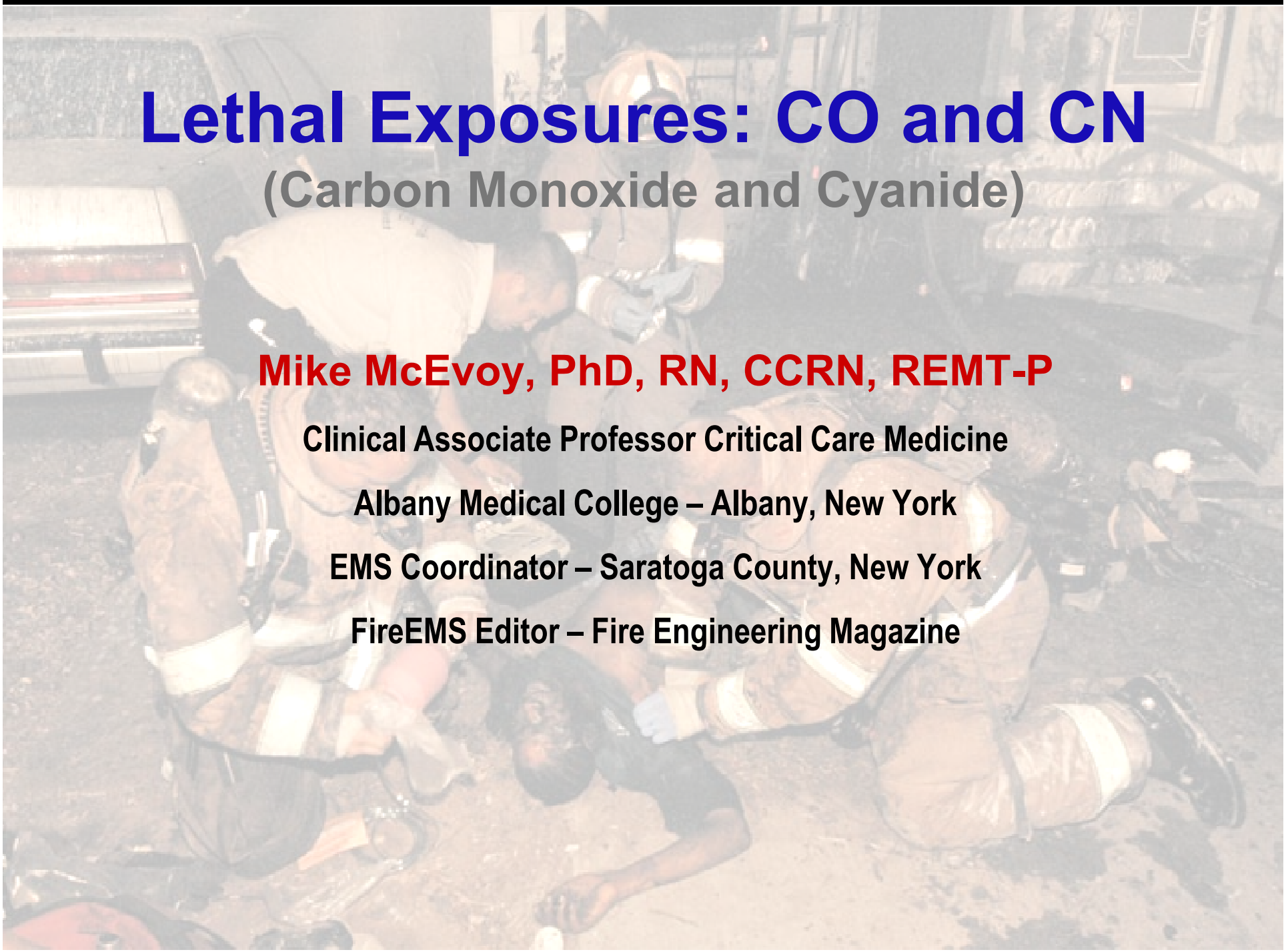

Lethal Exposures: CO and CN

(Carbon Monoxide and Cyanide)

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Albany Medical College – Albany, New York

EMS Coordinator – Saratoga County, New York

FireEMS Editor – Fire Engineering Magazine

Disclosures

- I am on the speakers bureau for Masimo Corpor
- I am the F MS nni dit r Fire Engineeri ag e.
- I do not in l to us eled or unapprov ses ug ts.

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?

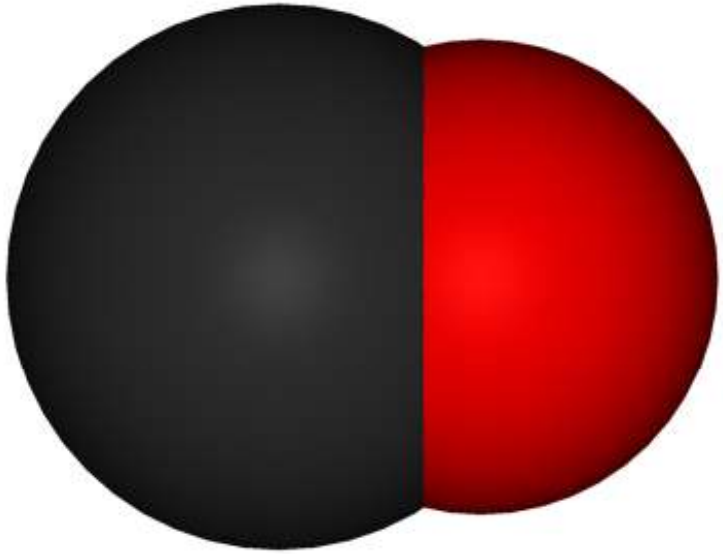
Carbon Monoxide (CO)

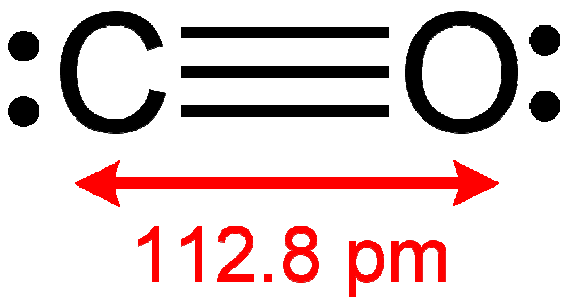
"The Great Imitator"

- Invisible
- Masquerades

Carbon Monoxide (CO)

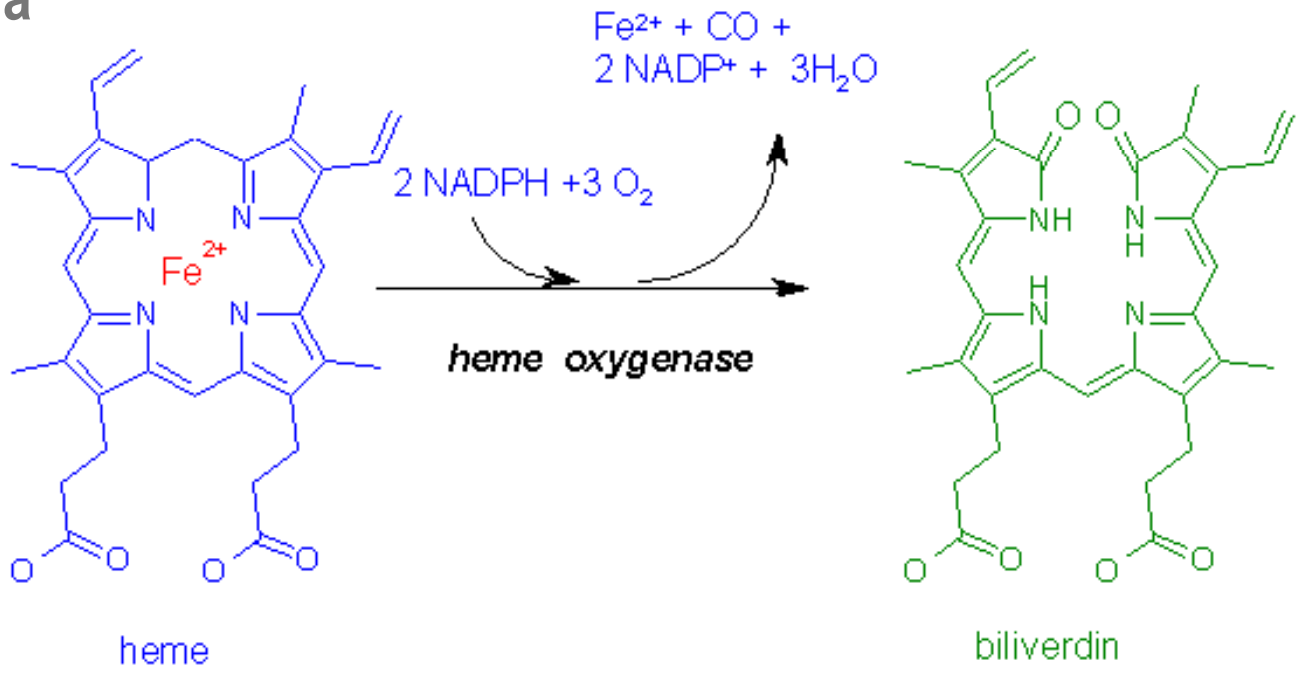
- **Gas:**
  - Colorless
  - Odorless
  - Tasteless
  - Nonirritating
- **Physical Properties:**
  - Vapor Density = 0.97
  - LEL/UEL = 12.5 – 74%
  - IDLH = 1200 ppm





Endogenous Sources of CO

- Normal heme catabolism (breakdown):
  - Only biochemical reaction in the body known to produce CO
- Hemolytic anemia
- Sepsis



Common Sources

1. **Incomplete combustion of any carbon-based material will produce carbon monoxide. Most commons 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







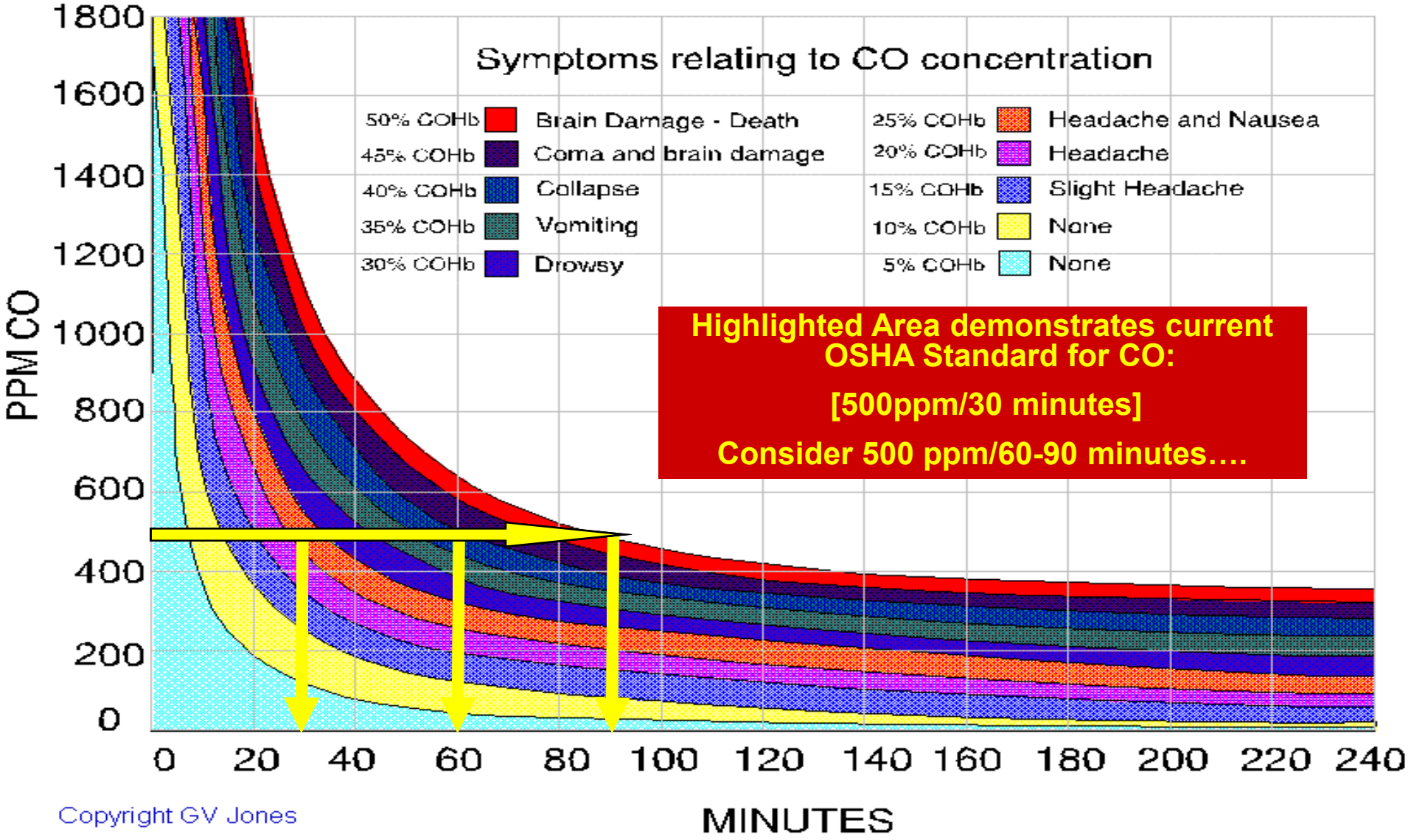

CO Exposure

Source	Exposure (ppm)
Fresh Air	0.06-0.5
Urban Air	1-30
Smoke-filled Room	2-16
Cooking on Gas Stove	100
Smoking a Cigarette	400-500
Automobile Exhaust	100,000

- 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



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

Prominent attorney dies from carbon monoxide poisoning

By Wendy Leonard
Deseret Morning News

After failing to show up for work Tuesday, a prominent Salt Lake attorney was found dead in his home.

