Acute Neurological Admission and Exposure to Carbon Monoxide

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Abstract

Carbon monoxide poisoning produces central nervous system hypoxia and frequently presents clinically in occult form. From December 1994 to March 1995 we screened 305 patients admitted for acute neurological problems to St. Luke's hospital for exposure to Carbon monoxide by determining blood carboxy-haemoglobin levels on admission. A questionnaire on indoor air pollution was also administered.

Three cases of acute intoxication are documented. The mean carboxy-haemoglobin of the other 302 patients was 3.24% (95% CL 0-7.4, n=43) for smokers and 0.73% (95% CL 0-1.90, n=259) for non-smokers. Five non-smokers and two smokers had levels higher than the upper limit. This level was probably only clinically significant in one case of a heavy smoker with transient ischaemic attack. There were no cases of exposure amongst strokes (n=141), epileptic fits (n=39), or episodes of loss of consciousness (n=87). However 3 out of 29 patients with impairment of consciousness without focal neurological signs had intoxication.

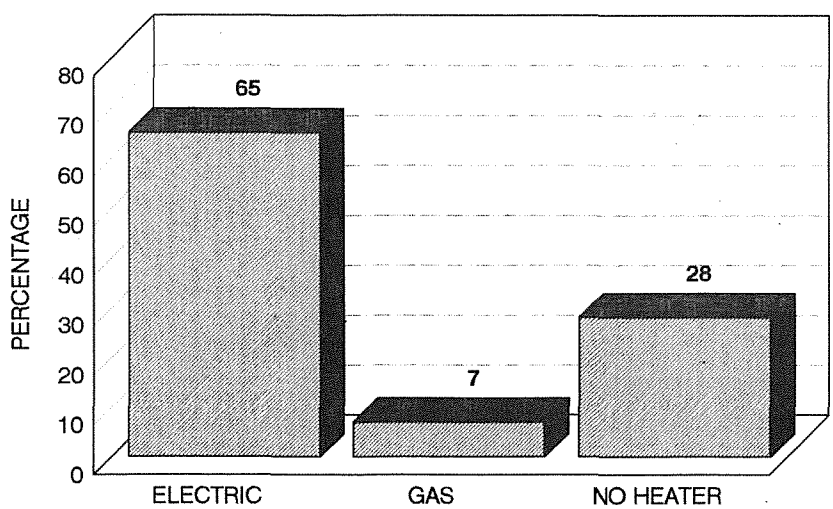
Although this intoxication does not appear to be very common, a high index of suspicion must be maintained, as 34% of the patients questioned had at least one potential source of carbon monoxide within their household. For this reason we suggest that, information on indoor heating should be part of routine history taking of acute neurological problems, particularly when impaired consciousness is persistent in the emergency room. Carboxy-Hb levels should be taken when there is even the slightest suspicion of exposure, as early treatment of this intoxication with high concentration oxygen therapy is very effective.

Introduction

Carbon monoxide is produced mainly by incomplete combustion of fossil fuel. Carbon monoxide (CO) binds to the haemoglobin molecule with much greater affinity than oxygen, and shifts the oxygen dissociation curve of the remaining haemoglobin to the left. The central nervous system and the myocardium, being the most susceptible tissues to hypoxia are most frequently affected[1].

The classical clinical picture of severe CO poisoning with carboxy-haemoglobin (CO-Hb) levels of above 50%, is that of unconsciousness with or without epileptic fits.[1,2] Lower levels i.e. 20-50% tend to cause fainting episodes, headache[3], vertigo, or exacerbation of angina pectoris[4]. The clinical diagnosis of this condition, for which treatment with hyperbaric oxygen therapy[5] is so effective, may not be easy.

