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Carbon monoxide poisoning

Carbon monoxide is a tasteless, odourless, colourless, and non-irritating gas produced by incomplete combustion of organic materials. It is also produced in man during the catabolism of haemoglobin. The normal endogenous production of carbon monoxide is sufficient to saturate 0·4-0·7% of the body's haemoglobin—that is, 0·4-0·7% carboxyhaemoglobinaemia—at rest,[1] but a national survey in North America found 1-2% carboxyhaemoglobinaemia in urban non-smokers as a result of environmental exposure and 5-6% in smokers (cigarette smoke contains about 4% carbon monoxide).[2]

Although mortality from carbon monoxide poisoning has fallen since natural gas was substituted for coal gas, about 1000 people die annually in England and Wales from this cause. Most do not reach hospital alive: in 1985, 1365 deaths from carbon monoxide poisoning were reported in England and Wales whereas only 475 admissions and 10 deaths were recorded in hospitals.[3,4] In Britain carbon monoxide is the main cause of death from poisoning in children.[5] In the United States more than 3800 people are estimated to die annually from carbon monoxide poisoning, and more than 10 000 people miss at least one day of work because of a sublethal exposure.[6]

Common sources of carbon monoxide are car exhaust fumes[7] (a lethal concentration can be reached in a closed one car garage in 10 minutes[1]); improperly maintained and ventilated heating systems[8]; smoke from all types of fires; and household gas (if appliances have not been converted for natural gas). Inhalation of methylene chloride (as in paint strippers) may also lead to carbon monoxide poisoning because of hepatic metabolism.[9]

Carbon monoxide has an affinity for haemoglobin 200-250 times that of oxygen, and the symptoms and signs that follow inhalation of carbon monoxide have been supposed to result from tissue hypoxia. Carbon monoxide combines with haemoglobin to form carboxyhaemoglobin, reducing the total capacity of the blood to carry oxygen and shifting the oxygen dissociation curve to the left.[10] The binding of one or more carbon monoxide molecules to haemoglobin also induces an allosteric modification in the remaining oxygen binding sites: the affinity for oxygen of the remaining haem group is increased, and the oxygen dissociation curve is distorted as well as being shifted to the left.[10] Tissue anoxia is thus far greater than would result simply from loss of oxygen carrying capacity.[11] More recent experimental evidence[12-19] suggests that carbon monoxide toxicity may also be due to inhibition of cellular respiration as a result of reversible binding to other haem proteins, particularly cytochrome oxidase (cytochrome a, a3), where it acts in the same way as cyanide. Although inhibition of cellular respiration has only been thought to occur in conditions of established tissue hypoxia (because the affinity of cytochrome oxidase for oxygen is much greater than that for carbon monoxide[20]), this view has recently been challenged.[19]

The clinical features of carbon monoxide poisoning are protean,[21] and the severity of poisoning depends on the concentration of carbon monoxide in the inspired air, the length of exposure, and the general health of the exposed person.[22-24] Infants, the elderly, and patients with cardiovascular disease, anaemia, lung disease, and an increased metabolic rate are at greater risk.[1] The symptoms of repeated exposure to low concentrations of carbon monoxide include headache, fatigue, difficulty in thinking, dizziness, paraesthesiae, chest pain, palpitations, visual disturbance, nausea, diarrhoea, and abdominal pain.[25] Not only may performance at work and school deteriorate but also the symptoms may be mistaken for other illnesses.[26] The diagnosis should thus be considered when patients present with vague and non-specific but persistent symptoms. Confirming the diagnosis analytically is often difficult as carboxyhaemoglobin concentrations (or the carbon monoxide content of exhaled air) may be low or undetectable because of the time between exposure and presentation. If there is any doubt about the relevance of individual carboxyhaemoglobin concentrations, particularly in heavy smokers, British Gas should be contacted so that gas appliances may be checked; flues and chimneys may also need to be swept. (British Gas produce an excellent video on carbon monoxide poisoning for medical and nursing audiences available from the British Gas Film and Video Library, Park Hall Road Trading Estate, London SE21 8EL.)

