Lethal Exposures: CO and CN
(Carbon Monoxide and Cyanide)

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Disclosures

• I am on the speakers bureau for Masimo Corporation and Dey, LLP.

• I am the Fire/EMS Technical Editor for Fire Engineering magazine.

• I do not intend to discuss any unlabeled or unapproved uses of drugs or products.
Learning Objectives

1. HCN and CO exposure, incidence, sources
2. Pathophysiology, clinical effects, sequelae
3. Diagnostic challenges, treatment issues
4. Solutions, assessment modalities
Fire Service Exposure Issues

- **Carbon Monoxide**
  - Leading cause of poisoning deaths worldwide
  - Commonly misdiagnosed (medical and fire personnel)
  - New evidence of harm from low level exposures
  - Potential role in FF cardiovascular events and deaths?

- **Cyanide**
  - Ubiquitous in occupied structures
  - Now leading cause of fire fatalities
  - North American fire fatality rate twice W. Europe & Japan
  - Potential role in FF deaths?
Carbon Monoxide (CO)

“The Great Imitator”
- Invisible
- Masquerades
Carbon Monoxide (CO)

- **Gas:**
  - Colorless
  - Odorless
  - Tasteless
  - Nonirritating

- **Physical Properties:**
  - Vapor Density = 0.97
  - LEL/UEL = 12.5 – 74%
  - IDLH = 1200 ppm
Endogenous Sources of CO

- Normal heme catabolism (breakdown):
  - Only biochemical reaction in the body known to produce CO
- Hemolytic anemia
- Sepsis
1. Incomplete combustion of any carbon-based material will produce carbon monoxide. Most common sources are:
   - Automobiles, trucks, buses, boats
   - Gas heaters and furnaces
   - Small gasoline engines
   - Portable / space heaters
   - Portable gas-powered generators
   - Barbecues / fireplaces
   - Structure / wildland fires
   - Cigarette smoke

2. Methylene chloride (paint stripper)
   - Liver converts to CO
### CO Exposure

<table>
<thead>
<tr>
<th>Source</th>
<th>Exposure (ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fresh Air</td>
<td>0.06-0.5</td>
</tr>
<tr>
<td>Urban Air</td>
<td>1-30</td>
</tr>
<tr>
<td>Smoke-filled Room</td>
<td>2-16</td>
</tr>
<tr>
<td>Cooking on Gas Stove</td>
<td>100</td>
</tr>
<tr>
<td>Smoking a Cigarette</td>
<td>400-500</td>
</tr>
<tr>
<td>Automobile Exhaust</td>
<td>100,000</td>
</tr>
</tbody>
</table>

- Environmental exposure typically <0.001% (10 ppm).
- Higher in urban areas.
- Sources:
  - Volcanic gasses
  - Bush fires
  - Human pollution
Severity of Intoxication: Morbidity Associated with COHb and Duration

Symptoms relating to CO concentration:

- 50% COHb: Brain Damage - Death
- 45% COHb: Coma and brain damage
- 40% COHb: Collapse
- 35% COHb: Vomiting
- 30% COHb: Drowsy
- 25% COHb: Headache and Nausea
- 20% COHb: Headache
- 15% COHb: Slight Headache
- 10% COHb: None
- 5% COHb: None

Highlighted Area demonstrates current OSHA Standard for CO:
[500ppm/30 minutes]
Consider 500 ppm/60-90 minutes....
Case Study: Even Low Exposure Levels Can Lead to Death

- **52 yo male**
  - Prominent attorney in Salt Lake City found dead in his home after failing to show up for work
  - Had complained to co-workers of nausea and other flu-like symptoms for several days
  - Upon discovery of his body, elevated levels of CO were discovered in the home—but levels were relatively low, only 130 PPM
  - Faulty boiler discovered

Even 130 Parts Per Million Over a Prolonged Period Can Kill You!
Carbon Monoxide Poisoning

