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AHC Media

Carbon Monoxide and Cyanide Poisoning in Smoke Inhalation Victims: A Review

Carbon monoxide and cyanide are highly lethal toxic compounds that can cause significant morbidity and mortality. Smoke inhalation victims present a unique challenge because they can be exposed to both substances. This article will review the most recent literature on carbon monoxide and cyanide diagnostic evaluation and treatment options for each poisoning. Practitioners need to maintain a high index of suspicion to identify and treat carbon monoxide and cyanide poisonings in smoke inhalation victims. Carbon monoxide poisoning can be identified with detection of carboxyhemoglobin levels in blood or bedside co-oximetry. Initial treatment is the administration of 100% normobaric oxygen. Hyperbaric oxygen is a treatment adjunct, although the benefits of this modality are controversial. For cyanide poisoning, there is no quick bedside or laboratory confirmatory test, and it remains a clinical diagnosis. Fire victims with soot in their mouth, altered mental status, and metabolic acidosis with extremely high lactate levels suggest cyanide poisoning. Treatment options are hydroxycobalamin, sodium thiosulfate, and amyl/sodium nitrite. Both hydroxycobalamin and sodium thiosulfate can be given in suspected cyanide and concomitant carbon monoxide poisonings. Amyl and sodium nitrites can cause methemoglobinemia and hypotension and therefore are not recommended if carbon monoxide poisoning is also suspected. Several papers advocate the superiority of hydroxycobalamin due to its quicker onset of action, but to date there exists no well-designed randomized, controlled trial comparing its efficacy to sodium thiosulfate and amyl/sodium nitrite.

— Ann M. Dietrich, MD, Editor

Introduction

Victims of fires are complicated patients who can have a multitude of injuries that can cause significant morbidity and mortality. Smoke inhalation can cause systemic toxicity from carbon monoxide (CO) and/or cyanide (CN) exposure. Both toxins can cause significant injury or death if unrecognized by clinicians. The purpose of this article is to review the epidemiology, pathophysiology, diagnosis, and management of carbon monoxide and cyanide poisonings.

EXECUTIVE SUMMARY

- Carbon monoxide poisoning is more likely to occur during the winter months in colder climates due to defective household furnaces or improper use of cooking equipment for heating in enclosed spaces.
- In fires, hydrogen cyanide gas is created from the combustion of products containing carbon and nitrogen. Most often this is in foam rubber, wool, plastics, and other common synthetic materials.
- Mild CO poisonings can cause headache, nausea, fatigue, irritability, confusion, vertigo, and flu-like symptoms. Severe poisonings can have profound effects such as delirium, ataxia, chest pain, dyspnea, seizures, coma, myocardial infarction (MI), stroke, and death.
- Patients with CN poisoning present with nonspecific symptoms including headache, nausea, vertigo, anxiety, altered mental status, tachypnea, and hypertension. More serious poisonings will result in dyspnea, bradycardia, hypotension, and arrhythmia, with the most severe poisonings presenting with unconsciousness, convulsions, and cardiovascular collapse, followed by shock, pulmonary edema, and death.
- Blood lactate levels greater than 8 mmol/L in non-smoke-inhalation victims and greater than 10 in smoke-inhalation victims should be considered diagnostic of CN toxicity.

Epidemiology

Smoke inhalation is estimated to be responsible for up to 75% of fire-related deaths in the United States.[1] Inhalation injuries are also an independent predictor of prolonged intensive care unit (ICU) care and mortality.[2] In a case review of patients with carbon monoxide poisoning, those with concomitant smoke inhalation had a higher risk of dying than those with CO exposure alone.[44] A recent meta-analysis demonstrated that the overall mortality rate among burn patients was 13.9% (4-28.3%), with the mortality rate for burn patients with inhalational trauma being 27.6% (7.8-28.3%).[3] In the United States and Canada, fire death rates are twice as high as in Europe and Japan.[4] Due to the numerous mechanisms of injury associated with fires and smoke inhalation, it is difficult to actually quantify the morbidity and mortality directly related to exposures to CO and CN. Most of the data focused on CO and CN are based on non-fire-related exposures.

