

## Carbon monoxide poisoning in the home: recognition and treatment

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Carbon monoxide poisoning should be considered in elderly or housebound patients with recurrent illness, especially during winter months.

It is now five years since Gemelli and Cattani described in the *BMJ* acute carbon monoxide poisoning presenting as gastroenteritis in children.<sup>1</sup> Although carbon monoxide poisoning is less common in the United Kingdom since the conversion from town gas to natural gas,<sup>2</sup> unrecognised cases still occur in the community mainly because of faulty appliances and inadequate ventilation.<sup>3</sup> In mild cases patients often present with a variety of non-specific symptoms and the true cause may go unrecognised. General practitioners and accident and emergency departments may be faced with such cases and should be aware of the circumstances in which they occur. We describe an incident affecting 14 members of a family and review the management of this condition.

### Case reports

An 84 year old woman (case 1) who lived in a flat with her daughter, son in law, and two grandsons was admitted to the local cottage hospital in January 1989 with a vague story of having been unconscious for about 15 minutes. Clinical examination gave normal results and the results of electrocardiography, full blood count, urea and electrolyte measurements, and liver and thyroid function tests were within normal limits for her age. A cerebral transient ischaemic attack was diagnosed and she was discharged home after three days. Six days later she collapsed at home and was found breathless with a few bilateral basal crepitations. Her mild left ventricular failure was treated at home with bed rest and oral diuretics.

At the beginning of February she became much more breathless and was readmitted to the cottage hospital with left ventricular failure, which was confirmed by radiography. She recovered after further bed rest and an increased dose of diuretic and she was discharged home a week later. The next day she developed severe dizzy spells with considerable lethargy but no pain or breathlessness. Physical examination at home showed only that she was tired and drowsy. The possibility of a further transient ischaemic attack was considered but hospital admission was not believed to be justified.

At a follow up visit two days later she was found semiconscious and dehydrated with considerable

tachycardia and hypotension. She was thought to have had a cerebrovascular accident and admission was arranged to Aberdeen Royal Infirmary. The patient's daughter (case 4) then asked for an opinion on her two sons, who she said had been vomiting and feeling very weak for one and a half hours. She said they had had several episodes in the past month, each lasting several hours and settling spontaneously. She attributed these to a "bug" and as examination gave normal results the general practitioner agreed.

An hour later the daughter herself was found lying unconscious on the kitchen floor by ambulancemen who had been called to transfer the first patient to hospital. She had become dizzy, had vomited, and had fallen, striking her head. She was taken to the cottage hospital, where she made a rapid recovery. By this time other members of the family had arrived at the house because of their concern for the condition of the grandmother.

A telephone call was then received at the health centre from a grandson of the first patient to say that he had visited his grandmother and discovered eight people in the house who were semiconscious. Three doctors, four ambulances, and several policemen rapidly converged on the house and found two women and one man deeply unconscious in the kitchen. Three people in the sitting room and two boys in the bedroom were found semiconscious and a man was wandering about the house in a confused state.

Twelve patients were taken to the cottage hospital, where all recovered consciousness. They were given oxygen and taken to the accident and emergency department of Aberdeen Royal Infirmary. The grandmother (case 1), who remained unconscious, had been admitted earlier with a provisional diagnosis of a stroke. Measurement of blood carboxyhaemoglobin concentrations confirmed a diagnosis of acute carbon monoxide poisoning in all patients. The results are shown in the table. A fourteenth patient attended the next morning because he had been unwell with headache, nausea, drowsiness, and dull central chest pain. His carboxyhaemoglobin concentration was 1.2% and as he was free of symptoms by this time no further treatment was given. Four patients were transferred to the hyperbaric medicine unit for treatment with hyperbaric oxygen. The remaining nine were treated in the accident and emergency department with high flow oxygen (10 l/min) using MC masks and were discharged the next day. All patients made a complete recovery.

