CARBON MONOXIDE POISONING

EDUCATIONAL CURRICULUM

Bryan E. Bledsoe, DO, FACEP
REVIEWS

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EDUCATIONAL OBJECTIVES

TERMINAL OBJECTIVE
Upon completing this presentation the student will be more aware of the importance of carbon monoxide detection and carbon monoxide treatment in the prehospital and fire-ground setting.

COGNITIVE OBJECTIVES
Upon completing this presentation the student will be able to:

1. Describe the physical characteristics of carbon monoxide.
2. Name the principal source of carbon monoxide.
3. Describe the makeup of the carbon monoxide molecule.
4. Identify endogenous sources and exogenous sources of carbon monoxide.
5. Describe methylene chloride and discuss its role in the production of carbon monoxide.
6. Summarize the incidence of carbon monoxide poisoning today.
7. List factors associated with accidental death from carbon monoxide poisoning.
8. Discuss why accidental deaths from carbon monoxide peak in winter months and following disasters and major storms.
9. Differentiate why a pregnant woman and her developing fetus are at increased risk of suffering acute carbon monoxide poisoning.
10. Summarize possible long-term outcomes following fetal exposure to carbon monoxide.
11. Define the typical amount of carbon monoxide present in the atmosphere.
12. List common sources of atmospheric carbon monoxide.
13. Differentiate factors that affect the severity and intensity of carbon monoxide exposure.


15. List NIOSH exposure limits for carbon monoxide.

16. Describe why firefighters are at increased risk for carbon monoxide poisoning.

17. Discuss the pathophysiology of carbon monoxide poisoning.

18. Illustrate the relationship of carbon monoxide and oxygen in hemoglobin binding.


20. Detail strategies to reduce the effective half-life of carboxyhemoglobin.

21. Describe the significance of carbon monoxide binding to myoglobin.

22. Describe nitric oxide and detail its relationship with carbon monoxide exposures.

23. Detail the physiologic and pathophysiologic effects of nitric oxide.

24. List normal carboxyhemoglobin levels encountered in the population as a whole.

25. Discuss the effects of carbon monoxide on the major body systems to include:
   a. Central nervous system
   b. Cardiovascular system
   c. Respiratory system
26. Describe the effects of carbon monoxide on multiple organ dysfunction syndrome (MODS).

27. Identify why the signs and symptoms of carbon monoxide are vague and nonspecific.

28. Explain why carbon monoxide poisoning is often misdiagnosed.

29. Discriminate between acute and chronic carbon monoxide poisoning.

30. Discuss signs and symptoms of acute carbon monoxide poisoning.

31. Relate the symptomatology of carbon monoxide poisoning to carboxyhemoglobin (COHb) levels.

32. Describe why the signs and symptoms of CO poisoning do not always correlate to COHb levels.


34. Identify what delayed neurologic syndrome (DNS) is and detail the risk factors and signs and symptoms.

35. Discuss the importance of carbon monoxide detectors.

36. Detail the recent progress in biological carbon monoxide detectors.

37. Describe the CO-oximeter and list the physiologic parameters it can measure and monitor.

38. List the types of hemoglobin detected by CO-oximetry.

39. Recognize why all fire and EMS personnel should be competent in use of the CO-oximeter.

40. Discuss the treatment strategies for carbon monoxide poisoning based upon exposure level and presenting signs and symptoms.
41. Identify CDC criteria for carbon monoxide poisoning.