Between 2004 and 2006, an estimated 20,636 nonfatal, unintentional, non-fire-related CO exposures were treated annually in the United States.[5] Carbon monoxide poisoning is more likely to occur during the winter months in colder climates due to defective household furnaces or improper use of cooking equipment for heating in enclosed spaces.[6,7,8] The CDC also has noticed spikes in carbon monoxide poisonings during disasters that result in power outages, resulting in improper use of generators in poorly ventilated areas.[9,10] Other causes of poisonings are from suicide attempts, older automobiles, use of power tools in enclosed spaces, and hookah smoking.[11]

According to the Toxic Exposure Surveillance System, there were 3165 human exposures to cyanide reported to Poison Centers from 1993 to 2002.[12] In 2011, only 246 cyanide exposures were reported to Poison Centers in the United States.[13,14] The number of actual cases is likely underreported. The most common cause of cyanide poisoning is smoke inhalation. It is speculated that most on-site fatalities in smoke inhalation victims are due to cyanide

Table 1. Contributors to HCN Production

Major Contributors to HCN Production During Combustion

- Insulation
- Furniture coverings
- Plastic furniture
- Draperies
- Carpets
- Appliances
- Many plastics
- Some clothing
- Products containing nylon, polyurethane, melamine, acrylonitrile

Minor Contributors to HCN Production During Combustion

- Grass clippings
- Green wood
- Tobacco
- Cotton
- Paper
- Wool
- Silk
- Weeds
- Animal carcasses

poisoning.[15] In fires, hydrogen cyanide gas is created from the combustion of products containing carbon and nitrogen. Most often this is in foam rubber, wool, plastics, and other common synthetic materials.[16] (*See Table 1.*) Cyanide is found in nature in the Prunus species, consisting of apricots, bitter almonds, cherries, and peaches. All of these fruits have pits containing the glucoside amygdalin. When ingested, amygdalin is biotransformed by intestinal d-glucosidase to glucose, aldehyde, and cyanide. Cyanide is also found in cassava root.[17] Cyanide salts are used in industrial settings, particularly in jewelry and textile industries.[18] In medical treatments, cyanide poisoning can result from sodium nitroprusside treatment for hypertensive emergency.[19]

Pathophysiology

Carbon monoxide (CO) is an odorless, tasteless, colorless gas that is created by the incomplete combustion of fuels (fossil fuels, oil, wood, etc.). CO is inhaled in the lungs, perfuses across the alveolar barrier, and binds to hemoglobin to form carboxyhemoglobin. CO has an affinity to hemoglobin 210 times greater than that of oxygen. Consequentially, there is a leftward shift of the hemoglobin-oxygen dissociation curve, decreasing hemoglobin's release of oxygen, resulting in cellular hypoxia. CO also impairs the oxygen supply to the mitochondria in myocardial cells, causing injury through the binding to intracellular myoglobin.[20-23]

CO poisoning can cause nonspecific symptoms. The most common symptoms are headache, dizziness, nausea and vomiting, loss of consciousness, and confusion.[24] Mild poisonings can cause headache, nausea, fatigue, irritability, confusion, vertigo, and flu-like symptoms. Severe poisonings can have profound effects such as delirium, ataxia, chest pain, dyspnea, seizures, coma, myocardial infarction (MI), stroke, and death. (*See Table 2.*) Chronic exposures have been associated with depression, confusion, and memory loss.[21-23]

The systems most affected by CO poisoning are those that are the most prone to hypoxia: the brain and the heart. In the cardiac system, CO poisoning can result in ischemia, infarction, and/or dysrhythmias. Patients with a history of coronary artery disease are more prone to such injuries, but ischemia can occur in patients with no underlying coronary artery disease.[25] Those patients with cardiac injury are more likely to have increased long-term mortality.[26] In the central nervous system (CNS), carbon monoxide causes acute intoxication, neurological dysfunction, and can lead to chronic neurological sequelae. The areas of the brain most susceptible to injury are the cerebral cortex, white matter, basal nuclei, and Purkinje cells of the cerebellum.[27] (*See Figure 1.*) In severe poisonings, other organs can also be injured, such as the kidneys and liver. Whether this is because of direct organ injury due to hypoxia or secondary to low perfusion due to acute heart failure in the setting of MI has not been established.[21]