A secretary at the downtown law firm called a neighbor around 10:30 a.m. when Bruce H. Jensen didn't show up as usual. The neighbor entered Jensen's home, near 1100 East and 1000 South, and found his body on the bed, said Salt Lake Fire spokesman Scott Freitag.

"I don't know how to describe it, to see your friend lying on the bed," neighbor Bill Dowse told KSL-TV.


Bruce H. Jensen

Jensen, 52, lived alone and no one else was in the home at the time. Freitag said he had been dead for a few hours and that the medical examiner took the body and performed an autopsy. It was determined Jensen died from carbon monoxide poisoning.

Crews returned to the home to test for the chemical and found it was at 130 parts per million, well above Qwestar's safety standard of 50 parts per million.

"There weren't extremely high levels of carbon monoxide in the home, but he was exposed to moderate levels over several days," Freitag said.

The source of the fumes is believed to be a leak in the boiler heating system.

"It was an older home with an old boiler that hadn't been inspected for a long time," Freitag said.

Had the boiler been working properly, he said, carbon monoxide would not have been a problem. However, Freitag said, a carbon monoxide detector, available for as little as $20, "would have alerted him there was a problem in the house."

Coworkers reported that Jensen had mentioned not feeling well since Friday. He had told them he was experiencing nausea and other flu-like symptoms, also symptomatic of the poisoning, which slowly takes over red blood cells in the body.

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 [1]
  - At least 3,800 deaths in the US annually [2]
  - 1,400-3,000 accidental deaths in the US annually [3,4]
- **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 [5]
  - Myocardial Injury is a common consequence of CO poisoning and can identify patients at a higher risk for premature death [6]

[1] Hampson NB, Weaver LK. Carbon Monoxide poisoning: A new incidence for an old disease. Undersea and Hyperbaric Medicine 2007;34(3):163-168.

[2] Mott JA, Wolfe MI, Alverson CJ, MacDonald SC, Bailey CR, Ball LB, Moorman JE, Somers JH, Mannino DM, Redd SC. National Vehicle Emissions policies and practices and declining US carbon monoxide-related mortality. JAMA 2002;288:988-995

[3] Hampson NB, Stock AL. Storm-Related Carbon Monoxide Poisoning: Lessons Learned from Recent Epidemics. Undersea Hyperb Med 2006;33(4):257-263

[4] Cobb N, Etzel RA, Unintentional Carbon monoxide-related deaths in the United States, 1979 through 1988. JAMA 1991;266(5):659.

[5] Weaver LK, et al. N Engl J Med, 2002;347(14):1057-067.

[6] Henry CR, et al. JAMA. 2006;295(4):398-402.

Unintentional Poisoning Deaths – US, 1999-2004

TABLE 2. Number of deat… 99 and 2004

Type of substance	e change (%)
Drugs	68.3
Nonopioid analgesics[§]	18.1
Psychotherapeutic drugs[¶]	83.5
Narcotics and hallucinogen…	54.6
Other drugs acting on the c…	-0.5
Other and unspecified drug…	87.3
Other substances	1.3
Alcohol	6.0
Organic solvents and halog…	2.0
Carbon monoxide and othe…	-1.7
Pesticides	—[§§]
Other and unspecified chem…	10.6
Total	62.5

* Age-adjusted rates per 10…
† *International Classificatio…*
§ Includes painkillers such a… …er drugs.
¶ Includes antiepileptic, sed…
** Includes heroin, opioid an…
†† Category used to classify … …algesic and a sedative) and deaths attril…
§§ Rates based on fewer tha…
¶¶ Includes corrosives, meta…

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.

Carbon Monoxide

NIOSH
Fire Fighter Fatality Investigation and Prevention Program

Death in the line of duty...

A Summary of a NIOSH fire fighter fatality investigation *March 31, 2005*

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, member

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

- *ensure that pre-incident planning is performed on commercial structures*

Carbon Monoxide

Firefighter Injuries – 2006 (United States):

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

- NFPA Survey of Fire Depts for U.S. Fire Experience, 2006.

seat of a fire at a furniture warehouse. His crew exited due to worsening conditions and a missing member announcement was made. At

- *ensure that pre-incident planning is performed on commercial structures*

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?

- 1
  (>
- 3
  d
- 1
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- 4
  H
- 146 h/a pts: 4 COHb > 10% (3%, all unrelated to smoking)
  Heckerling et al, *Ann Intern Med*, 1987;107:174-6.

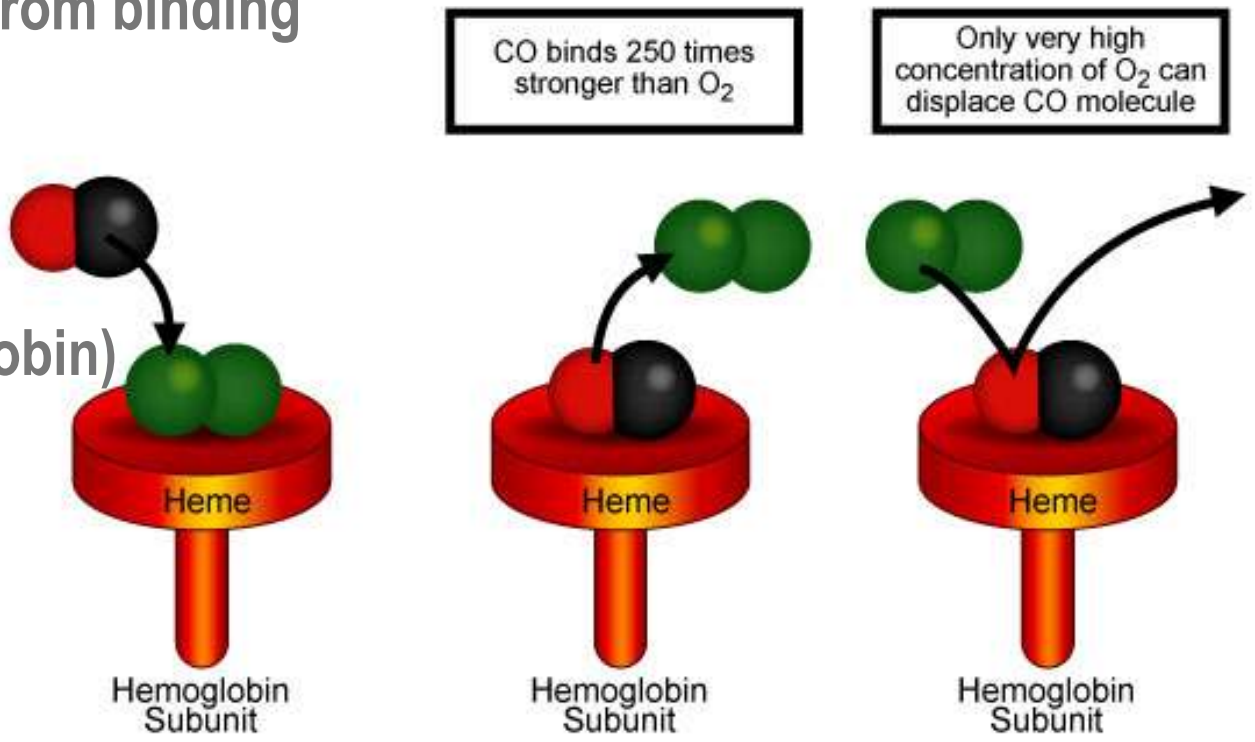
Up to 10% of UAP, ACS, seizure, and h/a admits have CO poisoning

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
  Heckerling et al, *Am J Med* 1988;84:251-6.
- 753 acute admits med-surg, neuro, psych: 2 w/ minor COHb ↑
  Heckerling et al, *Am J Emerg Med* 1990;8:301-4.
- Conclusion: **Widespread ED screening expensive, unproductive unless quick and cheap screening tool became available.**

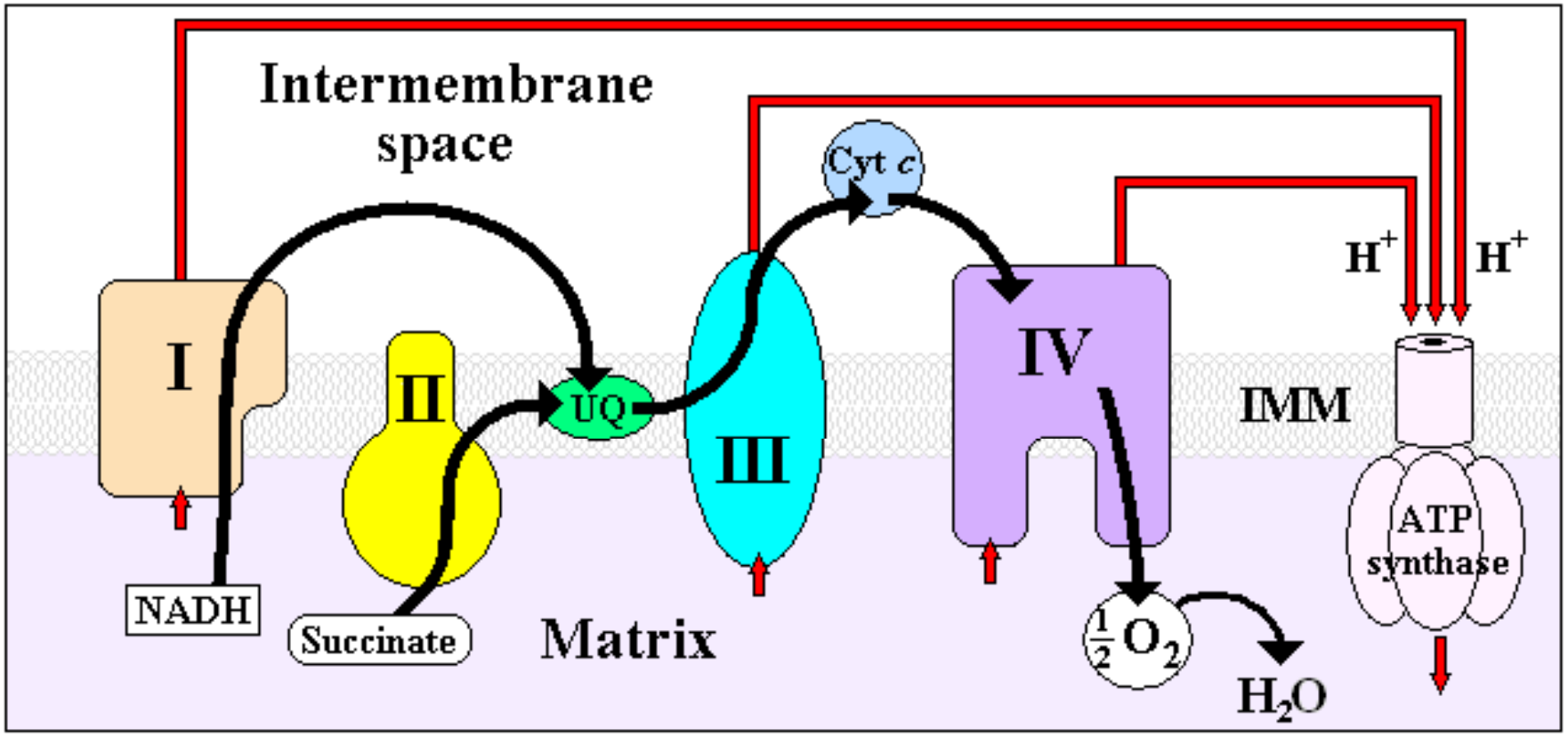
Pathophysiology

- CO displaces O_2 from hemoglobin binding sites (4)
- CO prevents O_2 from binding
- (carboxyhemoglobin)



