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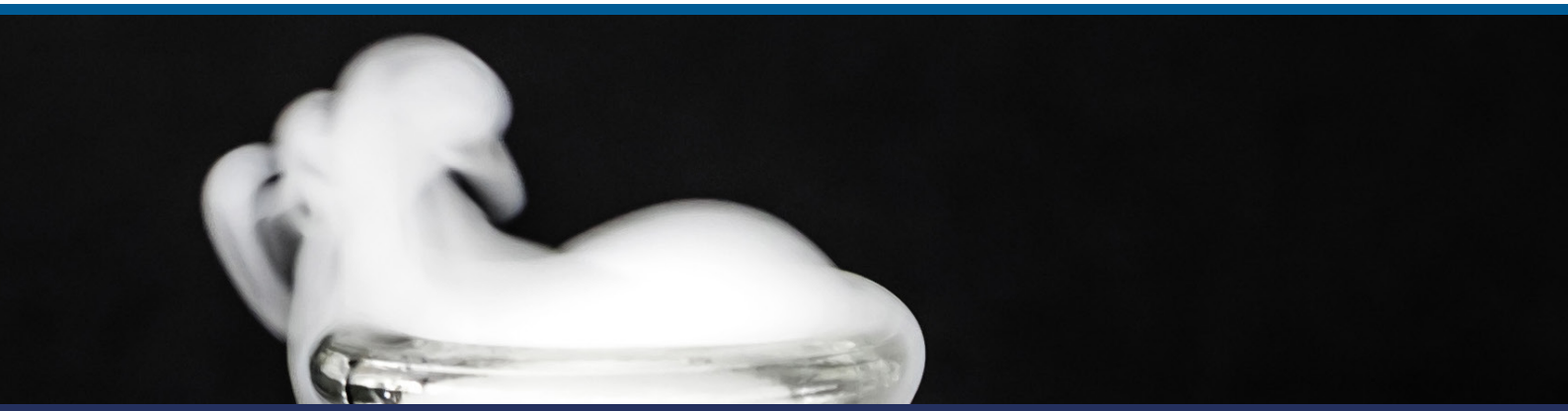

Clinical Communiqué

Next Edition: June 2021

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VICTORIAN INSTITUTE OF FORENSIC MEDICINE

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Editorial

Dr Nicola Cunningham

Welcome to the first edition of the Clinical Communiqué for 2021. In this edition, we look at deaths due to carbon monoxide (CO) poisoning – a gas produced by the incomplete combustion of carbon-based compounds and the most abundant pollutant (by mass) in the atmosphere. CO is often referred to as the 'silent killer' because it is colourless, odourless, tasteless, non-irritating, and extremely toxic. CO is also known as the 'great imitator', for its toxicity produces signs and symptoms that can be attributed to many common disease processes. It has been estimated that CO-exposed patients presenting to emergency departments are misdiagnosed in 30-50% of cases. This represents a substantial number of missed opportunities to prevent vulnerable patients (or their families and friends) from returning to potentially lethal environments. Thus, the lessons from the cases described in this edition are two-fold, firstly, they serve as a timely reminder to clinicians to consider the potential for toxic gas exposure when a patient presents with a vague constellation of symptoms, especially where others close to them (including pets) have reportedly fallen ill as well. Secondly, they reiterate an important public health message about the use of gas appliances, particularly as the winter season approaches. Portable generators, charcoal burners, wood heaters, gas heaters, hot water systems and stoves, have all been implicated in CO-related deaths. As is demonstrated, the risk of unintentional CO poisoning with gas appliances increases with faulty installation, inappropriate use, inadequate maintenance, and poor ventilation.

In this edition we have two new case summary authors, Ms Melanie Gordon, and Dr Janine Rowse, who each contribute important insights on CO poisoning. In addition to the case summaries, we have a new feature section of the Clinical Communiqués, titled 'More on the Matter', where we present case vignettes that demonstrate the wide-ranging scenarios where toxic exposure to CO may occur. From recreational activities such as boating, caravanning and scuba diving, to daily activities such as cooking, showering, and using refrigerator units, the risks remain ever-present.