Long-term neurological sequelae are varied both in deficit and intensity. The most common symptoms are disorientation, memory disturbance, attention disturbance, parkinsonism, ataxia, urinary incontinence, gait disturbance, and other neuropsychiatric manifestations.[27] Also, a lucid interval of recovery followed by a sudden and progressive recurrence or deterioration of neuropsychiatric symptoms sometimes occurs, which is known as delayed encephalopathy.[28]

Cyanide's mechanism of toxicity involves the disruption of the electron transport chain in the mitochondrial production of ATP by binding to and inhibiting cytochrome oxidase a_3. (*See Figure 2.*) With the cessation of aerobic cellular metabolism, cells are forced to rely on anaerobic fermentation for the production of ATP, with the subsequent production of lactate. The result is a profoundly acidotic state, quickly resulting in cellular death. Clinical manifestations reflect rapid dysfunction of oxygen-sensitive organs, with CNS and cardiovascular findings predominating. The time to onset of symptoms typically is seconds with inhalation of gaseous hydrogen cyanide (HCN).[17] Estimates of lethal dose vary, but exposures to gaseous HCN at concentrations above 300 ppm will likely result in death within a few minutes.[29] Mild cases of CN poisoning present with nonspecific symptoms including headache, nausea, vertigo, anxiety, altered mental status, tachypnea, and hypertension. More serious poisonings will result in dyspnea, bradycardia, hypotension, and arrhythmias, with the most severe poisonings presenting with unconsciousness, convulsions, and cardiovascular collapse, followed by shock, pulmonary edema, and death.[30,31] (*See Table 3.*)

Table 2. Symptoms of Carbon Monoxide Toxicity

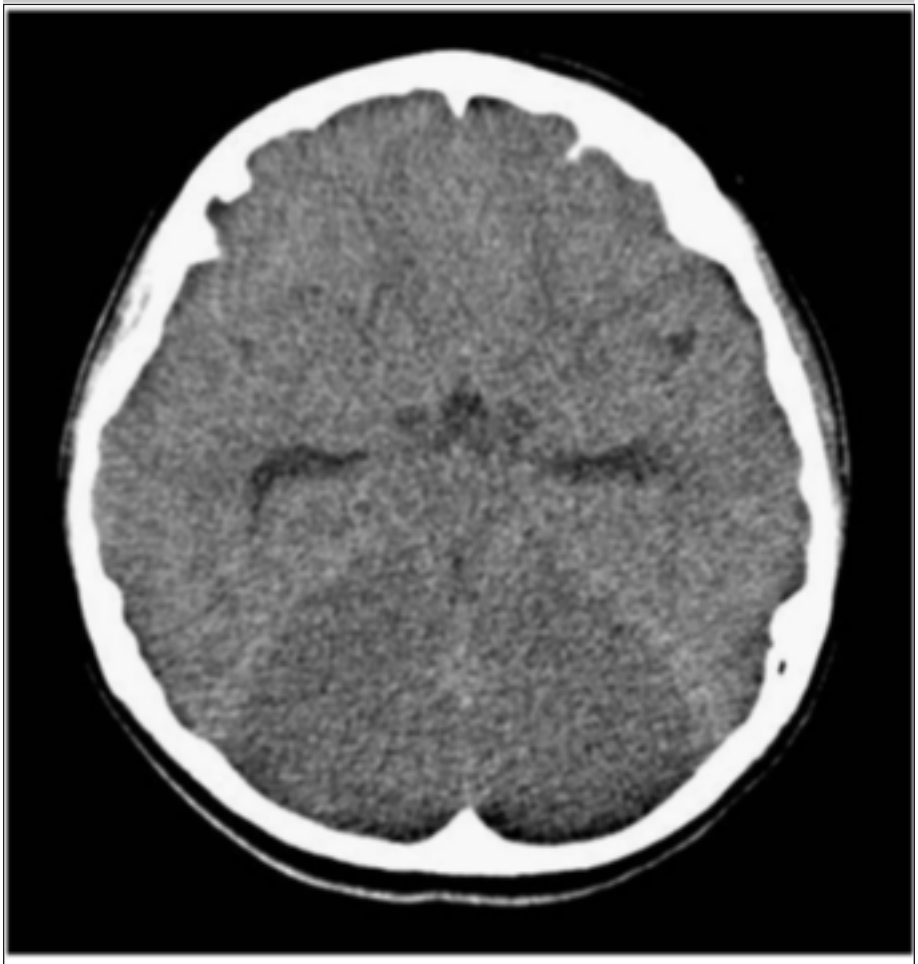
Mild Exposure	Severe Exposure
Headache	Delirium
Dizziness	Ataxia
Nausea	Loss of consciousness
Vomiting	Chest pain
Confusion	Dyspnea
Vertigo	Myocardial infarction
Irritability	Stroke
Flu-like symptoms	Coma
Fatigue	Death