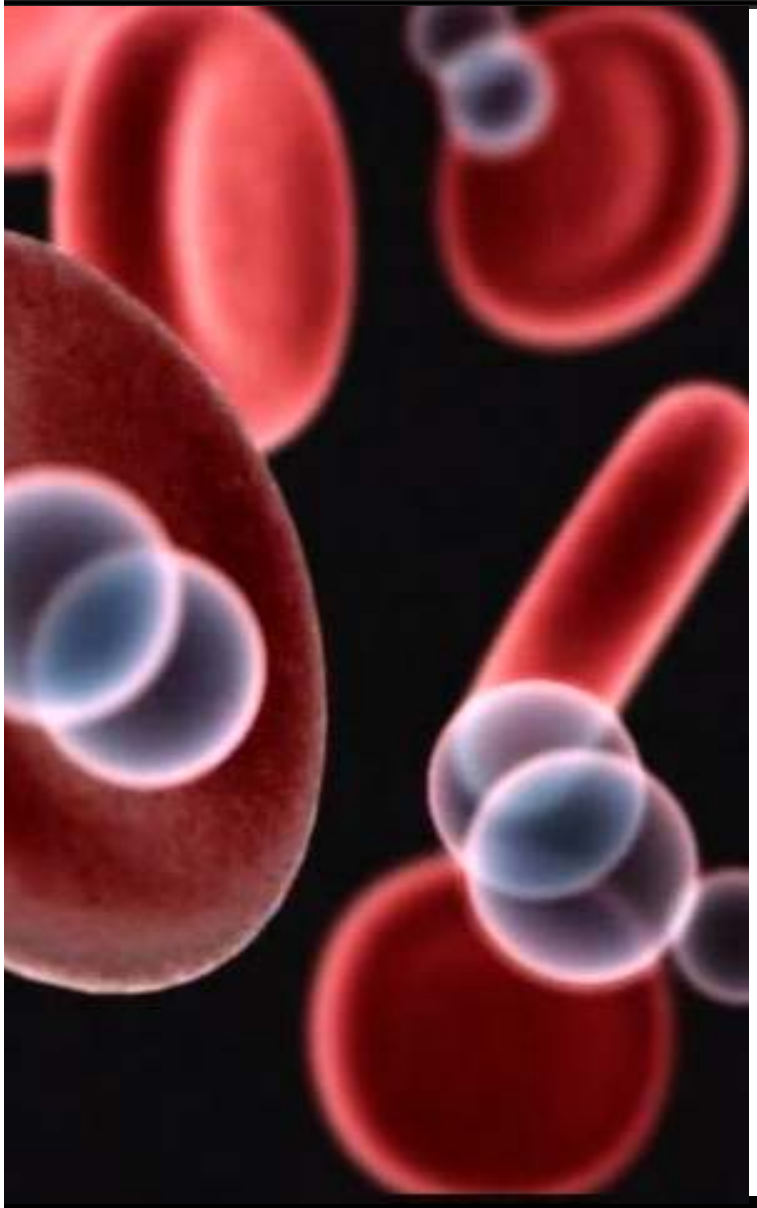
- COHb increases O_2 affinity, interfering with normal release

Messy Pathophysiology



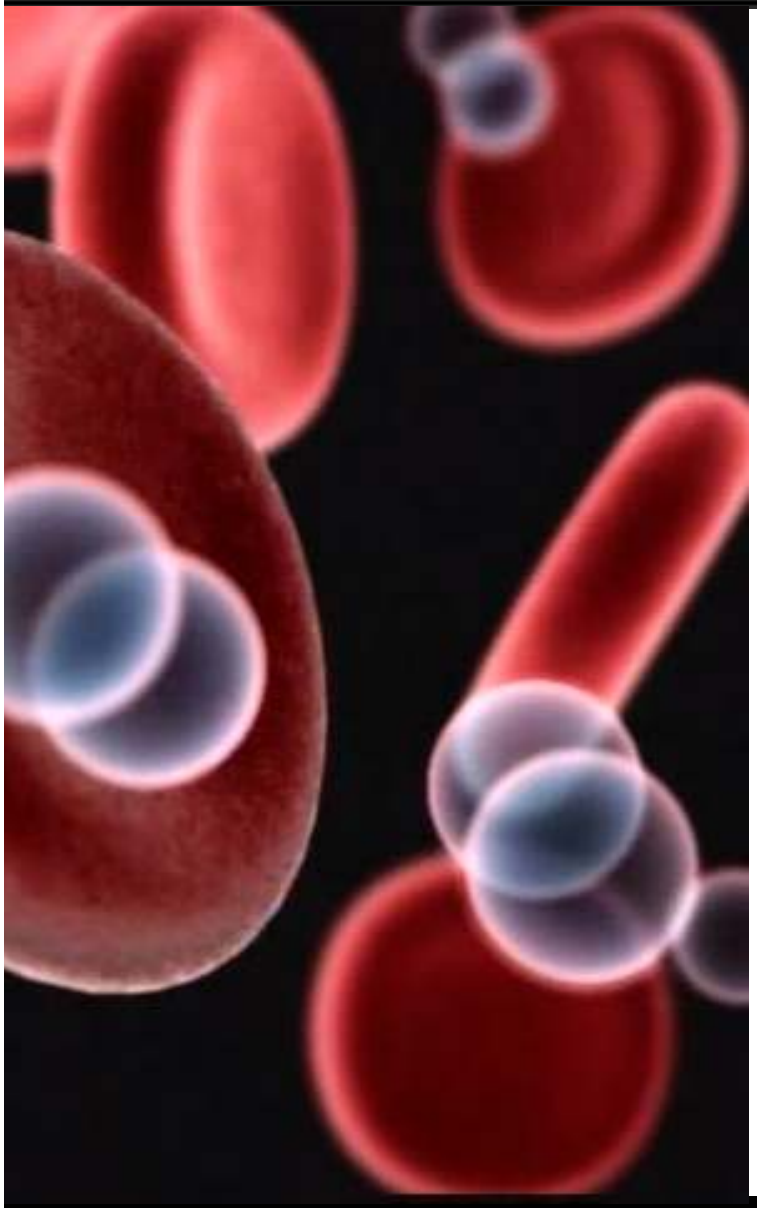
- Complex IV of Electron Transport Chain – binds cytochrome c oxidase
- CO does NOT bind with same affinity as O_2 (requires significant hypoxia)
- Delayed effect ATP synthesis produces lactic acidosis

Pathophysiology



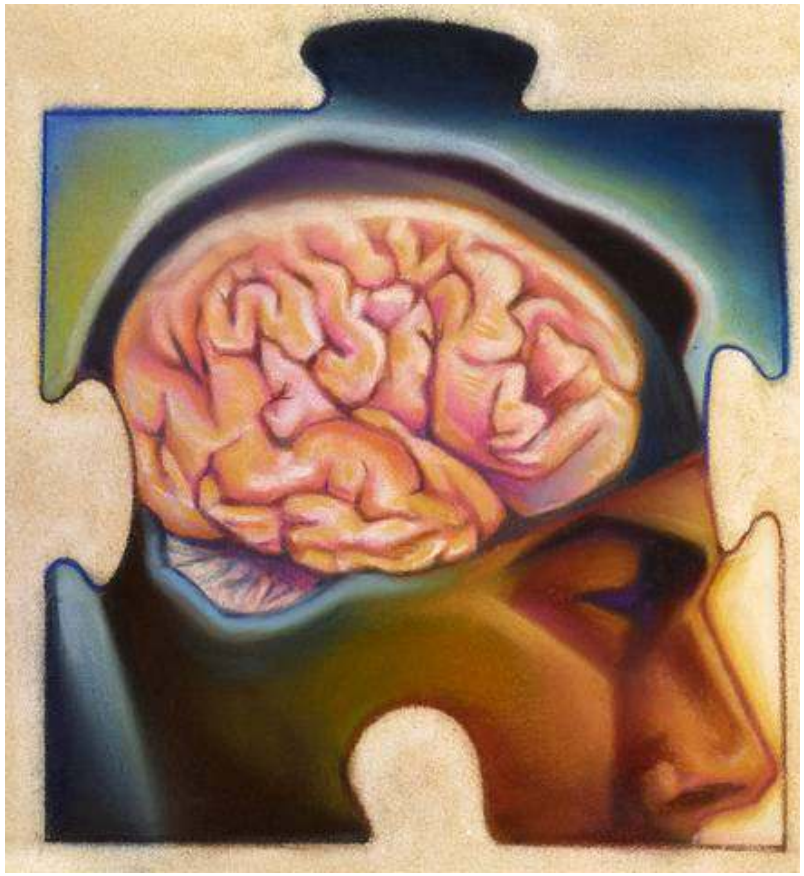
- **CO limits oxygen transport**
  - Greater affinity (>210 x) for hemoglobin
- **Inhibits oxygen transfer**
  - Interferes with normal unloading to tissues
- **Binds with myoglobin (muscle)**
  - Interferes with heart and skeletal muscle fxn
- **Binds to cytochrome oxidases**
  - Induces anerobic metabolism (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)

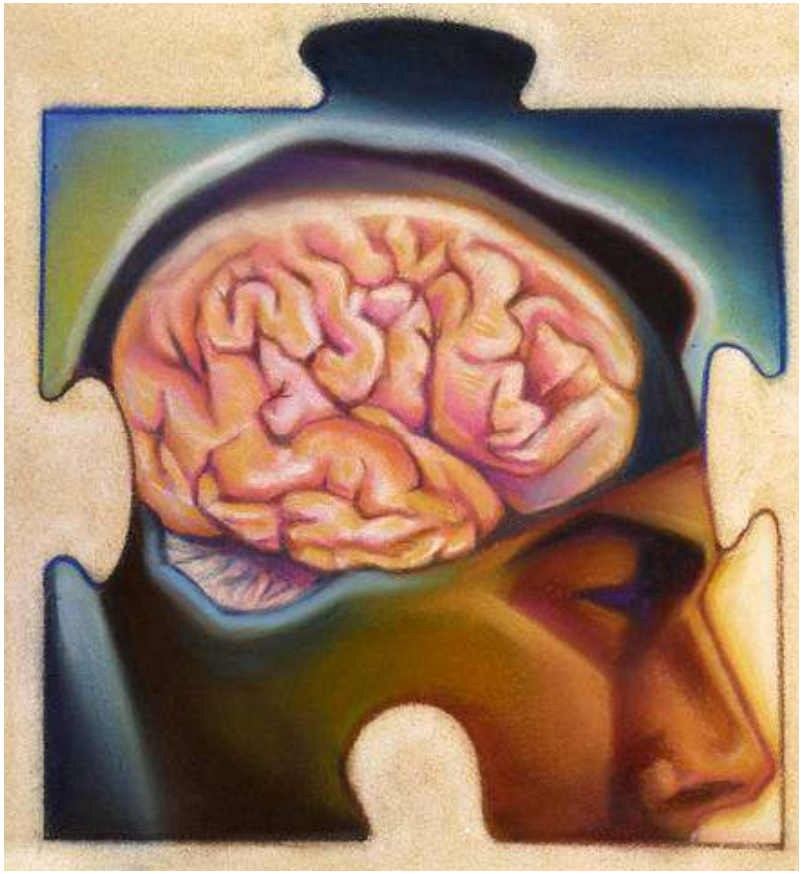
Harper A et. al, Age and Ageing. 2004;33(2):105-9.
Kao LW et. al, Emerg Med Clin North Am. 2004
Weaver LK, et al. N Engl J Med, 2002;347(14):1057-067

Long-Term/Chronic Sequelae

Cognitive and personality changes, dementia, seizures, psychosis, amnesia, parkinsonism, depression, short-term memory loss, incontinence.

