NON-INVASIVE RESPIRATORY GAS MONITORING:
A GUIDE FOR PARAMEDICS

EDUCATIONAL CURRICULUM

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ENDORSEMENTS

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EDUCATIONAL OBJECTIVES

TERMINAL OBJECTIVE
Upon completing this presentation the student will be able to clinically apply non-invasive respiratory gas monitoring in the out-of-hospital setting in order to enhance patient assessment skills and provide optimal interventions in patient care.

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

1. List the principal gases found in the earth’s atmosphere.
2. Discuss the concept of partial pressures in a gas mixture.
3. Describe Dalton’s law and relate it to the use of partial pressure measurements.
4. List the body’s two principal respiratory gases.
5. Identify the physical characteristics of oxygen.
6. Discuss why oxygen atoms must share electrons and ultimately exist in a diatomic state.
7. Identify the physical characteristics of carbon dioxide.
8. Differentiate the physical characteristics and biological importance of nitrogen.
9. Identify the physical characteristics of carbon monoxide.
10. Describe the two methods by which the human body transports oxygen.
11. Identify and describe the three methods by which the human body transports carbon dioxide.

12. Discuss the physical characteristics of hemoglobin and detail its function in respiratory gas transport.

13. Show and describe the heme structures on hemoglobin and their role in oxygen transport.

14. Discriminate between the ferrous and ferric states of iron as it relates to oxygen binding.

15. Discuss the physical changes in hemoglobin that result from oxygen binding.


17. Detail the chemical binding of carbon monoxide to hemoglobin and the effect of carbon monoxide on bound oxygen.

18. Describe methemoglobin.

19. Identify the causes of methemoglobinemia.

20. Discuss the relationship between the partial pressure of oxygen in the arterial blood as it relates to the saturation of hemoglobin.

21. List normal the normal value of oxygen in venous and arterial blood.
22. Describe four factors that decrease oxygen saturation.
23. Describe four factors that increase oxygen saturation.
24. Define 2,3-biphosphoglycerate (2,3-BPG) and discuss its role on oxygen transport.
25. Discuss the importance of the enzyme carbonic anhydrase in carbon dioxide transport and elimination.
26. Differentiate the binding characteristics of carbon dioxide and oxygen to hemoglobin.
27. Describe the Bohr Effect and its role in oxygen transport.
28. Describe the Haldane Effect and its role in carbon dioxide transport.
29. Detail the process of arterial blood gas sampling.
30. Compare and contrast arterial blood gas sampling with pulse oximetry.
31. List parameters usually monitored during arterial blood gas analysis.
32. Describe how pulse oximetry works.
33. Define oxygen saturation.
34. Describe the perfusion index and its role in prehospital respiratory gas monitoring.
35. Identify what a provider can learn from pulse oximetry.
36. Describe both the benefits and limitations of pulse oximetry.
37. List and describe the out-of-hospital contraindications for pulse oximetry.
38. List and detail the limitations of pulse oximetry.
39. Compare and contrast the limitations and benefits of first-generation and second-generation pulse oximeters.
40. Identify and discuss common myths associated with pulse oximetry.
41. Detail the physiological parameters of the patient discerned from pulse oximetry.
42. Categorize abnormal pulse oximetry findings and describe the clinical significance of each.
43. Define interventional strategies for abnormal pulse oximetry readings.
44. Describe the concept of capnography.
45. Detail available methods for carbon dioxide monitoring.
46. Discuss end-tidal carbon dioxide.
47. Identify information provided by capnography.
48. List the indications for capnography in the out-of-hospital setting.

49. Differentiate the clinical significance of rising and dropping carbon dioxide levels.

50. Describe the concept of transcutaneous carbon dioxide monitoring.

51. Describe the incidence and pathophysiology of carbon monoxide poisoning.

52. List the signs and symptoms of carbon monoxide poisoning.

53. Describe methods for detection of carbon monoxide levels in the blood including CO-oximetry.

54. Discuss the capabilities of co-oximetry to monitor the major types of hemoglobin and the respective quantities and saturations of each.