Our expert commentary is written by Dr Matthew Spotswood, an intensivist and emergency physician, who has compiled a concise and practical overview of the pathophysiology, clinical features, and treatment of patients with CO toxicity. In his commentary, Dr Spotswood deftly highlights a number of pitfalls and recommendations with regards to the assessment and management of these patients.

Limitations in coding data make it difficult to know the true number of deaths due to unintentional CO poisoning in Australia each year. Between 2011 and 2016 there were 15 deaths reported across Australia that were clearly attributed to using gas and solid fuel appliances in confined spaces without adequate ventilation.

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Editorial (continued)

It is likely that there are a greater proportion injuries and health problems related to CO poisoning that are 'near misses' and remain unreported.

Despite the existence in most Australian jurisdictions of coronial findings focussed on unintentional CO poisoning, the potential dangers remain somewhat unknown or under-estimated in residential and recreational fields. There are common themes to the comments and recommendations made by coroners in such cases, namely:

- Introduce mandatory installation of hard-wired CO detection alarms in residences, boats, and caravans where gas appliances are either permanently fixed or portable appliances are likely to be used.
- Perform all installations by licensed gas fitters to ensure compliance with the relevant regulations and safety standards.
- Conduct regular servicing of gas appliances.
- Never use gas appliances in enclosed spaces and always keep vents free of obstruction.

A brief note on measuring and reporting toxic CO levels

CO levels may be measured and reported as a percentage of carboxyhaemoglobin (COHb) in blood. On average, normal concentrations of COHb in non-smokers are 0-2%, with baseline concentrations up to 10% in smokers. Toxic CO exposure levels are sometimes also given in ppm (parts per million) as a measurement of the number of molecules of CO in air. The recommended upper level for atmospheric CO in an enclosed space is less than 5 ppm.

At low levels of exposure, CO may cause poor concentration, memory and vision problems, and loss of muscle coordination. At higher levels, it causes headaches, drowsiness and nausea. At very high levels, the symptoms intensify and can be life threatening within hours to minutes. See Table 1 for COHb and exposure levels corresponding to adverse health effects of CO.

The upcoming Carbon Monoxide Awareness Week (3-9 May 2021) is an annual national event run by the *Chase and Tyler Foundation* to raise awareness of fuel-burning appliance safety across Australia reducing illness, injury and death by unintentional carbon monoxide poisoning (see https://www.chaseandtyler.org.au). Carbon monoxide poisoning is an entirely preventable illness, and if identified early enough, is wholly reversible. It is imperative that clinicians and the general public learn to identify and mitigate the potential environmental hazards, and recognise the signs and symptoms of CO poisoning, to keep patients and those around them safe.

