PRACTICAL SUMMARIES IN

ACUTE CARE

A Focused Topical Review of the Literature for the Acute Care Practitioner

Carbon Monoxide Poisoning

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Introduction

Carbon monoxide (CO) poisoning is the leading cause of death and injury due to poisoning in the United States, and the worldwide incidence of CO poisoning is estimated to be largely under-diagnosed, with more than one-third of all cases going undetected.[1,2]

The Centers for Disease Control and Prevention reported that CO poisoning contributed to an average of 1902 unintentional deaths and 2385 intentional suicides per year in the United States from 1968 through 1998.[3,4] In the three-year-period from 2001 through 2003, there were more than 15,000 annual emergency department (ED) visits related to CO exposure/poisoning and 500 annual deaths attributed to unintentional, non-fire-related CO exposure.[5]

CO poisoning epidemics commonly occur during winter months with the misuse of non-electronic heating and cooking devices.[6,7] Use of these devices also has been shown to increase CO poisoning incidence during natural disasters like hurricanes when prolonged power outages are common.[8]

CO is the product of incomplete combustion of carbon-based products, such as gas or coal. Because it is colorless, odorless, and tasteless, CO is undetectable by human senses. While there are numerous potential sources for CO, the two most common are motor vehicle exhaust and smoke. In addition to the more common sources, methylene chloride—a chemical found in some automotive cleaners, spray paints, and other household products—is converted into CO in the liver after the compound is ingested or inhaled.[9]

The effects of CO become apparent after an exposure period of 20 hours to ambient levels as low as 100 ppm. Exposure to levels near 1000 ppm for greater than two hours can result in COHb levels of 50% or greater.[10]

Pathophysiology

CO toxicity is a result of tissue hypoxia and direct CO-mediated damage at the cellular level. CO binds competitively to hemoglobin; in fact, hemoglobin's affinity for CO is approximately 240 times that of oxygen. This is why, even with low ambient levels of CO, significant toxicity can result over time with prolonged exposures. In addition to hemoglobin binding with CO with such great affinity, the binding of CO to hemoglobin causes a leftward shift of the oxyhemoglobin dissociation curve (the Haldane effect). This results in a decrease in oxygen delivery to the peripheral tissues. The net results are impaired oxygen delivery and cellular hypoxia.

In addition to binding hemoglobin, carbon monoxide binds cardiac and skeletal myoglobin, with a three times greater affinity for cardiac myoglobin than hemoglobin.[11] Because carboxymyoglobin dissociation is slower

VOLUME 3 • NUMBER 1 • JANUARY 2008 • PAGES 1-8

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Statement of Financial Disclosure: Executive Editor, Ann M. Dietrich, MD, FAAP, FACEP, Dr. Khandelwal (author) and Dr. Winograd (peer reviewer) reported no financial relationships with companies having ties to this field of study. Dr. Kaide (author) reported that he is part owner and chief clinical consultant of Callibra, Inc.

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than that of COHb due to its increased affinity, it is possible to see a rebound effect with late release of CO from myoglobin and its subsequent binding to hemoglobin.[12]

During pregnancy, the fetus is particularly vulnerable to CO exposure because fetal hemoglobin binds CO with a greater affinity than hemoglobin A. This, combined with slow transplacental transport and the fetal oxyhemoglobin dissociation curve being naturally shifted to the left, can cause fetal CO levels to be deadly in exposures that typically would be nonfatal.[13,14]

Presenting Features

The signs and symptoms of CO exposure depend on the amount of CO in inspired air, minute ventilation, and duration of exposure to CO. The diagnosis of CO poisoning can be easily missed because the clinical findings of CO poisoning are highly variable and nonspecific and can mimic a viral syndrome.[15] The physician must include CO poisoning in the differential diagnosis of every patient who presents with any of the common viral symptoms. This is especially true during winter months, when both viral syndromes and accidental CO poisonings occur with greater frequency.