Diagnosis of Carbon Monoxide Poisoning

The key to diagnosis of CO poisoning is to have a high index of suspicion. Nonspecific symptoms and a history of household contacts with similar symptoms and circumstances surrounding the onset of symptoms should prompt consideration of exposure in the differential diagnosis.

CO poisoning causes cellular hypoxia. Standard pulse-oximetry does not

Figure 1. Diffuse Hypodensity of Cerebellum Due to Hypoxic-ischemic Injury



Axial nonenhanced cranial CT scan of an 8-year-old patient on the day of CO poisoning shows diffuse hypodensity of the cerebellum due to hypoxic-ischemic injury.
Reprinted with permssion Fan HC, Wang AC, Lo CP et al. Damage of cerebellar white matter due to carbon monoxide poisoning: A case report. *Am J Emerg Med* 2009;27:757.e5–757.e7.

distinguish between the wavelengths caused by the refractions of hemoglobin, carboxyhemoglobin, and methemoglobin. Previously, measuring plasma levels of carboxyhemoglobin (COHb) was the gold standard for diagnosing CO poisoning. Pulse Co-oximetry (Rad-57 Masimo Corporation, Irvine, CA) distinguishes between the wavelengths of hemoglobin, methemoglobin, and carboxyhemoglobin, and has become widely available, but the reliability is debatable. Several studies have shown co-oximetry to be highly reliable compared to COHb measurements.[32-36] Other studies have shown a high false-positive rate, and conclude that a positive screening for CO poisoning on co-oximetry warrants confirmation by COHb levels.[37-39] Studies have also shown that co-oximetry tends to overestimate actual COHb levels.[38,40] What can be concluded is that co-oximetry is a useful tool to quickly screen suspected CO poisonings, which can then be confirmed by laboratory analysis.

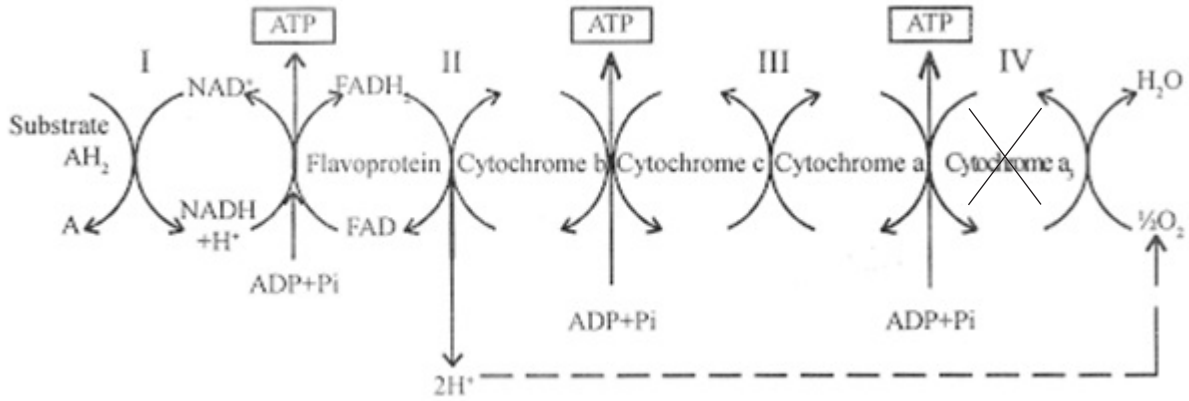
Normal carboxyhemoglobin levels are between 2% and 5% in nonsmokers and can range from 5-13% in chronic smokers. The presence of fetal hemoglobin, as high as 30% at 3 months, may be read as an elevation of HbCO level to 7%.[20,21,23] The level of carboxyhemoglobin in the blood does not correlate with the severity of toxicity.[41] Negative carboxyhemoglobin levels may be present in patients who have already received 100% oxygen or if sufficient time has passed since time of exposure.

