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UNINTENTIONAL CARBON MONOXIDE POISONING FROM CHARCOAL BARBECUES

George Jelinek

Summary

In two weeks in the winter of 1993 eight members oftwo families suffered paoisoning with carbon monoxide produced by charcoal barbecues used indoors to provide heating. Following these incidents a review of the literature was carried out. This paper presents clinical details and reviews the literature. Recommendations are made for public health measures to combat this unintentional form of CO poisoning.

Introduction

Carbon monoxide (CO) is an agent frequently used for deliberate self-harm. Poisoning with CO is the commonest cause of death by completed suicide in industrialised nations.[1-3] Unintentional CO poisoning is also common although the incidence is more difficult to determine. Many cases are probably unrecognised.[4-6] Dolan suggests that it is the protean nature of symptoms of CO poisoning which causes its true incidence to be grossly underestimated.[7] It is likely that many cases are also missed at autopsy because the pathological lesions are non-specific.[8] Common mechanisms include leaky motor vehicle exhausts, faulty home heaters, and inadequate ventilation around appliances producing CO.[1,9-11]

This paper describes two non-English speaking families who presented to the Emergency Department of Fremantle Hospital approximately two weeks apart in the middle of winter 1993. Eight people, six in one family and two in the other, suffered CO poisoning following indoor use of a charcoal barbecue for heating. There have been occasional reports of CO poisoning in similar circumstances in the medical literature. In January 1994, a large series of 79 patients was reported from Washington.[12] CO poisoning from charcoal barbecues has not previously been described in Australia.

The cases reported here illustrate the ease with which accidental CO poisoning can occur. This leads to a review of the epidemiology and effects of unintentional CO poisoning with particular emphasis on chronic exposure which has thus far not been discussed in detail in the literature.

Case reports

FAMILY A

This family of six (mother 24 years old, father 37 years old, two daughters 11 and 9 years old, and two sons, 10 and 6 years old) presented to the Emergency Department at Fremantle Hospital at 0400 hours on 30 June 1993. They had taken a charcoal barbecue indoors to use as a heater overnight after cooking the evening meal. In the early hours of the morning, the mother lost consciousness on the way to the toilet. On regaining consciousness after an estimated period of four minutes, she alerted the rest of the household. Her 11 year old daughter was roused but lost consciousness twice for a total of 15 minutes and vomited once while being taken outside the house. The six

year old boy lost consciousness once for approximately five minutes and the nine year old girl once for one minute. The father and the other boy left the house uneventfully.

On arrival in the Emergency Department, only the 10 year old boy was asymptomatic. The mother felt very tired and the father complained of a severe headache and felt drowsy. The 11 year old girl felt drowsy, and the nine year old girl complained of a headache and drowsiness. The six year old boy had a slight headache.

There was no significant past medical history apart from a history from the six year old boy of mild asthma treated with salbutamol inhaler. None of the family smoked cigarettes. The family was Vietnamese and had been in Australia six months. The children spoke sufficient English to allow communication.

TABLE 1

	Age	MMSE	COHb	LOC
Father	37	30	4 %	No
Mother	24	26	2 %	Yes
Girl	11	28	4%	Yes
Boy	10	30	*	No
Girl	9	30	4%	Yes
Boy	6	*	3 %	Yes

* not done

Age (years), mini-mental state examination results (out of 30), carboxyhaemoglobin levels (%), and whether there was loss of consciousness (LOC) for members of Family A.

Physical examination of all six patients was normal. Mini-mental state examinations (MMSEs) were performed with the assistance of the children and results are listed in Table 1. These are scored out of a possible 30 according to the standard format of Folstein et al.[13] Carboxyhaemoglobin (COHb) levels were done on five of the patients and are also listed in Table 1. No blood was taken from the 10 year old boy who was terrified of needles. Electrocardiographs performed on both adults were normal.

Treatment was commenced with 100% oxygen by tight-fitting mask, non-rebreathing valve and reservoir bag. All six patients were treated that morning with hyperbaric oxygen on a standard 18 m table, breathing 100% oxygen at an absolute pressure of 2.8 atmospheres. The indication for treatment was loss of consciousness for the three females and the six year old boy. The other two patients were treated at the same time as they were felt to have received a similar dose of CO, and the chamber could accommodate them. They received a second 18 m treatment as outpatients next day. All were discharged symptom-free.