42. Describe CDC diagnostic categories for carbon monoxide poisoning.

43. List the carboxyhemoglobin levels that generally indicate the need for carbon monoxide poisoning treatment.

44. Discuss the rationale for the use of high concentrations of oxygen in carbon monoxide poisoning.

45. Discuss the controversy surrounding the use of hyperbaric oxygen therapy in carbon monoxide poisoning.

46. Describe the benefits and indications for hyperbaric oxygen therapy in carbon monoxide poisoning.

47. Discuss the indications for hyperbaric oxygen therapy in carbon monoxide poisoning.

48. Describe the importance of ongoing treatment and monitoring of victims of carbon monoxide poisoning.

49. Describe the pathophysiology of methylene chloride.

50. Identify potential risks to the rescuer when caring for a patient poisoned with methylene chloride.

51. Describe the harmful effects of methylene chloride exposure.

52. Discuss the suggested treatment of methylene chloride exposure.

53. Explain the significance of concomitant carbon monoxide and cyanide poisoning.

54. Explain how cyanide poisoning may be wrongly attributed to carbon monoxide poisoning.
55. Describe the chemical and physical properties of cyanide.

56. Identify common sources of cyanide during combustion.

57. List the OSHA permissible level for cyanide exposure.

58. Discuss the pathophysiology of cyanide exposure.

59. List body tissues most susceptible to cyanide poisoning.

60. Identify the two currently available cyanide antidotes.

61. List the ingredients of the cyanide antidote kit.

62. Describe the function of the nitrites and sodium thiosulfate in the treatment of carbon monoxide poisoning.

63. Describe methemoglobin.

64. List untoward effects associated with nitrite administration.

65. Describe why methemoglobin cannot bind oxygen.

66. Detail concerns about the role of carboxyhemoglobin levels and methemoglobin levels in dual carbon monoxide/cyanide poisonings.

67. Discuss hydroxocobalamin and its relationship to Vitamin B₁₂.

68. Detail how hydroxocobalamin eliminates cyanide.

**AFFECTIVE OBJECTIVES**

Upon completing this presentation the student will be able to:

69. Value the role of CO-oximetry as an adjunct in the assessment of the patient with possible carbon monoxide poisoning.

70. Understand the uses and limitations of CO-oximetry.

71. Recognize the importance of screening for carboxyhemoglobin and methemoglobin in the out-of-hospital setting.
72. Demonstrate an appreciation for the role CO-oximetry plays in developing an accurate field diagnosis/impression in the patient with possible carbon monoxide or carbon monoxide/cyanide poisoning.

73. Understand the significance in how emergency services personnel are at increased risk of carbon monoxide poisoning.

74. Justify why it is important to have a high index of suspicion for both carbon monoxide and cyanide poisoning.

75. Detail why it is important for treatment strategies to be modified based upon the presentation in patients with mixed carbon monoxide/cyanide poisoning.

PSYCHOMOTOR OBJECTIVES

Upon completing this presentation the student will be able to:

76. Demonstrate the proper use of CO-oximetry.

77. Perform a comprehensive patient assessment of a patient with carbon monoxide and/or cyanide poisoning.

78. Demonstrate appropriate interventions for simulated abnormal CO-oximetry readings.

79. Troubleshoot problems that may arise when monitoring with CO-oximetry.

80. Demonstrate proper documentation of CO-oximetry readings.

81. Using a simulated cyanide antidote kit, prepare and administer the antidotes to a simulated patient.
PRESENTATION

I. Introduction:
   A. Overview of carbon monoxide and carbon monoxide poisoning.

II. Chemistry of carbon monoxide:
   A. Gas:
      1. Colorless
      2. Odorless
      3. Tasteless
      4. Non-irritating
   B. Results from incomplete combustion of carbon-containing fuels.
   C. Abbreviated as “CO.”
   D. Molecule consists of one carbon atom and one oxygen atom joined by a triple bond.
      1. Extremely stable molecule

III. Sources of carbon monoxide:
   A. Endogenous:
      1. Normal heme catabolism
      2. Hemolytic anemias
      3. Sepsis
   B. Exogenous:
      1. House fires
      2. Automobile exhaust
      3. Gas-powered generators
      4. Propane-powered vehicles (e.g., fork-lifts)
      5. Heaters
      6. Indoor stoves
      7. Camp stoves
      8. Boat exhaust
      9. Cigarette smoke
     10. Charcoal fires/cookers
   C. Methylene chloride:
      1. Hydrocarbon consisting of 2 chloride and 2 hydrogen molecules bound to carbon molecule.
      2. Used a paint remover and adhesive remover.
      3. Converted to CO in the liver after inhalation.