Often there is a mild leukocytosis in carbon monoxide poisoning. Arterial blood gases should show a normal PaO_2 level and a mild acidosis. Cardiac biomarkers can be elevated if the exposure has resulted in myocardial ischemia. Metabolic acidosis secondary to lactic acidosis can be seen if there is ischemia. In severe poisonings, there can be evidence of end organ injury, such as elevated BUN, creatinine, and creatinine kinase levels, as well as proteinuria due to rhabdomyolysis. There can be abnormal liver function tests due to hepatic failure.[21,23] Characteristic findings on imaging of the brain are petechial hemorrhages of the white matter, necrosis of the bilateral globi pallidi, spongy change, and progressive demyelination in the cerebral cortex, thalamus, and hippocampus.[42]

Diagnosis of Cyanide Poisoning

Due to its lethality and the delay in return of blood cyanide levels, cyanide poisoning is a clinical diagnosis. The diagnosis should be suspected in anyone who inhales smoke in a closed-space fire, particularly in the presence of soot in the mouth, altered mental status, and hypotension.[43] Blood lactate levels greater than 8 mmol/L in non-smoke-inhalation victims and greater than 10 in smoke-inhalation victims should be considered diagnostic. While severe burns, CO poisoning, and other traumatic injuries can cause lactic acidosis, they are unlikely to cause the profound lactic acidosis that is seen in CN poisoning.[44,45] Due to delay in results, laboratory cyanide levels should only be obtained for diagnostic confirmation and not for medical decision making. Toxic and lethal thresholds of cyanide are 1.0 mg/L and 3.0 mg/L, respectively.[46] Venous blood gases with very high PaO_2 levels (levels that would be anticipated on an arterial blood gas) can also be suggestive of cyanide poisoning. This is attributed to the inability of cells

Figure 2. Mitochondrial Electron Transport Chain



to extract and use oxygen, and so venous blood remains highly oxygenated.[43]

Treatment of Carbon Monoxide Poisoning

Treatment begins by removing the victim from the source of exposure. The antidote of carbon monoxide poisoning is oxygen. (*See Table 4.*) All victims suspected of toxicity should receive 100% oxygen via a nonrebreather mask, noninvasive positive pressure ventilation, or endotracheal ventilation. The theory of treatment is based on oxygen competitively displacing CO from hemoglobin. While breathing room air, this process takes about 300 minutes. While on a 100% oxygen by nonrebreather mask, this time is reduced to about 90 minutes; with hyperbaric oxygen treatment, the time is shortened to 32 minutes.[47] Once the diagnosis is confirmed, the use of hyperbaric oxygen should be considered. (*See Figure 3.)* The only absolute contraindications to hyperbaric therapy are an untreated pneumothorax, ongoing medical instability, and medication history of past bleomycin or cisplatin treatment, or current doxorubicin, disulfiram, or sulfamylon treatment.[47,48] The goal of initial intervention, whether hyperbaric or normobaric oxygen, is a carboxyhemoglobin level of less than 5%.[23] Once the patient is clinically stable, imaging of the brain and neuropsychological testing is required. MRI is the most sensitive modality to detect brain abnormalities, although it is unknown if MRI findings during acute poisonings can predict long-term neurological sequelae.[27,49]