FAMILY B

A 42 year old woman presented to the Emergency Department at Fremantle Hospital at midnight on 13 June 1993. She had cooked indoors with a charcoal barbecue and left it burning inside as heating. Her 13 year old son had also been in the house.

TABLE 2

	Age	MMSE	COHb	LOC
Mother	42	28	12.0%	Yes
Boy	13	23	4.4%	No

Age (years), mini-mental state examination results (out of 30), carboxyhaemoglobin levels (%), and whether there was loss of consciousness (LOC) for members of Family B.

After the barbecue had been inside about three and a half hours, she began to feel unwell with headache, dys-pnoea and pounding in her ears, and she vomited four times. She telephoned her sister and lost consciousness briefly on her arrival. There was a past history of bronchiectasis and asthma, treated with salbutamol by inhalation. She was a non-smoke born in El Salvador.

Physical examination was normal. MMSE result with interpreter assistance was 28/30 and COHb level was 12%. White cell count was 17.1×10^9/L.

She was treated with 100% oxygen by tight-fitting mask, non-rebreathing valve, and reservoir bag. While awaiting hyperbaric oxygen therapy, her 13 year old son was contacted by telephone. He complained of headache, palpitations, difficulty with coordination and generalised weakness. He was advised to present to the Emergency Department.

On arrival in the Emergency Department, there were no abnormal physical findings. MMSE result was 23/30 and COHb level was 4.4%.

For similar indications to Family A, both patients were treated with hyperbaric oxygen on an 18 m table, followed by a second 18 m treatment as outpatients next day. Both were discharged symptom-free.

Background and literature review

The literature on CO poisoning due to charcoal barbecuing is reviewed. To put this problem into perspective, the epidemiology of other causes of unintentional CO poisoning is examined. Accidental exposure to CO as described in this report can clearly occur regularly in

TABLE 3

REPORTS OF UNINTENTIONAL CO POISONING DUE TO CHARCOAL COMBUSTION

Authors	Year	Number of patients	COHb levels	Ethnic background
		Charcoal barbecues		
Anonymous[14]	1966	1 fatal	Not stated	Not stated
Wilson et al.[15]	1972	5 fatal	65%	Not stated
		2 non-fatal	71%	
			82%	
Fain & McCormich[16]	1988	2 fatal	30%	Not stated
			41%	
Baron et al.[11]	1989	1 fatal	Not stated	Not stated
Geehr et al.[17]	1989	Not stated (<22)	Not stated	Not stated
Gasman et al.[18]	1990	12 non-fatal	6.9%	Yes
			15.1%	
			17.1%	
			17.4%	
Sternbach & Varon[19]	1990	5 non-fatal	18.5%	Yes
			21.1%	
			25.5%	
			31.9%	
			38.3%	
Hampson et al.[12]	1994	79	Average 21.6%	73%
		Charcoal fires in enclosed spaces		
Finck[8]	1966	2 fatal	Not stated	Not stated
Wilson et al.[15]	1972	2 fatal on review	Not stated	Not stated

certain situations. This leads to a consideration of the circumstances in which such exposure to CO may occur, particularly for certain occupational groups, and its possible effects.

CO poisoning due to charcoal barbecuing

There have been several reports in the literature of CO poisoning due to charcoal briquets burned indoors. A report in 1966[14] described several fatalities from CO poisoning in confined spaces. One of these was the case of a man who was using a charcoal barbecue grill outside when a rain shower caused him to take the grill into his tool shed. He was found dead by his wife some time later.

In 1972, five fatal and two non-fatal cases of CO poisoning were reported following the use of charcoal barbecues in confined spaces.[15] The deaths occurred in a camper van, a station wagon, a trailer and in a cellar. The three victims on whom autopsies were performed had COHb levels of 65%, 71% and 82%. Review of the medical literature by the authors also highlighted five other deaths in similar circumstances, three of which are further described below.[8,14] Because the hot coals after cooking on a charcoal barbecue do not emit irritating fumes, the authors felt that they were a particularly attractive option for indoor heating for people not acquainted with the dangers of CO poisoning. They suggested that warnings be printed on such barbecues, as they are on packets of charcoal heat beads.