While there is a correlation between the degree of a patient's symptoms and a rise in measured COHb levels, the actual COHb level does not predict the degree of symptoms that a patient may experience. One can generalize about symptomatology at the extremes of COHb levels. COHb levels between 3% and 10% are common in asymptomatic cigarette smokers.[16] Levels greater than 25% are considered significantly elevated, with levels between 40% and 50% almost always causing overt symptoms.[10]

At lower COHb levels, acute CO poisoning usually presents with symptoms such as headache, dizziness, nausea, and weakness. As COHb levels increase, patients may become confused or have difficulty concentrating, which can proceed to lethargy and coma.[15,17,18] Tachycardia and tachypnea may develop in response to cellular hypoxia. Additionally, the sensation of air hunger and agitation may be seen, which can progress in later stages to hypotension, bradycardia, and decreased respiration.

The extremes of age are at higher risk for morbidity and mortality from CO exposure. Patients with coronary artery disease may have anginal symptoms or actual myocardial infarction after CO exposure, due to the relative hypoxia at the cellular level.[19] Patients with underlying pulmonary and cerebrovascular disease may witness an exacerbation of symptoms. The classically described cyanotic patient with cherry-red lips actually is rarely seen.[20]

It is not uncommon to have a patient awake and alert upon ED presentation, despite having been described by rescuers as unconscious or barely conscious at the scene. Despite their grossly normal cognitive status upon presentation, these patients sustained hypoxia sufficient to cause end-organ injury and syncope and require aggressive treatment. Secondary ischemic injury frequently is seen in severe CO poisoning.

Since patients can be considered "alert and oriented x 3" and still have some degree of cognitive impairment, a neuropsychologic (NP) testing battery has been developed to help assess subtle cognitive changes that may be overlooked during routine ED examination. The Carbon Monoxide Neuropsychologic Screening Battery, which takes up to 30 minutes to administer, consists of six different tests that together assess global cognitive function. Although some centers use this as criteria for HBO (hyperbaric oxygen) treatment, controversy exists about whether the testing has the ability to predict which patients will develop delayed neurologic sequelae (DNS). Furthermore, some feel that the testing doesn't allow for differentiation between cognitive impairment from CO and cognitive impairment from other possible coingestants. Finally, it may be

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Outside the United States
$329 per year plus GST (Student/Resident rate: $159.50 plus GST).

Practical Summaries in Acute Care, ISSN 1930-1103, is published monthly by AHC Media LLC, 3525 Piedmont Rd., NE, Bldg. 6, Suite 400, Atlanta, GA 30305.

SENIOR VICE PRESIDENT/PUBLISHER: Brenda L. Mooney
ASSOCIATE PUBLISHER: Lee Landenberger
SENIOR MANAGING EDITOR: Suzanne Thatcher
MARKETING MANAGER: Shawn DeMario
GST Registration Number: R128870672.
Periodical postage paid at Atlanta, GA.
POSTMASTER: Send address changes to ***Practical Summaries in Acute Care***, P.O. Box 740059, Atlanta, GA 30374.

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difficult to attribute abnormal NP testing to CO poisoning in patients with preexisting psychological and psychiatric illnesses.[21]

Specific Injury Patterns

There are three specific injury patterns that deserve special mention: cardiovascular sequelae, DNS, and symptoms associated with long-term exposure. As moderate to severe CO poisoning is associated with an increased risk of cardiovascular sequelae, an ECG and cardiac biochemical markers should be obtained upon presentation and followed throughout the patient's hospitalization.[22] Patients presenting with cardiac injury must be closely followed during hospitalization and at discharge as there does appear to be a correlation between myocardial injury at presentation and long-term mortality.[23]

A significant proportion of patients, up to 40%, with significant CO exposure develop a syndrome of DNS, characterized by variable degrees of cognitive deficits, movement disorders, personality changes, and focal neurological deficits. While onset of DNS usually is within 20 days of recovery from the initial insult, they have been shown to occur as many as 240 days afterward. These deficits may last for a year or longer, necessitating ongoing neurological and neuropsychiatric follow-up.[24-26]

Chronic CO poisoning, from long-term exposure at low levels, frequently is overlooked due to obscure and vague symptomatology, a wide range of presentations, and a general lack of awareness of the problem. The most commonly reported symptoms are: headache, dizziness, insomnia, anorexia, nausea, weight loss, apathy, and personality disturbance. Palpitations, impaired memory, decreased libido, increased sweating, impairment in sleep, and diminished alcohol tolerance also may be seen. Neurological signs, including hyperreflexia, altered pain perception, nystagmus, ataxia, weakness, tremors, myoclonus, hemiplegia, anosmia, aphasia, and facial nerve palsies, have been reported.[27,28]

Diagnosis

The diagnosis of acute CO poisoning requires diligence and an algorithmic approach. A history of potential exposure is the most reliable indicator, although this may be difficult to ascertain; therefore, a high index of suspicion on the part of the physician is paramount. In practical terms, this means that the physician must at least consider CO poisoning in any patient who presents with nausea, headache, dizziness, or lethargy.