Abelsohn A, CMAJ 2002:166 (13):1685-90

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

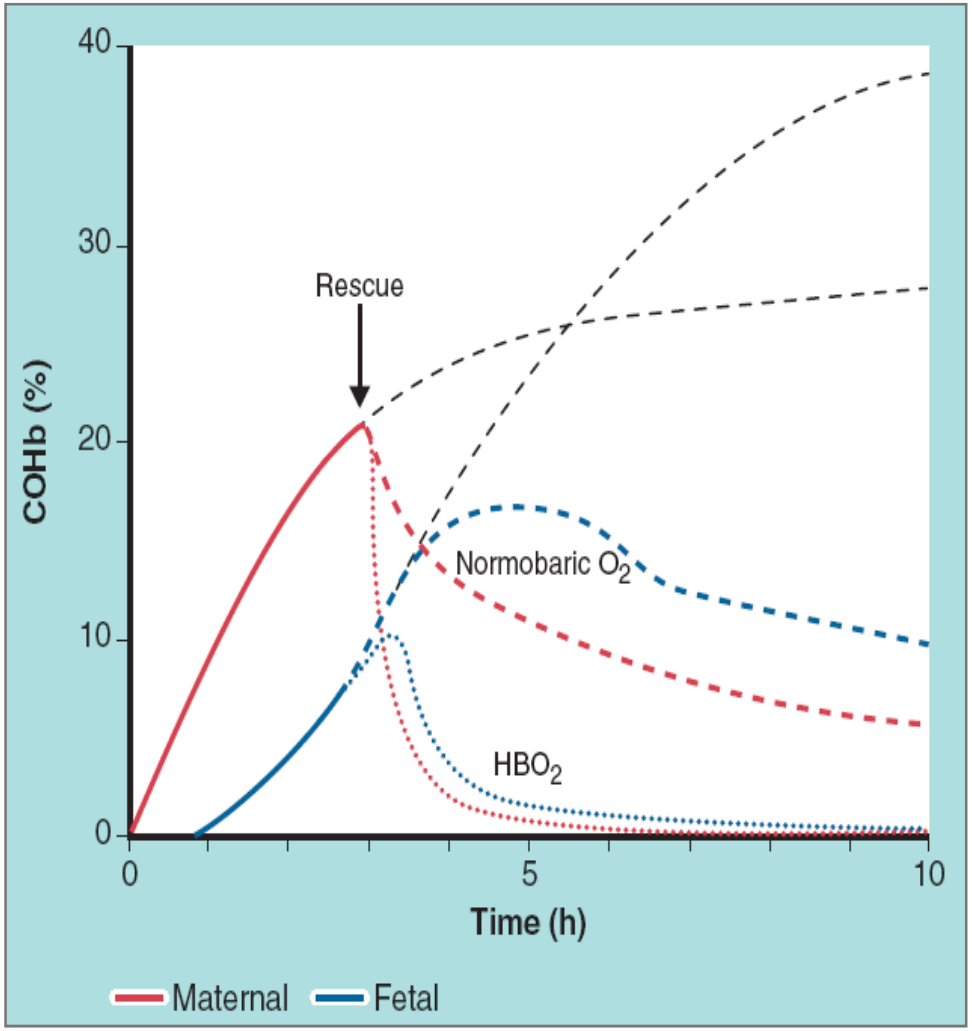
Myocardial Injury and Long-Term Mortality Following Moderate to Severe Carbon Monoxide Poisoning. Henry CR, Satran D, Lindgren B, Adkinson C, Nicholson C, Henry TD. JAMA. 2006;295(4):398-402

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%**

COHb% as a marker of cardiovascular risk in never smokers: Results from a population-based cohort study. Hedblad BO, Engstrom G, Janzon E, Berglund G, Janzon L. Scand J Pub Health. 2006;34:609-615.

Fetal Damage



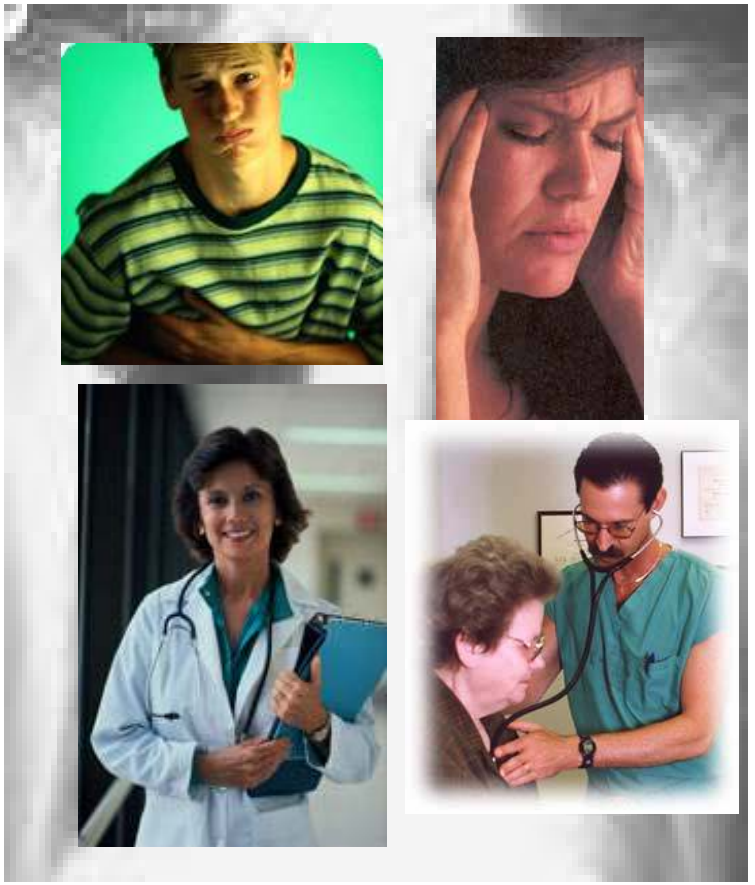
Theoretical effect of different treatments

on maternal and fetal COHb levels over time

Rucker J, Fisher J, Carbon Monoxide Poisoning, Chapter 63 Longo LD: The biological effects of carbon monoxide on the pregnant woman, fetus, and newborn infant. Am J Obstet Gynecol 1977;129: 69-103.

Signs and Symptoms

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



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

Barker MD, et al. J Pediatr. 1988;1:233-43

Barret L, et al. Clin Toxicol. 1985;23:309-13

Grace TW, et al. JAMA. 1981;246:1698-700

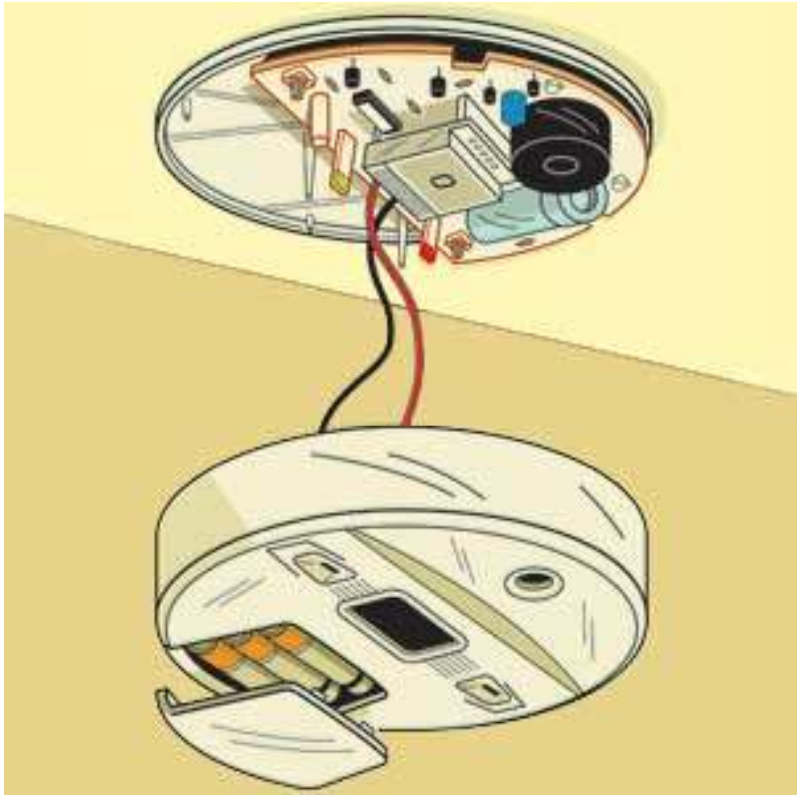
Diagnostic Problem

- **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

- Revised 1992, 1995, 1998
- 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%)

National Academy of Clinical Biochemistry:

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)

THE NATIONAL ACADEMY OF CLINICAL BIOCHEMISTRY

Blood Sampling for COHb

- A-COHb = V-COHb

Touger et al, *Ann Emerg Med,* 1995;25:481-3.

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), r^2 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

www.micro-direct.com

- Requires 20 second breath holding, measures E_TCO in PPM
- Present accuracy $\pm$ 2 PPM, COHb obtained from Haldane Equation (essentially = PPM ÷ 6)

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

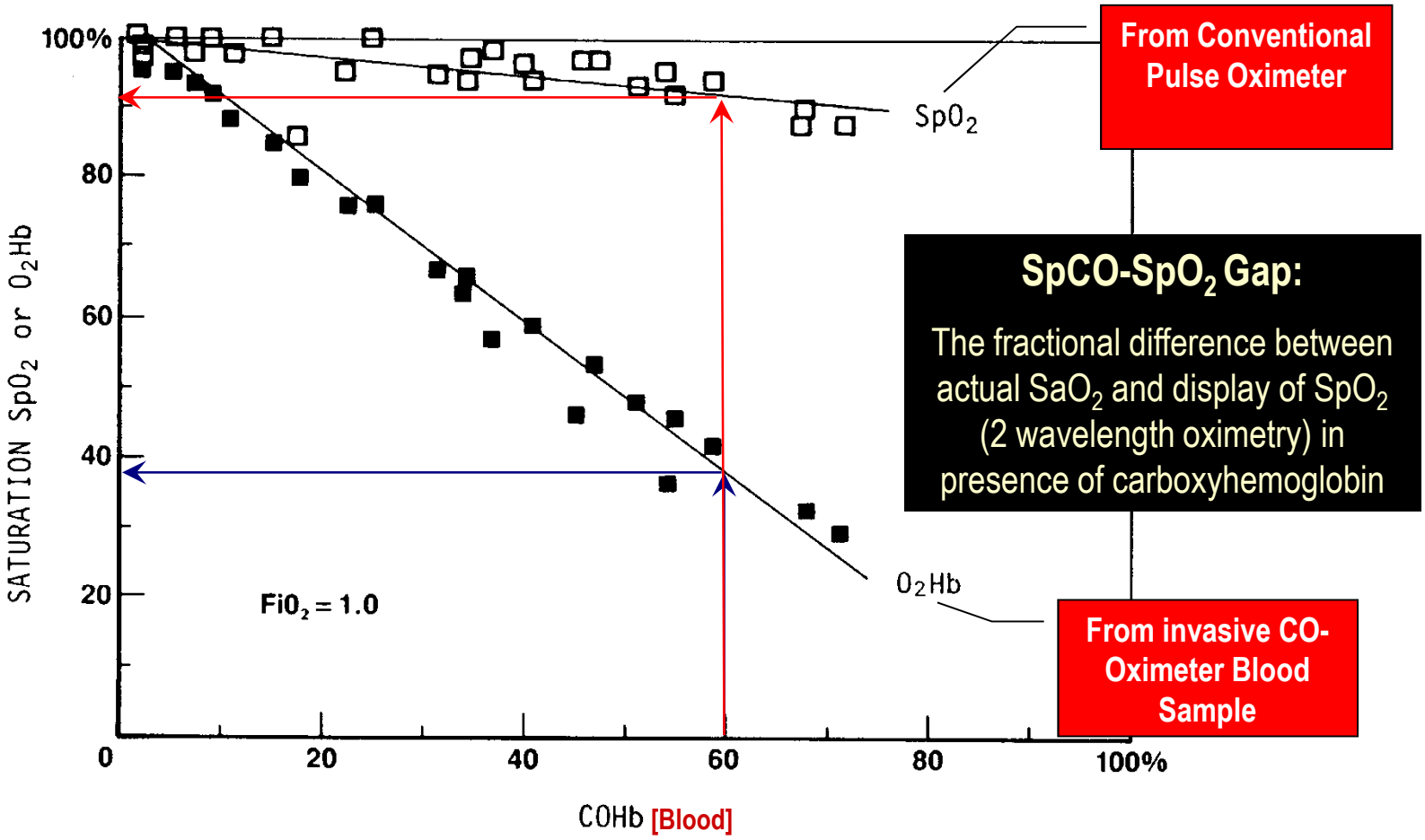
www.masimo.com

Pulse CO

- Fast, econom
- Can be used
  (including un
- No disposab
- No calibratio
- Use wider tha
  devices after
  in marketplace