Table 3. Symptoms of Cyanide Poisoning

Symptoms of Mild Cyanide Poisoning

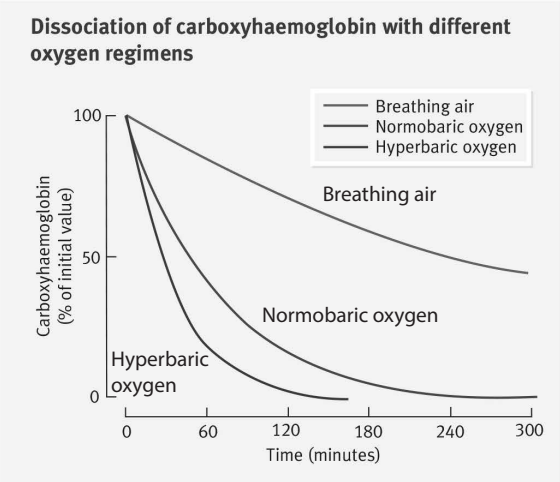
- Headache
- Nausea
- Vertigo
- Anxiety
- Altered mental status
- Tachypnea
- Hypertension

Symptoms of Serious Cyanide Poisoning

- Dyspnea
- Bradycardia
- Hypotension
- Arrhythmia
- Unconsciousness
- Convulsions
- Cardiovascular collapse

Hyperbaric treatment remains controversial because there is no global consensus of when it is appropriate to consider hyperbaric treatment and what hyperbaric protocols should be used. The Undersea and Hyperbaric Medical Society recommends hyperbaric oxygen therapy for patients with serious carbon monoxide poisoning — as manifested by transient or prolonged unconsciousness, abnormal neurologic signs, cardiovascular dysfunction, or severe acidosis — or patients who are 36 years of age or older, were exposed for 24 hours or more (including intermittent exposures), or have a carboxyhemoglobin level of 25% or greater.[48,50] (*See Table 5.*) Studies have indicated that hyperbaric oxygen can decrease the severity and incidence of long-term neurological sequelae.[48,51] Animal studies have shown that hyperbaric oxygen leads to more rapid improvement in cardiovascular status, lower mortality,

Figure 3. Dissociation of Carboxyhaemoglobin with Different Oxygen Regimens



Increasing the partial pressure of inspired oxygen accelerates elimination of carbon monoxide.
Reprinted with permission from Bateman DN. Carbon monoxide. *Medicine* 2012;40:115-116.

Table 4. Treatments for Carbon Monoxide Poisoning

- 100% oxygen by nonrebreather mask, noninvasive positive pressure ventilation, or endotracheal intubation
- Hyperbaric oxygen treatment

Table 5. When to Consider Hyperbaric Oxygen Treatment for CO Poisoning

- When the patient has a COHb level greater than 25-30%
- Evidence of cardiac involvement
- Severe acidosis
- Transient or prolonged unconsciousness
- Neurological impairment
- Abnormal neuropsychiatric testing
- Patient's age is 36 years or older
- Pregnant women

Source: Centers for Disease Control and Prevention

and lower incidence of neurological sequelae.[51] However, in February 2008, a clinical policy statement released by the American College of Emergency Physicians (ACEP) gave Level C recommendations to hyperbaric oxygen as a therapeutic option for CO-poisoned patients, but that its use cannot be mandated.[52]

Cyanide Treatment

Due to the lethality of cyanide, once a patient is removed from the potential source of exposure, initiation of antidote therapy must occur as soon as possible, preferably in the pre-hospital stage. There are numerous available options for treatment of cyanide poisoning. The most commonly selected treatments are hydroxycobalamin and sodium thiosulfate. (*See Table 6.*) Which treatment is superior remains controversial. To date, there are no well-designed randomized, controlled trials comparing the efficacy of hydroxycobalamin versus sodium thiosulfate with sodium and/or amyl nitrite.