When considering CO poisoning in families or groups of people, it is important to remember that the patients usually have parallel presentations. On the other hand, viral syndromes tend to cause illness in an index member of the family who then passes the disease on to other group members. These other family members present serially and independently to their physicians. When an entire family presents with similar symptoms that fit the demographics and patterns for CO poisoning, this diagnosis must be pursued and excluded.

COHb levels may in fact be normal at presentation, particularly if the patient has been removed from the CO source for some time period. Once CO poisoning is suspected, the COHb level can be measured in either venous or arterial blood samples, with venous usually being the preferred source.[29] It is important to note that pregnancy and hemolytic anemia can increase COHb levels to 5% and that heavy smoking can elevate levels to as high as 13%.[30]

Pulse oximetry has no role in the screening or diagnosis of CO poisoning because pulse oximeters can misread COHb for oxyhemoglobin and give falsely elevated oxygen saturation values. If a patient's COHb levels are normal at the time of evaluation, but the index of suspicion for CO toxicity remains high, it is not unreasonable to send police, emergency medical services, or the local gas company to test ambient CO levels at the site in question.

Treatment

Prompt removal of the patient from the source of CO poisoning and high-flow oxygen by nonrebreather mask at 15 L/min is the mainstay of treatment for acute CO poisoning. The use of 100% FiO_2 would be ideal to hasten the removal of CO from the hemoglobin molecule; however, this is only attainable in the operating room by way of an anesthesia circuit.[31] Breathing this concentration of oxygen effectively reduces the half-life of COHb from 300 minutes at ambient atmospheric conditions to 90 minutes. Seizures, cardiac ischemia, hypotension, and other complications of acute CO poisoning can be managed supportively.

The use of HBO (hyperbaric oxygen) in the treatment of CO poisoning is widely accepted due to a clear scientific rationale for its use, but controversy does exist in some circles regarding its clinical efficacy. At three atmospheres absolute (ATA), which is three times ambient atmospheric pressure, HBO decreases the half-life of COHb to less than 30 minutes.[32] HBO also increases the amount of oxygen dissolved in blood from 0.3 mL/dL with 100% FiO_2 to 6 mL/dL under hyperbaric conditions, which is enough to sustain life even in the absence of hemoglobin. In animal studies, HBO has been shown to

promote the dissociation of CO from cytochrome-c oxidase, inhibit leukocyte adhesion, and reduce brain lipid peroxidation.[33-35] These effects appear to play a role in limiting direct cellular toxicity and decreasing the incidence of DNS.

The debate of hyperbaric versus normobaric oxygen is ongoing in the scientific community. While HBO may not be appropriate in every patient, its potential clinical benefit, coupled with its minimal side-effect profile (primarily aural barotrauma), should result in its administration to patients who demonstrate any of the following: coma; acidosis with a pH below 7.1; history of loss of consciousness (even if awake upon presentation); any neurological abnormality; or evidence of cardiac dysfunction. Pregnant women would benefit from HBO with COHb levels above 15% or if they exhibit any signs of fetal distress. Patients with persistent neurological symptoms despite normobaric oxygen therapy, patients with neuropsychometric abnormalities, or patients with severely elevated COHb levels also may derive benefit from HBO.

Despite the general acceptance of HBO for the treatment of severe CO poisoning, no single, universally accepted protocol for level of pressure and duration of treatments has been established within the hyperbaric community. Protocols generally range from a single treatment for 60 minutes at a pressure of 2.8-3.0 ATA to multiple treatments at varying levels of pressure. There is evidence to support a multi-treatment regimen based on work by Gorman and Runciman that showed lower mortality and less residual neurological deficits with multiple treatments.[36]

Conclusion

Every physician should expect to encounter a patient with CO poisoning. It is vital to maintain a high index of suspicion because this complicated and often-lethal entity is associated with a highly variable clinical presentation. Intervention in the ED may inhibit or decrease the pathophysiologic consequences that are mediated by multiple mechanisms at the cellular level.