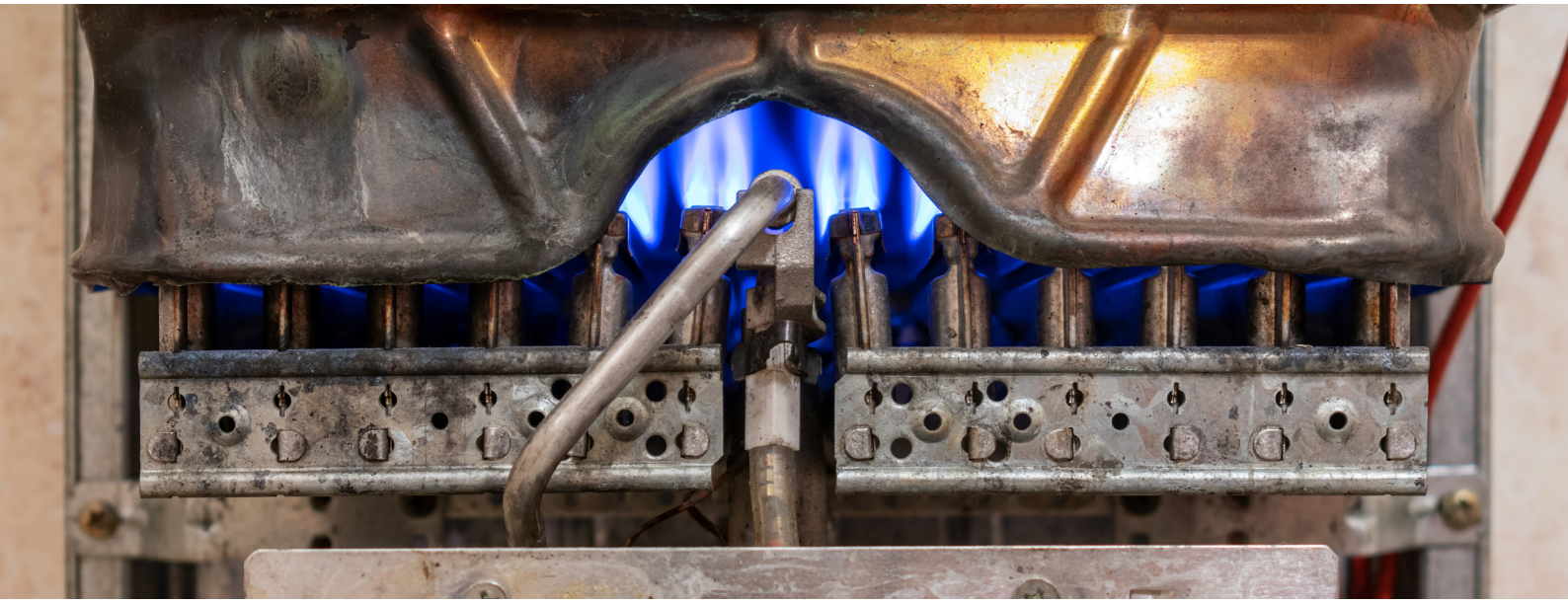
Table 1. Blood COHb Levels Corresponding to Adverse Health Effects of Carbon Monoxide

Effect	COHb[a] (percent)	Exposure[b] (ppm)
Endogenous production	<0.5	0
Typical level in nonsmoker	0.5–1.5	1–8
Increased risk of arrhythmias in coronary artery disease patients and exacerbation of asthma (epidemiological studies)	0.3–2[b]	0.5–10[b]
Neurodevelopmental effects on the auditory system in rats	2–4[b]	12–25[b]
Enhanced myocardial ischemia and increased cardiac arrhythmias in coronary artery disease patients	2.4–6	14–40
Decreased exercise stamina in healthy adults	5–8	30–50
Neurobehavioral/cognitive changes, including visual and auditory sensory effects (decreased visual tracking, visual and auditory vigilance, visual perception), fine and sensorimotor performance, cognitive effects (altered time discrimination, learning, attention level, driving performance), and brain electrical activity	5–20	30–160
Acute and delayed onset of neurological impairment (headache, dizziness, drowsiness, weakness, nausea, vomiting, confusion, disorientation, irritability, visual disturbances, convulsions, and coma) and pathology (basal ganglia legions)	20–60	160–1,000
High risk of death	>50	>600

[a]Reported value, unless otherwise denoted as predicted.

[b]Predicted from the Cobum-Forster-Kane (CFK) model (unless otherwise denoted as reported).

Source: Agency for Toxic Substances and Disease Registry (ATSDR). 2012. Toxicological profile for Carbon Monoxide. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Chapter 2 p22. Available at: https://wwwn.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=1145&tid=253.



Case #1 A poisonous adventure

Case Number
2017 TASCD 531 Tas
And
2017 TASCD 532 Tas

Case Précis Author
Melanie Gordon
RN
Quality Manager, Epworth HealthCare

i. Clinical Summary

Mr GB and Mr BD, along with their friend Mr BL, and Mr BL's young daughter, commenced a short sea trip in a 'Double B' boat they jointly owned. On the first night, the boat was anchored in a bay with all four persons on board. Close to midnight Mr BL's daughter began fitting. Mr BL made an emergency call to 000 and his daughter was retrieved by helicopter and taken to a large metropolitan hospital. Following blood tests and a period of observation in hospital, Mr BL's daughter was discharged into the care of her parents, with the plan for a follow up CT brain scan. She did not have a prior history of seizures.