In 1988, two deaths were reported from CO poisoning due to charcoal barbecues being used in enclosed camping facilities.[16] The two patients, a 53 year old man and a 12 year old boy, had COHb levels of 41% and 30% respectively. In 1989 a large series of fatal CO poisoning from non-vehicular sources was described.[11] One of the deaths was listed as due to charcoal use in a confined enclosure although further details of that case were not provided.

In 1989, the emergency health impact of a severe storm in New York was detailed.[17] Five hospital emergency departments in the region, accounting for 190,000 annual patient visits, were surveyed. Most attendances were for injuries such as fractures, abrasions, and lacerations. Surprisingly, the most common non-surgical reason for attendance was CO poisoning which caused 22 attendances. The precise number of cases of CO poisoning

due to charcoal barbecues was not stated, however it was noted that common to all such cases was indoor use of barbecues in poorly ventilated areas and that two incidents involved multiple family members.

In 1990, 12 members of a non-English speaking family were reported to have developed CO poisoning from indoor use of a charcoal barbecue.[18] They had initially presented over several hours in groups of four with non-specific symptoms suggestive of food poisoning. COHb levels on the four patients present in the emergency department were 6.9%, 15.1%, 17.1% and 17.4%.

This prompted a report of two 31 year old, non-English speaking women who presented to the Emergency Department at Stanford Medical Centre, and three other family members at home, all poisoned with CO from indoor cooking on a charcoal barbecue.[19] COHb levels on these patients were 38.3%, 31.9%, 25.5%, 21.1% and 18.5%.

A large series of patients with CO poisoning due to charcoal barbecues was reported in 1994.[12] Seventy-nine patients were treated over 14 years to October 1993, referred from 10 counties in the state of Washington. The criteria used for treatment with hyperbaric oxygen (HBO) were a COHb level of 25% or more, angina or ischaemic changes on ECG, or neurological impairment including transient loss of consciousness.

These 79 patients represented 16% of all cases of unintentional CO poisonings treated during the study period. As expected, most occurred in winter, and ethnic minorities were over-represented (73%). Most incidents (69%) involved more than one individual. COHb levels averaged 21.6% (SD 9.6%), and a third of patients lost consciousness. Headache was the most common (67%) symptom. Two other deaths from CO poisoning due to a charcoal fire in a foxhole have also been reported.[8]

Interestingly, levels of COHb above 5% have been found in over half of studied non-smoking workers in charcoal grilling occupations and in over 80% of smoking workers.[20] It is possible that charcoal burning in confined spaces may be particularly liable to cause CO poisoning due to the high levels of CO released in charcoal combustion. Charcoal barbecues have been shown to produce an air stream containing CO at a level of 20 to 2,000 ppm, with 75% producing levels of 200 ppm or more. This is the level defined as the maximum safe level by the US Department of Labor's Occupational Safety and Health Administration.[21]

CO poisoning from appliances used for heating and cooking

There have been many case reports of individuals or groups poisoned with CO due to heating with other appliances in enclosed spaces. In 1978, three incidents of CO poisoning from blockage of flues for home heaters were reported, with three deaths and nine other casualties.[22] Seventeen deaths were reported from Switzerland in the early 1980s due to CO poisoning from gas water heaters without proper ventilation.[23] In 1983, two reports from Denmark detailed several deaths and near-fatalities from hot water systems.[24,25] Caplan et al. reported 11 incidents of CO poisoning from home heating systems in 1986 in which 16 patients died.[26] A study in Brussels in 1987/88 confirmed the danger of faulty water heaters.[27] It showed that about two thirds of all incidents of unintentional CO poisoning in the homes surveyed were due to hot water systems.

In 1988, two mountain climbers were reported to have succumbed to CO poisoning after cooking inside a tent at 14,200 feet.[28] It should be noted that there is some experimental animal work suggesting that high altitude results in higher COHb levels for a given exposure[29] and that it exacerbates some cardiovascular effects of CO poisoning.[30] The same year, several deaths from gas water heaters were reported from Copenhagen.[31] A 1989 report described a family of three poisoned from a central heating system running on butane gas.[32] The three family members had COHb levels of 4.8%, 6% and 8.5%. All three developed ECG abnormalities which improved after hyperbaric oxygen therapy. In the same year, Wharton et al. detailed five people at a hotel poisoned by CO drawn into the air conditioning from gas heaters for the indoor swimming pool.[33] One died. A report in 1991 described six civilians poisoned with CO during the Gulf War after being advised to stay inside in sealed rooms due to the risk of chemical weapon attack.[34]

Several instances of mass CO poisoning from home heating during a severe storm in Washington were reported in 1993.[35] Because of low temperatures and power failures, many residents of the area developed CO poisoning from improvised indoor heating. Recently, Rudge provided details of 300 patients treated at a military hyperbaric facility.[36] Infants were shown to be particularly likely to have been affected by faulty home heaters.