### Source of poisoning

The source of the poisoning was a gas central heating boiler in the kitchen, and several factors contributed to the production of carbon monoxide. The wall mounted boiler had been installed about four years earlier with a conventional flue with an offset determined by the original construction of the house. This arrangement is less safe than a room sealed appliance incorporating a balanced flue system. The offset of the flue impeded the exit of gases and the acute incident had been precipitated by a gale force wind of 123 knots, the highest ever recorded at a low level station (Metoro-

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Blood carboxyhaemoglobin levels and treatment of 13 patients with acute carbon monoxide poisoning

Case No	Age (years)	Sex	Initial carboxyhaemoglobin concentration (%)	Treatment	Reasons for hyperbaric oxygen
1	84	F	29.6	Hyperbaric oxygen	Comatose on admission
2	49	F	22.0	Hyperbaric oxygen	Unconscious at scene, headache, nausea, vomiting
3	52	M	28.0	Hyperbaric oxygen	Unconscious at scene, chest pain, ischaemia on electrocardiography
4	46	F	23.9	Hyperbaric oxygen	Unconscious at scene, headache, nausea, vomiting
5	50	M	23.3	High flow oxygen	
6	15	M	21.9	High flow oxygen	
7	54	M	27.2	High flow oxygen	
8	53	F	20.6	High flow oxygen	
9	59	M	27.3	High flow oxygen	
10	39	M	27.1	High flow oxygen	
11	25	M	10.7	High flow oxygen	
12	19	M	9.6	High flow oxygen	
13	10	M	15.2	High flow oxygen	

logical Office, Edinburgh, personal communication). The boiler had not been serviced for several years and was burning gas inefficiently and producing carbon monoxide. The danger was exacerbated by double glazing, which considerably reduced ventilation in the kitchen, and the closure of the night vent in the kitchen owing to the high winds. The grandmother spent most of her time in the kitchen and had thus suffered repeated exposure to carbon monoxide.

This type of installation is common and as a result of this incident health visitors and community nurses in the Grampian area have been asked to pay attention to heating and ventilating arrangements when visiting the elderly and infirm and to advise inspection by British Gas if there are potential problems.

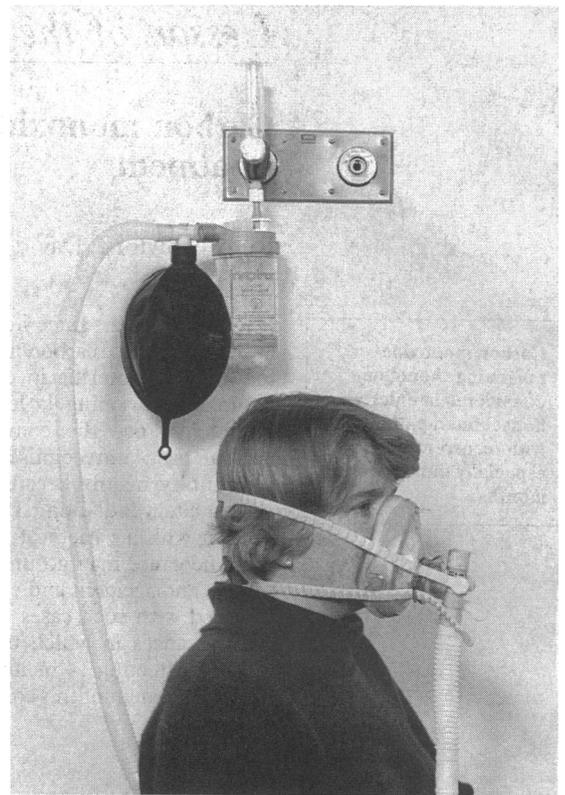
### Discussion

Numbers of deaths from acute carbon monoxide poisoning have fallen since the introduction of natural gas (methane), which does not contain carbon monoxide.<sup>2</sup> Most people, however, fail to appreciate that natural gas requires twice the volume of air for combustion as town gas and that carbon monoxide can be produced by incomplete combustion of natural gas if appliances are poorly installed or maintained or if there is inadequate ventilation or blocking of the flue of the appliance. The condition is underdiagnosed by general practitioners and accident and emergency departments because of the vague non-specific symptoms, especially in people with chronic exposure to low concentrations of carbon monoxide.<sup>3</sup>