The newest drug available in the United States, hydroxycobalamin (Cyanokit-R), was approved by the FDA for treatment of known or suspected cyanide poisoning in 2006. Hydroxycobalamin (vitamin B12a) contains a cobalt ion that binds cyanide with greater affinity than cytochrome oxidase, to form the nontoxic metabolite cyanocobalamin (vitamin B12 family), which is excreted in the urine. The starting dose of hydroxycobalamin is 5 g given as an IV infusion over 15 minutes. A second dose of 5 g can be given.[53] In animal studies, hydroxycobalamin showed anecdotal efficacy for treatment of cyanide poisoning.[54] In a swine model of cyanide-induced cardiac arrest, hydroxycobalamin improved mean arterial blood pressures and pH, decreased lactate and cyanide levels in the blood when compared to epinephrine, and hydroxycobalamin showed increased survival in pigs compared to sodium thiosulfate.[55,56] In humans, a prospective study showed 28 out of 42 (66.7%) confirmed cyanide poisonings survived after treatment with hydroxycobalamin.[57] A retrospective study of hydroxycobalamin given empirically pre-hospital for suspected cyanide poisoning showed a survival to hospital discharge in 30 out of 72 patients in whom survival status was known (41.7%), and showed a pre-hospital return of spontaneous circulation in 21 out of 38 (55%) of patients in cardiac arrest.[58] The most common

Table 6. Treatments for Cyanide Toxicity

Treatment	Mechanism of Action	Delivery	Side Effects
Hydroxycobalamin	Binds to CN to form cyanocobalamin	5 g IV infusion over 15 minutes Additional 5 g may be given	Chromaturia Red skin discoloration Transient hypertension
Sodium thiosulfate	Uses rhodanase to form thiocyanate	12.5 g IV infusion over 30 minutes Given after sodium nitrite Given as sole agent if suspected CO poisoning May repeat at half dose after 1 hour	Hypotension (infusion rate dependent)
Sodium nitrite	Oxidizes hemoglobin to methemoglobin, combines with cyanide to form cyanomethemoglobin	300 mg IV infusion at 75-150 mg/min	Methemoglobinemia Hypotension
Amyl nitrite	Oxidizes hemoglobin to methemoglobin, combines with cyanide to form cyanomethemoglobin	0.3 mL ampule crushed and contents poured onto a gauze and placed in front of patient's mouth, or endotracheal tube if patient intubated, to inhale over 15-30 sec; repeat qMin until IV sodium nitrite available	Methemoglobinemia Hypotension

side effects of hydroxycobalamin are chromaturia and red skin discoloration, which are asymptomatic, self-limiting, and typically resolve in 72 hours. Another side effect is a brief hypertension that returns to baseline an average of 4 hours after administration. The most serious adverse effect documented in healthy volunteers was an allergic reaction in 2 out of 136 volunteers, both of which did not decompensate to full anaphylaxis.[57,59,60] Hydroxycobalamin can cause alterations in laboratory values, including elevated renal and hepatic function and uninterpretable dipstick urinalysis. This also typically resolves by 72 hours after administration.[53,57] The use of hydroxycobalamin can also interfere with measurement of carboxyhemoglobin levels by co-oximetry.[61] There is also a case report of hydroxycobalamin interfering with hemodialysis, causing the dialysis machine to shut down due to internal sensors detecting a "blood leak," which the authors attributed to hydroxycobalamin.[62]

Sodium thiosulfate removes cyanide through the action of rhodanase, an enzyme located in the liver, kidneys, and skeletal muscle. It acts to add a sulfur atom to cyanide to form thiocyanate, which is then excreted in the urine.[30] Compared to hydroxycobalamin, sodium thiosulfate has a slower onset of action.[63] The recommended dosing according to manufacturer is to administer 12.5 g (250 mg/kg in pediatrics, not to exceed 12.5 g) immediately after administration of sodium nitrite over 30 minutes.[64] No clinical trials of sodium thiosulfate are available, and efficacy has been extrapolated from case studies and series of acute cyanide poisoning.[54]

Amyl nitrite and sodium nitrite both are part of the cyanide antidote kit, which had been the treatment of choice for cyanide poisoning in the United States prior to the introduction of hydroxycobalamin in 2006. Sodium nitrite is given IV. Amyl nitrite perles are crushed up and inhaled and are generally reserved for cases in which IV access is delayed or not possible. Nitrites oxidize the iron in hemoglobin from the ferrous to the ferric state, converting hemoglobin into methemoglobin. Cyanide then combines with methemoglobin to form cyanomethemoglobin. It also causes vasodilation, leading to hypotension. The major side effects are hypotension and cellular hypoxia secondary to methemoglobinemia.[30,65] There is a black box warning for the use of nitrites because of hypotension and methemoglobinemia.[66] Use of amyl or sodium nitrite is not recommended in smoke inhalation victims who may also have carboxyhemoglobinemia secondary to carbon monoxide poisoning.[57]