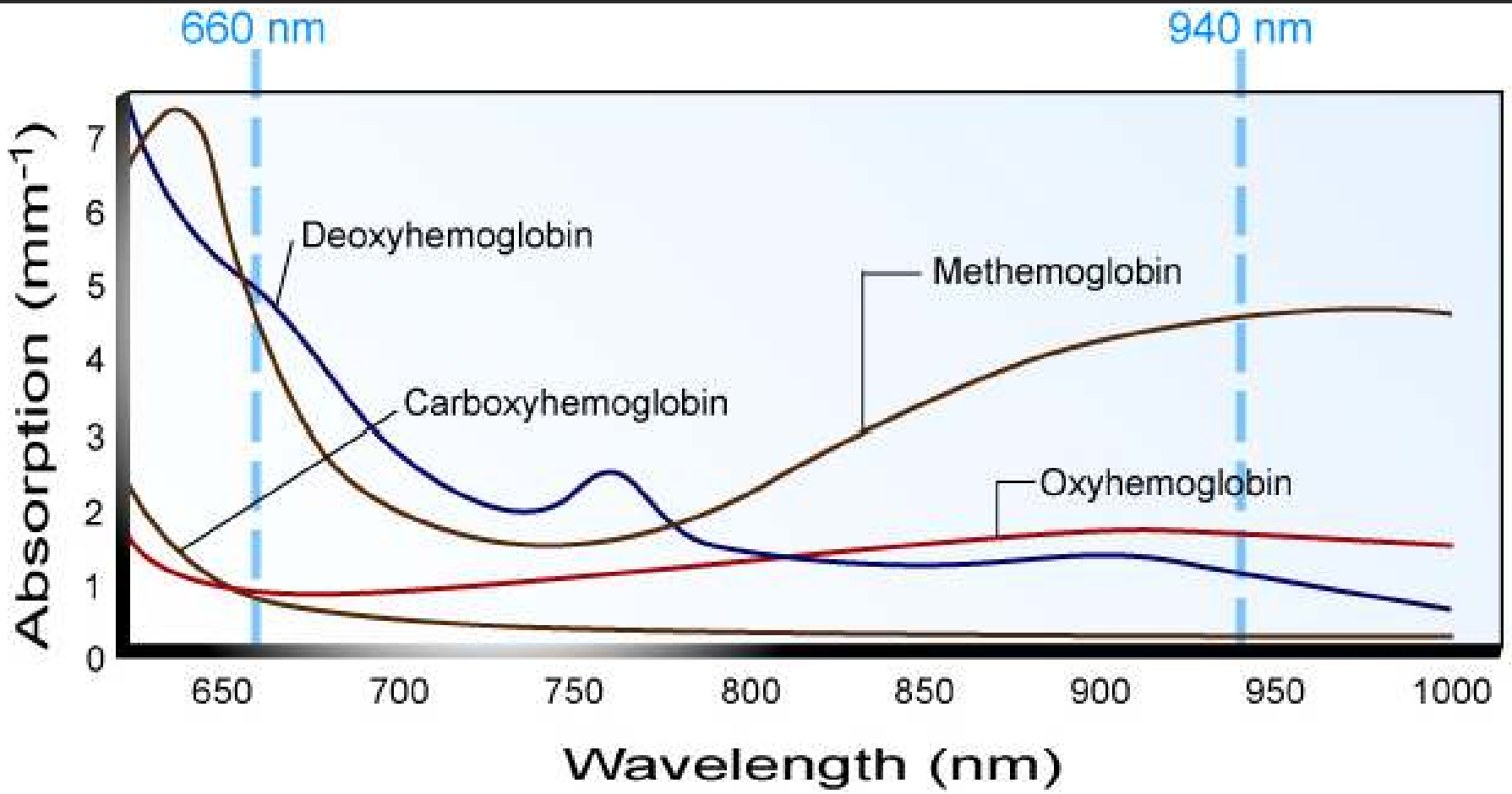
Limitations of Pulse Oximetry

Conventional pulse oximetry can not distinguish between COHb, and O_2Hb



Barker SJ, Tremper KK. The Effect of Carbon Monoxide Inhalation on Pulse Oximetry and Transcutaneous PO_2. *Anesthesiology* 1987; 66:677-679

How Noninvasive Pulse CO-Oximetry Works

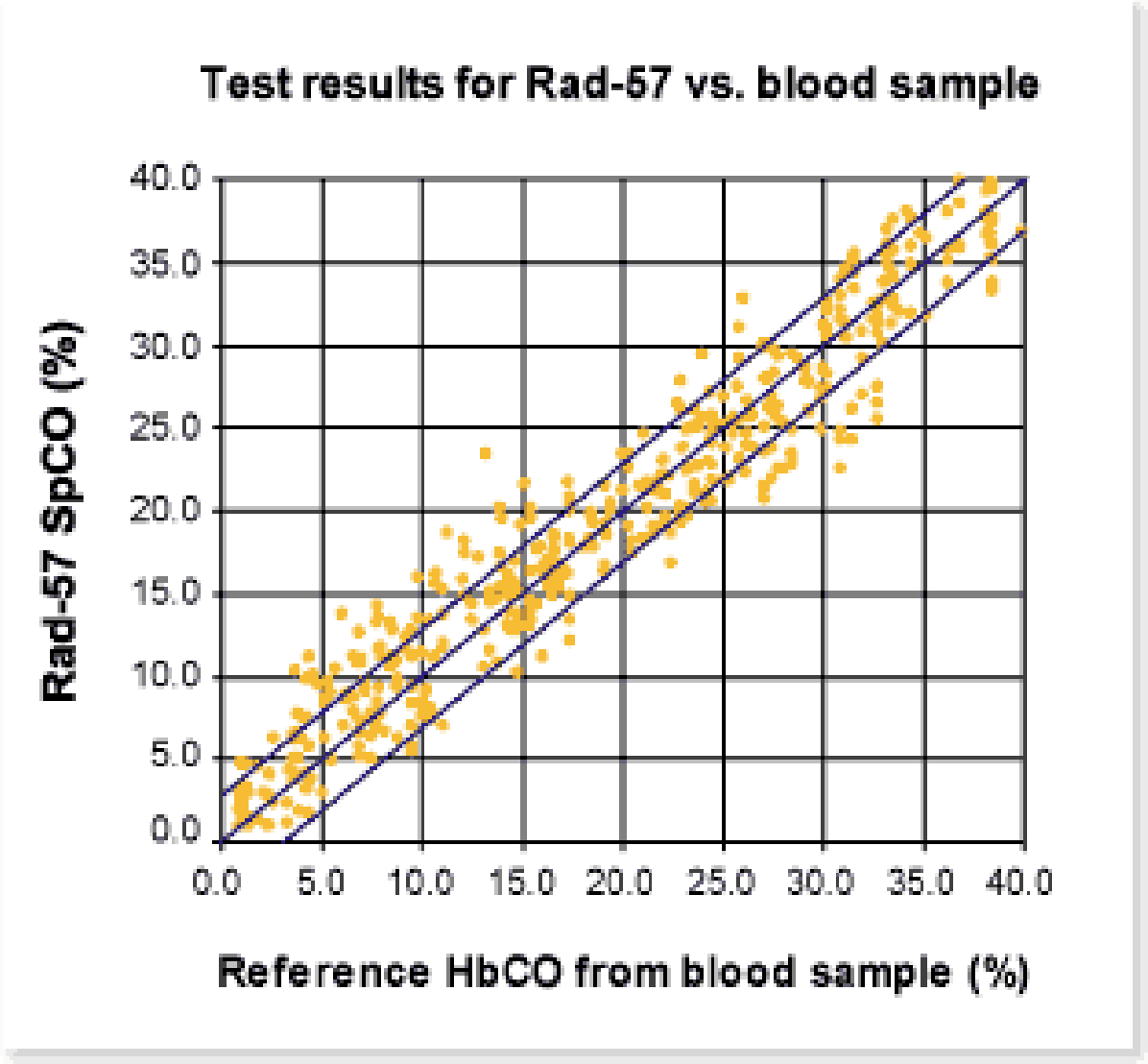


Oxygenated Hb and reduced Hb absorb different amounts of Red (RD) and Infrared (IR) Light

(Two-wavelength oximeters cannot measure dyshemoglobins)

FDA Validation

Masimo Rainbow SET Compared to Reference Methodology

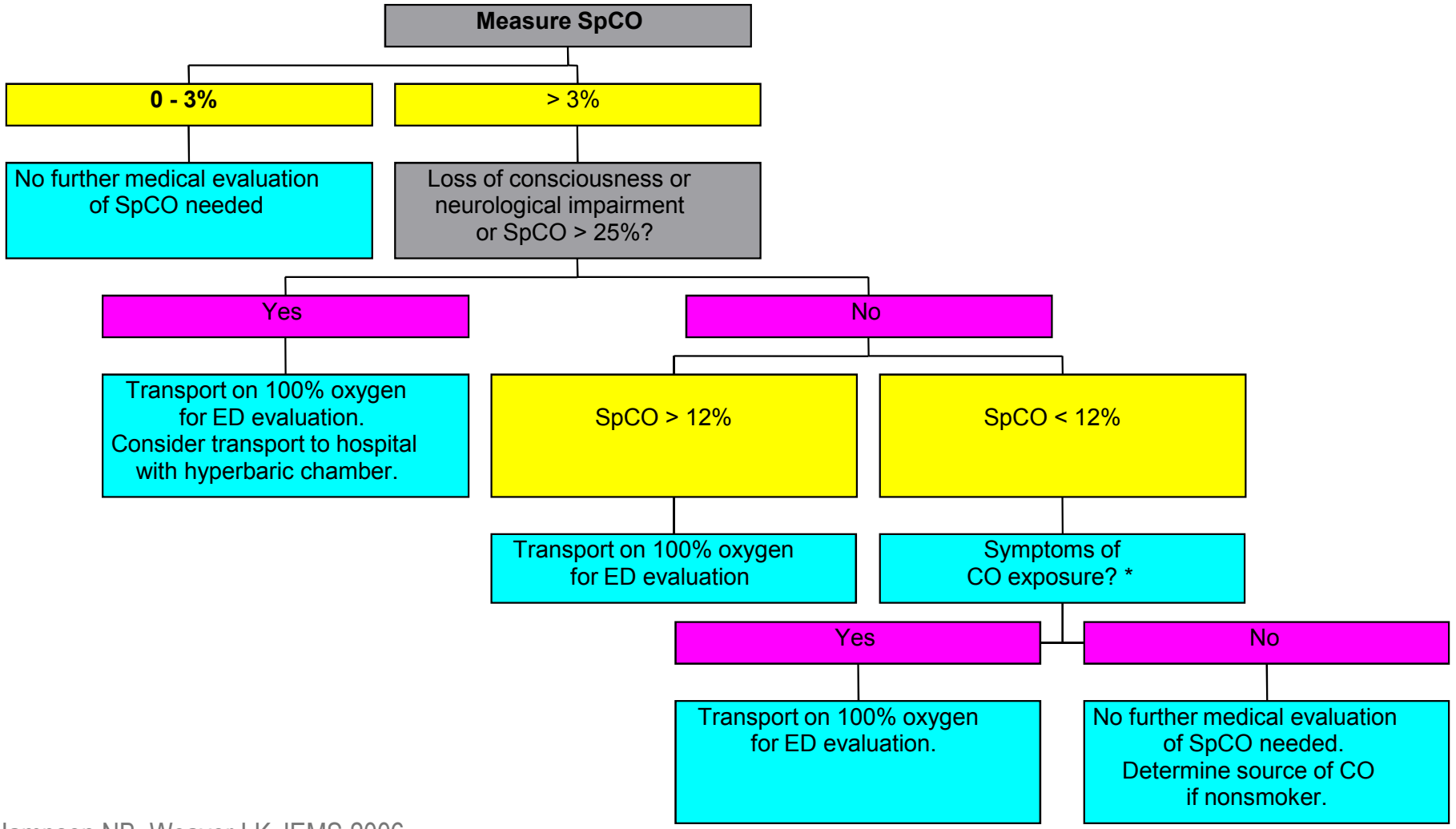


5,000 Patient Brown University Study

- 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



Hampson NB, Weaver LK JEMS 2006

Categorizing Symptoms

Headache:	None	Mild	Moderate	Throbbing	Severe
LOC:	Alert	Slight confusion	Very confused	Syncope or unconscious	Seizures
SOB:	None	Exertional		At rest	
GI:	None	Mild nausea	Nausea	Vomited or vomiting	