Epidemiology of unintentional CO poisoning

These reports highlight the dangers of using certain appliances indoors for heating or cooking. There are however other, more common causes of unintentional CO poisoning. Motor vehicle exhaust is the most common, followed by exposure to CO during fires or burns. There are many other causes also reported in the literature, with home heating and cooking appliances contributing significantly to overall incidence.

A 1991 study reviewed 10 years of death certificate reports in the United States.[9] The number of cases where

unintentional CO poisoning had contributed to death was determined. Of 56,133 deaths in which CO was a contributing factor, 25,889 (46%) were suicides, 15,523 (28%) were associated with fires or burns, and 11,547 (21%) were unintentional. They found that the number of unintentional deaths was gradually decreasing. Most of these unintentional cases (57%) were due to motor vehicle exhaust, usually while stationary, and most were elderly males in winter in colder climates. It was felt that improvements in motor vehicle emission systems and home heating appliances had contributed to the fall in incidence. A further study analysing the contribution of weather conditions to the incidence of unintentional CO poisoning confirmed that winter was the peak season.[37] The authors suggested that during certain high-risk periods such as particularly cold weather, health authorities could play a preventative role by alerting physicians and the community to these risks.

A three year study in France[10] found that 735 cases (17.5 per 100,000) had been reported to emergency organisations or laboratories. There were 291 incidents responsible, with 12% due to fires and 4% to car exhausts. Of the remaining 196 domestic causes, most were due to water heaters (57%), followed by boilers (20.5%), coal stoves (9%), braziers (4%), cookers (2%) and heating devices (1.5%). The types of cooking and heating devices were not specified. There were a few other miscellaneous causes.

A study from West Virginia showed that, from 1978-1984, there were 62 unintended fatalities not involving a motor vehicle.[11] This represented nearly half (42%) of all unintentional CO fatalities in that time. The deaths were predominantly due to faulty or poorly vented or ventilated cooking or heating appliances.

Other studies have shown a similarly high rate of CO poisoning from motor vehicles. Fatalities have been recorded not only from faulty motor vehicle exhaust systems but also from cars running in enclosed spaces.

Further West Virginian studies detailed 82 vehicle-related CO-caused fatalities over seven years.[38] Of the 64 incidents responsible for the deaths, 50 were due to defective exhausts and 14 due to poor ventilation in enclosed garages. In Florida, 15 cases over five years were reported of unintentional poisoning from motor vehicle exhaust, mostly due to running the vehicles in enclosed spaces.[39]

It is likely that the incidence of non-fatal CO poisoning from motor vehicle exhaust is higher than reported. For example, seven of 18 drivers in one United States region tested after road traffic crashes had COHb levels of 25% or more, and a salesman driving a car with defective exhaust had four serious crashes in 10 weeks.[14]

After eight schoolchildren developed CO poisoning in a school bus in Seattle, CO levels in such buses were measured.[40] A number had CO levels which were above acceptable limits. After a period of idling in the parking area, over a third had levels which were too high. Interestingly, CO levels in vehicles on busy highways have been shown to be higher with increased traffic volumes and reduced speeds.[41] It is also likely that smoking by drivers in motor vehicles contributes to the higher incidence of crashes in smokers through the effects of CO poisoning.[42]

Occupational CO poisoning

Unintentional CO poisoning has been identified as occurring commonly from car exhaust and fires, and in the home from faulty cooking and heating appliances. However, many occupational groups may also be at risk from exposure to CO. Those studied to date include fire fighters,[43] motor vehicle examiners,[44] charcoal grillers,[20] bus drivers,[40] manufacturing workers,[45] blast furnace workers,[46] and workers using propane-fuelled forklifts in warehouses.[47]