Measuring the COHb level, using a venous sample or CO-oximeter, can help the physician confirm a suspected diagnosis of CO poisoning. Normobaric, high-flow oxygen is the standard treatment, with hyperbaric oxygen reserved for select cases.

There have been a few significant changes in how clinicians look at CO poisoning over the past several years, with the majority of the focus on whether on not to treat with HBO. In the following section, the authors will review several key questions that face the acute care clinician when assessing an acute carbon monoxide poisoned patient.

Should hyperbaric oxygen be used in CO poisoning to prevent delayed neurologic sequelae?

Source: Weaver LK, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. *N Engl J Med* 2002;347:1057-1067.

Authors randomly assigned patients with symptomatic acute carbon monoxide poisoning (76 patients in each treatment group) to either 3 sessions with HBO therapy or normobaric oxygen with sham HBO therapy to maintain blinding. Enrolled patients included half with a reported loss of consciousness and 8% requiring intubation. Follow up rate was 95%. Neurologic sequelae were determined by performance on 6 neuropsychological tests. At 6 weeks, HBO was associated with a 21.1% absolute reduction in the rate of neurologic sequelae. At 12 months, the effect was reduced but still present at a 14.5% absolute reduction.

Commentary

While there are definitely conflicting opinions on this issue, Weaver and coworker's study is the only randomized, double-blind, placebo controlled clinical trial that addresses long-term neurologic sequelae. This trial (Class I in design and execution) demonstrates statistical significance with regard to the reduction of the incidence of neurological sequelae measured 6 weeks, 6 months, and 1 year after CO poisoning using HBO treatment. Post-hoc subgroup analysis of patients from the Weaver et al trial demonstrated that HBO therapy improved outcome specifically in patients with loss of consciousness, metabolic acidosis as defined by a base excess of less than -2 mmol/liter, and COHb > 25%. In addition, a more recent cohort analysis also demonstrated a statistically significant reduction in the incidence of neuro-cognitive sequelae with the use of HBO therapy in CO poisoned patients older than age 36 and in those with exposure intervals greater than 24 hours.[37]

Critics of HBO in CO poisoning often cite the study performed by Scheinkestel's group from Australia.[38] However, the numerous flaws in the design and execution of the trial significantly impair the study's conclusions that HBO therapy was no more effective than normobaric oxygen. These flaws include, but are not limited to the following: A primary outcome measure not intended to look at delayed neurologic sequelae; the use of an unconventional placebo arm; the continuous application of normobaric oxygen for a minimum of 72 hours in both groups (which may

result in pulmonary oxygen toxicity); an unusually high incidence of neurologic sequelae in both arms (not consistent with the previously established incidence of neurologic sequelae); atypical patient population with a high percentage of patients with intentional exposure as well as a high co-ingestant rate; and finally, very poor follow up rates.

Although one paper should not create a "standard of care" policy, the Weaver et al study is the best study to date that reviews this topic; therefore, it should be given considerable weight when the question of HBO for CO poisoning arises.

When should cardiac testing be done in a patient presenting after carbon monoxide poisoning?

Source: Satran D, et al. Cardiovascular manifestations of moderate to severe carbon monoxide poisoning. *J Am Coll Cardiol* 2005; 45:1513-1516.

The authors reviewed the cardiovascular manifestations of 230 consecutive patients treated for moderate to severe poisoning in the hyperbaric chamber at Hennepin County Medical Center. Indications for HBO therapy included any history of loss of consciousness, seizure, focal neurologic deficit, ischemic chest pain, ECG changes, new dysrhythmias, or hypotension. In addition, carboxyhemoglobin levels greater than 40% or 25% with a history cardiovascular disease, cerebrovascular disease, age $\geq$ 60, exposure $\geq$ 2 hours, or a Hgb $\leq$ 10 mg/dL also were indications. In the group, 72% were men and the mean age was 47.2 years. In terms of baseline cardiac risk factors, 56% were smokers, 23% had hypertension, 7% had diabetes, and 7% had a previous myocardial infarction. Myocardial injury, assessed by biomarkers (cardiac troponin I level $\geq$ 0.7 ng/mL or creatine kinase-MB level $\geq$ 5.0 ng/mL) or ECG changes, were demonstrated in 37% of patients. There were two patterns of myocardial injury: 1) severely poisoned (GCS < 15) younger patient (average age, 43) with few cardiac risk factors and an echocardiogram demonstrating global left ventricular dysfunction consistent with a stunned myocardium; and 2) older patients (average age 64, 50% with a normal GCS) with a higher frequency of cardiac risk factors along with regional wall motion abnormality seen by echocardiogram, usually unmasking underlying CAD.