IV. Incidence of carbon monoxide poisoning.
A. CO leading cause of poisoning deaths in industrialized countries.

B. CO may be responsible for half of all poisonings worldwide.

C. Approximately 5,000-6,000 people die annually in the United States as a result of CO poisoning:
   1. Most are suicides
   2. Accidental poisoning deaths declining:
      a. Improved motor vehicle emission policies.
      b. Use of catalytic converters.
      c. Home CO detectors.
   3. Most accidental CO deaths due to:
      a. House fires
      b. Automobile exhaust
      c. Gas-powered generators
      d. Indoor heating systems
      e. Stoves and other appliances
      f. Charcoal grills
      g. Camp stoves
      h. Water heaters
      i. Boat exhausts
   4. Increased accidental CO deaths seen in:
      a. Patients > 65 years of age.
      b. Male
      c. Ethanol intoxication
   5. Accidental deaths peak in winter:
      a. Use of heating systems
      b. Closed windows
   6. Significant increase in CO poisonings following disasters and storms:
      a. Relates to loss of utilities.
      b. Use of gasoline generators.
      c. Use of fuel-powered heaters.

D. Approximately 40,000-50,000 emergency department visits annually due to CO poisoning.

E. Pregnancy is a particular risk for CO poisoning:
   1. Fetal hemoglobin (HgF) has a much greater affinity for CO than adult hemoglobin (HgA).
   2. Following CO exposure pregnant mothers may exhibit mild to moderate symptoms, yet the fetus
may have devastating outcomes (i.e., seizures, cerebral palsy, and death).

V. Exposure to CO:
   A. Environmental exposure typically <0.001% (10 ppm).
   B. Higher in urban areas.
   C. Sources:
      1. Volcanic gasses
      2. Brush fires
      3. Human pollution
   D. CO exposure is a function of:
      1. Minute ventilation ($V_{min}$)
         a. Minute ventilation:
            i. Tidal Volume $\times$ Respiratory rate
      2. Duration of exposure
      3. Concentration of CO in the environment.
      4. Concentration of O$_2$ in the environment.
   E. Exposure limits:
      1. OSHA:
         a. 50 ppm (as an 8-hour time-weighted average)
      2. NIOSH:
         a. 35 ppm (as an 8-hour time-weighted average)
   F. Firefighter risks:
      1. CO is significant and deadly risk factor for firefighters.
      2. Sources:
         a. Structure fires
         b. Apparatus fumes
         c. Portable equipment fumes (i.e., generators, K-12 saw, chain saws, rescue tools).
         d. Underground utility fires
         e. Closed-space rescue situations
      3. CO is slightly lighter than dry air, yet tends to accumulate in low-lying areas in humid environments.

VI. The pathophysiology of CO poisoning:
   A. Pathophysiology of CO poisoning first described in 1857 by French physician Claude Bernard.
   B. Pathophysiology of CO poisoning is complex.
   C. CO binds to hemoglobin with approximately 250 times the affinity as oxygen.
1. Combination of CO and hemoglobin is called carboxyhemoglobin (COHb).
2. CO displaces O₂ from the hemoglobin binding sites (4).
3. CO prevents O₂ from binding.
4. COHb cannot carry O₂.
5. COHb causes the premature release of the remaining O₂ into the tissues.