Treating CO Poisoning - Oxygen

Hyperbaric Oxygen Treatment

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

Weaver LK et al, Hyperbaric Oxygen for Acute Carbon Monoxide Poisoning, N Engl J Med 2002;347(14) :1057-067

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

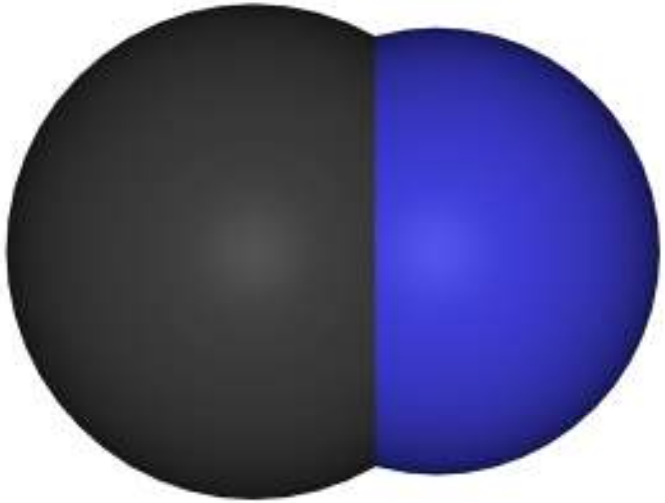
CO Assessment

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

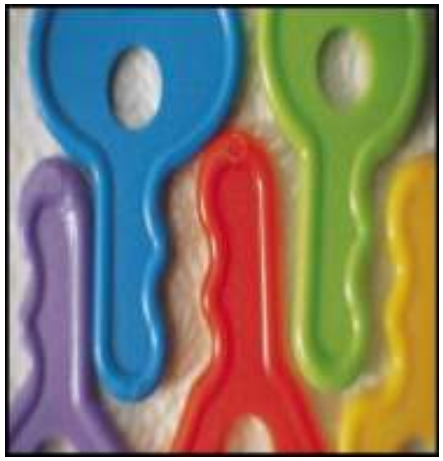
Cyanide in Fire Smoke

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) =

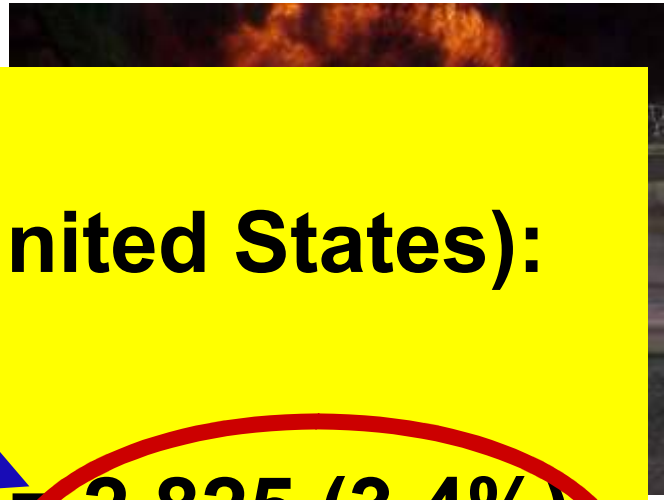
- Burn 2 – 3 times hotter and ignite 2 – 3 times faster
- Emit significant HCN during pyrolysis

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



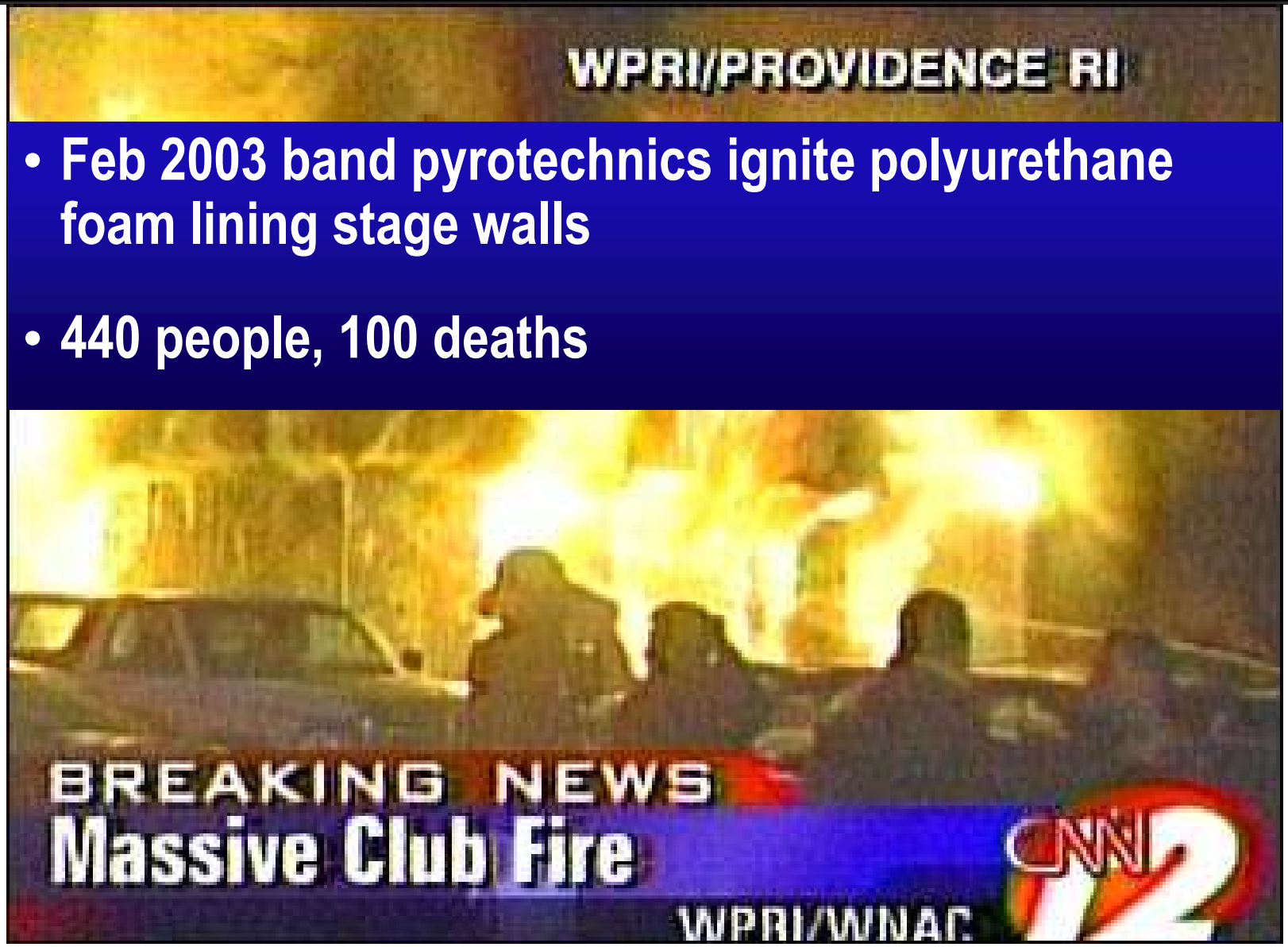
Firefighter Injuries – 2006 (United States):

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

= 3,555 (4.3%)

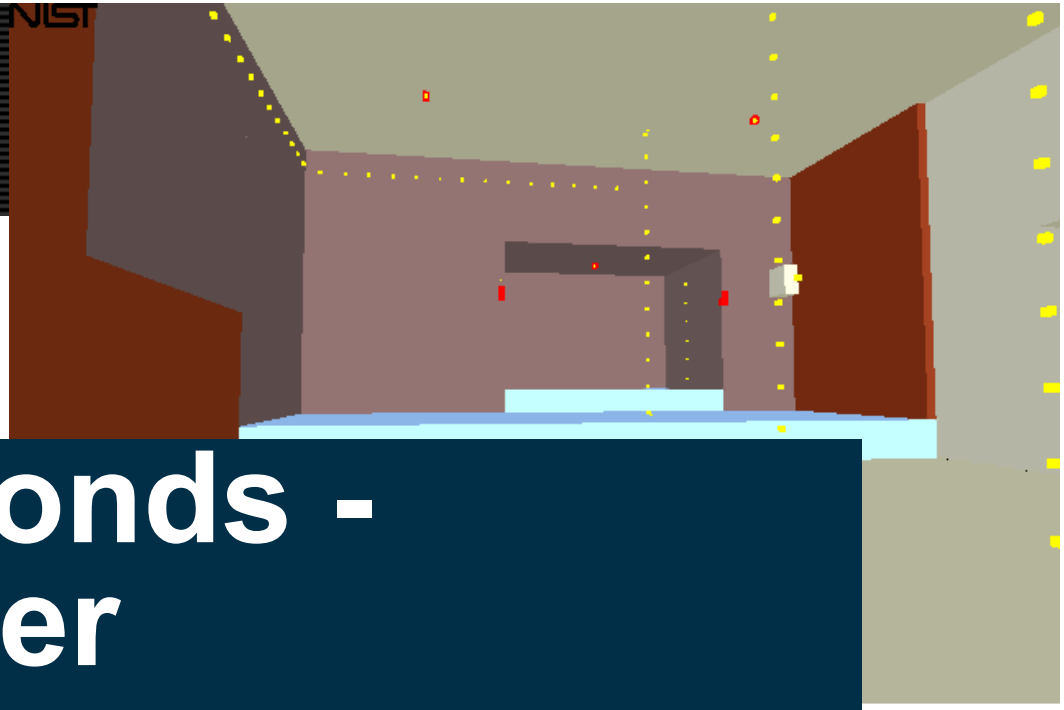
- NFPA Survey of Fire Depts for U.S. Fire Experience, 2006.

