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CARBON MONOXIDE POISONING

Alan Wood

Here is an outline of the problem of carbon monoxide (CO) poisoning in Tasmania. These figures do not cover accidental deaths.[1,2] It should be noted that a lethal concentration of CO can be reached in a closed garage in ten minutes.[3]

	1981	**1984**
Total Suicides	63	52
Gassing and other Vapours	14	N/A
Male to Female	5 to 1	3 to 1

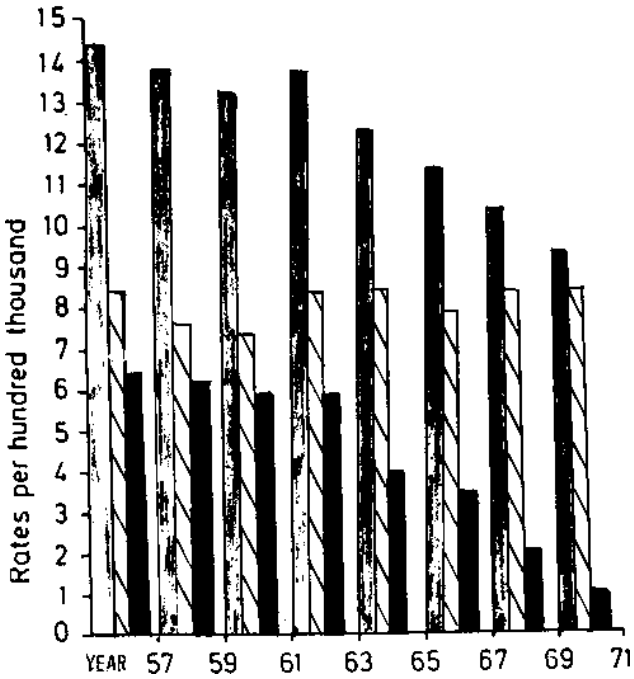
Figure 1 shows the reduction of the number of people using gassing as a means of suicide in Britain since the introduction of North Sea gas[4]. It makes the point that the availability and lethality of methods of suicide are important determinants of the numbers dying by these means.

More surprising is that when one looks through the older literature to determine outcome of survivors of poisoning with carbon monoxide it is very difficult to find good articles. This poses a problem as there is not a good yardstick by which the outcome of populations treated by hyperbaric oxygen can be measured. A commonly quoted article[5] states that of 63 survivors (no hyperbaric treatment) followed up at three years, 43 % had memory impairment, 33 % had deteriorated personality, 11 % had what was described as "Gross damage".

Reviewing more recent articles serveral themes emerged as I went through the data. The first issue that arises is the usefulness of carboxyhaemoglobin levels. It seems clear from the literature that there is a lack of correlation between carboxyhaemoglobin levels and clinical status[6]. Hence these levels should not override a full clinical assessment, and it may be quite dangerous to allow this one parameter to dictate treatment. Tissue levels of carbon monoxide are what needs to be measured and this cannot be performed in a clinical laboratory. Higher cognitive functions (such as memory, attention, concentration, calculation, etc.) may be more easily assessed and reflect impairment of the high metabolic rate tissue of the central nervous system (CNS).

FIGURE 1

UK SUICIDE RATES BY MODE OF DEATH 1957-1971



Solid columns are carbon monoxide deaths, hatched columns are non-carbon monoxide deaths and stippled columns are total deaths from suicide. The graph has been constructed from data in reference 4.

A second point raised in the literature is the great usefulness of hyperbaric oxygen both for acute and delayed presentation. The ethical considerations of a control group comparison of such an effective treatment have been addressed to some extent.[7]. In 1985, 215 poisoned patients were divided into good and poor groups. Those in the good group were conscious, with lower carboxyhaemoglobin levels and well enough to be psychometrically tested. They were given normobaric oxygen. The poor group were unconscious with high carboxyhaemoglobin levels and were treated with hyperbaric oxygen.

As a medical student I recall being taught Haldane's view that the toxicity of carbon monoxide was due entirely to its power to combine with haemoglobin in the red blood cells thus putting them out of action as oxygen carriers. This could be called the simple hypoxia theory. However it does not fully explain all of the clinical findings. A key point in the literature is the work of Goldbaum.[9] He transfused carboxyhaemoglobin into dogs with little ill effect. This highlights the point that tissue toxicity is probably one of the major determining factors in outcome and there is no easy way of measuring that.