The features of acute carbon monoxide poisoning are better known and more easily recognised than those of chronic exposure to low concentrations. The diagnosis is

often suggested by the circumstances in which the patient is found. The symptoms, signs, and prognosis of acute poisoning correlate poorly with the degree of carboxyhaemoglobinaemia measured on arrival at hospital.[27 28] Nevertheless, carboxyhaemoglobinaemia of less than 10% is not usually associated with symptoms, and 10-30% may cause only headache and mild exertional dyspnoea,[24] though presentation with gastroenteritis should not be forgotten.[29] Coma, convulsions, and cardiorespiratory arrest may occur if carboxyhaemoglobinaemia exceeds 60%.

Neuropsychiatric problems may develop insidiously over weeks after recovery from carbon monoxide intoxication.[30 31] These problems include intellectual deterioration, memory impairment, cerebral, cerebellar, and mid-brain damage—for example, Parkinsonism and akinetic mutism—and changes in personality typified by increased irritability, verbal aggressiveness, violence, impulsiveness, and moodiness.[32-46] Up to two fifths of patients develop memory impairment, and a third suffer late deterioration of personality.[34] Although there are no pathognomonic features of carbon monoxide poisoning to be found at necropsy,[37] certain areas of the brain are particularly vulnerable. Bilateral necrosis of the globus pallidus is common[38] and may be shown by computed tomography[39 40] and magnetic resonance scanning.[41] The cerebral cortex, hippocampus, and substantia nigra may also be affected.[38]

Patients who have been poisoned by carbon monoxide should be removed from exposure and given 100% oxygen using a tightly fitting facemask. Endotracheal intubation and mechanical ventilation may be needed if the patient is unconscious. Carboxyhaemoglobin readily dissociates when the partial pressure of carbon monoxide in the alveolar air falls below that in mixed venous blood: the elimination half life of carbon monoxide is reduced from about 250 minutes when breathing air to just under 50 minutes when 100% oxygen is given at sea level.[42] Hyperbaric oxygen at 2·5 atmospheres pressure (250 kPa) further reduces the half life to 22 minutes[42] and also increases oxygen dissolved in the blood to a concentration sufficient to meet the needs of the body even without functioning haemoglobin.[43 44] Oxygen may also protect against inhibition of cytochrome oxidase and accelerate reactivation—as it does in experimental cyanide poisoning.[45 46]

Treatment with hyperbaric oxygen was first used successfully in Glasgow in the 1960s,[47] and subsequent clinical experience has confirmed that it can reduce morbidity (especially that due to delayed neuropsychiatric charges) from 43%[34] to less than 5%.[27 35 48] It should be used even if patients present late in those with carboxyhaemoglobinaemia of more than 40% or a history of loss of consciousness, persistent neurological deficits (including coma), or cardiac abnormalities.[27 48-50] Unfortunately this policy has only rarely been adopted in Britain because of the few centres with suitable hyperbaric facilities. It is most important, however, that all accident and emergency and medical staff should know the location of the nearest compression chamber. (This information can be acquired urgently in Britain by contacting the duty diving medical officer on 0705 822351 extension 41769 during working hours, or the duty staff officer to flag officer, Portsmouth, on extension 22008 outside working hours.)

Oxygen must be given (by whatever means) until the degree of carboxyhaemoglobinaemia is less than 5%. General symptomatic and supportive measures may also be required. Corticosteroids,[51] mannitol,[51] and hyperbaric oxygen[52] have all been advocated for treating cerebral oedema but are not of proved value in humans when the oedema results from carbon monoxide poisoning.