First of all the gas is colourless and odourless and the patient, his family, ambulance and emergency room staff may not be aware of the exposure. None



ACUTE NEUROLOGICAL ADMISSIONS TYPE OF BATHROOM WATER HEATING USED. (N=176) MEAN AGE 66.1 (SD 18.1)

of the presenting clinical features are unique and the patient might be admitted to hospital with a working diagnosis of 'stroke' or 'unexplained unconsciousness' or 'confusion'[6,7,8]. Furthermore it has been shown that patients with underlying cerebrovascular or cardiovascular disease could present a different and atypical clinical picture with CO-Hb levels as low as 10-20%. These patients could be admitted to hospital with 'epileptic fit'[9,10] or 'unstable angina'[4] without this toxic exposure being recognized.

The aim of this study was to screen a population of acute neurological admissions to a general hospital during the winter months so as to (i) identify and describe cases of CO poisoning, particularly trying to identify 'occult' presentations, (ii) to determine the indoor fossil fuel utilization of this group, so as to establish what percentage of this patient population has a potential source of intoxication within the household.

Patients and methods

St Luke's hospital is the only acute medical facility serving the whole of the island of Malta (population around 330,000). From the 5th December 1994 to the 31st March 1995 all acute neurological admissions were to be included in the study. Diagnostic categories were (i) presumed stroke i.e. patients presenting with recent onset of dysphasia, dysarthria, or unilateral weakness or numbness. (ii) Generalized or focal motor seizures; (iii) Episodes of loss of consciousness, with full recovery. If the cause was evident e.g. hypoglycaemia, heart block, drug overdosage the patient was still included; (iv) Any patient with unconsciousness, obtunded sensorium or confusion but with no focal neurological signs on admission and (v) history of recent headaches. Patients with anaemia were excluded. (<12g/dl females, <14g/dl males).

Blood CO-Hb was determined on admission with an IL 482 CO oximeter (Instrumentation laboratories, Lexington, Massachusetts). Two CO-Hb calibration standards were used regularly to check the calibration of the instrument. An effort was made to make the result available to the attending clinician within one hour of admission so as to be able to provide treatment where necessary.

When calculating peak CO-Hb levels, a standard reference chart was used[1]. The possible shortening of the half life of CO-Hb by oxygen given by ambulance or emergency room personnel was not included in the calculation. This could have led to an underestimation of peak CO-Hb levels. It was assumed that there was no further decay in CO-Hb levels after blood sampling[1].

The morning following admission a standardized questionnaire was administered to the patient or one of his household if he was unable to communicate. Only two of the authors of the paper collected this information. Besides basic demographic information, the average quantity of cigarettes smoked per day in the week prior to admission was noted. Patients were questioned on the use of gas and kerosene space heaters and bathroom water heaters in the household and exposure to fires or car exhaust. In the absence of questionnaire data, smoking data was obtained from the patient's case notes.

	SMOKERS	NON-SMOKERS	TOTAL
Mean age (years)	52.8 (S.D. 21.4)	69.0 (S.D. 18.3)	66.7 (S.D. 19.6)
Mean CO-Hb (%)	3.24 (S.D. 2.12)	0.73 (S.D. 0.59)	1.09 (S.D. 1.09)
Male (n)	29	99	128
Female (n)	14	160	174
Total	43	259	302

Table 1. Acute neurological admissions; control group
CO-Hb=Carboxy haemoglobin

Results

336 patients were eligible for inclusion into the study. CO-Hb was determined in 307 (91%) patients. 2 patients were excluded because of anaemia. Three patients had clear evidence of CO poisoning. (Cases 1,2 and 3). The other 302 patients were used to determine a control value for mean CO-Hb in both smokers and non smokers. 141 (46.7%) patients had presumed stroke or transient ischaemic attack, 87 (29.8%) an episode of loss of consciousness, 39 (12.9%) epileptic fits, 26 (8.6%) impaired consciousness and 9 (3.0%) headaches. Table 1, shows the characteristics of the whole group. There was no significant difference between the mean CO-

SOURCE OF CO	NUMBER	%
Kerosene Heater	31	(14.4%)
Kerosene Stove	27	(12.5%)
Gas space heater	17	(7.9%)
Gas water heater	12	(5.6%)