D. CO ultimately removed from the circulation and destroyed:
1. Half-life:
   a. Room air: 240-360 minutes
   b. 100% O₂: 80 minutes
   c. Hyperbaric O₂ (HBO): 22 minutes

E. CO also binds to other iron-containing proteins:
1. Myoglobin
   a. Binding reduces O₂ available to the heart:
      i. Ischemia
      ii. Dysrhythmias
      iii. Cardiac dysfunction
2. Cytochrome
   a. Important in cellular energy production
   b. Part of electron transport chain
      i. CO binds to cytochrome.
      ii. Decreases energy production
      iii. Effects very similar to those of cyanide

F. CO causes an increase in circulating levels of nitric oxide (NO):
1. NO is a highly-reactive gas that participates in numerous biochemical reactions.
2. Oxygen free radical
3. Causes cerebral vasodilation through relaxation of smooth muscle in arterioles:
   a. Syncope
   b. Headache
4. Causes peripheral vasodilation:
   a. Syncope
5. May result in oxidative damage to the brain:
   a. Probable cause of delayed neurologic sequelae (DNS).
G. Normal COHb levels:
   1. Endogenous: 0.4-0.7%
   2. Tobacco smokers:
      a. 1 pack/day: 5-6%
      b. 2-3 packs/day: 7-9%
      c. Cigars: up to 20%
   3. Urban commuter: 5%
   4. Methylene chloride (100 ppm for 8 hours): 3-5%
H. Impact of CO on major body systems:
   1. Neurologic:
      a. CNS depression resulting in impairment:
         i. Headache
         ii. Dizziness
         iii. Confusion
         iv. Seizures
         v. Coma
      b. Long-term effects
         i. Cognitive and psychiatric problems
   2. Cardiac:
      a. Decreased myocardial function:
         i. Hypotension with tachycardia
         ii. Chest pain
         iii. Dysrhythmias
         iv. Myocardial ischemia
         v. Most CO deaths are from ventricular fibrillation.
      b. Long-term effects:
         i. Increased risk of premature cardiac death
   3. Metabolic:
      a. Respiratory alkalosis (from hyperventilation)
      b. Metabolic acidosis with severe exposures
   4. Respiratory:
      a. Pulmonary edema (10-30%)
         i. Direct effect on alveolar membrane
         ii. Left-ventricular failure
         iii. Aspiration
         iv. Neurogenic pulmonary edema
   5. Multiple organ dysfunction syndrome (MODS):
      a. Occurs at high-levels of exposure
I. Pathophysiology summary:
1. CO limits O₂ transport:
   a. CO more readily binds to Hb forming COHb

2. Inhibits O₂ transfer:
   a. CO changes the structure of Hb and causes premature release of O₂ into the tissues.

3. Tissue inflammation:
   a. Poor perfusion initiates an inflammatory response.

4. Poor cardiac function:
   a. Decreased O₂ delivery can cause dysrhythmias and myocardial dysfunction.
   b. Long-term cardiac damage reported after single CO-exposure.

5. Increased activation of nitric oxide (NO):
   a. Peripheral vasodilation

6. Vasodilation:
   a. From NO increase.
   b. Cerebral vasodilation and systemic hypotension can cause reduced cerebral blood flow.
   c. NO oxidizes oxyhemoglobin (O₂Hb) to methemoglobin (METHb).

7. Free-radical formation:
   a. NO accelerates free-radical formation (highly-reactive molecules that take part in various types of chemical reactions)
   b. Free-radicals can cause endothelial and oxidative brain damage.

J. Patient groups at risk for CO poisoning:
   1. Children
   2. Elderly
   3. Persons with heart disease
   4. Pregnant women and their unborn child
   5. Patients with increased oxygen demand
   6. Patients with decreased oxygen-carrying capacity (i.e., anemias, blood cancers)
   7. Patients with chronic respiratory insufficiency.

VII. Signs and symptoms of CO poisoning:
   A. Signs and symptoms usually vague and non-specific:
      1. Closely resemble those of other diseases
2. Often misdiagnosed as:
   a. Viral illness (e.g., the “flu”)
   b. Acute coronary syndrome
   c. Migraine
3. Estimated misdiagnosis occurs in 30-50% of CO-exposed patients.
4. CO often called “The Great Imitator”.