Station Nightclub Fire - RI



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

Simulation of platform area

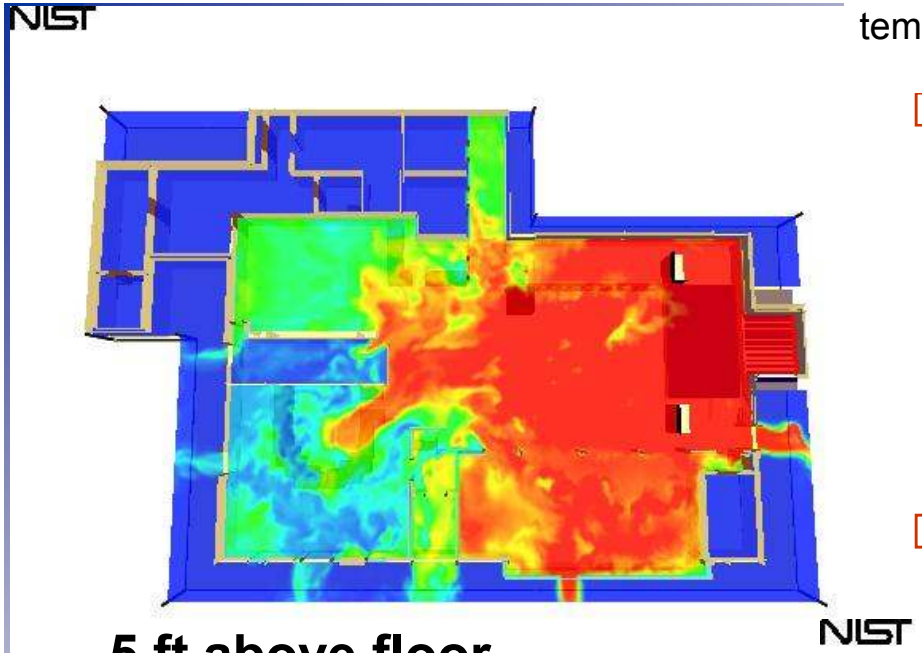


- 60 seconds - flashover
- 90 seconds – CO, CN, O2 incompatible with life

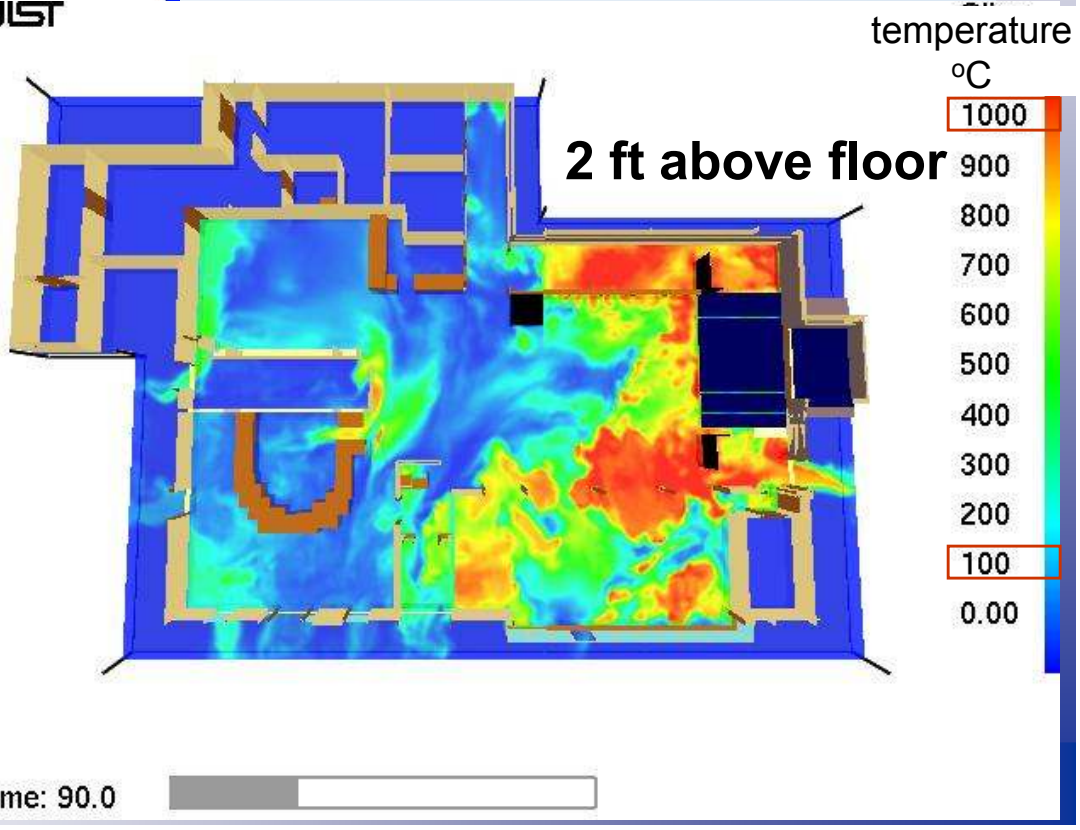
NIST Simulation

Temperatures in The Station at 90 seconds

(from computer simulation)



5 ft above floor



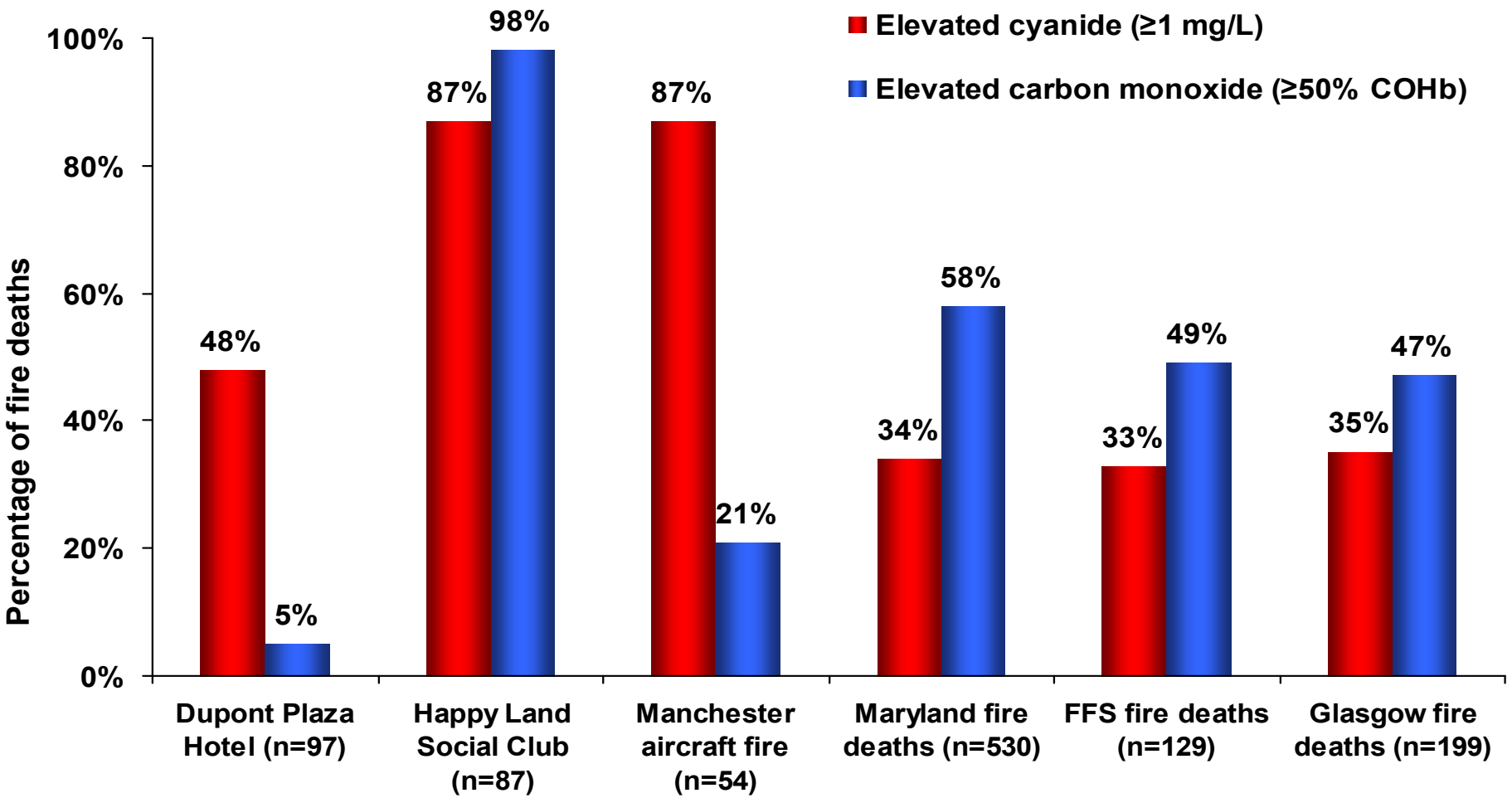
2 ft above floor

Peak temperatures in red exceed 1000 °C (1832 °F)

Light blue indicates temperatures around the boiling point of water (100 °C, or 212 °F)

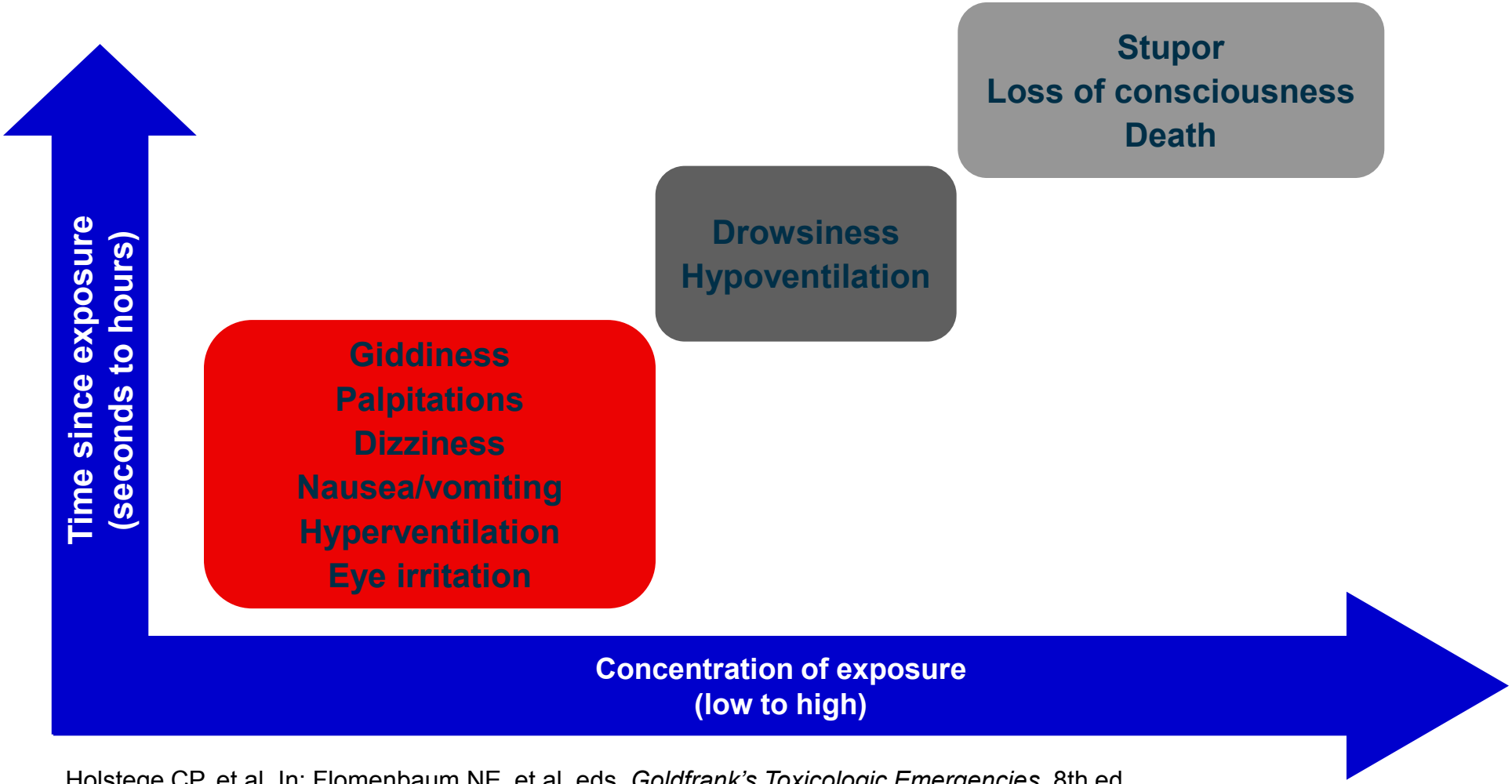
NIST

CN & CO: Exposure in Fire Deaths



COHb, carboxyhemoglobin; FFS, Foundation for Fire Safety.
Adapted from Alarie Y. *Crit Rev Toxicol*. 2002;32:259-289.

Progression of CN Poisoning



Holstege CP, et al. In: Flomenbaum NE, et al, eds. *Goldfrank's Toxicologic Emergencies.* 8th ed. New York, NY: McGraw-Hill; 2006:1712-1757.