Mr GB and Mr BD continued the voyage to their destination, arriving the following evening at a marina where they intended to moor and spend the night on the boat. Mr GB called his wife to let her know about their plans, and Mr BD invited Mr BL to come onboard and bring some alcohol. Mr BL declined to join them.

When Mr BL arrived at the marina the next morning, he was unable to raise either of the men by phone. Mr BL sought the assistance of the marina owner and accessed the boat where he found both men dead in the cabin.

Emergency services arrived quickly at the scene and one paramedic reported feeling light-headed after being in the cabin of the boat. Fire personnel also attended due to concerns about gas, and it was noted that no smoke detector or similar device was fitted in the cabin.

ii. Pathology

Following toxicological analysis of blood samples taken from Mr GB and Mr BD at autopsy, it was found that Mr GB had a carbon monoxide saturation level of 59%. The same analysis found alcohol to have been present in GB's body at the level of 0.149 mg per 100 mL of blood. Mr BD had a carbon monoxide level of 69%, with an alcohol level of 0.187mg per 100 mL of blood.

iii. Investigation

Investigations revealed that at the time Mr GB contacted his wife, there was no mention of either men feeling unwell.

However, Mr BD had sounded intoxicated during the call he made to Mr BL a few hours later.

In light of the toxicological findings, the coroner was satisfied that the cause of deaths was carbon monoxide poisoning and their cases did not proceed to inquest. With alcohol concentrations at the levels found in both men, the alcohol consumption would have masked the effect of carbon monoxide concentration. There would have been a critical loss of judgement, coordination, impaired balance, sedation and sleep, symptoms common to both carbon monoxide poisoning and alcohol intoxication.

Further investigation by an officer of Marine and Safety Tasmania found the source of the carbon monoxide was from a portable power generator which had been bought and installed by Mr GB and Mr BL in the starboard aft (right rear) machinery space area, which was an enclosed, but not an airtight space. The generator was in an enclosed space and was connected to a long home-made extension pipe that had a defect in it. The pipe was fitted to the muffler which was obstructed on the end by a shower fitting. Investigators determined that the obstructed muffler would have created back pressure on the make-shift exhaust system, propelling exhaust gases into the machinery space area and into the cabin. The hole in the piping would have also allowed gases to escape into those areas.

One final area of investigation following the deaths of Mr GB and Mr BD, was a review of Mr BL's daughter's blood. Toxicological analysis revealed that high levels of carbon monoxide were present in her blood at the time she presented to hospital with a seizure.

iv. Coroner's Findings

The coroner found that Mr GB and Mr BD died as a result of carbon monoxide intoxication due to inhalation of exhaust from a petrol generator on a boat. The coroner deemed both deaths to be, *'entirely avoidable due as they were, to a poorly installed and maintained petrol driven generator and the absence of any device to warn as to the presence of carbon monoxide in the cabin.'*

The generator was in an enclosed space and was connected to a long home-made extension pipe that had a defect in it.

The coroner recommended that all petrol driven generators only be used in accordance with manufacturer's recommendations, and in particular, should not be installed in a confined space and should not have the exhaust system modified in any way.

In addition, the coroner recommended that all boats with enclosed cabins which have petrol driven motors of any type installed be fitted with a carbon monoxide detector.

Importantly, there was no blame attached to those involved with the medical care of Mr BL's daughter.

In the absence of any clinical history or any reason to consider carbon monoxide poisoning, the coroner acknowledged that it was not reasonable to have expected that diagnosis to have been made when she presented to hospital with fitting.

v. Author's Comments

As we head into autumn and winter it becomes increasingly important to be aware of the symptoms of carbon monoxide poisoning since more cases present during these seasons compared to spring and summer. The cases of Mr GB and Mr BD are not uncommon, with carbon monoxide being one of the leading causes of unintentional poisonings each year. Therefore, it is invaluable for clinicians to have an index of suspicion for environmental poisonings, particularly in the winter months.