Groups particularly at risk include the elderly, housebound, and chronically sick or disabled, who may develop unexplained recurrent vague illnesses, especially during winter months.<sup>4</sup> The cases described here show both the difficulty in diagnosing chronic carbon monoxide poisoning and the potential for mass casualties if the problem is not recognised, as when the condition of the grandmother deteriorated, attracting her relatives to the house, where they were also poisoned.

An incident like this places considerable strain on resources. Four ambulances and one police vehicle were required to take the patients 37 miles to hospital and this removed the emergency ambulance cover for a rural area and tied up medical and police manpower for a long time. Nine patients had to be accommodated in the accident and emergency department and treated overnight in the theatre recovery area and this required extra nursing staff. Limited hyperbaric facilities would have had difficulty in accommodating so many patients at once and there may be no facilities for monitoring acutely ill patients in oxygen chambers. The financial implications of such treatment must also be considered.

This incident highlights some typical features of carbon monoxide poisoning. It tends to occur in bad weather, recurrent episodes are common, several people are often affected simultaneously, and spontaneous recovery occurs when the patient is outside the home. Clinical manifestations are protean,<sup>4,5</sup> but nausea, vomiting, and diarrhoea are common, possibly due to bowel ischaemia.<sup>6</sup> Headaches and dizziness are also found, muscular weakness is common,<sup>7</sup> and cardiovascular symptoms may be present with ischaemic changes shown by electrocardiography.<sup>8</sup> Poisoning is particularly likely to develop when ventilation is blocked to prevent draughts. Ten people died from industrial carbon monoxide poisoning between 1981 and 1985; it was the commonest cause of death by asphyxia or gassing and was usually associated with the use of appliances or compressors in confined spaces.<sup>9</sup> If carbon monoxide poisoning is suspected detailed questions about the heating and ventilation arrangements at home should be asked and blood taken



*Non-rebreathing circuit with non-return valve, oxygen reservoir, and tight fitting mask suitable for treating carbon monoxide poisoning*

for measuring carboxyhaemoglobin concentration. Carboxyhaemoglobin levels above 10% indicate carbon monoxide poisoning. There is a poor correlation, however, between carboxyhaemoglobin concentrations and the severity of the patient's condition because of both elapsed time between exposure and sampling and the effects of administration of varying amounts of oxygen on the elimination of carbon monoxide. Patients with long term exposure to low concentrations of carbon monoxide are much more likely to have normal or low carboxyhaemoglobin concentrations.<sup>7</sup>

The clinical condition of the 13 patients in this study varied considerably despite most of them having similar carboxyhaemoglobin levels. The decision to treat patients with hyperbaric oxygen was therefore based entirely on their clinical state (table).

Carboxyhaemoglobin in the blood reduces the amount of circulating oxyhaemoglobin and also shifts the oxyhaemoglobin dissociation curve to the left, so reducing oxygen availability.<sup>10</sup> This is particularly dangerous in the myocardium, where under normal conditions 60-70% of oxygen is extracted from the blood in the coronary circulation compared with an average of 25% in most other tissues.<sup>11</sup> Carbon monoxide also has a direct toxic effect on cellular respiration as it competes with oxygen for cytochrome  $a_3$ ,<sup>12</sup> and this may help to explain the poor correlation between blood carboxyhaemoglobin concentrations and the actual severity of poisoning.<sup>13</sup> The tissues which are most sensitive to the toxic effects of carbon monoxide are those with the highest metabolic requirements—that is, the nervous system and myocardium—and this explains why cardiac, neurological, and psychiatric problems are common clinical manifestations of carbon monoxide poisoning.<sup>7,8,14</sup>