Table 2. Source of CO in acute neurological admissions.
(n=215, mean age 65.4 s.d. 18.9, female 116, 54%)
CO=Carbon monoxide:
**some patients have more than one source*

Box 1

Clinical features of CO poisoning and CO-Hb levels*

<10%	Asymptomatic
10-20%	Unstable angina in coronary artery disease
20-30%	Headache, Vertigo
30-50%	Severe headache, Vomiting, Disturbance of Consciousness
>50%	Coma, Convulsions, respiratory depression death

**Broad individual variation*

Hb of all 5 diagnostic categories in smokers and non-smokers respectively.

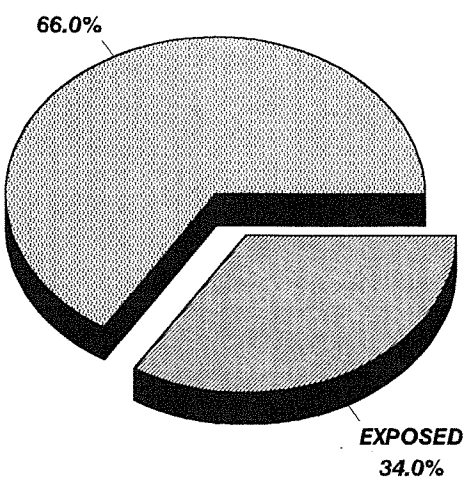
5 non-smokers had a CO-Hb level outside the 95% confidence limits. As the CO-Hb levels recorded were all between 2-3% none was considered clinically significant. Two smokers had a CO-Hb level outside the 95% confidence interval. One had cerebral haemorrhage and a CO-Hb level of 8%, and it was assumed that this was of no clinical relevance to the neurological diagnosis. A second patient with a CO-Hb of 8.9% will be described as case 4.

Complete information on indoor heating and indoor pollution was only obtained in 215 patients (71.2%) as seen on Table 2. However, the age and gender distribution mirrored the whole 302 patient group very faithfully. 73 (34%) had at least one potential source of intoxication in their house hold.

Case 1. A 28 year old man attempted to commit suicide. He switched on his car and stayed inside the car which was lying in a communal three car garage. Inadvertently he had left the garage door ajar. After about 1 hour an owner of a second car inside this garage found him unconscious. The car was switched off and he was carried out of the garage into the open air. Within twenty minutes of exposure he was in the emergency room. He was deeply comatose with a CO-Hb level of 60.2%. Peak CO-Hb levels probably reached 70%. Electrocardiogram was normal apart from sinus tachycardia. He was started on 100% oxygen and transferred to a hyperbaric chamber in 20 minutes. He was given 100% oxygen at 2.8 atmospheres for 90 minutes by the end of which he was fully conscious. He was also treated with dexamethasone 2mg 8 hourly and cimetidine 200mg twice daily intravenously. Two other courses of hyperbaric therapy were administered every 8 hours. By the third day he was up and about with some headache. Dexamethasone was tailed off over 10 days.

Case 2. A 75 year old man was found unconscious by his wife in a one car garage. According to his wife he had been attempting to repair his car. He had switched on his car about 10 minutes before. The door was closed. He arrived in the emergency room, about 20 minutes later. His CO-Hb was 25.2%, and probably peak level had been around 30%. H was semi-conscious and responded to name. His reflexes were brisk but there were no focal signs. The electrocardiogram only showed a partial right bundle branch block. He was started on 100% oxygen and about 30 minutes later received 100% oxygen at 2.8 atmosphere for about one hour in the hyperbaric chamber.

At the end of this treatment he was fully conscious. He received dexamethasone 2mg 8 hourly and cimetidine 200mg twice daily intravenously. However over the next three days he complained of severe headache and a cerebellar gait was noted. He improved rapidly and could walk unaided after 10 days. Dexamethasone was tailed off.



ACUTE NEUROLOGICAL ADMISSIONS. (N=215)
PROPORTION OF PATIENTS HAVING A SOURCE OF CARBON MONOXIDE INDOORS

Box 2

Difficulties in the Diagnosis of CO poisoning

*Gas is colourless, odourless and non-irritant.

*Patient may be unaware of the exposure.

*Ambulance staff, co-habitants unaware of exposure.

*Patient with impaired consciousness unable to give history.

*No pathopneumonic feature of the intoxication. (Textbook cherry red colour, a rare finding).

*Mimics common conditions such as unstable angina, drug overdosage, status epilepticus.

*Patients with underlying cardiovascular and cerebrovascular disease can have an atypical presentation.

Case 3. A 78 year old lady, who suffered from diabetic nephropathy and retinopathy and was previously already in a frail condition was referred to hospital with severely obtunded sensorium. She was a non-smoker and at 9.00 am her CO-Hb was 4.5%, and blood glucose 11.2 mmol/l. She has been well until the previous day. Her daughter had than used a kerosene heater in the patient's bedroom from 10.00 am to 9.00 pm. She had noticed her mother to be very sleepy and decided to switch off the heater. By 7.00 am next day she found her mother to be unarousable.

In casualty she was noted to respond to pain but not to name. Reflexes were absent but plantars were upgoing. There were no lateralising signs. CT scan of the brain was not performed. She was rehydrated and given intravenous cefuroxime, but she died of bronchopneumonia 6 days later. A metabolic screen did not reveal any alternative explanation for her deterioration. It was calculated that the peak CO-Hb levels 12 hours before admission would have been around 15%.

Case 4. A 43 year old man was seen in the emergency room for right hemiparesis with dysarthria. CO-Hb on admission was 8.9%. He had no source of indoor pollution in his household but had been smoking up to 40 cigarettes per day. Two hours before admission he had had the same neurological deficit but his resolved completely within 15 minutes. He had not smoked a cigarette since that moment so that his peak levels were probably around 12-14%. However, the same symptoms occurred again one hour later.

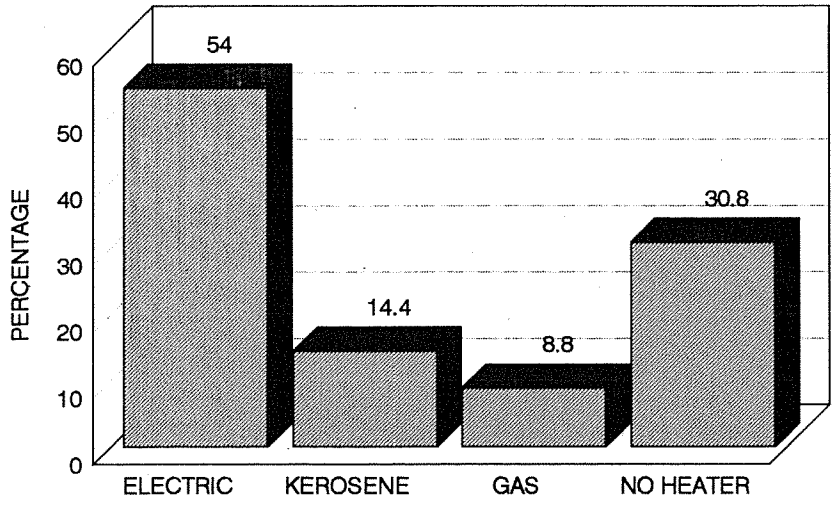
CT scan of the brain was normal and within 24 hours of onset there was no neurological deficit. There was no source of emboli in his heart and carotids. However, this was considered a case of cerebrovascular thrombosis or embolism with ischaemia exacerbated by high CO-Hb levels from cigarette smoking.

Discussion

Out of 305 patients screened, only three patients had evidence of intoxication. In two patients the diagnosis was made by ambulance staff who immediately recognised the intoxication. Only one case, was 'occult' or discovered as a result of the study.

None of the patients with stroke had evidence of intoxication, while none of the patients with intoxication had focal neurological signs. All three patients had impaired or obtunded sensorium. If one had to look just at patients with obtunded sensorium with no focal neurological signs, then 3 out of 29 had intoxication, that is around 10%.

Out of 39 patients admitted with epileptic fits, none had evidence of intoxication and the mean CO-Hb level was not



ACUTE NEUROLOGICAL ADMISSIONS (N=176)
TYPE OF INDOOR HEATING USED. MEAN AGE 66.1 (SD 18.1)
N.B. some patients have more than one type of heater

Box 3

Tips for the clinical diagnosis of CO poisoning

Why is it important to diagnose?

*To identify those patients likely to need 100% or hyperbaric oxygen treatment so as to prevent long term neuro-psychiatric sequelae.

*To recognise a source of CO intoxication before fatal or life threatening exposures can result.

When to suspect it?

*Impaired consciousness or coma in casualty with no focal or lateralizing neurological signs.

*Epileptic fits.

*Headaches, or flue-like illness in the emergency room.

*Unstable angina.

What to ask about?

*Accident sites likely to result in exposure. Garage, warehouse, bathroom, car, fire.

*Multiple simultaneous complaints in co-habitants.

*Type of indoor heating used.

*Take CO-Hb levels blindly when the patient is unconscious and no history is available.

Pitfalls

*Chronic metabolic conditions e.g. diabetes, uraemia, opiate addiction, do not exclude CO intoxication.

*Patients being discharged from the emergency room for minor complaints, rather than being admitted to hospital, could also have CO intoxication.

different from other groups. It is possible in theory that levels of around 20% could cause a fit in susceptible epileptic patients[9]. Heckerling et al. in a similar study described two cases of sub-acute CO poisoning out of 43 cases with epilepsy. However this could have been co-incidental.

The four cases presented here show an interesting spectrum of the neurological presentation of CO poisoning. Case one is a typical, classical presentation[11,12] of an individual with coma, very high CO levels, and quick response to treatment. Case two is very similar in presentation although this time is dealing with a 75 year old man. The exposure was much shorter i.e. 10 minutes instead of sixty, the peak CO-Hb levels much lower, and the recovery much slower, clearly illustrating the elderly man's greater vulnerability to central nervous system hypoxia. This vulnerability is further illustrated in the chronically ill 78 year old woman who lost consciousness probably with peak CO-Hb levels as low as 15%. Probably these chronically ill patients are the patients most likely to have 'occult' presentations.

Case four is a man who sustained two transient ischaemic attacks. Indeed the probable cause was cerebral artery embolism or thrombosis. CO-Hb levels of 10% are usually of no consequence in smokers[13], however undoubtedly during those crucial moments of cerebral ischaemia caused by acute cerebral artery occlusion, a raised CO-Hb level may exacerbate the ischaemia precipitating infarction. To date there appear to be no studies documenting the effects of 100% oxygen therapy in heavy smokers with transient ischaemic attacks.

This study was carried out in an island population with only one acute medical facility. However, it is possible that a number of patients were either not referred to hospital and treated at home, or else they could have been discharged from the emergency department. For this reason a number of cases could have been missed.

Indeed the frequency of carbon monoxide poisoning will depend on the use of fossil fuel made by the community providing the catchment area of the hospital. In this study 34% of patients screened had a potential source of CO within the household. This does not in any way necessarily represent the patterns in the community. However, knowing that fossil fuel use in this hospital population is very widespread shows that one should always keep a high index of suspicion for CO poisoning[14,15], particularly when consciousness is impaired on admission. The type of indoor heating used should be included in routine history taking.

CO-Hb levels should be determined where there is the slightest suspicion of intoxication, particularly in those cases with impaired consciousness when no information is available, as treatment with high concentration oxygen therapy in good time can greatly limit the extent of long term neurological sequelae.

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