TABLE 1

	No sequelae	**Died**	**Sequelae**	**Sequelae after delayed hyperbaric therapy**
Poor group (Treated with hyperbaric oxygen)	89 %	9 %	2 %	Not applicable
Good group (Treated with normobaric oxygen)	88 %	0	*12 %	0

* This group of the good population was then given delayed hypoerbaric oxygen and all recovered

Two points should be made. Firstly giving "mildly" poisoned cases normobaric oxygen produces a large number of sequelae. Secondly, the sequelae so produced can be successfully treated by hyperbaric oxygen therapy.

A similar article[8] said the same sort of thing, pointing out that long delays in starting treatment were not crucial although they are not advantageous. Purely psychiatric presentations of carbon monoxide poisoning offer a wide range of symptoms. Reviewing the literature revealed a lobe by lobe picture of problems. More frequently listed were dementia, psychosis and Parkinsonian syndromes. More rarely Korsacoff like syndromes (an amnestic disorder usually associated with alcoholism, with profound short term memory disturbance) were mentioned while other presentations included cortical blindness and multiple sclerosis-like pictures. The list is quite extensive and often these patients present in quite a bizzare fashion. For instance an elderly man was twice taken to casualty by neighbours complaining of visual hallucinations and disorganised behaviour. He was sent home on two consecutive evenings, and then on the third evening he was admitted. It transpired that he was being poisoned each night by his caravan's gas water heater.

The picture of chronic or sub-acute poisoning seems to be quite complex. The relatively slow induction would seem to be the mode of poisoning most likely to result in neuro-psychiatric presentations and again carboxyhamoglobin levels are not useful (Table 2).

A complex point is that the relative affinity of tissue cytochrome systems for oxygen is higher than for carbon monoxide. Hence tissue hypoxia plus carbon monoxide are required to poison the tissue cellular system. Simply having carboxyhaemoglobin bound tightly to haemoglobin in red blood cells may not necessarily mean that the tissues become poisoned. This may explain the complexity of clinical pictures encountered. So simple hypoxia due to carbon monoxide and tissue toxicity acting together is better able to explain the clinical findings including the value of hyperbaric oxygen in treatment. This provides the most rapid

TABLE 2

DIFFERENCE BETWEEN AVERAGE CARBOXYHAEMOGLOBIN LEVELS IN UNCONSCIOUS PATIENTS WHO DIED AND THOSE WHO SURVIVED[6]

		Average COHb	**Range of COHb**
Died	11	30.8 %*	19.7 - 49 %
Survived	33	29.3 %*	1.1 - 53 %

*****The difference does not reach statistical significance.

and logical method to displace carbon monoxide from the tissues as well as from haemoglobin. This will minimise tissue damage to high metobolic rate tissues. Clinically most cases present with damage to the high metabolic rate tissues of the CNS or the myocardium.

Case history

I would like now to present a case history of a twenty seven year old male with no past psychiatric history, physically very fit and in good health. My involvement started 24 hours after admission when consultation was requested with Liaison Psychiatry. The history, which was pieced together from police reports and contacts with friends and relatives, revealed that he was last seen at 9 a.m. and was found unconscious in his car in an isolated place at 5 p.m. He was slumped in the car with the ignition turned on, but the engine not running and the petrol tank half full.

There was a vacuum cleaner hose from the exhaust into the car. The rescuers did not note a strong smell of exhaust fumes in the car interior which is most unusual in these cases. When one opens a car door in this situation usually the fumes are so overpowering that one almost faints at the first breath. So the question was raised as to the duration and intensity of exposure to exhaust fumes.

In casualty he was found to be agitated, irritable and combatitive when examined or interferred with. He was lapsing in and out of consciousness but always capable of responding purposefully to pain. The carboxyhaemoglobin level was low at 2.5 %. This did not tie in with the CNS clinical state. He was placed on normobaric oxygen. Psychiatric management the next day focussed on the request to manage this serious suicide attempt. His mental state had not improved, and now he was not responding purposefully to pain and had decorticate posturing with extensor plantar responses. The confusion at this time centered around the question of poisoning with other substances and perhaps it was considered by non-psychiatrists that he may have a dissociative disorder of some sort. Hyperbaric therapy was suggested. However because of the clinical confusion which was hoped to be resolved by investigation, as well as problems with the family who initially rejected the idea of hyperbaric therapy because it was unproven, it took eight days before he came to treatment. Logistical problems arose because this man had been agitated in casualty and had to be restrained so the question of safety of patient and attendant in our small tank arose.

As a result of these and other problems the time lag between what we would have liked to happen and what did happen was eight days. In response to treatment his level of alertness and arousal improved a little, however there was no return of cortical function. Further problems with the family resulted in a further six days elapsing before the next treatment. There was no significant change after this treatment. He was eventually placed in a rehabilitation hospital. The outcome five months after the poisoning was that he is able to vocalise unintelligable sounds. He is said by the staff to recall, very infrequently, their names. He can not communicate any of his needs nor care for himself. He is violent occasionally, if uncomfortable with a full bowel or bladder, and hits out at the staff. Previously his CAT scan was normal but now it reveals marked cortical atrophy and increased ventricle to brain ratios and infarction of the basal ganglia.

This man has profound dementia and long term placement out of the rehabilitation setting in a nursing home is being looked for. So despite hyperbaric treatment (which was delayed) this patient did not do at all well. It is a very interesting example of how the very clear literature often does not seem to reflect how things are in real life.

Summary

The diagnosis of carbon monoxide poisoning requires a fairly high index of suspicion particularly with neuropsychiatric presentations. However most cases have organic features of cognitive impairment, impaired attention, concentration and memory. Carboxyhaemoglobin levels do not indicate the severity of the poisoning as this case shows well. Carbon monoxide levels in plasma might be more important but cannot be measured, but dysfunction in high metabolic rate areas such as the brain and myocardium can be measured[10], but by different means, such as cognitive testing and mental state examination. Poor emergency room state does not preclude full recovery and it is claimed that minor delays in instituting treatment are not crucial, although I have my doubts about that. Hyperbaric therapy produces fewer end point defects and is also effective on defects left by normobaric treatment.

Perhaps the final point is that a crucial part of assessment of a patient is the assessment of higher mental functions. These may be one of the most sensitive indicators of tissue toxicity that we have available. The other indicator, the ECG, seems less sensitive.

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TREATMENT OF CARBON MONOXIDE POISONING

Chris Lourey

Introduction.

The subject of carbon monoxide poisoning really has a rich history and its toxic affects were actually first noted by one of the founding fathers of modern medicine, Claude Bernard, in 1857.[1,2] Most of what I say this morning is really a compilation of evidence and recommendations that have been progressively made over the last sixty years which probably indicates that medicos are pretty slow learners.

My personal interest in carbon monoxide was enhanced when the Victorian section of the South Pacific Underwater Medicine Society gathered all the necessary data to make the ministerial submission to get the hyperbaric facilities for the State of Victoria established at the Alfred Hospital. This required an assessment of projected case load, clinical efficacy and cost effectiveness of hyperbaric oxygen (HBO) therapy pertinent to the State of Victoria. The clinical indications were category 1 indications as recognized by most authorities.[1]

The analysis of the data revealed a significant disparity in the incidence of carbon monoxide poisoning in Victoria when compared with other demographic centres, and unless Victoria is very different (which it is, if you listen to New South Welshmen) at best carbon monoxide poisoning in the State of Victoria was being inadequately treated. But what I think was that it was not even being recognized. A conservative estimate depending on the literature source and areas is a case load of somewhere between 20-50 cases per million people per year. In 1985 in Victoria there were 3 reported cases. In addition accruing clinical evidence challenges the clinical efficacy of treatment with normobaric oxygen as is recommended in standard texts. So in this context I will present a brief overview.

The disease

Carbon monoxide is a colourless, odourless, non irritating gas produced by the incomplete combustion of carbonaceous materials. The commonest sources are fire, automobile exhaust, petrol or propane engines, especially when operating in confined spaces, and generally, the colder the climate the greater the danger of carbon monoxide poisoning when operating engines in confined spaces, charcoal burners and faulty furnaces. Natural gas does not contain carbon monoxide, however if combustion is incomplete because of a bird's nest or a dead possum or accrued rubbish in the flue, carbon monoxide production will occur. Most of the commercial paint strippers contain methylene chloride which is readily absorbed through the skin and mucus membranes and metabolised into carbon monoxide.

The classical text book description of carbon monoxide poisoning, cherry red mucus membranes and skin, is not commonly seen and therefore is a most unreliable clinical aid. Because the initial symptoms of toxicity mimic other disorders such as influenza, acute confusional states and coma, a very high index of suspicion is necessary for diagnosis.

The strong possibility of dual pathology such as cardiac disease also must be considered. What is very interesting is that the highest percentage of deaths in fires (which the general population think is due to burns) is in fact due to carbon monoxide poisoning, and carbon monoxide poisoning in combination with coronary artery disease and burns. I ask the question rhetorically "In how many patients when admitted to a hospital for burns is a carboxyhaemoglobin level done ?"

Pathology

The primary toxic effects of carbon monoxide are those of tissue hypoxia. The biochemical lesion involves a preferential binding of carbon monoxide with haemoglobin, myoglobin and cytochrome oxidase A3, all are pyrole iron complexes.

Evidence also suggests that, in the brain, altered mitochondrial activity is not immediately reversible with re-perfusion and re-oxygenation.[3] This explains some of the symptoma-