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Psychiatry: private and public provision

Conditions seem favourable for further expansion in private acute psychiatric care. The government is encouraging managers of district and regional health authorities to seek closer cooperation between the National Health Service and private providers, and the generally low standard of NHS provision is undoubtedly an incentive for those who can afford it to seek private treatment. Counselling for people with psychiatric problems is already highly commercialised. Three questions arise from the expansion in private psychiatry: What is happening in the private psychiatric sector? What effects are current resource constraints having on NHS psychiatry? What form of coexistence between private and public provision will prove most effective and efficient?

The market in private acute psychiatric treatment is still small,[1] largely because people with mental illness descend down the social scale. In addition, insurance risks are high, and until recently private treatment was provided mainly in a few hospitals remote from most of the population. At the same time, private practice among NHS consultants has remained undeveloped, focusing mainly on the outpatient treatment of patients with neuroses.

Now the number of consultant psychiatrists working in the private sector and the number of private hospitals are increasing, predominantly in the more affluent south. Unlike in the NHS, morale is high and the facilities are usually impressive. In 1980 there were 25 full time private psychiatrists in England and Wales; by 1986 there were 60. In addition, 300 consultants had by 1986 developed a substantial commitment to private practice.[2] There are over 20 private psychiatric hospitals with almost 2000 beds (10% occupied by chronically ill patients).[2] In all, about 4% of beds for patients with acute mental illness are private (compared with about 10% of beds for elective surgery).[1]

Commercial and financial information on private medicine is difficult to obtain,[3,4] but it has been estimated that in 1984 private acute psychiatry was generating a return of around £25m annually (compared with about £500m for acute general hospital care).[1] Since the early 1980s the domination of this market by British charitable institutions has been increasingly challenged, particularly by a few American multinational companies, which are now responsible for about half the total cash turnover.[3] Their investment and the resulting competition has affected the pattern of provision. For example, consultant private practice has moved away from the conventional "closed" model of psychiatric inpatient care, with treatment being provided entirely by full time hospital staff, to an "open" model in which local consultants have admitting rights.

How resource constraints are affecting NHS psychiatry is uncertain and likely to remain so—mainly because the necessity for, and sufficiency of, psychiatric treatment is hard to quantify. Furthermore, ideological differences among both providers and consumers lead to contradictory judgments,[5] as can be illustrated by events at the Bethlem Royal Hospital and the Maudsley Hospital and at the Institute of Psychiatry. Last year a group of doctors and academics wrote to *The Times*: "The government's conjoint undermining both of the universities and the NHS constitutes an immensely threatening pincer movement on this centre [and] is a devastating attack on the core of British psychiatry and on the future care of a vulnerable sector of the community. Our financial crisis has meant that of the last eight academic posts to fall vacant at the Institute, only one could be filled, and three university chairs have been lost. The Bethlem/Maudsley are at the same time faced with a £400 000 per year deficit which will require drastic cuts in patient services."[6] This year the chairman of the Joint Hospital Special Health Authority replied to subsequent similar criticism[7]: "The financial crisis is not the same and the deficit has been reduced from £1.4 million to £367 000. Cuts are not inevitable and are indeed not foreseen."[8] He added that since 1979 the joint hospital had opened four new units at a total revenue increase of £3·3m; in addition, a new unit for computed tomography opened last year, and building has started on a first block of new wards (at a capital cost of £3·7m).

The eventual mix between private and public mental health care will be resolved by market forces and empirical means. Meanwhile, a key NHS objective continues to be to provide cost effective treatments that are flexible and responsive to changing circumstances. Psychiatric treatment is heterogeneous, and the therapeutic value of most of its components has not been proved. The NHS thus cannot hope to provide all specialist psychiatric services, and Professor Sydney Brandon asked in his recent report on a "subversive foray into private practice": "Do we have to recognise that certain kinds of care such as some or all of secure accommodation, the management of the severely head injured, psychosexual counselling, intensive psychotherapy or long term social skills training cannot be provided as basic care and have to be contracted out or sought privately?"[2]

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