LUCAS COUNTY

January 13, 2011

TO: ALL LUCAS COUNTY PARAMEDICS

FROM: Brent Parquette, NREMT-P
LCEMS Continuing Education Program

RE: **Continuing Education – February 2011**

The new Life Pak 15's are here! In anticiptation of field deployment in early March, CE sessions for the month of February will be dedicated to in-service training on our new equipment. While offering many of the same monitoring and therapy options we have had in the past, the LP15 will aslo have the capability of CO oximetry and an updated transmission solution for 12-Lead ECG's.

With CO oximetry being a new diagnostic tool for us, material will be presented during class specific to carbon monoxide monitoring. A "new protocol" has just been completed which references parameters for CO monitoring, assessment, treatment and fire rehab considerations.

In-service training on the LP15 will take place during skill stations, which will allow for "hands-on practice." We anticipate having eight (8) identical stations that will run for approximately 1 hour.

I ask that you review the attached material referencing CO poisoning/monitoring to help better prepare you for class participation.

As always, if you have any questions or comments, please fee free to contact me. I look forward to seeing you in the month of February!

Brent
(419) 213-6508
bparquette@co.lucas.oh.us

CARBON MONOXIDE POISONING

CO Oximetry

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 heavier than air and tends to accumulate in low-lying areas.

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 O_2 from the hemoglobin binding sites (4).
  3. CO prevents O_2 from binding.
  4. COHb cannot carry O_2.
  5. COHb causes the premature release of the remaining O_2 into the tissues.
- D. CO ultimately removed from the circulation and destroyed:
  1. Half-life:
     a. Room air: 240-360 minutes
     b. 100% O_2: 80 minutes
     c. Hyperbaric O_2 (HBO): 22 minutes
- E. CO also binds to other iron-containing proteins:
  1. Myoglobin
     a. Binding reduces O_2 available to the heart:
        i. Ischemia
        ii. Dysrhytnmias
        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 O_2 transport:
    a. CO more readily binds to Hb forming COHb
2. Inhibits O_2 transfer:
    a. CO changes the structure of Hb and causes premature release of O_2 into the tissues.
3. Tissue inflammation:
    a. Poor perfusion initiates an inflammatory response.
4. Poor cardiac function:
    a. Decreased O_2 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 (O_2Hb) 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 the 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 gasses
            ii. CO
            iii. O_2
            iv. H_2S (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 (O_2Hb)

c. Carboxyhemoglobin (COHb)
d. Methemoglobin (METHb)

2. Values reported as:
a. SpO_2
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 O_2.

1. Maximizes hemoglobin O_2 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 SpO_2 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 SpO_2 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 O_2
        a. O_2 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 4^{th} 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 nitrites:
        i. METHb does not transport O_2
        ii. Conversion of Hb to METHb changes the state of the heme molecules where O_2 binds.
        iii. METHb has heme in the ferric (Fe^{3+}) state and not the ferrous (FE^{2+}) state
        iv. O_2 can only bind to heme when in the Fe^{2+} state.
        v. Concomitant CO and cyanide poisoning can significantly decrease the O_2 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 B_{12})
    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

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Carbon Monoxide Suggested Treatment Algorithm