55. List normal carboxyhemoglobin levels for smokers and non-smokers.

56. Describe treatment strategies for carbon monoxide poisoning.

57. Identify why the fetus of a pregnant woman is at increased risk of serious carbon monoxide poisoning.
58. Describe how routine CO-oximetry can be a risk management tool in EMS and the fire service.

59. Discuss the significance of methemoglobin and methemoglobinemia in out-of-hospital care.

60. Identify the signs and symptoms encountered with increasing levels of methemoglobin.

61. List substances associated with an increased risk of methemoglobinemia.

62. Detail the treatments of methemoglobinemia including methylene blue.

63. Identify the importance of detecting concomitant cyanide and carbon monoxide poisoning.

64. Discuss the role of cyanide poisoning treatment and methemoglobinemia.

65. Detail why hydroxocobalamin is preferred over the nitrites in mixed carbon monoxide/cyanide poisonings.

AFFECTIVE OBJECTIVES

Upon completing this presentation the student will be able to:

67. Appreciate the uses and limitations of pulse oximetry.

68. Understand the value of screening for carboxyhemoglobin and methemoglobin in the out-of-hospital setting.

69. Demonstrate an appreciation for the role additional respiratory gas information plays in developing an accurate field diagnosis/impression of the out-of-hospital patient.

PSYCHOMOTOR OBJECTIVES

Upon completing this presentation the student will be able to:

70. Demonstrate a comprehensive patient assessment using pulse oximetry.

71. Demonstrate a comprehensive patient assessment using CO-oximetry.

72. Demonstrate a comprehensive patient assessment using capnography.

73. Demonstrate appropriate interventions for simulated abnormal oximetry readings.

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

75. Troubleshoot problems occurring when monitoring with pulse oximetry.
76. Troubleshoot problems occurring when monitoring with pulse CO-oximetry.

77. Demonstrate proper documentation of oximetry and CO-oximetry readings.
I. Introduction:
   A. The respiratory gasses are essential for life. The ability of
      out-of-hospital emergency care providers to monitor
      these gasses is essential to quality out-of-hospital
      care. Pulse oximetry has been the standard method
      for monitoring peripheral arterial oxygen saturation.
      Newer methodologies now allow the monitoring of
      carbon dioxide through capnography and carbon
      monoxide through CO-oximetry.

II. Respiratory gas physiology:
   A. The earth’s atmosphere contains several gasses:
      1. Oxygen (O₂).
      2. Carbon dioxide (CO₂).
      4. Water vapor (H₂O).
      5. Trace gasses:
         a. Argon (Ar).
         b. Neon (Ne).
         c. Helium (He).
   B. When gasses are present in a mixture, such as occurs in
      the earth’s atmosphere, the individual gasses are
      often represented based upon their partial pressures:
      1. Dalton’s Law.
      2. Determining partial pressure using Dalton’s law.
   C. Biological life depends primarily upon three gasses:
      1. Oxygen (O₂)
         a. Odorless.
         b. Tasteless.
         c. Colorless.
         d. Supports combustion.
         e. Present as a diatomic gas.
         f. Oxygen atoms must share electrons for
            stability.
         g. Necessary for animal life.
         h. Derived from plant photosynthesis:
            i. Algae (75%).
            ii. Terrestrial plants (25%).
2. Carbon Dioxide (CO₂)
   a. Colorless.
   b. Sour taste at high concentrations.
   c. Very low concentrations in fresh air.
   d. Asphyxiating.
   e. Waste product of animal life.
   f. Contains 2 atoms of oxygen and 1 atom of carbon.

3. Nitrogen (N₂)
   a. Most abundant gas in atmosphere (78%)
   b. Extremely stable
   c. Not a respiratory gas—but important in biological systems
   d. N₂ is very stable and must be converted to other nitrogen forms—usually by nitrogen-fixing bacteria in the soil.

Abnormal respiratory gases:
4. Carbon monoxide (CO).
   a. Colorless.
   b. Odorless.
   c. Tasteless.
   d. Results from incomplete combustion of carbon-containing compounds.

D. Respiratory gas transport:
   1. Oxygen:
      a. 97% reversibly bound to hemoglobin.
      b. 3% dissolved in plasma.
   2. Carbon dioxide:
      a. 70% as bicarbonate (HCO₃⁻).
      b. 23% reversibly bound to hemoglobin.
      c. 7% dissolved as plasma.