Other antidotes not available in the United States include 4-dimethylaminophenol (4-DMAP) and dicobalt edentate. 4-DMAP is used outside of the United State instead of sodium or amyl nitrite. It generates methemoglobin more rapidly than sodium nitrite, with peak methemoglobin concentrations at 5 minutes after administration compared to 30 minutes after sodium nitrite administration. Like nitrites, the major side effects are methemoglobinemia and hypotension. Cobalt in the form of dicobalt edetate has been used as a cyanide chelator, but its usefulness is limited by serious adverse effects such as hypotension, cardiac dysrhythmias, decreased cerebral blood flow, and angioedema.[17]

Conclusions

Practitioners need to maintain a high index of suspicion to identify and treat carbon monoxide and cyanide

poisonings in smoke inhalation victims. Carbon monoxide poisoning can be identified with detection of carboxyhemoglobin levels in blood or bedside co-oximetry. The level of carboxyhemoglobin does not correlate with the level of toxicity. In addition to 100% normobaric oxygen, hyperbaric oxygen is a treatment option, although there is controversy regarding its benefits. Conversely, there is no quick test to confirm cyanide poisoning. Upon initial evaluation, fire victims with soot in their mouth, altered mental status, and metabolic acidosis with highly elevated lactate levels are highly suggestive of cyanide poisoning. Once cyanide toxicity is suspected, treatment must begin immediately. Further study is needed to determine the efficacy of hydroxycobalamin compared to sodium thiosulfate. If carbon monoxide poisoning is suspected, amyl and sodium nitrite are contraindicated.

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CME/CNE Questions

1. A patient brought from a house fire may be at risk for which of the following?
   A. trauma
   B. smoke inhalation
   C. carbon monoxide poisoning
   D. cyanide poisoning
   E. all of the above
2. Which of the following is a non-invasive way to screen for carbon monoxide poisoning?
   A. pulse co-oximetry
   B. pulse oximetry
   C. fetal hemoglobin screen
   D. clinical CO score
3. What is the first and most important treatment for carbon monoxide poisoning?
   A. cyanocobalamin
   B. hydroxycobalamin
   C. sodium thiosulfate
   D. amyl nitrite
   E. oxygen
4. What is the source of carbon monoxide's toxicity?
   A. a higher affinity to hemoglobin than oxygen
   B. impaired oxygen delivery to tissues
   C. cellular hypoxia
   D. multi-organ dysfunction
   E. all of the above
5. Which of the following patients may be particularly susceptible to carbon monoxide toxicity?
   A. men
   B. women
   C. smokers
   D. pregnant patients
6. Cyanide's mechanism of toxicity in humans involves which of the following?
   A. disruption of oxygen-hemoglobin affinity
   B. disruption of electron transport and ATP production
   C. direct cytotoxic injury
   D. direct neurotoxic injury
7. Since there is currently no rapid test for cyanide poisoning, which of the following is highly concerning for

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CME Objectives

Upon completing this program, the participants will be able to:

- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients; and
- identify possible complications that may occur with traumatic injuries.

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To earn credit for this activity, please follow these instructions:

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cyanide poisoning in a smoke exposure victim?
A. WBC > 30 k/uL
B. Hgb < 8 g/dL
C. pO_2 > 60 mmHg
D. lactate > 8 mmol/l
E. sodium < 125 mmol/L

8. Hydroxycobalamin selectively binds cyanide, resulting in the formation of which of the following?
A. free radicals
B. methemoglobin
C. cyanocobalamin
D. lactic acid
E. hydroxyurea

9. Amyl nitrite and sodium nitrite, when used to treat cyanide poisoning, may have which of the following serious side effects?
A. hypotension and methemoglobinemia
B. hypertension with crisis
C. hypernatremia
D. cobalt poisoning
E. cyanide deficiency

10. Cyanide, in the form of hydrogen cyanide gas, is formed from the combustion of carbon and nitrogen commonly found in:
A. liquid propane
B. coal
C. gasoline
D. oil
E. foam rubber, wool, plastics

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