B. CO poisoning classifications:
   1. Acute:
      a. Results from short exposure to a high level of CO
   2. Chronic:
      a. Results form long exposure to a low level of CO

C. Acute signs and symptoms:
   1. Malaise
   2. Flu-like symptoms
   3. Fatigue
   4. Dyspnea on exertion
   5. Chest pain
   6. Palpitations
   7. Lethargy
   8. Confusion
   9. Depression
   10. Impulsiveness
   11. Hallucination
   12. Confabulation
   13. Agitation
   14. Nausea
   15. Vomiting
   16. Diarrhea
   17. Abdominal pain
   18. Headache
   19. Drowsiness
   20. Dizziness
   21. Weakness
   22. Confusion
   23. Visual disturbances
   24. Syncope
   25. Seizures
26. Fecal incontinence
27. Urinary incontinence
28. Memory disturbances
29. Gait disturbances
30. Bizarre neurological symptoms
31. Coma
32. Death

D. Chronic signs and symptoms:
   1. Same as with acute CO poisoning except that the onset and severity may be extremely varied.

E. Signs and Symptoms by COHb levels:
   1. Mild (15-20%):
      a. Headache
      b. Nausea
      c. Vomiting
      d. Dizziness
      e. Blurred vision.
   2. Moderate (21-40%):
      a. Confusion
      b. Syncope
      c. Chest pain
      d. Dyspnea
      e. Weakness
      f. Tachycardia
      g. Tachypnea
      h. Rhabdomyolysis
   3. Severe (41-59%):
      a. Palpitations
      b. Dysrhythmias
      c. Hypotension
      d. Myocardial ischemia
      e. Cardiac arrest
      f. Respiratory arrest
      g. Pulmonary edema
      h. Seizures
      i. Coma
   4. Fatal (>60%):
      a. Death

F. Signs and symptoms of CO poisoning do not always correlate to COHb levels.
G. The bright red skin coloration often reported with CO poisoning is unreliable and generally a late sign in severe poisoning.

H. Long-term complications:
   1. Delayed neurologic syndrome (DNS):
      a. Recovery seemingly apparent
      b. Behavioral and neurological deterioration occurs 2-40 days later.
      c. True prevalence uncertain (estimates vary from 1-47%)
      d. Patients more symptomatic initially appear to be more apt to develop DNS.
      e. DNS more common when there is an LOC initially
      f. Signs and symptoms of DNS:
         i. Memory loss
         ii. Confusion
         iii. Ataxia
         iv. Seizures
         v. Urinary incontinence
         vi. Fecal incontinence
         vii. Emotional lability
         viii. Disorientation
         ix. Hallucinations
         x. Parkinsonism
         xi. Mutism
         xii. Cortical blindness
         xiii. Psychosis
         xiv. Gait disturbances
         xv. Other motor disturbances
   2. Cardiac complications:
      a. Early cardiac death more common with those who suffered myocardial injury from CO during initial exposure.
   3. Other effects:
      a. Depression and anxiety can exist for up to 12 months following CO exposure.
      b. Depression higher at 6 weeks for those who attempted suicide by CO.
c. No difference in rates of symptoms between those with accidental and suicidal exposure at 12 months post-exposure.

VIII. CO detection:
A. CO detectors widely available for more than a decade.
B. Vastly underutilized.
C. Underwriters Laboratories (UL) revised guidelines for CO detectors in 1998.
   1. Units made before 1998 should not be used.
D. Handheld CO detectors available:
   1. Personal (CO only)
   2. Commercial (common in fire departments)
      a. Measures:
         i. Combustible gases
         ii. CO
         iii. O₂
         iv. H₂S (hydrogen sulfide)
D. Handheld CO detectors available:
   1. Personal (CO only)
   2. Commercial (common in fire departments)
      a. Measures:
         i. Combustible gases
         ii. CO
         iii. O₂
         iv. H₂S (hydrogen sulfide)
E. Biological detection previously required hospital-based arterial blood gas or venous blood analysis.
F. Technology now available to detect biological COHb levels in the prehospital and ED setting:
   1. Uses oximetry-type technology.
   2. Referred to as CO-oximetry
G. CO-oximetry:
   1. Can detect:
      a. Deoxyhemoglobin (Hb)
      b. Oxyhemoglobin (O₂Hb)
      c. Carboxyhemoglobin (COHb)
      d. Methemoglobin (METHb)
   2. Values reported as:
      a. SpO₂
      b. SpCO
      c. SpMET
      d. Pulse rate
   3. Uses finger probe similar to pulse oximetry.
      a. Utilizes 8 different wavelengths of light instead of 2.
   4. Readings closely correlate with COHb levels measured using hospital-based technologies.
5. Should be routine for all fire service and EMS personnel.