- **Leading cause of poisoning deaths in industrialized countries:**
  - 50,000 emergency room visits in the US annually
  - At least 3,800 deaths in the US annually
  - 1,400-3,000 accidental deaths in the US annually

- **Even a single exposure has the potential to induce long-term cardiac and neurocognitive/psychiatric sequelae:**
  - Brain damage at 12 months after exposure is significant
  - Myocardial Injury is a common consequence of CO poisoning and can identify patients at a higher risk for premature death

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Unintentional Poisoning Deaths – US, 1999-2004

TABLE 2. Number of deaths – US, 1999 and 2004

<table>
<thead>
<tr>
<th>Type of substance</th>
<th>1999</th>
<th>2004</th>
<th>Rate change (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drugs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonopioid analgesics§</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychotherapeutic drugs‖</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcotics and hallucinogens</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Other drugs acting on the central nervous system</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other and unspecified drugs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other substances</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Organic solvents and halogenated hydrocarbons</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbon monoxide and other gases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pesticides</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other and unspecified chemicals</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
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</tr>
</tbody>
</table>

1. Drugs
2. CO and other gases
3. Alcohol
4. Organic solvents & halogenated hydrocarbons
5. Pesticides

MMWR, 9 Feb 2007 / 56 (05);93-96
Incidence

- **Increased accidental CO deaths:**
  - Patient > 65 years of age.
  - Male
  - Ethanol intoxication.

- **Accidental deaths peak in winter:**
  - Use of heating systems.
  - Closed windows.

- **Significant increase seen following disasters:**
  - Related to utility loss.
Career Fire Fighter Dies of Carbon Monoxide Poisoning after Becoming Lost While Searching for the Seat of a Fire in Warehouse - New York

SUMMARY
On December 16, 2003, a 30-year-old male fire fighter (the victim) died after he became separated from his crew members while searching for the seat of a fire at a furniture warehouse. His crew exited due to worsening conditions and a missing member announcement was made. At one point while inside the warehouse, members

NIOSH investigators concluded that to minimize the risk of similar occurrences, fire departments should

• ensure that pre-incident planning is performed on commercial structures
Firefighter Injuries – 2006 (United States):
• Total injuries = 83,400
  • Smoke or Gas Inhalation = 2,825 (3.4%)
  • Burns & Smoke Inhalation = 730 (0.9%)

Case Study

- 26 yo female visits PMD c/o severe headaches unrelieved by repeated doses of Excedrin® - has been home alone, 2 children visiting with ex-husband

- Neuro exam WNL, no other findings. Dx sinusitis. Tx Amoxil and T3’s

- Next day: h/a worse, now vomiting, calls EMS, transport to ED

- MD evaluates, no specific findings. Tx IV fluids, antiemetic, analgesic, head CT (neg). Given phenergan Rx, f/u with PMD

- Arrives home by taxi, ex-husband waiting to return children
Case Study continued…

- Next morning, same headache. Children difficult to awaken, once awake both have trouble walking, stumble and fall.

- EMS summoned, FD also dispatched. CO metering finds 1,200 to 1,600 ppm from bedroom space heater. Dead kitten found in children’s room. All three transported.

- Mom 29% COHb – sent for HBO, home 48 hours later. 4 yo son 14%, 2 yo daughter 17% - both admitted to regional children’s hospital for 24 hours observation.

- Mom with permanent neurological deficit, children no sequelae.

- Litigation considered against PMD, ED, and EMS.
Show me the money... Is this real?

- Up to 10% of UAP, ACS, seizure, and h/a admits have CO poisoning

- 146 h/a pts: 4 COHb > 10% (3%, all unrelated to smoking)
Attempts to Develop a Model

- Heckerling et al apply criteria to validate a predictor model for identifying CO poisoned pts. In ED.