vi. Resources

Deniz T, et al. Carbon monoxide poisoning cases presenting with non-specific symptoms. *Toxicol Ind Health* 2017; 33(1): 5-60, DOI: 10.1177/0748233716660641.

viii. Keywords

carbon monoxide, poisoning, inhalation of exhaust, petrol generator, boat

Case #2 The paradox of negative pressure

Case Number
COR 2017 3566 Vic

Case Précis Author
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i. Clinical Summary

Mrs S was a 62-year-old woman who was active and in generally good health with a history of diabetes mellitus, osteoporosis and arthritis. Mrs S lived alone in a public housing unit. In the month prior to her death, Mrs S felt unwell and attended a general practitioner, who organised a series of blood tests that were not completed.

Mrs S was found deceased in her home, having last been seen alive the day prior. On entry into her unit, intense heat was noted, and the gas heater was on high. It was apparent she had showered just prior to her collapse. A saucepan was burning on low heat on the stovetop in the kitchen.

ii. Pathology

Following an autopsy, toxicological analysis of Mrs S' blood detected an elevated carboxyhaemoglobin level of 64%. No other drugs or poisons were detected. The forensic pathologist determined Mrs S' cause of death to be carbon monoxide toxicity.

iii. Investigation

An inquest was held to examine the circumstances of Mrs S' unexpected death. The court heard that a carboxyhaemoglobin level of 64% would indicate high environmental concentrations of carbon monoxide, with death occurring almost immediately. The likely source of carbon monoxide was thought to be a 25-year old open flued gas (OFG) heater in Mrs S' unit.

The inquest focused on risk mitigation and prevention opportunities, given the potential safety issues relevant to the broader community. Issues explored at inquest included the cause and extent of carbon monoxide spillage from the heater, maintenance and service history of the heater, and the existing regulatory system.

Mrs S was described as a person who always used the exhaust fans in the bathroom and kitchen, when showering and cooking respectively. This was likely the case at the time of her death. Testing in Mrs S' unit revealed that with exhaust fans operating, and with external doors and windows closed, a negative pressure environment was created.

Negative pressure is of relevance in drawing air back down the flue of the heater into the room, taking with it the by-products of combustion (including carbon monoxide). Additionally, Mrs S' unit had been upgraded since the heater installation, including seals on doors, and aluminium framed windows. Vents installed vented mainly to a sealed roof space, without adequate ventilation to the atmosphere. The maintenance and service history of the heater was explored in detail. Despite servicing, the OFG heater was found to spill carbon monoxide into the living space when tested whilst the kitchen and bathroom exhaust fans were turned on. Furthermore, all heaters of the same type, installed in the other units at the complex, also failed spillage testing when the exhaust fans in those units were in operation.

Expert witnesses reported that although OFG heaters may have complied with design standards at the time of their installation, a significant risk currently exists. With the increasing energy efficiency of Australian homes as well as the use of more powerful internal exhaust fans, a deadly environment may result with OFG heaters in operation. The coroner investigated the lack of mandatory continuous professional development for gas fitters, as well as a lack of awareness of modifications to relevant standards of carbon monoxide testing methods.

iv. Coroner's Findings

The coroner found that the source of the toxic level of carbon monoxide was the OFG heater in Mrs S' unit.

A combination of factors was deemed to have contributed to her death. Lack of adequate ventilation and failure of the heater's draft diverter in a negative pressure environment created by other exhaust fans operating, ultimately resulted in lethal levels of carbon monoxide being introduced into the room air.

v. Author's Comments

The warning signs of life-threatening carbon monoxide poisoning can be subtle and include a constellation of non-specific symptoms which have multiple possible causes. This presents a challenge for clinicians assessing undifferentiated patients, particularly when differential diagnoses such as flu-like viral syndromes, migraines and depression may be more frequently encountered. Awareness of the symptoms of carbon monoxide toxicity by medical practitioners may lead to the uncovering of a history of potential exposure, and prevention of a lethal outcome.

The warning signs of life-threatening carbon monoxide poisoning can be subtle and include a constellation of non-specific symptoms which have multiple possible causes.