A French study[48] showed that CO poisoning was common in poorly ventilated automobile garages. Pedestrians and workers in city streets may also be at risk, as levels ranging from 10-50 ppm have been found in ordinary city streets in a large urban area. Much higher levels have been recorded in poorly ventilated underpasses and underground carparks.[49]

Discussion

Studies of CO poisoning due to charcoal barbecues

The literature review identified a number of case reports of small numbers of patients with similar poisonings to those reported here. Most involved patients of ethnic background using the barbecue indoors in winter. The paper by Hampson et al.[12] confirmed these observations. It suggested that CO poisoning by indoor use of charcoal barbecues is an important public health problem in industrialised countries with cold climates. Although the cases presented here did not occur during power failures, Hampson's large series contained many such cases, suggesting that public warnings at such times may be appropriate.

In contrast to the reported literature, the COHb levels in the cases reported here were low, and not all of the patients may have been treated under criteria adopted in other studies. The indication for treatment with hyperbaric oxygen for most of the patients reported here was loss of consciousness, although the father and 10 year old boy from family A and the son from family B did not lose consciousness. Nevertheless it was felt that all had suffered a similar exposure to the majority who did lose

consciousness, and warranted treatment at the same time, particularly as this did not entail additional cost. Given that four of the six members of family A lost consciousness despite COHb levels of 4% or less, it may be appropriate to re-examine the COHb level criterion of 25% on which treatment has been based in the absence of other indications.[12] This is important in cases such as these with prolonged exposure to comparatively low levels of CO, in which neurological damage is more likely.[50]

Recognition of unintentional CO poisoning

A mounting body of literature suggests that the problem of CO poisoning from accidental exposure is common and frequently unrecognised. Heckerling assessed 37 patients attending an emergency department in winter with a complaint of headache.[51] Seven (18.9%) had COHb levels greater than 10%. When further information was obtained or gas company officials investigated their homes, six of the seven were found to have suffered toxic CO exposure. It was noted that three had cohabitants at home who also had headache. Most importantly, the diagnosis of CO poisoning had not been suspected by treating doctors in any of the cases. Heckerling and colleagues[52] later derived a model for predicting whether a patient's symptoms were due to CO poisoning and showed that the presence of similarly affected cohabitants was the most reliable method. The model was then validated prospectively.[53]

Dolan and colleagues[54] studied all patients attending an emergency department with flu-like symptoms and found that 23.6% of such patients had COHb levels of 10% or more. Again none of the patients had been diagnosed as having CO poisoning.

The pathological effects of chronic exposure to low-dose CO

The literature review has revealed that both accidental CO poisoning occurs commonly. Clearly this may be an acute, emergent problem, as in this and other case reports, or a chronic problem, particularly in occupational exposures.

The effects of acute exposure to substantial levels of CO are well described in the medical literature and have been comprehensively reviewed by Mark.[1] Clinically the acute effects are principally on the nervous and cardiovascular systems with delayed neurological sequelae in up to 40% of untreated patients.[55]

However, prolonged exposure to low doses of CO may also be hazardous and may present a significant problem in terms of overall incidence as identified in the literature review.

ANIMAL DATA

A considerable body of experimental work suggests that exposure of animals to CO at low doses for long periods may cause pathological changes in many tissues.

A variety of changes in several animals has been noted, on birthweights[56] and behavioural development of animals exposed in the prenatal period, and on the cardiovascular, respiratory, hepatobiliary, nervous, endocrine and haematopoietic systems of young animals chronically exposed to CO. Additionally, higher stillbirth rates in herds of pigs exposed to CO from faulty gas heaters and inadequate ventilation have been reported.[57]

Piglets exposed to 200 to 250 ppm of CO have been reported to have developmental delays and to perform poorly in behaviour tests.[58] Those exposed to CO prenatally have had low haemoglobin levels at birth. Mice exposed to CO in utero have also been shown to perform poorly at tasks involving learning.[59]

Chronic low-dose exposure to CO in laboratory rats has produced myocardial hypertrophy,[60-62] altered coronary vessel growth,[63] altered cardiac adaptation to aortic constriction,[64] accelerated development of hypertension[65] and pulmonary hypertrophy.[62] Aortic intimal injury has occurred in rabbits.[66]