- 61 patients tested, model only detects 3 of 4 pts with ↑ COHb

- 753 acute admits med-surg, neuro, psych: 2 w/ minor COHb ↑

- Conclusion: Widespread ED screening expensive, unproductive unless quick and cheap screening tool became available.
Pathophysiology

- CO displaces O$_2$ from hemoglobin binding sites (4)
- CO prevents O$_2$ from binding

- (carboxyhemoglobin)

- COHb increases O$_2$ affinity, interfering with normal release
• Complex IV of Electron Transport Chain – binds cytochrome c oxidase
• CO does NOT bind with same affinity as O$_2$ (requires significant hypoxia)
• Delayed effect ATP synthesis produces lactic acidosis
Pathophysiology

- **CO limits oxygen transport**
  - Greater affinity (>210 x) for hemoglobin

- **Inhibits oxygen transfer**
  - Interferes with normal unloading to tissues

- **Binds with myoglobin (muscle)**
  - Interferes with heart and skeletal muscle function

- **Binds to cytochrome oxidases**
  - Induces anaerobic metabolism (cellular & tissue)

- **Increases nitric oxide (NO) formation**
  - Accelerates free radical formation
Pathophysiology - Effects

- **Hypoxia**
  - Cellular, cardiac and cerebral

- **Intracellular toxicity**
  - Cardiac and skeletal muscle dysfunction

- **Inflammatory response**
  - Secondary to hypoxia

- **Vasodilation**
  - Induced by NO release (hypotension)

- **Free radical formation**
  - Endothelial and oxidative cerebral damage
Neurologic Effect

Delayed Neurologic Syndrome
Experienced by 11-30% of patients with CO Poisoning (DNS)


Long-Term/Chronic Sequelae
Cognitive and personality changes, dementia, seizures, psychosis, amnesia, parkinsonism, depression, short-term memory loss, incontinence.

Case Studies: Neurological Sequelae

- **51 yo female Physical Therapist**
  - Iditarod racer stopped to change wet socks in a tent
  - Experienced nausea, then lost consciousness
  - Inhaled CO from a faulty propane heater for 3 hours
  - Prolonged recovery, IQ fell from 140 to 76, had to relearn reading & writing

- **32 yo female & 35 yo male Attorneys**
  - CO from inadequately ventilated furnace
  - Both unable to function as attorneys
Cardiac Effect

• “Myocardial injury occurs frequently in patients hospitalized for moderate to severe CO poisoning and is a significant predictor of mortality”

• Odds ratio’s from recent study demonstrate that a patient has a 3 times higher likelihood of cardiac death (within a 7 year follow-up period) from even one moderate to severe toxic CO exposure, when compared to a control group

Cardiac Effect

• 19 year study 8,333 Swedish males ÷ smokers, non-smokers, never smokers.

• Never smokers split into quartiles:
  • 0.13 – 0.49% COHb
  • 0.50 – 0.57%
  • 0.58 – 0.66%
  • 0.67 – 5.47%

• Relative risk CV event 3.7, death 2.2 highest to lowest quartiles

• Incidence CV disease & death in non-smokers related to COHb%

Fetal Damage

Theoretical effect of different treatments on maternal and fetal COHb levels over time

# Signs and Symptoms

<table>
<thead>
<tr>
<th>SpCO%</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;5%</td>
<td>None</td>
</tr>
<tr>
<td>5-10%</td>
<td>Mild headache, tire easily</td>
</tr>
<tr>
<td>11-20%</td>
<td>Moderate headache, exertional SOB</td>
</tr>
<tr>
<td>21-30%</td>
<td>Throbbing headache, mild nausea, dizziness, fatigue, slightly impaired judgment</td>
</tr>
<tr>
<td>31-40%</td>
<td>Severe headache, vomiting, vertigo, altered judgment</td>
</tr>
<tr>
<td>41-50%</td>
<td>Confusion, syncope, tachycardia</td>
</tr>
<tr>
<td>51-60%</td>
<td>Seizures, unconsciousness</td>
</tr>
</tbody>
</table>

Carbon Monoxide Poisoning Presents Like the Flu!
Haunted Houses or CO Poisoning?

- Wilmer W. “Mr. and Mrs. H.” Amer J Opthamology. 1921

- Purchased new home, c/o headaches & fatigue. Heard bells and footsteps during nights with sightings of mysterious figures.

- Investigation revealed prior owners had similar experiences.

- Furnace chimney found blocked, venting CO into home.
CO Poisoning: The Great Imitator

30-50% of CO-exposed patients presenting to Emergency Departments are misdiagnosed

Diagnostic Problem

- **Vague symptoms**
  - Food poisoning
  - Viral illness
  - Migraines
  - Drug abuse
  - ACS

- **Current diagnostic methods slow, invasive, costly**

- If the proper diagnosis is not made the patient is often inadvertently returned to the toxic environment
CO Alarms

• 61,100 CO incidents in 2005

• Increase 9% each year (= 77,597 in 2008)

• Peak December & January and 6-10 PM

• 92% residential

Source: NIFRS
UL 2034: listings for CO alarms


- Presently:
  - 30 PPM for 30 days
  - 70 PPM for 1 – 4 hours
  - 150 PPM for 10 – 50 minutes
  - 400 PPM for 4 – 15 minutes (6 min reset > 70 PPM)
  - Non-alarm status CO$_2$ < 5,000 PPM
  - Non-alarm limits for methane, butane, heptane, ethyl acetate and isopropyl alcohol

- NFPA 720
Treatment of CO Poisoning

- **Chemical Half-life of Carbon Monoxide bound to Hemoglobin**
  - 4 hours on room air
  - 45 minutes on 100% oxygen
  - 22 minutes on 100% in Hyperbaric Chamber at 2-4 atmospheres
Laboratory CO-oximetry

- CO-oximetry capability found in 50% of hospital laboratories
- Standard ABG cannot differentiate carboxy from oxyhemoglobin
- Invasive—need compelling reason to order, repeated tests to monitor tx
- Variable time to analysis (can take from minutes to hours to get results)
- Golden Standard—for measurement and/or detection of COHb (± 2%)
COHb Recommendations

“We recommend that clinicians routinely provide POCT of HbCO by CO-oximetry to screen patients with flu-like symptoms or headache in the emergency department for occult CO poisoning, particularly in communities where combustion is used for heating during the heating season. We found at least fair evidence that POCT of HbCO by CO-oximetry will lead to a correct and timely diagnosis of CO poisoning in patients who otherwise would have been missed”

(Weight of Evidence = Fair; Net Benefit = Substantial; Recommendation = B)
Blood Sampling for COHb

A-COHb = V-COHb


61 suspected CO poisoning patients @ Bronx Municipal Hospital ED, simultaneous A and V sampling COHb. Correlation r value 0.99 (95% CI, 0.99 to 0.99), r² value 0.98.

CONCLUSION: “Arterial and venous COHb levels only rarely differ by more than 1% to 2%.”
Exhaled CO Meters

- Estimation COHb from alveolar CO concentration first described in 1948 (Sjostrand T. Acta Physiol Scand 16:201-7)

- Predominantly used to monitor smoking cessation

- Compact, portable, well validated

- Requires 20 second breath holding, measures $E_T\text{CO}$ in PPM

- Present accuracy $\pm 2$ PPM, COHb obtained from Haldane Equation (essentially $= \text{PPM} ÷ 6$)

www.micro-direct.com
Exhaled CO Meters

- Fast, economical, portable
- CPT Code (94250)
- Requires 20 second breath hold (awake, alert patient)
- Disposable mouthpieces
- Regular gas calibration
- Despite widespread availability since 1970’s utilization very low
Exhaled CO Demo
Noninvasive Pulse CO-Oximetry

- FDA approved January 2006
- Compact, portable, well validated
- CPT Code (82375 SpCO, 83050 SpMet)
- Continuous carboxyhemoglobin measurement
- Present accuracy ± 3 % COHb
- Also measures oxyhemoglobin (SpO2), methemoglobin (SpMet), perfusion index (PI), approval for hemoglobin (Hgb) pending.
- No calibration needed

www.masimo.com
Pulse CO-Oximetry

- Fast, economical
- Can be used on any patient (including unconscious)
- No disposables
- No calibration necessary
- Use wider than exhaled devices after only 20 months in marketplace
Limitations of Pulse Oximetry

Conventional pulse oximetry can not distinguish between COHb, and O₂Hb

SpCO-SpO₂ Gap:
The fractional difference between actual SaO₂ and display of SpO₂ (2 wavelength oximetry) in presence of carboxyhemoglobin

How Noninvasive Pulse CO-Oximetry Works

Oxygenated Hb and reduced Hb absorb different amounts of Red (RD) and Infrared (IR) Light

*(Two-wavelength oximeters cannot measure dyshemoglobins)*
FDA Validation
Masimo Rainbow SET Compared to Reference Methodology

Test results for Rad-57 vs. blood sample

Rad-57 SpCO (%)

Reference HbCO from blood sample (%)
5,000 Patient Brown University Study

- Partridge and Jay (Rhode Island Hospital, Brown University Medical School), **assessed carbon monoxide (CO) levels of nearly 5,000 ED patients**

- **9 unsuspected cases of CO Toxicity (COT) were discovered.** 13 false positives, 0 false negatives

- **Extrapolated to all US hospitals, this would equal 50,000 cases of unsuspected COT annually**

- They concluded “**unsuspected COT may be identified using noninvasive COHb screening and the prevalence of COT may be higher than previously recognized**”

*Non-Invasive Carboxyhemoglobin Monitoring: Screening Emergency Department Patients for Carbon Monoxide Exposure. Partridge R, Chee KJ, Suner S, Sucov A, Jay G. Department of Emergency Medicine, Rhode Island Hospital, Brown Medical School, Providence, RI.*
Pulse CO-Oximeter Treatment Algorithm

Measure SpCO

0 - 3%
- No further medical evaluation of SpCO needed

> 3%
- Loss of consciousness or neurological impairment or SpCO > 25%?
  - Yes
    - Transport on 100% oxygen for ED evaluation. Consider transport to hospital with hyperbaric chamber.
  - No
    - SpCO > 12%
      - Transport on 100% oxygen for ED evaluation
    - SpCO < 12%
      - Symptoms of CO exposure? *
        - Yes
          - Transport on 100% oxygen for ED evaluation
        - No
          - No further medical evaluation of SpCO needed. Determine source of CO if nonsmoker.

Hampson NB, Weaver LK JEMS 2006
# Categorizing Symptoms

<table>
<thead>
<tr>
<th></th>
<th>None</th>
<th>Mild</th>
<th>Moderate</th>
<th>Throbbing</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LOC:</strong></td>
<td>Alert</td>
<td>Slight confusion</td>
<td>Very confused</td>
<td>Syncope or unconscious</td>
<td>Seizures</td>
</tr>
<tr>
<td><strong>SOB:</strong></td>
<td>None</td>
<td>Exertional</td>
<td></td>
<td>At rest</td>
<td></td>
</tr>
<tr>
<td><strong>GI:</strong></td>
<td>None</td>
<td>Mild nausea</td>
<td>Nausea</td>
<td>Vomited or vomiting</td>
<td></td>
</tr>
</tbody>
</table>
Treating CO Poisoning - Oxygen
Rate of cognitive sequelae was nearly twice as high when hyperbaric treatment was not used

**Methods**
- Random assignment of symptomatic patients with CO poisoning into one of two groups
  - Group 1: 3 hyperbaric oxygen treatments in a 24 hour period
  - Group 2: 1 normobaric oxygen treatment and two normobaric room air treatments

**Results**
- Group 1: 25% sequelae at 6 weeks, 18% at 12 months
- Group 2: 46% sequelae at 6 weeks, 33% at 12 months

Challenges to Detecting CO Poisoning

- Endogenous CO – we all have some level of COHb
- Kinetics CO uptake and excretion very complex, toxicity mechanism unclear
- Pollution: atmospheric vs. smoking
- Symptoms ambiguous, flu-like
- COHb levels poorly correlate with clinical condition
- Testing limitations: Lab CO-Oximetry, pulse oximetry, no biochemical marker
- Paucity of research
CO Assessment

1. Every patient, every time.

2. All occupants at CO alarm calls.

3. Firefighters.
NEED CASH FOR ALCOHOL RESEARCH
Hydrogen Cyanide (CN)

- **Toxic Gas:**
  - Colorless or pale blue
  - Faint bitter almond odor*
  - Irritating
  - Burning taste

- **Physical Properties:**
  - Vapor Density = 0.94
  - LEL/UEL = 5.6 – 40%
  - IDLH = 50 ppm

*Up to 40% of population unable to detect HCN odor
Cyanide in Fire Smoke

Natural Substances:
- Wool
- Silk
- Cotton
- Paper

Synthetic Substances:
- Plastics
- Nylons
- Styrofoam
- Polyurethane foam
Trends in Construction

Shift from wood & natural to lightweight materials (read synthetics and petroleum based products) =

- Burn 2 – 3 times hotter and ignite 2 – 3 times faster
- Emit significant HCN during pyrolysis

1. Less time to escape (shorter time to flashover)
2. Toxic gases incapacitate
3. Increased risk of injury (thermal, inhalation)
Fire in the United States - 2006

- 1,642,500 reported fires
  - 3,245 civilian deaths
  - 16,400 civilian fire injuries
  - 89 firefighter deaths
    - 11 during interior firefighting

- US fire fatality rate remains double those of Western Europe and Japan where smoke exposures are aggressively treated.
Fire in the United States - 2006

Firefighter Injuries – 2006 (United States):

- Total injuries = 83,400
- Smoke or Gas Inhalation = 2,825 (3.4%)
- Burns & Smoke Inhalation = 730 (0.9%)

= 3,555 (4.3%)

Station Nightclub Fire - RI

- Feb 2003 band pyrotechnics ignite polyurethane foam lining stage walls
- 440 people, 100 deaths
Simulation of platform area

- 60 seconds - flashover
- 90 seconds – CO, CN, O2 incompatible with life
Temperatures in The Station at 90 seconds (from computer simulation)

Peak temperatures in red exceed 1000 °C (1832 °F)
Light blue indicates temperatures around the boiling point of water (100 °C, or 212 °F)
CN & CO: Exposure in Fire Deaths

Percentage of fire deaths

- Elevated cyanide (≥1 mg/L)
- Elevated carbon monoxide (≥50% COHb)

Dupont Plaza Hotel (n=97): 48%, 5%
Happy Land Social Club (n=87): 98%, 87%
Manchester aircraft fire (n=54): 87%, 21%
Maryland fire deaths (n=530): 58%, 34%
FFS fire deaths (n=129): 49%, 33%
Glasgow fire deaths (n=199): 47%, 35%

COHb, carboxyhemoglobin; FFS, Foundation for Fire Safety.
Progression of CN Poisoning

Cyanide?
March 21, 2003 – Cincinnati, Ohio

• Flashover @ single family house fire

• 3 interior crew members, 2 proceeded out front door.

• FF Oscar Armstrong III, aged 25, seen walking towards door, fumbling with SCBA, turned and walked back towards fire.

• Cause of death = massive 3rd degree burns.