The coroner made eight recommendations: the first being that OFG heaters be phased out across Victoria. Some of the other recommendations focused on:

- Targeted information dissemination for the community, focusing on the dangers of OFG heaters, particularly in conjunction with negative pressure and insufficient ventilation.
- Improved testing of carbon monoxide leakage.
- Improved compliance systems to assure quality of heater servicing is in line with industry standards.
- A system of mandatory continuous professional development be considered for gas appliance plumbers and fitters.
- Improvements in information dissemination of changes to industry standards to relevant stakeholders.

Mrs S was a mother and a grandmother and, as the coroner acknowledged, there is nothing to relieve the grief endured by her family. The significant number of coroner's recommendations across both promotional, educational and regulatory domains, is a powerful reminder of the important function of the coronial process in improving systems, in order to prevent deaths in similar circumstances.

vi. Keywords

Carbon monoxide, carboxyhaemoglobin, gas heaters, poisoning, negative pressure, general practitioner

vii. Resources

https://www.bettersafercare.vic.gov.au/publications/heater-associated-carbon-monoxide-toxicity

More on the Matter
Outing the problem

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1995/09 NSW

Mr AM was a 43-year-old man who was cooking with charcoal briquettes on the verandah of his apartment. He then transferred the tray containing the burning briquettes into the apartment for warmth. He was later discovered deceased in his apartment.
At autopsy, Mr AM's blood concentration of CO was 61%. He was also found to have significant, but previously undiagnosed, coronary artery disease. The apartment he lived in was situated beneath a flight path of a nearby airport so had been insulated to minimise noise pollution, which also reduced the inflow of fresh air. Mr AM's cause of death was carbon monoxide poisoning.

2013 TASCD 060 Tas

Mr TB was a 39-year-old man who was found deceased in a caravan along with two other men also deceased. The three were friends who had travelled to a reserve to set up camp for a weekend. They were found the next morning by a fourth man, a friend who had also joined them for the weekend but had brought along his nine-year-old child, so had chosen instead to sleep with his child in a nearby tent. Investigations into the men's deaths revealed that the caravan belonged to Mr TB and had been refurbished by him a couple of years earlier. The vents in the cladding of the caravan did not align with any ventilation openings in the refurbished lining, and all the windows and doors had new seals in place. The roof vent was in a closed position.

A portable outdoor refrigerator that had always been kept outside the caravan when in use, was found inside the caravan connected to a 4.5kg LPG bottle and turned on. The gas bottle was also connected to a two-burner gas cooktop and a leak was found in the connector tubing prior to the cut-off valves for the cooktop. A pillow was resting on top of the ventilation grates on the refrigerator.

Testing confirmed that within the sealed caravan, with only the portable refrigerator in operation, the CO level after 75 minutes was 353 ppm, and would have been 1200 ppm after four hours. The coroner found that the men's deaths were due to unintentional CO poisoning while sleeping in a caravan with a portable gas-operated refrigerator in operation.

016/201801 NSW

Mr NB was a 23-year-old experienced sailor who had planned to spend a weekend on his yacht with his girlfriend. After a night and a day together on the yacht, his girlfriend woke feeling confused and unwell. She managed to call for help and when emergency services arrived, she was in a critical condition and Mr NB was already deceased. The investigation into the incident found that all the hatches on the yacht were sealed shut, and a liquified petroleum gas (LPG) stove appeared to have been turned on during the night to keep the cabin warm. Post-mortem toxicological analyses showed that Mr NB had a carboxyhaemoglobin level of 60%. At inquest, the court heard that Mr NB and his girlfriend would have felt increasingly light-headed, dizzy, nauseous and fatigued as the CO levels rose, and their capacity to reason and problem solve would have been progressively impaired. Mr NB's cause of death was CO poisoning caused by the incomplete combustion of LPG from the burner in the sealed cabin.

2016/3336 Qld

Mr AT was a 44-year-old recreational diver who had completed over 300 dives. He spent a day on the water with a group of experienced divers from their scuba club, using cylinders he had filled with the club's compressor. The environmental conditions were considered perfect for diving that day and his first dive was uneventful. Ten minutes into the second dive, he indicated to his dive partner that he felt unwell and intended to surface.