E. Hemoglobin:
   1. Protein-iron complex.
   2. Transports oxygen to peripheral tissues.
   3. Removes a limited amount of carbon dioxide from peripheral tissues.
   4. Made of various sub-units:
      a. 2 α sub-units (normal hemoglobin).
      b. 2 β sub-units (normal hemoglobin).
      c. 4 iron-containing heme structures.
5. Binding sites
   a. Heme structures:
   b. Non-oxygen-containing hemoglobin called deoxyhemoglobin (Hb):
      i. Heme in ferrous (Fe$^{2+}$) state.
   c. Oxygen-oxygen containing hemoglobin called oxyhemoglobin (O$_2$Hb):
      i. Heme in ferric (Fe$^{3+}$) state.
   d. Binding of oxygen to hemoglobin changes the shape (conformation) and color of the molecule.

F. Abnormal hemoglobin states:
   1. Carboxyhemoglobin (COHb):
      a. Results from binding of carbon monoxide to hemoglobin.
      b. Results from carbon monoxide exposure.
      c. Some level always present in smokers.
      d. Hemoglobin has approximately 250 times the affinity for carbon monoxide as it does for oxygen.
      e. Carbon monoxide displaces oxygen from hemoglobin.
      f. Carbon monoxide can be displaced from carboxyhemoglobin by high concentrations of oxygen.
      g. Half-life 4-6 hours.
   2. Methemoglobin (METHb):
      a. Hemoglobin in the ferric (Fe$^{3+}$) state.
      b. Oxygen cannot bind.
      c. Normally present in low concentrations (<1%) resulting from old hemoglobin.
      d. High levels can lead to hypoxemia.
      e. Causes:
         i. Hereditary:
            a. Hemoglobin M.
            b. Enzyme deficiencies.
         ii. Acquired:
            a. Nitrates.
            b. Nitrites.
            c. Dyes
d. Sulfonamides.
e. Lidocaine.
f. Benzocaine.

G. Oxygen saturation:
1. Oxygen saturation of oxygen is directly related to the partial pressure of oxygen in the blood (PO₂).
2. Venous blood saturation normally 30-40 mm Hg.
3. Arterial oxygen saturation normally 85-100 mm Hg.
4. Factors that decrease oxygen saturation:
   a. Decreased pH (acidosis).
   b. Increased carbon dioxide.
   c. Increased temperature.
   d. Increased 2,3-biphosphoglycerate.
5. Factors that increase oxygen saturation:
   a. Increased pH (alkalosis).
   b. Decreased carbon dioxide.
   c. Decreased temperature.
   d. Decreased 2,3-biphosphoglycerate.

H. 2,3-biphosphoglycerate (2,3-BPG).
1. Formerly called 2,3-diphosphoglycerate (2,3-DPG).
2. Found in hemoglobin.
3. Aids in oxygen release in the peripheral tissues.
4. Higher levels found in people who live at high altitudes.
5. Helps mitigate the effects of hypoxemia.

I. Carbon dioxide transport:
1. Majority transported as bicarbonate ion (HCO₃⁻).
2. Enzyme carbonic anhydrase necessary for rapid conversion of water and carbon dioxide to bicarbonate ion.
3. Some carbon dioxide transported bound to hemoglobin.
4. Carbon dioxide does not bind to heme as oxygen.
5. Binds to an amino acid in the protein sub-units.
6. When carbon dioxide is bound to hemoglobin the resultant compound is called carbaminohemoglobin (CO₂Hb).

J. Bohr effect:
1. Increases in carbon dioxide levels (respiratory acidosis) cause oxygen to be displaced from hemoglobin.
2. Facilitates oxygen transport.

K. Haldane effect:
1. Binding of oxygen (as occurs in the respiratory capillaries) tends to displace carbon dioxide.
2. Oxyhemoglobin is more acidic than deoxyhemoglobin:
   a. Promotes carbon dioxide elimination in the alveoli.
   b. Promotes release of hydrogen ions ($H^+$) which combine with bicarbonate ions to form water and carbon dioxide which are eliminated.