• ? Disorientation secondary to cyanide inhalation
Paris Fire Brigade Protocol

1. Known smoke exposure in enclosed space
2. Altered mental status
3. Soot in nares or mouth
Paris Fire Brigade Protocol

ROSC = 50%
Cyanokit® (hydroxocobalamin)

- 5 grams IV over 15 minutes
- Second dose if needed (clinical condition)
Cyanokit® (hydroxocobalamin)

- Vitamin B12 precursor
- Binds with cyanide in blood, forms cyanocobalamin (vitamin B12)
- Cyanocobalamin is not toxic and is excreted in the urine
- Significant side effects:
  - Turns mucosa, skin & urine red
  - Transient hypertension
  - Interferes with colorimetric lab values
- No other cyanide antidote is safe for field use!

Skin Redness in Healthy Volunteers

After 10 g treatment, photos on Day 1 and on Day 8.

*No flash photography. †Flash photography used.

HCO 5 g: Systolic Blood Pressure

Efficacy of Hydroxocobalamin for the Treatment of Acute Cyanide Poisoning in Adult Beagle Dogs
Other Antidotes Available in the US

Cyanide Antidote Kit

• Was the only commercially available FDA-approved US antidote

• Also known as the Taylor Kit, Lilly Kit, and Pasadena Kit

• Sulfur donor/methemoglobin inducer

• Three active components:
  • Amyl nitrite (perles)
  • Sodium nitrite (IV)
  • Sodium thiosulfate (IV)
Cyanide Antidote Kit:

• Amyl nitrite (inhalation)
  ▪ Converts Hb (Fe$^{2+}$) to metHb (Fe$^{3+}$) leads to variable metHb levels usually in the range of 3 to 5%

• Sodium nitrite
  ▪ Converts HbO$_2$ (Fe$^{2+}$) to metHb (Fe$^{3+}$) increases metHb levels to < 20%

• Sodium thiosulfate
  ▪ Enzymatically reacts with cyanide to form thiocyanate (SCN$^-$) and sulfite (SO$_3^{2-}$)

Hb, hemoglobin; metHb, methemoglobin; HbO$_2$, oxygenated hemoglobin.
Cyanide Antidote Kit: Contraindication

Not indicated for use in smoke-inhalation victims due to methemoglobin formation. (methemoglobin and COHb cannot transport oxygen)

Baskin SI, Brewer TG. Medical aspects of chemical and biological warfare. Office of the Surgeon General, Department of Army, US. 1997;271-286.
Antidotes Available in Europe

- 4-dimethylaminophenol (DMAP)
  - Methemoglobin inducer
  - Used in Germany for documented severe cyanide poisoning
  - Adverse effects severe; may result in high metHb levels

- Dicobalt edetate (Cobalt EDTA)
  - Fixates the cyanide ion and transforms into nontoxic derivative
  - Used in the UK and France
  - Side effects numerous; allergic responses common and compound can be extremely toxic in absence of cyanide ions

"Of course, I'm feeling uncomfortable. I'm not allowed on the couch."
**Hydroxocobalamin: Characteristics**

- Does not reduce oxygen-carrying capacity of blood
- Does not cause hemodynamic instability
- Transient side effects
  - Red discoloration of urine, skin, and mucous membranes
  - Transient increases in blood pressure
  - Interference with colorimetric laboratory tests
    - AST, total bilirubin, creatinine, glucose
    - Mg$^{2+}$, Fe$^{2+}$, creatinine kinase, phosphorus

Risk:benefit ratio renders it suitable for prehospital empiric use and use in smoke-inhalation victims

Take Home Message
1. Our customer death rate is excessive

2. CN antidote is a fire service role

3. Suspect CN in smoke inhalation (role of CN in FF deaths)
Fire Service Exposure Issues

- **We’re missing Carbon Monoxide poisonings**
  - Leads poisoning deaths worldwide, harms at low levels
  - Commonly misdiagnosed (medical and fire personnel)
    - Screen every patient every time
    - Screen people as well as buildings at CO calls
    - Assess firefighters (rehab, routinely, research)

- **Cyanide kills**
  - We need to aggressively treat our customers
  - Research on role in FF deaths
Thank You

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