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

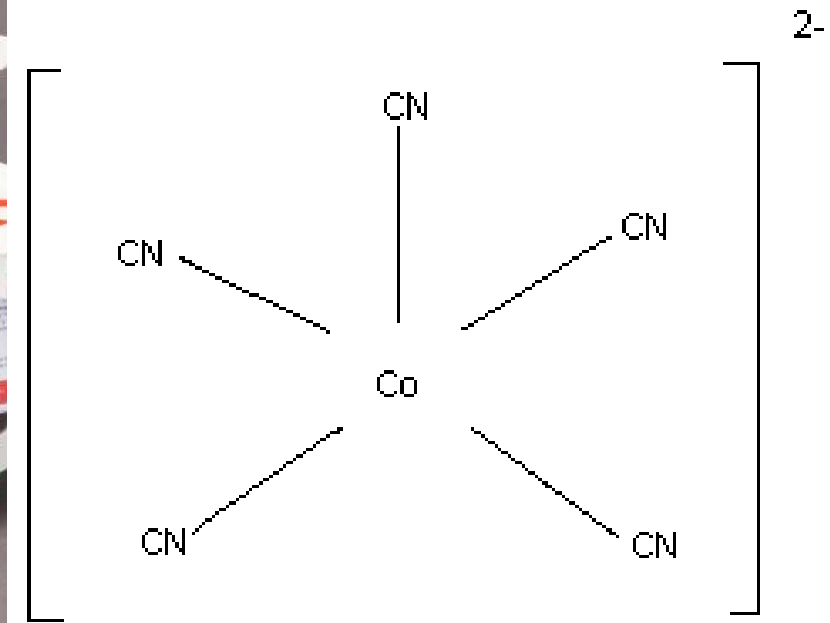
Paris Fire Brigade Protocol

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

Paris Fire Brigade Protocol

Cyanokit® (hydroxocobalamin)

- **5 grams IV over 15 minutes**
- **Second dose if needed (clinical condition)**



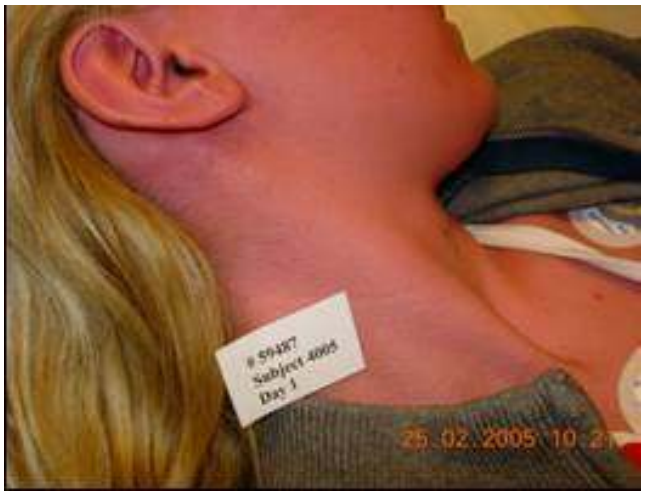
Cyanokit® (hydroxocobalamin)

- 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!**

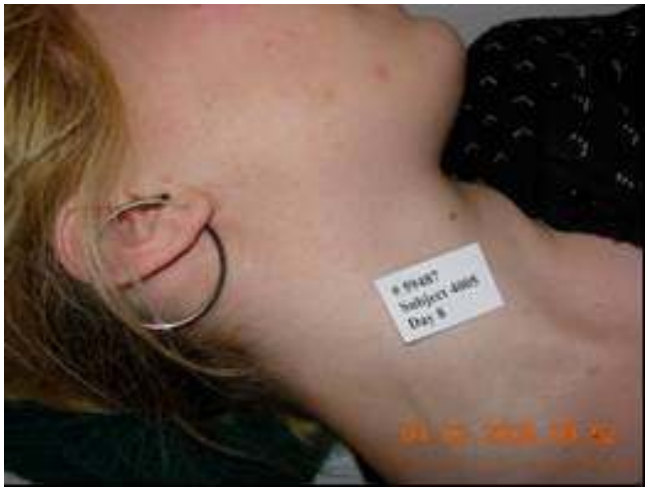
Sauer SW, Keim ME. Ann Emerg Med 2001;37:635-641.

Skin Redness in Healthy Volunteers

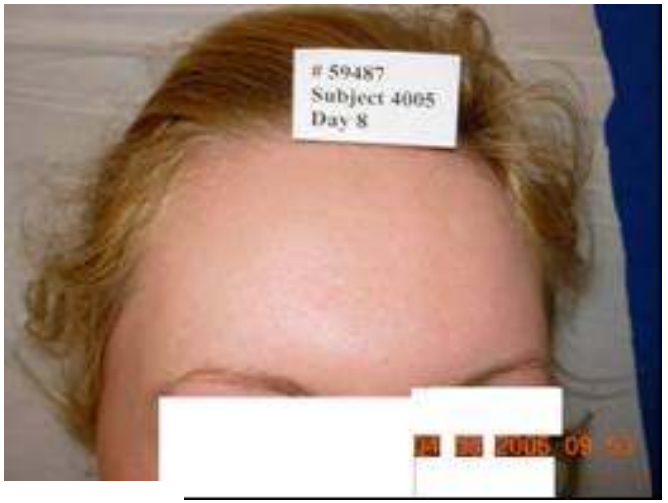
After 10 g treatment, photos on Day 1 and on Day 8.



Day 1*

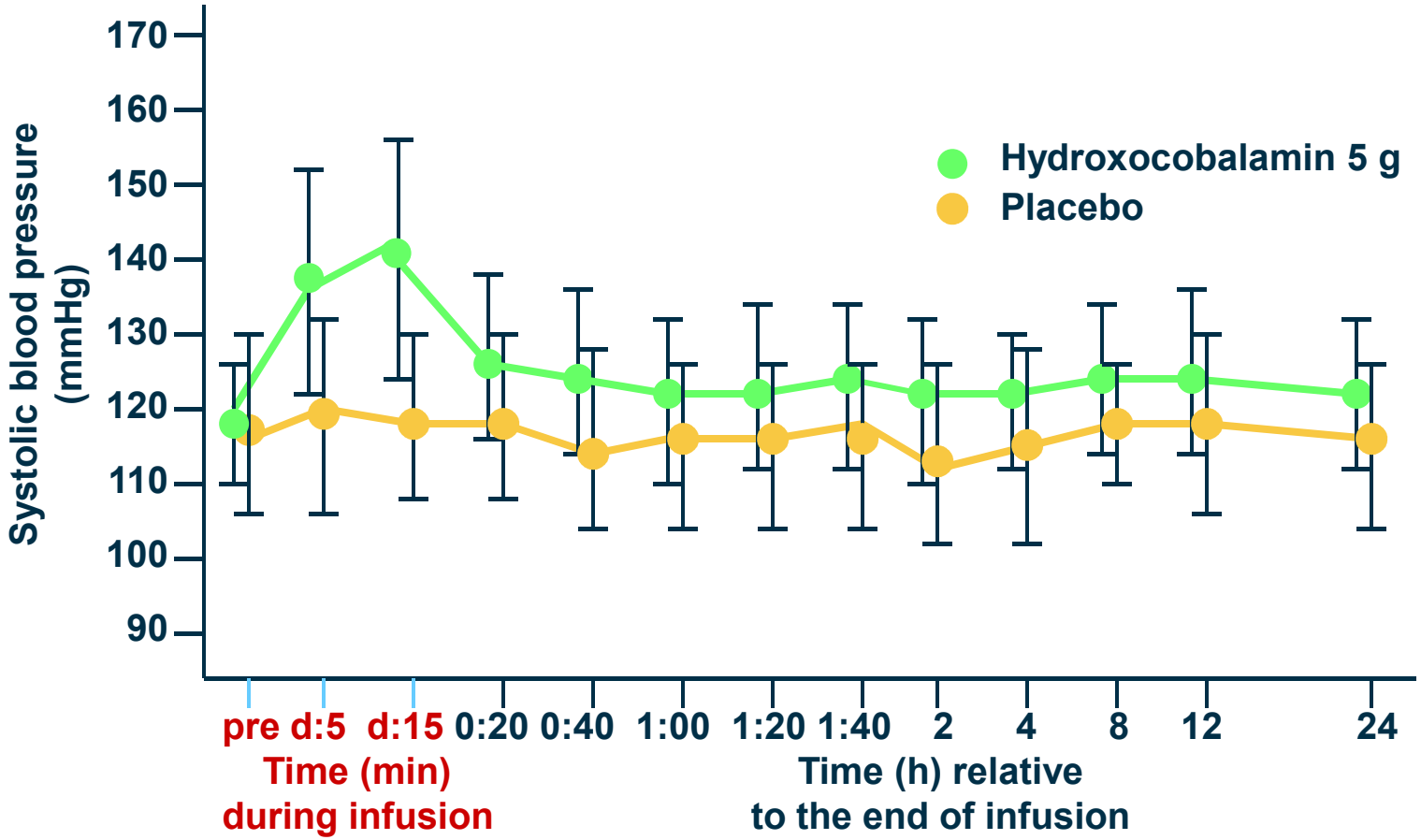


Day 8†



*No flash photography. †Flash photography used.
OHCo, hydroxocobalamin. Uhl W, et al. *Clin Toxicol*. 2006;44:17-28.

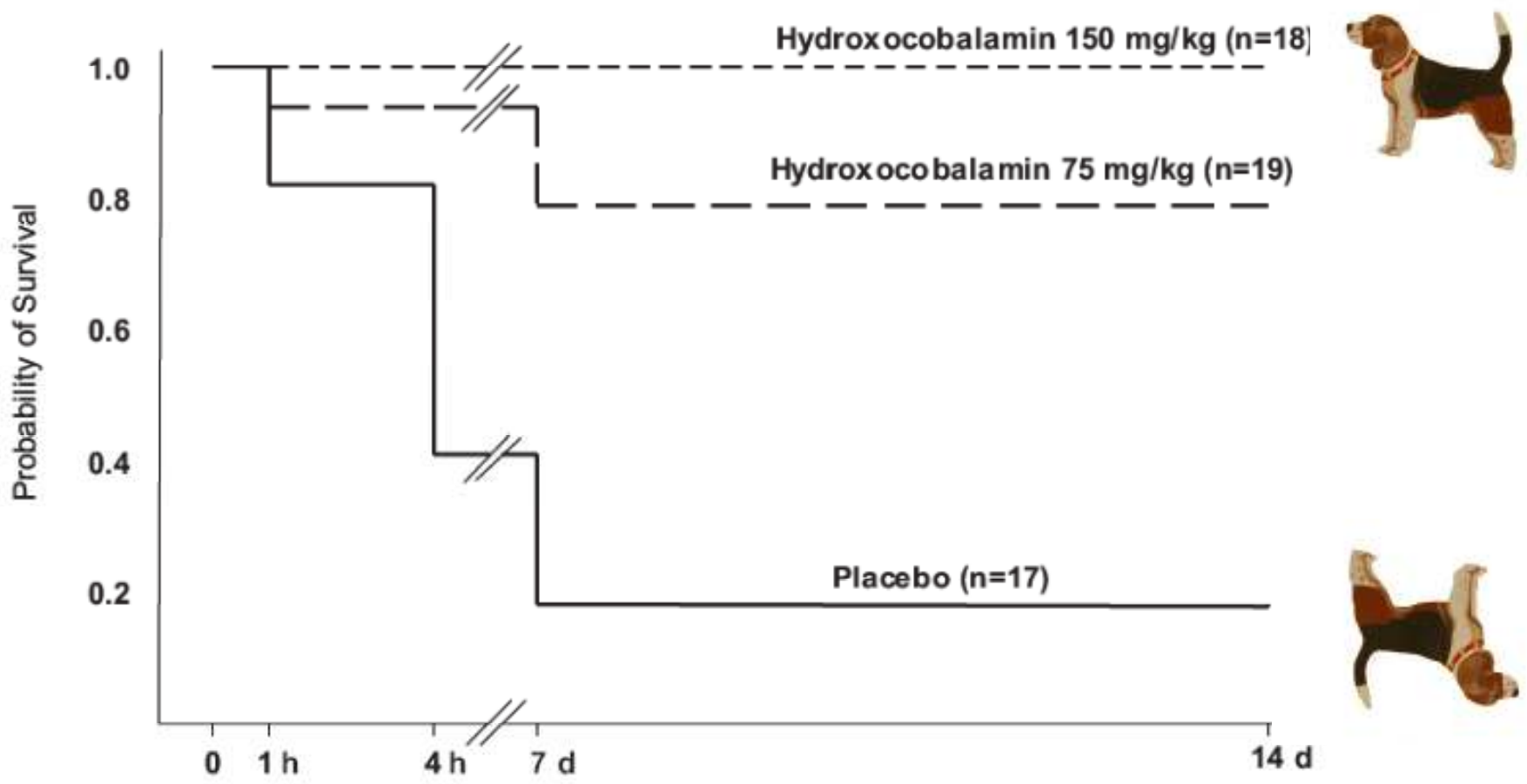
HCO 5 g: Systolic Blood Pressure



Uhl W, et al. *Clin Toxicol* (Phila). 2006;44 Suppl 1:17-28.

ARTICLE

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
  - Coverts 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.

Gracia R, Shepard G. *Pharmacotherapy*. 2004;24:1358-1366.

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.
Gracia R, Shepherd G. *Pharmacotherapy*. 2004;24(10):1358-1365.

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

Flomenbaum NE, et al. *Goldfrank's Toxicologic Emergencies*. McGraw-Hill. 2006.
Megarbane B, et al. *J Chin Med Assoc.* 2003;66:193-203.

"Of course, I'm feeling uncomfortabl
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

Flomenbaum NE, et al. *Goldfrank's Toxicologic Emergencies*. McGraw-Hill. 2006.
Megarbane B, et al. *J Chin Med Assoc.* 2003;66:193-203.

Take Home Message

CN Treatment

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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