The elimination half life for carbon monoxide is about 240 minutes when breathing air, 60 minutes when breathing 100% oxygen, and 23 minutes at three atmospheres absolute of oxygen.<sup>15</sup> Most of the masks used in accident and emergency departments are the plastic rebreather type and deliver a maximum

fractional inspired oxygen concentration of 50-60%, which has little effect on elimination of carboxyhaemoglobin and therefore they are inadequate for treating acute carbon monoxide poisoning.<sup>16,17</sup> Nine of our 13 patients were treated using these masks because leak tight masks were not available.

Patients who are treated with high flow oxygen should be given 100% oxygen using a leak tight mask with a circuit which minimises rebreathing (figure) and should have their carboxyhaemoglobin values measured hourly until two consecutive samples contain less than 5%.<sup>18</sup> If recurrent symptoms develop such as headache, nausea, dizziness, or poor memory or concentration then the patient should be referred for hyperbaric oxygen even at a late stage.<sup>18</sup>

Early recognition of carbon monoxide poisoning can be achieved only if clinicians have a high index of suspicion and an awareness of the circumstances in which it is likely to occur.<sup>5</sup> People who visit patients at home should look for risk factors such as blocked vents in rooms or cupboards containing gas appliances, or obstruction to air inlets at the bottom of gas fires by carpets, plinths, or other objects. Falls of soot or birds' nests in chimneys suggest obstruction of the flue. Long floppy yellow flames in gas fires indicate oxygen starvation, and carbon deposition on the radiants of gas fires is the result of incomplete combustion. Carbon monoxide poisoning is preventable by regular maintenance and servicing of all appliances by British Gas, which is willing to carry out inspections. Increased professional awareness is also important, and British Gas has produced an excellent video on carbon monoxide poisoning, which is available on loan to medical practitioners from British Gas Film and Video Library, Park Hall Road Trading Estate, London SE21 8EL.

Chronic carbon monoxide poisoning can be difficult to diagnose, but failure to recognise it may cause substantial disruption of medical services. Treatment consists of administering 100% oxygen using a leak tight non-rebreathing circuit with supportive therapy. Hyperbaric oxygen treatment should be used when appropriate. Failure to use existing facilities has resulted in successful litigation by patients in the United States.<sup>19</sup>