IX. CO poisoning treatment:
   A. CDC diagnostic criteria:
      1. Biological
         a. COHb > 5% in nonsmokers.
         b. COHb > 10% in smokers
      2. Environmental:
         a. No confirmatory test
   B. CDC diagnostic categories:
      1. Suspected:
         a. Potentially exposed person, no credible threat
      2. Probable:
         a. Clinically-compatible case where credible threat exists.
      3. Confirmed:
         a. Clinically-compatible cases where biological tests have confirmed exposure.
   C. Treatment based on severity of symptoms.
   D. Generally indicated when SpCO > 12-15%.
   E. Be prepared to treat complications:
      1. Seizures
      2. Cardiac dysrhythmias
      3. Cardiac ischemia
   F. Administer high-concentration O₂.
      1. Maximizes hemoglobin O₂ saturation.
      2. Can displace some CO from hemoglobin.
      3. Associated with improvement in neurological and cardiac complications.
      4. Consider CPAP
   G. Reviews of effectiveness of HBO mixed:
      1. Studies fail to demonstrate an improvement in outcome.
      2. Still commonly used—especially for severe poisonings.
      3. May aid in alleviating tissue hypoxia
      4. Significant decreases COHb half-life.
      5. Indications for HBO therapy:
         a. Strongly consider for:
            i. Altered mental status
ii. Coma
iii. Focal neurological deficits
iv. Pregnancy with COHb > 15%
v. History of LOC

b. Possibly consider for:
i. Cardiovascular compromise
ii. Metabolic acidosis
iii. Extremes of age

H. Ongoing treatment:
   1. Continue to monitor SpO₂ and SpCO.
   2. Obtain 12-lead ECG (if ALS) and monitor ECG.
   3. Document findings and plot trends.
   4. First-generation pulse oximeters may give falsely elevated SpO₂ levels due to elevated carboxyhemoglobin levels.

X. Methylene chloride:
   A. Slowly metabolized to CO.
   B. Victims do not pose contamination risks to rescuers.
   C. Contaminated clothing, skin and vomitus can secondarily contaminate rescuers

D. Effects of methylene chloride:
   1. Acute CNS depression
   2. Respiratory depression
   3. Cardiac dysrhythmias
   4. Respiratory tract irritation (at high levels)
   5. Non-cardiogenic pulmonary edema (at high levels)

E. Treatment:
   1. No antidote available
   2. Support respiratory and cardiac functions.
   3. Administer high-concentration O₂
      a. O₂ is antagonist of metabolically-released CO.

XI. CO and cyanide:
   A. Incidence of cyanide exposure more common than once thought.
   B. Effects of CO and cyanide are cumulative
   C. Symptoms of cyanide toxicity often attributed to CO because of lack of a high index of suspicion.
   D. Chemistry of cyanide:
      1. Gas:
         a. Colorless
b. Faint bitter almond smell
   i. Nearly 40% of population cannot smell cyanide.

2. Sodium cyanide and potassium cyanide are white powders
3. Molecule consists of 1 atom of carbon and 1 atom of nitrogen held together by a triple bond.
4. Cyanide anion extremely toxic

E. Cyanide source:
   1. Hydrogen cyanide is a product of combustion.
   2. High levels in:
      a. Plastics
      b. Wool
      c. Silk
      d. Synthetic rubber
      e. Polyurethane
      f. Asphalt

F. Toxicity varies by chemical form.
   1. OSHA permissible levels are 10 ppm as an 8-hour time weighted average.

G. Can be ingested or inhaled.

H. Cyanide pathophysiology:
   1. Inhibits enzyme cytochrome oxidase in the 4th complex of the electron transport chain.
      a. Found on the shelves of the mitochondria
   2. Cyanide stops electron transport and thus energy (ATP) production by the cell.
   3. Tissues affected are those dependent upon aerobic metabolism:
      a. Heart
      b. Central nervous system

I. Cyanide antidotes:
   1. Cyanide antidote kit:
      a. Ingredients:
         i. Amyl nitrite
         ii. Sodium nitrite
         iii. Sodium thiosulfate
      b. Nitrites induce the formation of METHb.
         i. Cyanide has a greater affinity for METHb than cytochrome oxidase.
ii. Binding of cyanide to METHb frees cytochrome oxidase so that cellular energy production is resumed.

c. Sodium thiosulfate binds to cyanide forming thiocyanate.
   i. Less toxic than cyanide
   ii. Excreted renally.

d. Problems related to nitrates:
   i. METHb does not transport O2
   ii. Conversion of Hb to METHb changes the state of the heme molecules where O2 binds.
   iii. METHb has heme in the ferric (Fe³⁺) state and not the ferrous (FE²⁺) state
   iv. O2 can only bind to heme when in the Fe²⁺ state.
   v. Concomitant CO and cyanide poisoning can significantly decrease the O2 carrying capacity of the blood.
   vi. Children are at particular risk for methemoglobinemia.
   vii. Sodium nitrite should be avoided for combination CO/cyanide poisonings where the SpCO > 10%.

2. Hydroxocobalamin
   a. Precursor to cyanocobalamin (Vitamin B₁₂)
   b. Combines with cyanide to form cyanocobalamin which is eliminated through the kidneys.
   c. FDA approval 12/2006
   d. Marketed as Cyanokit™
   e. Preferred in mixed CO/cyanide poisonings
REFERENCES


Course Evaluation Form

Instructor Name_________________________________________

Date__________________ Time ___________________________

Course Location ________________________________________

Directions:
Circle the number that applies. 1) Needs improvement 2) Satisfactory
3) Good 4) Excellent.

The Course:

Provided enough time to clearly present all lesson objectives

1  2  3  4

Provided enough time for students to learn material or learn and
practice the skills

1  2  3  4

Contained information necessary for students to be knowledgeable in
the lecture topics/practical skills presented

1  2  3  4

Was logical in the progression of instruction

1  2  3  4

Correlated well to the field application of the topics

1  2  3  4
Kept students interested/involved in the course

1  2  3  4

Assessment-based philosophy received a positive reaction from students

1  2  3  4

Assessment-based thinking helped differentiate between critical/non-critical patients.

1  2  3  4

Assessment-based style provided a satisfactory approach to patient care.

1  2  3  4

Prepared the students to properly manage patients

1  2  3  4

Provide any additional comments in the space below:
Carbon Monoxide Suggested Treatment Algorithm

1. Measure SpCO

2. If SpCO > 3%
     - Yes: No further evaluation of SpCO required.
     - No: Transport on 100% oxygen. Consider CPAP. Transport to hospital with HBO capabilities.

3. Transport on 100% oxygen for Emergency Department evaluation

4. SpCO > 12-15%
   - Yes: Symptoms of CO exposure?
     - Yes: Unexplained shock/hypotension?
       - Yes: No further evaluation of SpCO needed. Determine source of CO if nonsmoker.
       - No: Consider concomitant carbon monoxide and/or cyanide poisoning.
     - No: Symptoms of CO exposure?

5. No: Symptoms of CO exposure?
   - No: No further evaluation of SpCO needed. Determine source of CO if nonsmoker.