III. Respiratory gas measurement:
A. Arterial blood gas sampling.
2. Invasive.
3. Expensive.
4. Painful.
5. Difficult.
7. Excellent diagnostic test.
8. Provides:
   a. pH (normal 7.35-7.45)
   b. $PO_2$ (normal 80-100 mm Hg)
   c. $PCO_2$ (normal 35-45 mm Hg)
   d. $HCO_3^-$ (normal 22-26 mmol/L)
   e. Base excess (-2-+3)
   f. $SaO_2$ (normal > 95%)
   g. COHb available on most machines.
   h. Total hemoglobin available on most machines.

B. Pulse oximetry
1. Introduces in 1980s
2. Non-invasive.
3. Safe.
4. Inexpensive.
5. How it works:
   a. Probe is placed over vascular bed (finger, earlobe).
b. Light-emitting diodes (LEDs) emit light of 2 different wavelengths:
   i. Red = $\lambda$ 660 nm
   ii. Infrared = $\lambda$ 940 nm
c. Light absorbed by:
   i. Arterial blood.
   ii. Venous blood.
   iii. Other tissues.
d. Light that passes through the vascular bed is detected by a photodetector.
   i. Photodetectors on opposite side of vascular bed.
   ii. Specialized reflective photodetectors can be on the same side as the LEDs.
e. Deoxyhemoglobin and oxyhemoglobin each absorb light at different rates due to color and conformation:
   i. Deoxyhemoglobin absorbs more red light than infrared light.
   ii. Oxyhemoglobin absorbs more infrared light than red light.
   iii. Difference in absorption is measured:
   \[
   R = \frac{AC_{\lambda R}}{DC_{\lambda R}} \cdot \frac{AC_{\lambda IR}}{DC_{\lambda IR}}
   \]
   Where:
   $AC =$ arterial circulation.
   $DC =$ venous and other tissue pigments.
   $\lambda R =$ red light (660 nm)
   $\lambda IR =$ infrared light (940 nm)
f. Only the pulsatile component of arterial blood flow is measured (hence the name “pulse oximetry”).
g. Absorption readings are compared with validated SpO$_2$ levels in the database for the oximeter.
h. Oxygen saturation:
i. \( SpO_2 = \frac{Oxygen \ Content}{Oxygen \ Capacity} \)

ii. Thus:

\[
SpO_2 = \frac{HbO_2}{Total \ Hb} \times 100
\]

iii. Thus

\[
SpO_2 = \frac{Fractional \ Saturation}{100 - (\%COHb + \%MetHb)} \times 100
\]

i. Perfusion Index (PI):
   i. Found on second-generation oximeters.
   ii. Reflects strength of the pulse at monitoring sites.
   iii. Ranges from 0.02% (very weak) to 20% (very strong).
   iv. Helps determine best site for probe placement.

j. Terminology:
   i. SaO\(_2\) is used for oxygen saturations determined by arterial blood gas analysis.
   ii. SpO\(_2\) is used for saturations determined by pulse oximetry.
   iii. Readings are normally similar.

6. What pulse oximetry can tell you:
   a. SpO\(_2\).
   b. Pulse rate.

7. What pulse oximetry cannot tell you:
   a. Oxygen content of the blood.
   b. Amount of oxygen dissolved in the blood.
   c. Respiratory rate.
   d. Tidal volume
   e. Ventilation adequacy.
   f. Cardiac output
   g. Blood pressure
   h. Amount of dyshemoglobinemia

8. Who should use pulse oximetry?
a. Any level of EMS provider who administers oxygen and has been educated in pulse oximetry usage:
   i. First Responders.
   ii. Emergency Medical Technicians.
   iii. Advanced Emergency Medical Technicians (EMT-Intermediates).
   iv. Paramedics
b. Always document pulse oximetry readings and communicate these to emergency care providers.

9. Who should not use pulse oximetry:
   a. Persons not trained in use and application of the device.

10. Out-of-hospital indications for pulse oximetry:
    a. To monitor the adequacy of arterial hemoglobin saturation (SpO₂).
    b. To quantify the SpO₂ response to an intervention.
    c. To detect the presence and quality of pulsatile blood flow in an endangered body region (i.e., extremities).