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- Gemelli F, Cattani R. Carbon monoxide poisoning in childhood. *Br Med J* 1985;291:1197.
- Meredith T, Vale A. Carbon monoxide poisoning. *Br Med J* 1988;296:77-8.
- Myers RAM, Goldman B. Planning an effective strategy for carbon monoxide poisoning. *Emergency Medicine Reports* 1987;8:193-200.
- Meredith TJ, Vale JA, Proudfoot AT. Poisoning caused by inhalation agents. In: Weatherall DJ, Ledingham JGG, Warrell D, eds. *Oxford textbook of medicine*. 2nd ed. Oxford: Oxford University Press, 1987:53-9.
- Grace TW, Platt FW. Subacute carbon monoxide poisoning. Another great imitator. *JAMA* 1981;246:1698-700.
- Janes S, Lock B. Carbon monoxide poisoning in childhood. *Br Med J* 1985;291:1725.
- 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.
- Dwyer EM Jr, Turino GM. Carbon monoxide and cardiovascular disease. *N Engl J Med* 1989;321:1474-5.
- Health and Safety Executive. *Blackspot construction: a study of five years' fatal accidents in the building and civil engineering industries*. London: Library and Information Services, 1988:16.
- Roughton FJW, Darling RC. The effect of carbon monoxide on the oxyhaemoglobin dissociation curve. *Am J Physiol* 1944;141:17-31.
- Comroe JH. The transport of oxygen by blood. In: *Physiology of respiration*. 2nd ed. Chicago: Year Book Medical Publishers Inc, 1974:183-96.
- Goldbaum LR, Ramirez RG, Absalon KB. What is the mechanism of carbon monoxide toxicity? *Aviat Space Environ Med* 1975;46:1289-91.
- Somogyi E, Balogh I, Rubanyi G, Sotonyi P, Szegedi L. New findings concerning the pathogenesis of acute carbon monoxide (CO) poisoning. *Am J Forensic Med Pathol* 1981;2:31-9.
- Smith JS, Brandon S. Morbidity from acute carbon monoxide poisoning at three-year follow-up. *Br Med J* 1973;i:318-21.
- Piantadosi CA. Carbon monoxide, oxygen transport, and oxygen metabolism. *Journal of Hyperbaric Medicine* 1987;2:27-44.
- Clark CJ, Campbell D, Reid WH. Blood carboxyhaemoglobin and cyanide levels in fire survivors. *Lancet* 1981;i:1332-5.
- Kindwall EP. Hyperbaric treatment of carbon monoxide poisoning. *Ann Emerg Med* 1985;14:1233-4.
- Myers RAM, Snyder SK, Emhoff TA. Subacute sequelae of carbon monoxide poisoning. *Ann Emerg Med* 1985;14:1163-7.
- Kindwall EP, Goldman RW. Currently accepted: carbon monoxide. In: *Hyperbaric medicine procedures*. 5th ed. Milwaukee: Department of Hyperbaric Medicine, St Luke's Hospital, 1984:90-8.

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## Correction

### The BMA in Edinburgh

An editorial error occurred in this article by Dr Fiona Godlee. The picture entitled "University of Edinburgh Medical School" was in fact a picture of the Court of the Quadrangle, Edinburgh University. Both pictures appear in a series of watercolours by P W Adam that were presented to Joseph Lister in 1895 and are now housed in the Royal College of Surgeons, London.

## MATERIA INDOMEDICA

### Market values

We face a host of problems in trying to keep our people healthy. As poverty, illiteracy, and superstition are still widespread government agencies, themselves plagued by various inadequacies, cannot cope. Enlightened individuals and groups are therefore supplementing the effort to provide "health for all by 2000 AD." Agencies set up by volunteers for helping the underprivileged soon found that in order to be effective it was necessary to interact with the government, which wields immense power.

We have large numbers with severe deformities of the limbs owing to leprosy. They cannot compete with the able-bodied for jobs, be the tasks ever so humble. The state government, therefore, set up sheltered workshops for them. One such workshop in a neighbouring city was handed over to a voluntary organisation and has flourished. A section of the workshop produces cloth, which is of excellent quality and meets the requirements of certain government institutions. Since the workshop was set up by the government, it was expected that the output could be sold to these institutions. Officials were approached and the voluntary organisation was advised that it should obtain a directive from the ministry concerned ordering the institutions to purchase cloth from this workshop. This was

done. All went well for a year. The orders were then discontinued even though the directive remained in effect. When the voluntary organisation approached the institutions it was told that under another directive—this time from the Ministry of Finance—it was mandatory to purchase cloth only after floating a tender and purchasing at the lowest cost. Failure to do so would invite strictures from the auditors. All efforts by the voluntary organisation to get the issue reconsidered failed. It is at a grave disadvantage when it fills in the tender for it finds itself outwitted by unscrupulous agencies that quote absurdly low figures to obtain the contract and then fudge on the quality of cloth supplied in order to make a profit.

This organisation continues striving to make the government see the light. The Ministry of Social Welfare of the same government gives large sums as grants to various agencies for the rehabilitation of the handicapped. Yet this voluntary organisation, running a workshop founded by the government, helping those afflicted by a ruinous disease, and granted the right to sell a product at competitive rates to government institutions, finds itself edged out by the unscrupulous. —SUNIL PANDYA