His partner lost sight of him during the ascent, and he was not seen again until his body was retrieved from the seabed the next day. Data from Mr AT's dive watch established that shortly after 12 minutes, he had been close to the surface for less than 30 seconds, after which time he had descended to 29 metres where he remained. High levels of CO and CO2 were subsequently detected in his dive cylinder. Autopsy findings were subarachnoid haemorrhage and a COHb level of 56%.

An expert in diving and hyperbaric medicine considered that the haemorrhage was a consequence of severe CO toxicity, and even if rescued prior to his final descent, Mr AT may have suffered irreversible damage. His cause of death was listed as CO toxicity, which caused fatal drowning. Cylinders from the scuba club were seized and tested and found to contain varying levels of contaminants. Mr AT's second dive cylinder had 2,366 ppm of CO. The source of contamination was determined to be the electrically powered air compressor at the club that had been poorly configured and maintained. Club members were unaware of the potential risks of contamination when using air compressors to fill their dive cylinders.

23/2019 SA

Ms HP was a 47-year-old woman who visited a rural property with her husband and two sons. The onsite accommodation was infrequently used and had a converted concrete water tank for a bathroom. The night they arrived, Ms HP's son went to have a shower, spending approximately 15 minutes in the bathroom. Her husband showered next, spending another 15 minutes in the bathroom.

Ms HP followed but never left the bathroom. Her husband found her unresponsive on the bathroom floor approximately 15 minutes later with the shower and the sink tap still running. He pulled her out of the bathroom, but she could not be revived. Investigations into her death found that the gas water heater in the bathroom was at least 45 years old, and had been installed without a hood or funnel. A metal plate was attached to the top of the heater instead of a flue, which led to the production of very high levels of CO in the poorly ventilated room. The cause of death was CO toxicity.

Expert Commentary
Carbon monoxide toxicity

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Carbon monoxide (CO) is a colourless, odourless, and tasteless gas formed during the incomplete combustion of carbon compounds. Common sources include fires, engine exhaust systems, and faulty gas or combustion heating or cooking appliances. CO therefore has the potential to be present in the majority of households, especially during the colder months when heaters are being used and rooms are less ventilated.[1] CO exposure occurs in three main circumstances: 1) intentional self-poisoning; 2) occupational exposures due to fires or the operation of machinery in confined and poorly ventilated spaces, with industries at greatest risk being mining, firefighting, and fishing; and 3) accidental exposure from faulty or poorly installed heating and cooking devices.[2] The two case summaries in this edition of the Clinical Communiqué describes two such unintentional poisonings, attributable to faulty installation and inadequate ventilation.

Pathophysiology

Once inhaled, CO binds with high affinity to haemoglobin, displacing oxygen and reducing oxygen carrying capacity, particularly in hypoxic environments. CO diffuses into extravascular tissue and interrupts mitochondrial respiration by binding to and inhibiting haem proteins responsible for production of adenosine triphosphate (ATP), slowing down oxidative phosphorylation, and generating oxygen free radicals, causing intracellular damage.

The organ systems most affected by this state of oxidative stress and cellular inflammation are those most reliant on ATP synthesis, namely the cardiovascular and central nervous systems.[3,4] The patient groups most at risk include the elderly, those with chronic ischaemic cardiac and respiratory disease, and the foetus due to the relatively hypoxic foetal circulation and the increased affinity for CO of foetal haemoglobin.

Diagnosis

Symptoms therefore commonly include headache, dizziness, fatigue, nausea, vomiting, altered mental state, chest pain, breathlessness, and loss of consciousness. It must be noted that these are non-specific, and therefore the context of the presenting history becomes paramount in raising clinical suspicion of CO toxicity as a diagnosis.

