	Seizure	Flu
Positive	Confirmed	4	1	17	0	3
	Probable	26	19	5	3	2
	Total*	30	20	22	3	5
Negative	Possible case	15	4	11	8	2
	Non case	870	241	317	135	110
	Total*	885	245	328	143	112
Grand total*		915	265	350	146	117

*Some patients presented with more than one complaint. COPD, chronic obstructive pulmonary disease.

597 homes in Greater London and South East England found that 22% had at least one defective gas appliance installation that was deemed to be unsafe and 7% of the 1414 appliances tested were reported to be immediately unsafe.³⁵ A follow-up study funded by the CORGI Trust, with a stricter methodology for examining gas fires, found 4% of 209 homes with at least one unsafe gas appliance installation.³⁶

CO poisoning frequency

This study has confirmed that patients present to EDs with raised COHb levels and non-specific symptoms that could be attributable to CO poisoning. As will be discussed later, a raised COHb level per se does not prove CO toxicity. Overall in this study, the percentage of CO poisoning in patients attending the ED with chest pain, headache and fits are largely consistent with those reported in the literature.^{23 25–27} However, 7.5% of positive cases among COPD patients were found in this study, which was far less than that in the study by Calverley *et al*,²¹ which reported 17.6%. The difference may be due differences in sample size (Calverley n=91, our study n=265) and study population; their patients (attending a respiratory medicine out-patients' clinic) were more likely to be stable compared to our population who were suffering from an exacerbation of their disease. Therefore, our patients were less likely to have smoked recently and were more likely to have been administered supplemental oxygen than in the previous study. Similarly, a lower proportion was noted in patients with flu-like symptoms compared with a previous study (4.3% vs 23.6%),²² despite the previous study using a much higher $\geq 10\%$ COHb as a marker for CO poisoning. The reasons for this discrepancy could be multiple:

the earlier study may have contained bias because only 9% (55/637) of potentially eligible patients were recruited, there is likely to be differences between the public's awareness of CO today compared to when this study was conducted in 1985 and patient demographics and healthcare-seeking behaviour may have been different. Our study period coincided with the H1N1 influenza outbreak, when people with flu-like symptoms were being encouraged to stay at home, which meant that a smaller than expected number of patients with flu-like symptoms were recruited.

It was interesting to note that cases were found throughout the summer months. CO poisoning is considered to be a seasonal condition with the early winter months being a particular risk, when people switch on their heating systems. Data collection did not occur over a full year, so a comparison of different months cannot be made; however, it is important for clinicians to maintain vigilance for CO poisoning at all times. CO poisoning has also been associated with older people;³⁷ however, we found no evidence that risk was greater in any age group; indeed, although not significant, there was a slightly higher proportion of cases in children.

Median COHb levels

COHb levels were highest in positive patients presenting with headache (8.7%; range 4–33.3%), which would suggest that it would be a useful trigger symptom to alert clinicians to the possibility of CO poisoning. Again this agreed with the findings of a previous study.²⁷ Headache was also the most common complaint among patients where CO poisoning was previously suspected.

All the patients diagnosed with seizures who met the case definition had COHb levels lower than those typically associated with such severe symptoms as seizures.³⁸ Unlike chronic cardiorespiratory disease, there is no evidence to suggest that seizure threshold is affected by exposure to low levels of CO. The expert group (the emergency physician, toxicologists and epidemiologist from the COED group) decided to classify seizure patients with COHb levels between 2.5% and 5% (non-smokers) or 5% and 8% (smokers) as non-exposed cases for the purposes of this study, in particular where seizure was described as tonic-clonic. However, it was

Table 4 Results by age

Age (years)	Number	Positive (%)
≤18	57	4 (7.0)
19–65	1159	54 (4.7)
≥66	537	18 (3.4)

χ^2 /Fisher's exact tests show non-significant differences between all age groups.

Table 5 Monthly recruitment

	January	February	March	April	May	June	July	August	September	October
Positive cases	6	4	10	2	9	17	11	7	7	3
Positive (%)	3.0	3.6	3.6	0.6	3.6	9.0	4.8	13.5	10.4	4.1

noted that one patient (with COHb of 0.7%) in the seizure group had a domestic source of CO discovered (this patient arranged an inspection of their gas boiler as a result of being recruited into the study in spite of a low COHb level); whether this was a coincidental finding or caused the fit cannot be determined from the study.

Our finding that the majority of positive patients—in both smokers and non-smokers—had COHb levels <10% is important as many clinicians use ≥10% as an indicator of CO poisoning. Indeed, 56% of the patients with confirmed sources of CO exposure had COHb levels below 10%; if these patients had been sent home, they could have been reexposed. The importance of involving public-health officials in the follow-up to patient care is therefore vital.

Clinical and public health lessons

CO poisoning was not suspected by patients nor emergency crew in 80% of positive cases. Retrospective review of the cases from one hospital site suggested that for 6 of the 11 (54%) confirmed cases the diagnosis of CO exposure was not immediately obvious. All of them were found to have faulty boilers that were leaking CO. It is not possible to state how many of these patients would have been sent home to continued exposure if it was not for the study, but it is very likely that some of these patients may have been discharged without appropriate public health follow-up. In addition, following the completion of this study, two cases of confirmed CO exposure have been identified early because of increased vigilance by nursing staff in the department at Frimley. Both had malaise, fatigue and vague headache which are likely to have been attributed to viral illness before the study. This agrees with previous evidence that heightened awareness of occult CO poisoning can be achieved both in healthcare staff and members of the public,^{8 9} although it is not certain how this level of vigilance can be maintained. Public health professionals have an education role to play in preventing CO exposure, assisting clinicians with diagnose poisoning³⁹ and identifying causes of poisoning. The links established between EDs and the HPA have led to improved surveillance of CO poisoning and identification of new trends, such as the dangers of shisha pipe smoking.⁴⁰

An additional unexpected outcome of the study was increased awareness of CO in patients, which led to the possible identification of 'false negatives'. Many negative patients (ie, with symptoms suggestive of CO poisoning but normal COHb levels) subsequently arranged for home gas service inspections. Two patients reported

back to the study team that sources of CO had been identified in their homes; retrospective consideration of their records showed it likely that one patient's symptoms were caused by CO, while for the other patient it was less clear due to the timing of onset and resolution of symptoms, dose-response relationships and lack of activation of a CO alarm. Despite the relatively low COHb threshold chosen to indicate CO exposure, this finding shows that CO poisoning can still be missed, which was most likely due to delayed patient presentation and/or delayed COHb testing after presentation.

CO alarms

It was interesting to note from the questionnaires that smoke alarms were installed more commonly than CO alarms (88% and 16%, respectively) in patient homes. There have been repeated, high-profile campaigns to promote the use of smoke alarms over a number of years and building regulations were introduced in 1992 which required that all newly built dwellings should have hard-wired smoke alarms.⁴¹ This has resulted in 85% of all domestic properties in England having smoke alarms, which has coincided with a reduction in deaths from house-fires.⁴¹ Similar campaigns to promote the use of suitable CO alarms could have similar important benefits for the health of the population.

Limitations

COHb levels are difficult to interpret in the clinical environment.⁸ COHb levels reach equilibrium at a slow rate which is affected by levels of activity and by variability in ambient concentrations at the scene of exposure.¹ Levels decline with time at a relatively predictable rate which is dependent on inspired oxygen concentration and activity level of the individual (higher levels of both reduce the elimination half-life of CO). Unfortunately, these factors are not easily assessed in the clinical environment.¹ This study found that the time since exposure, as well as timing and concentration of supplemental oxygen therapy, although collected on the questionnaires, were often poorly recorded or recalled. Also, no note of physical exertion before arrival in the ED was made. Obviously, the longer the delay in measuring COHb levels, the more difficult it becomes to interpret the significance of the level. In this study the mean time for a COHb measurement to be taken after presentation at the hospitals was 46 min (range 0 min–10 h). This was lower for positive patients than negative ones (33 vs 46 min).

The number of cigarettes smoked and time since last smoked influence the COHb level measured. Significantly more smokers than non-smokers had

elevated COHb, a finding which is not unexpected as chronic smokers are known to have higher resting COHb levels. Although researchers did try to control for this using detailed questionnaires, it is likely that this was subject to some responder bias. Patients may be reluctant to admit to health professionals that they smoke.^{42 43} Indeed during this study a number of patients changed their story between the triage questionnaire and full questionnaire once the COHb level was known, and only 2 of the 10 patients who were exposed to CO from charcoal-burning shisha pipes indicated that they were smokers. Uncertainties about smoking status could have affected the results of this study with some interchange between the 'probable' (positive) and 'possible' (negative) subcategories.

Although the delay in COHb measurements would tend to underestimate the incidence found in the study while questionable smoking status would tend to have the opposite effect, the overall influence of the confounding factors cannot be determined.

The finding of raised COHb levels in this study does not necessarily give any direct information on the incidence of clinical poisoning by CO. Although there are validated formulae to predict COHb levels from different ambient CO levels, most notably the Coburn-Forster-Kane model,⁴⁴ these contain physiological parameters that are not measurable in clinical practice. In addition, it takes between 8 and 12 h for COHb levels to reach equilibrium in the presence of stable ambient CO levels^{8 45} duration of exposure is rarely known with any degree of certainty and CO levels may vary at the scene with patients moving between areas of different concentrations. Lastly, exertion will increase CO uptake⁴⁶ but again this is difficult to quantify in clinical practice. This, together with the uncertainties about the timing of COHb measurements, means that ambient CO levels cannot be calculated retrospectively. The study did not have access to the results of the appliance checks and environmental investigation. Without these ambient levels the dose-response effect of CO cannot be determined with any certainty; therefore, it was only possible to evaluate whether CO poisoning caused the symptoms, even when a source of CO was discovered, by indirect factors such as onset and duration of symptoms in relation to the site of exposure, and the presence of cohabitants with symptoms.

The results of this study should not be extrapolated to give an estimation of incidence of CO poisoning in other regions or nationally. However, in spite of these limitations, this study discovered otherwise unexplained raised COHb levels in 4.3% of the study population with external sources of CO exposure being identified in 1.3%. What was of particular note was that cases occurred throughout the duration of data collection, which extended into the summer months, a time where risk of CO exposure from domestic sources is considered to be low. However, boilers are used throughout the year to heat water, so one important message from this study is

that clinicians should be vigilant about CO poisoning at all times, not just during the winter months.³⁸

CONCLUSIONS

This study showed that CO exposure does result in patients presenting to EDs with non-specific symptoms. Clinical suspicion is required to recognise that CO may be the cause of the non-specific symptoms, in particular headache. COHb levels can be difficult to interpret and clinicians need to understand that a normal level does not exclude CO exposure. If there is clinical suspicion, then appropriate public-health follow-up should be arranged and the patient advised to have their fuel-burning appliances checked by appropriately qualified and registered engineers. Efforts to increase use of domestic CO monitors are likely to assist the public to recognise CO exposure and seek appropriate medical assistance. Future studies must include systematic assessment of fuel-burning appliances and measurements of ambient CO levels at the scenes of possible exposure if incidence of CO poisoning (rather than raised COHb levels) is to be calculated more precisely.

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Contributors COED Group—The members of the COED group contributed to the initial development of the study. Professor David Baker was a member of the expert review group who discussed the 'probable' and 'possible' definitions (with the authors Ms Catherine Keshishian, Professor Virginia Murray and Dr Simon Clarke). All authors assisted with planning of the project. SC was chief investigator for the study and principle investigator for Frimley. He completed the funding and ethics proposals, assisted with all aspects of data collection and interpretation, wrote the first draft and collated the subsequent drafts of the manuscript. CK was epidemiologist, undertook the sample size calculations, developed and managed the database, undertook data interpretation, assisted with the all drafts of the manuscript. VM was principle investigator for the HPA and assisted with all drafts of the manuscript. GK undertook sample size calculation and statistical analysis of the data and assisted with all drafts of the manuscript. RR developed the public health response and training and assisted with all drafts of the manuscript.

EC, SO and DE were research nurses and led staff training and data collection at each site. They assisted with all drafts of the manuscript. PW and SB were principle investigators at London and Leeds, respectively. They set up the project locally, assisted with data collection and interpretation and assisted with all drafts of the manuscript. CP assisted with the initial literature search, with data collection and interpretation, and with all drafts of the manuscript.

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REFERENCES

- Wright J. Chronic and occult carbon monoxide poisoning: we don't know what we're missing. *Emerg Med J* 2002;19:386–90.
- Anderson E, Andelman R, Strauch J, et al. Effect of low-level carbon monoxide exposure on onset and duration of angina pectoris. A study in ten patients with ischemic heart disease. *Ann Intern Med* 1973;79:46–50.
- Aronow W, Ferlinz J, Glauser F. Effect of carbon monoxide on exercise performance in chronic obstructive pulmonary disease. *Am J Med* 1977;63:904–8.
- Allred E, Bleecker E, Chaitman B, et al. Short-term effects of carbon monoxide exposure on the exercise performance of subjects with coronary artery disease. *N Eng J Med* 1989;321:1426–32.
- Burnett R, Dales R, Brook J, et al. Association between ambient carbon monoxide levels and hospitalizations for congestive heart failure in the elderly in 10 Canadian cities. *Epidemiol* 1997;8:162–7.
- Morris R, Naumova E. Carbon monoxide and hospital admissions for congestive heart failure: evidence of an increased effect at low temperatures. *Environ Health Perspect* 1998;106:649–53.
- Chief Medical Officer. Carbon monoxide: the forgotten killer. Department of Health. 2002. PL/CMO/2002/2, PL/CNO/2002/2.
- Green E, Short S. *Indoor air quality in the home (2): carbon monoxide*. Institute for Environment and Health, 1998. Assessment A5. <http://www.cranfield.ac.uk/health/researchareas/environmenthealth/ieh/ieh%20publications/a5.pdf> Cranfield University, UK (accessed November 2010)
- Barret L, Danel V, Faure J. Carbon monoxide poisoning, a diagnosis frequently overlooked. *Clin Toxicol* 1985;23:309–13.
- Raub J, Mathieu-Nolf M, Hampson N, et al. Carbon monoxide poisoning—a public health perspective. *Toxicol* 2000;145:1–14.
- Grace T, Platt F. Subacute carbon monoxide poisoning. Another great imitator. *JAMA* 1981;246:1698–70.
- Kirkpatrick J. Occult carbon monoxide poisoning. *West J Med* 1987;146:52–6.
- Volans V. *Neuropsychological effects of chronic exposure to carbon monoxide in indoor air*. Final Report to the Department of Health, London Carbon Monoxide Group. 2006.
- Messier LD, Myers RA. A neuropsychological screening battery for emergency assessment of carbon monoxide poisoned patients. *J Clin Psychol* 1991;47:675–84.
- Myers RAM, DeFazio A, Kelly MP. Chronic carbon monoxide exposure: a clinical syndrome detected by neuropsychological tests. *J Clin Psychol* 1998;54:555–67.
- Amitai Y, Zlotogorski Z, Golan-Katzav V, et al. Neuropsychological impairment from acute low-level exposure to carbon monoxide. *Arch Neurol* 1998;55:845–8.
- Heckerling PS, Leikin JB, Maturen A, et al. Screening hospital populations from the emergency department for occult carbon monoxide. *Am J Emerg Med* 1990;8:301–4.
- Whincup P, Papacosta O, Lennon L, et al. Carboxyhaemoglobin levels and their determinants in older British men. *BMC Public Health* 2006;6:189.
- Partridge R, Chee KJ, Sunar S, et al. Non-invasive carboxyhaemoglobin monitoring: screening emergency department patients for carbon monoxide exposure. *Resp Care, Open Forum Abstr* 2006;51:1332.
- Hampson N. Emergency department visits for carbon monoxide poisoning in the Pacific Northwest. *J Emerg Med* 1988;16:695–8.
- Calverley P, Leggett R, Flenley D. Carbon monoxide and exercise tolerance in chronic bronchitis and emphysema. *Br Med J* 1981;283:878–80.
- Dolan M, Haltom T, Barrows G, et al. Carboxyhaemoglobin levels in patients with flu-like symptoms. *Ann Emerg Med* 1987;16:782–6.