11. Out-of-hospital contraindications for pulse oximetry:
    a. Usage by personnel with inadequate education in pulse oximetry use.
    b. Persons not prepared or incapable of obtaining, documenting, or communicating results of a pulse oximetry assessment of a patient with a real or potential medical emergency.

12. Limitations of pulse oximetry:
    a. Oximetry is not a measure of ventilation.
    b. Oximetry may lag behind hypoxic events.
    c. Oximetry is not a substitute for physical examination.
    d. Very low saturation states may cause low oximetry readings due to an absence of measured SpO₂ levels in the database.
e. Pulse oximetry cannot detect abnormal forms of hemoglobin (e.g., COHb and METHb).

13. First-generation oximeters:
   a. First-generation oximeters relied primarily on raw absorption measurements and correlated these with known SpO$_2$ reference values in the database.
   b. Limitations found with first-generation technology:
      i. Hypotension can cause false readings.
      ii. Carboxyhemoglobin can falsely elevate SpO2 readings.
      iii. Oximetry unreliable during helicopter transport because of movement and vibration.
      iv. Dyes and pigments (e.g., nail polish) can cause abnormal readings.
      v. Vasoconstriction can cause low or absent readings.
      vi. Hypothermia can cause low or absent readings.
      vii. Bright ambient lighting can cause readings in the absence of a pulse.
      viii. Shivering can cause readings in the absence of a pulse.
      ix. Movement can cause readings in the absence of a pulse.

14. Second-generation oximeters:
   a. Second-generation oximeters use various technologies to minimize artifacts and to minimize the possibility of false readings:
      i. Adaptive filters (noise attenuation).
      ii. Signal processing algorithms.
      iii. Improved sensors.
   b. Problems prevented with second-generation technology:
      i. Motion artifact.
      ii. False readings during low-flow states.
      iii. False bradycardias.
      iv. False hypoxemias.
v. Missed desaturations.
vi. Missed bradycardias.
vii. Data dropouts.
viii. Effects of abnormal hemoglobin.

15. Myths often associated with pulse oximetry:
   a. Age affects oxygen saturation.
   b. Gender affects oxygen saturation.
   c. Anemia affects oxygen saturation.
   d. Oxygen saturation inaccurate in dark-skinned individuals.
   e. Jaundice affects oxygen saturation.

16. Out-of-hospital usage of pulse oximetry:
   a. Assure scene safety.
   b. Perform initial (primary assessment).
   c. Apply oxygen when appropriate (either with or after oximetry).
   d. Perform secondary assessment.
   e. Provide ongoing monitoring.
   f. Always follow local protocols.

17. What do oximetry readings mean?
   a. $\text{SpO}_2 = 95-100\% \rightarrow \text{Normal}$
   b. $\text{SpO}_2 = 91-94\% \rightarrow \text{Mild hypoxemia}$
   c. $\text{SpO}_2 = 86-90\% \rightarrow \text{Moderate hypoxemia}$
   d. $\text{SpO}_2 < 85\% \rightarrow \text{Severe hypoxemia}$

18. Interventional response:
   a. Normal:
      i. Change inspired oxygen (FiO$_2$) to maintain saturation:
         a. Increase
         b. Decrease
   b. Mild hypoxemia:
      i. Increase inspired oxygen (FiO$_2$) to increase saturation.
   c. Moderate hypoxemia:
      i. Increase inspired oxygen (FiO$_2$) to increase saturation.
      ii. Assess and increase ventilation.
   d. Severe hypoxemia:
i. Increase inspired oxygen (FiO₂) to increase saturation.
ii. Increase ventilation.

19. Pulse oximetry to assess circulation in endangered tissues (off-label indication):
   a. Oximeter probe can be placed on tissues distal to a possible vascular injury.
   b. Oximetry can monitor distal circulation with fractures, dislocations, and crush injuries.
   c. Clinical correlation always needed.