The diagnosis is confirmed by measuring elevated carboxyhaemoglobin (COHb) levels in blood: most modern point-of-care blood gas analysers include co-oximetry, and routinely report COHb on an arterial or venous blood gas printout. Normal COHb level is considered to be <2% in non-smokers, and <5% in smokers, although some heavy smokers may have a baseline exceeding 10%.[1] It is important to note that conventional pulse oximetry cannot distinguish between COHb and oxyHb, and that a so-called "normal" SpO2 reading may mask significant COHb levels or miss profound hypoxia.[3]

Cardiac effects occur in up to one third of CO poisonings, and can manifest as myocardial ischaemia, QTc prolongation, cardiac arrhythmias, cardiac failure, and cardiogenic shock.

Clinical Syndromes

CO toxicity occurs across a spectrum of severity, ranging from mildly symptomatic to critically ill with rapidly progressive brain injury and cerebral oedema requiring intensive care unit (ICU) management. The predictors of short-term mortality are: metabolic acidosis with pH<7.2; loss of consciousness; need for endotracheal intubation; fire as the source of CO inhalation; and high COHb level. The level of clinical severity does not necessarily correlate with the measured COHb level.[3]

Cardiac effects occur in up to one third of CO poisonings, and can manifest as myocardial ischaemia, QTc prolongation, cardiac arrhythmias, cardiac failure, and cardiogenic shock.

The risk of myocardial infarction is increased both acutely and long-term.[3]

Neurologic manifestations can be divided into the acute brain injury, and the development of long-term neurocognitive symptoms such as impaired memory, anxiety/depressive symptoms, vestibular and balance problems, Parkinsonian-like syndromes, peripheral neuropathies and cognitive impairment.[3,4] These symptoms become evident by six weeks post-exposure, and may persist for years. The severity of the initial poisoning episode does not clearly correlate with the development of these symptoms, and they may occur in settings of low-level chronic exposures.[3]

Other clinical syndromes include rhabdomyolysis due to CO binding to myoglobin and causing tissue hypoxia.

Treatment

The overarching principle of treatment lies in removing the patient from the exposure, and providing organ supports as clinically indicated.

Specific treatment involves the delivery of 100% oxygen to promote the rapid removal of COHb.

The half-life of COHb in adults breathing room air at sea level is 5-6 hours, which is reduced to ~80 minutes by breathing 100% oxygen. This can be further reduced to 22 minutes by the delivery of hyperbaric oxygen (HBO) at 3 atmospheres. HBO therapy also may play a role in reducing mitochondrial dysfunction, reducing inflammation, and decreasing the incidence of long-term neurological sequelae.[1,3,4,5]

In general, hospital admission should be sought for: myocardial ischaemia; cardiac dysrhythmias; COHb levels >20% (or >10% in pregnancy); neurological signs and symptoms; metabolic acidosis; persisting symptoms despite several hours of 100% O2 therapy; suicidal intent.[6]

It is advisable to contact the regional hyperbaric unit early in the process to determine suitability for HBO therapy. Treatment criteria vary between centres, so it is worth referring promptly, as the benefits of HBO are best seen if administered early in the course of the poisoning.[6]

Neuropsychological testing and follow-up is also recommended.

Pitfalls and Recommendations

- Unintentional CO poisoning is a difficult clinical diagnosis owing to the non-specific symptoms. It should be considered in patients with vague, recurrent or prolonged symptomatology, or when more than one household member is affected.[1]
- History-taking should focus on potential for exposure, e.g. heating, ventilation, confined spaces.
- SpO2 monitoring will be misleading. COHb levels must be taken to confirm both diagnosis and degree of hypoxia.
- 100% oxygen should be delivered regardless of SpO2 readings – therapy should NOT be titrated to the SpO2 level as this will result in underdosing.
- Early consultation with hyperbaric specialists is advised to determine course of management.
- Treatment thresholds are much lower in pregnancy given the risk to the foetus.
- Severity of toxicity will not necessarily correlate to COHb levels: treatment decisions should be based on the clinical syndrome.

CO poisoning is preventable and treatable. Safe installation and servicing of appliances and ventilation systems can save lives.

Thanks to Dr David Cooper – Director of Hyperbaric Medicine, Royal Hobart Hospital, Tasmania.

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