- Balzan M, Cacciottolo J, Mifsud S. Unstable angina and exposure to carbon monoxide. *Postgrad Med J* 1994;70:699–702.
- Balzan M, Agius G, Debono A. Carbon monoxide poisoning: easy to treat but difficult to recognise. *Postgrad Med J* 1996;72:470–3.
- Heckerling P, Leikin J, Maturen A, et al. Predictors of occult carbon monoxide poisoning in patients with headache and dizziness. *Ann Intern Med* 1987;107:175–6.
- Heckerling P. Occult carbon monoxide poisoning: a cause of winter headache. *Am J Emerg Med* 1987;5:201–4.
- Heckerling P, Leikin J, Maturen A. Occult carbon monoxide poisoning: validation of a prediction model. *Am J Med* 1988;84:251–6.
- Heckerling P, Leikin J, Terzian C, et al. Occult carbon monoxide poisoning in patients with neurological illness. *Clin Toxicol* 1990;28:29–44.
- Department of Health. <http://gp.dh.gov.uk/2011/09/27/carbon-monoxide-poisoning-alert/> (accessed November 2010)
- Barker SJ, Curry J, Redford D, et al. Measurement of carboxyhaemoglobin and methemoglobin by pulse oximetry. *Anesthesiol* 2006;105:892–7.
- Chee KJ, Nilson D, Partridge R, et al. Finding needles in a haystack: a case series of carbon monoxide poisoning detected using new technology in the emergency department. *Clin Toxicol* 2008;46:461–9.
- Health Protection Agency. Compendium of chemical hazards: carbon monoxide. http://www.hpa.org.uk/webc/HPAwebFile/HPAweb_C/1194947421806 (accessed November 2010)
- Mindlin M, Ruggles R. A carbon monoxide 'action card' for public health practitioners. *Chem Hazards Poisons Rep* 2010;10:34–6.
- Mandal S, Ruggles R, Leonardi G, et al. Developing best practice response to carbon monoxide incidents: a toolkit for health protection frontline staff. *Public Health* 2011;125:148–56.
- Croxford B. Gas appliance check project. Bartlett School of Graduate Studies, University College London. 2007. <http://www.hse.gov.uk/GAS/domestic/uclgasfinal.pdf> (accessed November 2010)
- Croxford B. Carbon monoxide exposure and neurological symptoms (CENSY Project). Bartlett School of Graduate Studies, University College London. 2009. <http://www.gas-safety-trust.org.uk/censy-report> (accessed November 2010)
- CORGI Carbon Monoxide Report. 2008. <http://www.gas-safety-trust.org.uk/corgi-carbon-monoxide-report-2008> (accessed November 2010)
- Keles A, Demircan A, Kurtoglu G. Carbon monoxide poisoning: how many patients do we miss? *Eur J Emerg Med* 2008;15:154–7.
- Chief Medical Officer. *Carbon monoxide poisoning: needless deaths, unnecessary injury*. Department of Health, London, UK, 2010. PL/CMO/2010/02, PL/CNO/2010/02.
- Zenner D, Dar O, Clarke S, et al. The water-pipe—an increasingly common source of carbon monoxide poisoning? *Chem Hazards Poisons Rep* 2011;19:10–12.
- Hansard Report. 15th Jan 2010. Column 712–713. <http://www.publications.parliament.uk/pa/ld200910/ldhansrd/text/100115-0002.htm> (accessed November 2010)
- Jones R, Commins B, Cernik A. Blood lead and carboxyhaemoglobin levels in London taxi drivers. *Lancet* 1972;300:302–3.
- Russell M. Blood carboxyhaemoglobin changes during tobacco smoking. *Postgrad Med J* 1973;49:684–7.
- Coburn RF, Forster RE, Kane PB. Consideration of the physiological variables that determine the blood carboxyhaemoglobin concentration in man. *J Clin Invest* 1965;44:1899–910.
- Hill EP, Hill JR, Power GG, et al. Carbon monoxide exchanges between the human fetus and mother. *Am J Physiol* 1977;232:H311–23.
- Forbes WB, Sargent F, Roughton FJW. The rate of carbon monoxide uptake by normal men. *Am J Physiol* 1945;143:594–608.