Cardiomegaly, splenomegaly, and elevated haemoglobin and haematocrit have been demonstrated in rats.[65] Increased numbers of erythrocytes cause increased blood volume.[67] Platelet count is altered in rabbits chronically exposed to low-dose CO,[68] and platelet aggregation increased in pigs.[69] CO exposure has been shown to enhance liver cell necrosis in rats with ethanol-induced liver damage.[70]

Guinea pigs exposed to 200 ppm of CO prenatally and as infants exhibited changes in QRS vector loops implying myocardial damage.[71] Prenatal exposure to low doses of CO (75-150 ppm) caused changes in peripheral nervous system activity in rats, some of which were irreversible.[72] Similar central effects have been observed in rats exposed in utero to levels up to 300 ppm, with disruption of neostriatal development.[73] It has also been demonstrated that CO exposure in rats caused negative effects on evoked potentials in a complex discrimination learning experiment.[74] Low-dose chronic exposure to CO produced a mild stress reaction in rats, with elevated catecholamine levels[75] and increased serum steroid and brain serotonin levels.[76]

The effect of combined CO exposure and high altitude has also been investigated. It has been shown that chronic exposure to CO at 35 ppm when combined with high altitude produced no additional cardiovascular effects

in rats over and above the two insults singly.[77] However at 500 ppm, there was a significant interactive effect on mean electrical axis in rats, as well as on haematocrit.[78]

HUMAN DATA

Translating such animal data to humans is difficult, and there is little published research on such effects.

Mortality rates from atherosclerotic heart disease were shown to be higher by a factor of 1.35 in New York bridge and tunnel workers chronically exposed to CO compared with a standardised comparison group not similarly exposed.[79] Toxic effects of CO on humans have also been demonstrated in those with compromised coronary or peripheral circulation.[80] It has been reported that two workers with coronary artery disease died following occupational exposure to CO, as a result of exacerbation of their pre-existing heart disease.[81] The cardiovascular endurance of a group of Canadian fire-fighters has been shown to be lower than expected, and it was suggested that this might have resulted from chronic exposure to CO.[82]

A historical prospective cohort study of mortality among motor vehicle examiners in the United States was undertaken by the National Institute for Occupational Health and Safety.[44] Such workers were estimated to be exposed chronically to a time-weighted average CO concentration of 10-24 ppm. The only significant finding was that cancer deaths (all types) were increased in these people. The significance of this finding is uncertain.

However, two major reviews of the cardiovascular effects of chronic exposure to CO have not provided conclusive evidence on the case for a direct cardiotoxic effect.[83,84]

There is little else reported in the literature regarding the effects of occupational CO exposure although many papers have described the effects of pollutants attributed to smoking, of which CO is one, both in animals[85] and in humans.[86-90]

Despite the paucity of direct evidence that chronic low-dose CO poisoning produces tissue damage in humans, the animal data strongly suggest that such effects are likely. This accords well with the pathophysiology of CO poisoning, with long term exposure causing more CO to be bound to mitochondrial enzymes[91-94] in the presence of comparatively low COHb levels.[95]

The toxic effects of chronic CO exposure are likely to be greater in smokers, who have been shown to have higher CO levels than non-smokers and to have occupationally-induced elevations of those levels above those currently considered safe.[96,97]

Conclusions

CO poisoning is common. Although the majority of such poisonings result from deliberate self-harm, a significant proportion is unintentional. Most of the unintentional poisonings are due to motor vehicle exhaust, fires, and home appliances used for cooking or heating. Additionally, there is likely to be a number of workers exposed to CO in the course of their employment. While the effects of this long-term exposure are as yet unknown, animal and limited human data suggest a number of pathological changes, particularly to the cardiovascular system. This area requires further research in humans.

The reported literature suggests that unintentional CO poisoning is significantly underdiagnosed. Emergency department medical and nursing staff, particularly the triage nurse,[98] need to be vigilant for CO poisoning which can result in very subtle presentations. Suspicion should be high during cold, inclement weather and in those whose occupations may expose them to significant concentrations of CO.