Commentary

It appears appropriate to obtain a baseline ECG and serial cardiac markers in those patients presenting with anything more than mild poisoning. The problem for the practicing physician is to separate mild poisoning from moderate/severe poisoning. Perhaps the easiest way to determine moderate or severe poisoning is to look at whether the patient in question is a candidate for hyperbaric oxygen therapy. Although this may vary from center to center, only moderate and severely poisoned individuals fit that criteria. For instance, some centers would include the presence of abnormal neuropsychologic testing in the moderate poisoning group. Also, most centers would include the presence of a metabolic acidosis as defined by a base excess of less than -2 mmol/liter in the moderately/severely poisoned group. Interestingly, these same authors found that myocardial injury in the subset of patients tested was a significant long-term predictor of mortality as well.[39]

Can COHb levels be accurately assessed with a venous blood gas sample?

Source: Touger M, et al. Relationship between venous and arterial carboxyhemoglobin levels in patients with suspected carbon monoxide poisoning. *Ann Emerg Med* 1995;25:481-483.

The objective of this paper was to determine whether venous blood sampling for COHb accurately predicted arterial COHb. To achieve this, the authors performed a prospective comparison of simultaneously sampled arterial and venous blood in 61 CO poisoned patients. The accuracy of venous carboxyhemoglobin (COHb) as a predictor of arterial carboxyhemoglobin was determined by measures of both correlation and agreement between the two variables. The mean arteriovenous difference was 0.15% (95% CI, -0.13% to 0.45%).

Commentary

There are very sound theoretical reasons to believe that venous and arterial COHb levels would be very close. Chief among these is that COHb is not utilized or metabolized by tissues and no CO would be extracted along the path from the arterial side to the venous side of the circulatory system. This correlation, however, has never been studied in humans until this article was published. Venous blood sampling is less invasive and significantly less painful than an arterial stick and usually can be obtained more quickly. This makes venous COHb determination more desirable than arterial puncture. The results suggest that the agreement between venous and arterial COHb is unlikely to be greater than 1-2%. This difference is very unlikely to influence the deter-

mination between who might receive nonrebreather oxygen and who would need HBO. Additionally, the similarity between arterial and venous pH is close enough to allow for accurate determination of acidosis with only a venous blood gas.

Can noninvasive co-oximetry be used in place of blood gas analysis for CO detection?

Source: Barker SJ, et al. Measurement of carboxyhemoglobin and methemoglobin by pulse oximetry: A human volunteer study. *Anesthesiology* 2006;105:892-897.

In this study, the authors looked at a relatively new technology that uses 8 wavelengths of light to distinguish between various forms of hemoglobin, including: oxyhemoglobin, deoxyhemoglobin, carboxyhemoglobin, and methemoglobin. The device is noninvasive and works much like a standard pulse oximeter, with the added ability to measure other abnormally bound hemoglobin. The authors had 10 volunteers breathe 500 parts per million of carbon monoxide until their arterial COHb (sampled from an indwelling arterial catheter) reached 15%. They additionally looked at 10 volunteers who had methemoglobinemia induced with sodium nitrite. Our review of this study will pertain only to the carbon monoxide group. They analyzed the arterial samples on 3 standard laboratory co-oximeter devices, and then used a noninvasive pulse co-oximeter (NIPC) device made by Masimo Inc. (Rainbow SET Rad-57 Pulse Co-oximetry) to evaluate the COHb. At press time, this device was the only commercially available noninvasive pulse co-oximeter. These readings were done simultaneously with the arterial blood draws. The arterial sample and the noninvasively obtained levels were compared. As an indicator of measurement uncertainty, the authors used a Bland-Altman precision. This showed a 2.2% uncertainty for the NIPC measurement of COHb.

