Severe parkinsonism secondary to carbon monoxide poisoning

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Carbon monoxide from domestic appliances continues to present a hazard, especially in old people whose declining cognitive function makes them forgetful.

CASE HISTORY

A man aged 82 was found collapsed in his house with the gas fire switched on but unlit. There was evidence of coffee-ground vomit. He consumed 25 units of alcohol per week and smoked a pipe. His medical history was unremarkable and he was on no medication. Before this episode he had been completely independent, living in his own first-floor flat. On admission he was semiconscious (Glasgow coma scale 7); he was apyrexial, with a tachycardia of 100 beats per minute and blood pressure of 150/80 mmHg. Abdominal examination was unremarkable and his chest was clear on auscultation. The central nervous system was not examined.

A random blood glucose was 1.4 mmol/L, which was corrected immediately with intravenous dextrose. He was breathing spontaneously with an oxygen saturation of 98% on 15 L/min oxygen by mask. Carboxyhaemoglobin was 42%. The initial full blood count revealed a haemoglobin of 15 g/dL and his coagulation screen was normal. Intensive care staff suggested continued treatment with high-flow oxygen by mask.

The working diagnosis was carbon monoxide poisoning. Later that day he became hypotensive (80/40 mmHg) and was treated with intravenous colloid. Within 48 hours of admission his condition had stabilized, with a Glasgow coma scale of 13/15. He was normotensive and his pulse rate was 62 and regular. He was speaking a few words but was still drowsy. His haemoglobin had now fallen to 12.8 g/dL. He received oxygen by mask for the next couple of days and underwent physiotherapy. His mobility continued to improve and he was discharged ten days after admission, mobilizing with a stick.

Six days after hospital discharge his condition deteriorated. He was less responsive, poorly mobile and drowsy. He was admitted to a medicine for the elderly assessment ward where he was seen to have developed signs of parkinsonism, together with both faecal and urinary incontinence. He was bradykinetic, exhibited marked rigidity and had poverty of facial expression. He required help with transfers, dressing, grooming and bathing. The only other significant findings were slight blurring of the optic disc margins and a score of 1/10 on Mental Status Questionnaire examination.

A computed tomographic scan of the head showed mild atrophic changes and a magnetic resonance imaging scan revealed an increased signal in the periventricular white matter in keeping with ischaemia or toxic demyelination (see Figure 1). The basal ganglia appeared unaffected. The working diagnosis was carbon-monoxide-induced parkinsonism.

He was started on both Madopar (levodopa/benserazide) and pergolide up to maximum dosages with no clear therapeutic benefit. Dexamethasone was also tried without obvious effect. The case was discussed with the local specialist in hyperbaric oxygen therapy and the patient was...

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Figure 1 Magnetic resonance imaging scan showing increased signal in periventricular white matter
given a four-week course of daily sessions of oxygen at 2 atmospheres for one hour, six days a week.

His rigidity was less marked following hyperbaric oxygen treatment. However, the mild improvements were sustained for only a few hours and the signs of severe parkinsonism returned shortly after treatment. Eventually, he was transferred to a continuing-care ward, dependent in all aspects of nursing care.

COMMENT
This case illustrates the hazards of carbon monoxide poisoning and in particular delayed neurological deterioration. The severity of carbon monoxide poisoning should be judged from clinical findings since the level of carboxyhaemoglobin is a poor indicator. This patient had clear early evidence of carbon monoxide poisoning; immediate treatment with hyperbaric oxygen therapy\(^1,2\) might have prevented subsequent neurological deterioration. Several studies have shown that hyperbaric oxygenation can reverse both the acute\(^3\) and the delayed\(^4\) effects of carbon monoxide poisoning. The time course in this patient was characteristic for the delayed effects of carbon monoxide poisoning. Nearly all such patients resume full activity in four or five days, followed by a clear and seemingly normal interval of two to ten days. Patients then become abruptly apathetic and confused, with walking changing to a halting shuffle and diffuse skeletal muscle rigidity\(^5\). Deaths may occur as late as one month after poisoning.

Hyperbaric oxygen therapy is 100% oxygen delivered at two to three times the atmospheric pressure at sea level\(^6\). It is the fastest method of reversing the potentially life

## Excision of a false left ventricular aneurysm

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Myocardial rupture complicates 3% of all myocardial infarctions and is the cause of death in 17% of fatal infarcts\(^1\). By comparison, false left ventricular aneurysm is uncommon, with an unknown prevalence and natural history\(^2\). It results from rupture of a densely adherent full

thickness infarct, with containment of the resulting haematoma.

## CASE HISTORY
A man aged 49 was referred in April 1997 with a three-week history of progressive dyspnoea on exertion and epigastric pain radiating to his neck and back. During the subsequent 18 months he became more troubled, with paroxysmal nocturnal dyspnoea and easy fatigability. He had undergone 4-vessel coronary bypass surgery in February 1990.

On admission in October 1998 electrocardiography showed T wave inversion in the lateral leads and a chest radiograph revealed an asymmetrically enlarged left ventricle. Echocardiography demonstrated a large inferolateral aneurysm with a layer of mural thrombus. On cardiac angiography his native coronary arteries were occluded but a left internal mammary artery graft to the left anterior descending artery was patent with good

## REFERENCES