People in certain occupations, such as mechanics in garages, may be at risk, especially if they smoke cigarettes. Others at risk are those with faulty home heating appliances and those using equipment not designed for heating, such as outdoor barbecues, for that purpose. As identified in the literature review and highlighted by the cases reported here, migrants may be at particular risk in this regard. This was recognised by the California Department of Health Services when it issued a public health warning in 1990 concerning the indoor use of charcoal.[99] Asian immigrants were especially targeted. With the progressive development of a multicultural society in Australia, it may be prudent for public health authorities to consider warning newly arrived migrants of some of the dangers associated with activities which may be acceptable in their countries of origin.

In many developing countries, the same implements are often used for cooking and heating, and are used indoors. This is inappropriate when the materials used, such as barbecue charcoal beads, combust slowly and relatively inefficiently, producing large amounts of CO. Chen et al have warned that several hundred million people in developing countries are at risk of poisoning with CO and other products of combustion from indoor heating and cooking.[100]

It has been noted that public health interventions can drastically reduce the mortality from CO poisoning. The Office of the Medical Examiner of New York, for instance, applied stringent inspection rules for gas refrigerators, reducing the number of accidental deaths from this form of poisoning from 168 in 1951 to two in 1964.[14] Although CO poisoning from charcoal barbecues is uncommon in Australia, there is a strong case for warn-

ings about indoor use to be carried on such barbecues as well as the charcoal beads, preferably in several languages.

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THE WORLD AS IT IS

UNUSUAL DECOMPRESSION ILLNESS
A case report

Peter Cardon

A 37 year old male student diver came to me on a Tuesday, referred by his diving instructor. On the Saturday and Sunday he had been training for an Open Water certificate. He took part in a pool session on Saturday morning and an open water dive to 15 m for 40 minutes in the afternoon. At the completion of the day's diving he was tired and had a mild headache but was otherwise well.

The following morning he took part in a further open water session and after some difficulty with weights he undertook a "tired diver" tow, an emergency ascent from about 6-10 m and an ascent using a secondary air source. The remainder of the dive was given to a successful hunt for crayfish.

The dive plan was for 40 minutes to a maximum depth of 15 m, however his depth gauge showed the maximum depth for the dive was 29 m. He had been working against some surge and was a bit cold toward the end of the dive. He made a comfortable ascent up an anchor warp and felt normal on arrival at the surface.

Within one minute of surfacing and releasing the warp to make his entry onto the dive boat he found his hands would not work for him. He had difficulty hanging onto the boat and managing his gear. Eventually he was helped aboard and fell into the boat where he lay helpless and vague. He described being unable to organise himself, he could not get his gloves off and his hands were clawed. He felt uncoordinated with "spasticity" of his limbs and an unpleasant feeling of entrapment in his wetsuit which verged on panic. He became aware of other divers in the water but his perception was impaired and he was troubled by glare. He felt numbness in his arms and chest. His distress was seen by another diver and the dive instructor was called.

A distinct improvement occurred over a few minutes after removal of the student's hood, however he had difficulty with speaking and complained that his legs felt heavy. It took about ten minutes to take the boat to the beach and by then he had recovered so much that he was able to hold the boat while gear was unloaded. However, ten minutes later he had nausea and vomiting so that the car had to be stopped twice during the half hour drive to town.

When he got home he went to bed. He got up once two hours later to vomit then felt better but had a frontal headache which lasted three hours more. No medication was taken. The following day (Monday) he was aware of slight fogging of his peripheral vision, a mild impairment of mental focus and his ears felt strange.

He had had a full medical for a Pilot's Licence in April 1993 and had been found to be quite fit.

On examination on Tuesday he gave a clear description of the events. He moved normally with no apparent impairment of gait, balance or co-ordination. Basic psychometrics showed some difficulty with simple arithmetic but a good performance with immediate recall, including having no difficulty with the Babcock sentence. A quite detailed neurological examination was within normal clinical limits.

Had I been able to find even a minor neurological abnormality he would have been sent off to Christchurch. However I felt that, as he was now symptom free and normal neurologically, there was a reasonable case to be made for resting and observing him over the next few days, as referral to the nearest Hyperbaric Medicine Unit would have involved many hours of travel and considerable disruption to his life. In the event he produced no sequelae.

At follow-up two months later the patient was well with no residual symptoms and stated that he had taken the personal decision not to dive again.

My interpretation of this history is that this student diver suffered an acute form of decompression illness, possibly cerebral arterial gas embolism, with significant transient cerebral ischaemia and no measurable residual impairment.

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