Commentary

NIPC is a very useful advance in technology. It allows rapid and painless testing for CO exposure. The biggest advantage that is conferred is that the barrier for testing for possible CO is lowered immensely. Now, any suspicion of CO poisoning can be quickly evaluated and either confirmed or refuted in less than a minute, with no needles and no lab testing needed. This study reviews simultaneous measurements of CO performed by the lab on arterial blood and by the hand-held NIPC device. A 2.2% uncertainty was calculated. This should be interpreted in the context of an approximately 2% uncertainty that is readily published for the measurement of oxyhemoglobin saturation as measured by standard pulse-oximetry.

There has been some criticism of the accuracy of NIPC in an ED triage screening study. This clinical study showed a number of falsely elevated results on the NIPC and some falsely low readings.[40] Another group described a similar problem initially when conducting a screening of ED patients but found that with proper placement of the device (over the nail bed and not over the adjacent skin), the number of false positives decreased significantly. They enrolled more than 14,000 patients in their protocol and now routinely use the device at triage for CO screening.[41]

Overall, the use of this device seems to be reliable and effective for screening purposes. The use of confirmatory studies is recommended with equivocal readings or when the clinical picture strongly suggests CO poisoning and the NIPC reading is normal.

Should routine CO poisoning screening be performed?

Source: Chee K, et al. Noninvasive carboxyhemoglobin monitoring: screening emergency department patients for carbon monoxide exposure. *Acad Emerg Med* 2006;13(5 suppl 1):s179.

The objective of this paper was to assess patients for occult carbon monoxide exposure and to attempt to correlate elevated levels with clinical and demographic data. In their ED triage area, they utilized a noninvasive pulse co-oximeter (Rainbow SET Rad-57 Pulse Co-oximetry, Masimo, Inc.). Over a 12-day period 2950 patients presented to their triage area, of which 1756 had COHb levels documented. The authors also reviewed vital signs, gender, age, mode of transportation, and smoking to attempt to correlate these with COHb levels. Smoking was the only factor that was consistently correlated with higher levels COHb levels. In the group of screened patients, they found 3 unsuspected cases of significantly elevated COHb levels that were high enough to indicate true CO toxicity. These cases were confirmed by elevated blood carboxyhemoglobin levels.

Commentary

While there has been some criticism of this study in the form of letters to the editor, along with a series report of some false-negative testing using noninvasive co-oximetry, the authors were, nonetheless, able to identify 3 patients of 1756 screened who had carbon monoxide poisoning. In an ED that sees 50,000 patients yearly, these numbers suggest that up to 85 patients with occult CO poisoning would be annually

identified. In many cases the patients who are identified are being exposed to low-level CO and have not yet received enough CO to cause them to become symptomatic. If in fact they were the victims of a faulty furnace, there is a possibility that some may have gone on to severe poisoning as the furnace issues became worse over time. Patients driving vehicles that have exhaust leaks may build up CO levels high enough to cause syncope during long drives. This has tragic potential in a moving vehicle. Even if the co-oximeter missed some patients by under reporting true elevated levels or caused a few additional negative blood screening tests to be performed, it nonetheless will find patients who otherwise may have been sent back to a dwelling that could eventually cause their demise.

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CME QUESTIONS

1. **During pregnancy, the fetus is particularly vulnerable to CO exposure because:**
   A. fetal hemoglobin binds CO with a greater affinity than hemoglobin A.
   B. slow transplacental transport and the fetal oxyhemoglobin dissociation curve being naturally shifted to the left
   C. Fetal CO levels may be deadly in exposures that typically would be nonfatal.
   D. All of the above

2. **Which of the following about CO is correct?**
   A. It interferes with peripheral oxygen utilization
   B. It impairs tissue perfusion
   C. It creates oxidative stress on cells
   D. All of the above

3. **Delayed neurologic sequelae (DNS) in CO exposure patients:**
   A. usually occur within 20 days of recovery
   B. frequently occur up to 3 years later.
   C. Are uncommon
   d. Have no effect on cognition.

4. **Physicians must at least consider CO poisoning in any patient who presents with which of the following?**
   A. Nausea
   B. Headache
   C. Dizziness or lethargy
   D. All of the above

5. **At three atmospheres absolute (ATA), which is three times ambient atmospheric pressure, HBO decreases the half-life of COHb to:**
   A. 24 hours
   B. less than 30 minutes
   C. 18 hours
   D. 9 hours

Answers: 1. D; 2. D; 3. A; 4. D; 5. B