C. Capnography:
   1. Carbon dioxide cannot be measured by oximetry methods:
      a. Carbon dioxide binds to a different site on hemoglobin than oxygen.
      b. Carbaminohemoglobin does not change color or conformation when carbon dioxide bound.
      c. Carbaminohemoglobin cannot be distinguished from deoxyhemoglobin by oximetry.
   2. Carbon dioxide can be monitored through measurement of exhaled air.
      a. By use of pH-sensitive paper.
      b. Infrared spectography.
         i. Sensor can be placed on ventilation circuit for intubated patients.
         ii. Sensor available for patients who are not intubated, but who can breathe through a sensor tube.
   3. Measurement of exhaled carbon dioxide through the respiratory cycle results in a capnogram.
      a. End-tidal carbon dioxide (EtCO₂) is the maximum amount of carbon dioxide exhaled at the end of respiration.
   4. Information provided by capnography:
      a. End-tidal carbon dioxide levels (which can be used to estimate arterial carbon dioxide levels [PaCO₂]).
      b. Provides information about ventilation.
      c. Normal values:
i. Approximately 5%.
ii. Approximately 35-37 mm Hg.
d. Difference between PaCO₂ and EtCO₂ is called the CO₂ gradient:
i. Normally 5-6 mm Hg.

5. Indications for capnography.
a. To verify endotracheal tube placement.
b. To determine the adequacy of ventilation.
c. To continuously monitor a critical patient where arterial blood gasses may not be available.
d. To maintain a specific carbon dioxide level (e.g., brain injury).

6. Interpretation of capnography:
a. Elevated or raising carbon dioxide levels:
i. Increased carbon dioxide production (e.g., fever, seizures).
ii. Decreased alveolar ventilation (e.g., CNS depression, decreased minute ventilation, muscular disorder).
iii. Equipment malfunction (e.g., bad sensor).
b. Low or falling carbon dioxide levels:
i. Decreased carbon dioxide production (e.g., hypothermia, cardiac arrest, pulmonary embolism).
ii. Increased alveolar ventilation (e.g., tachypnea, hyperpnea, increased minute ventilation).
iii. Equipment malfunction (e.g., obstruction of ventilation system, misplaced endotracheal tube, bad sampling head).

D. Transcutaneous carbon dioxide monitoring.
1. Technology available for approximately 30 years.
2. Primarily used in neonates.
3. Transcutaneous oxygen levels often available.
4. Uses heated probe that measures changes in pH.
a. Probe must be frequently moved.
5. pH changes correlated to changes in carbon dioxide levels.

IV. Carbon monoxide and carbon monoxide poisoning:
   A. Carbon monoxide is the leading cause of poisoning deaths in industrialized countries.
   B. At least 3,800 people in the United States die annually from carbon monoxide poisoning.
   C. Carbon monoxide results from the combustion of fossil fuels and carbon-containing compounds.
   D. Tends to accumulate in poorly-ventilated areas.
   E. Carbon monoxide displaces oxygen from hemoglobin.
      1. Hemoglobin has an affinity for carbon monoxide that is 200-250 times that of oxygen.
      2. Only high concentrations of oxygen can displace carbon monoxide from hemoglobin.
   F. Symptoms of carbon monoxide poisoning:
      1. Carboxyhemoglobin levels do not always correlate with symptoms nor predict sequelae.
      2. Carbon monoxide poisoning often called the great imitator.
         a. Symptoms:
            i. Mild Severity (COHb <15-20%)
               a. Headache
               b. Nausea
               c. Vomiting
               d. Dizziness
               e. Blurred vision
            ii. Moderate severity (COHb 21-40%)
               a. Confusion
               b. Syncope
               c. Chest pain
               d. Dyspnea
               e. Tachycardia
               f. Tachypnea
               g. Weakness
               h. Rhabdomyolysis
            iii. Severe severity (COHb 41-59%)
               a. Dysrhythmias
               b. Hypotension
               c. Cardiac ischemia
               d. Palpitations
e. Cardiac arrest
f. Respiratory arrest
g. Pulmonary edema
h. Seizures
i. Coma
iv. Fatal severity (COHb > 60%)
   a. Death
v. Red skin color usually a late finding and unreliable.

G. Detection:
1. Formerly required hospital-based arterial blood gas sampling.
   a. Clinically requires a high index of suspicion.
   b. Technology available (CO-oximetry) to detect COHb levels in the out-of-hospital and emergency department setting.
      i. Uses 8 wavelengths of light to detect 4 different hemoglobin moieties:
         a. Oxyhemoglobin
         b. Deoxyhemoglobin
         c. Carboxyhemoglobin
         d. Methemoglobin
   ii. Provides:
      a. SpO₂
      b. SpCO
      c. SpMET
      d. Pulse rate
2. Carbon monoxide evaluation should be routine at all levels of EMS and the fire service.
   a. All field personnel should be educated in use of the oximeter and CO-oximeter.

H. Normal carbon monoxide levels (persons ages 3-74 years):
   1. Nonsmokers = 0.83 ± 0.67%
   2. Smokers = 4.30 ± 2.55%
   3. All persons combined = 1.94 ± 2.24%

I. Treatment of carbon monoxide poisoning:
   1. Treatment is based on severity of symptoms.
   2. Treatment generally indicated with SpCO > 12-15%.
3. High-concentration oxygen should be administered to displace carbon monoxide from hemoglobin.
4. Be prepared to treat complications (e.g., seizures, cardiac ischemia).
5. Efficacy of hyperbaric oxygen therapy controversial:
   a. Generally reserved for severe poisonings.
   b. May aid with tissue hypoxia.
J. Significant and evolving body of scientific literature showing there are numerous long-term and permanent sequelae from CO poisoning.
K. Fetal hemoglobin (HgF) has a much greater affinity for carbon monoxide than adult hemoglobin (HgA).
   1. Pregnant mothers may exhibit mild symptoms, yet the fetus may have devastating outcomes from carbon monoxide poisoning.
L. Risk management:
   1. Carbon monoxide poisoning symptoms are non-specific and easy to miss and can lead to death and disability.
   2. Missed carbon monoxide poisoning is a significant legal risk for out-of-hospital and emergency department personnel.
   3. Carbon monoxide poisoning is a particular occupational risk for firefighters.
   4. A simple COHb reading can be life-saving.
   5. Screening can be quickly performed.
V. Methemoglobin and methemoglobinemia.
   A. Not a respiratory gas—but has implications with regard to other respiratory gasses and toxins.
   B. Methemoglobin is hemoglobin with the heme in the ferric (Fe$^{3+}$) state.
      1. Cannot bind oxygen.
      2. Reflects hemoglobin at the end of its functional life.
      3. Results in dark reddish-brown blood.
      4. Most frequently seen in children < 4 months of age.
   C. As methemoglobin levels increase, a functional anemia results (hemoglobin amounts normal but a significant amount of hemoglobin nonfunctional).
D. Symptoms:
1. Cyanosis begins around lips with SpMET >10-15%
2. Organs with high oxygen demands manifest toxicity first.
3. Symptoms vary with SpMET concentration:
   a. SpMET = 1-3%:
      i. Normal, asymptomatic.
   b. SpMET = 3-15%:
      i. Slight grayish blue skin discoloration.
   c. SpMET = 15-20%:
      i. Asymptomatic, but cyanotic.
   d. SpMET = 25-50%:
      i. Headache
      ii. Dyspnea
      iii. Confusion
      iv. Chest pain
      v. Weakness
   e. SpMET = 50-70%:
      i. Altered mental status
      ii. Delirium

E. Treatment:
1. Administer oxygen at high concentrations.
2. Remove offending agents.
3. Consider methylene blue as an antidote:
   a. Accelerates the enzymatic degradation of METHb.

F. Carbon Monoxide and Cyanide
1. Parts of cyanide kit (amyl nitrite and sodium nitrite) induce methemoglobinemia.
2. Cyanide antidotes (nitrites) and carbon monoxide poisoning can lead to elevated levels of COHb and METHb significantly reducing oxygen-carrying capacity of the blood.
3. Sodium nitrite should not be administered when combination cyanide/carbon monoxide poisonings when SpCO > 10%.
4. Hydroxycobalamin converts cyanide to cyanocobalamin (Vitamin B_{12}) which is cleared renally.
   a. Preferred over nitrites in mixed poisonings.
b. Hydroxycobalamin is the antidote of choice in mixed carbon monoxide/cyanide poisonings,
REFERENCES


Kansas State Board of EMS. “Monitoring Pulse Oximetry”. [Available at: http://www.ksbems.org/curricula.htm]


Non-Invasive respiratory Gas Monitoring:
Out-of-Hospital Implications

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: