

Inhalational Injury

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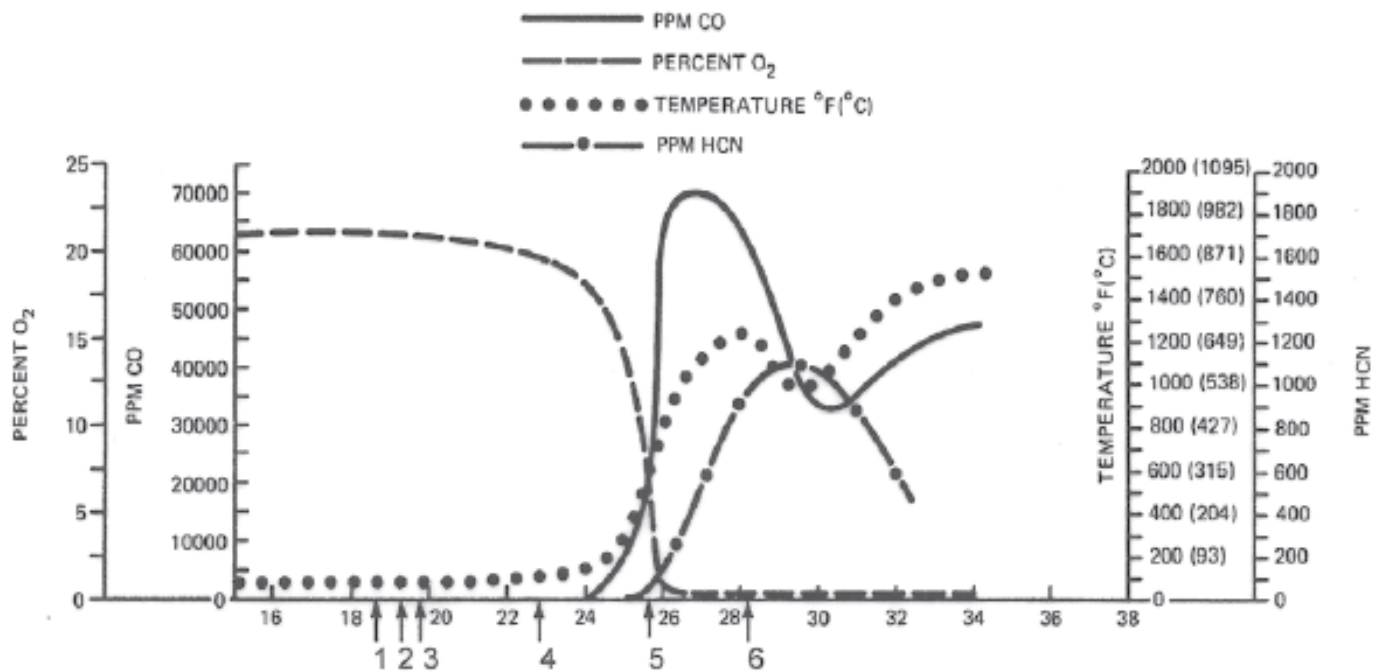
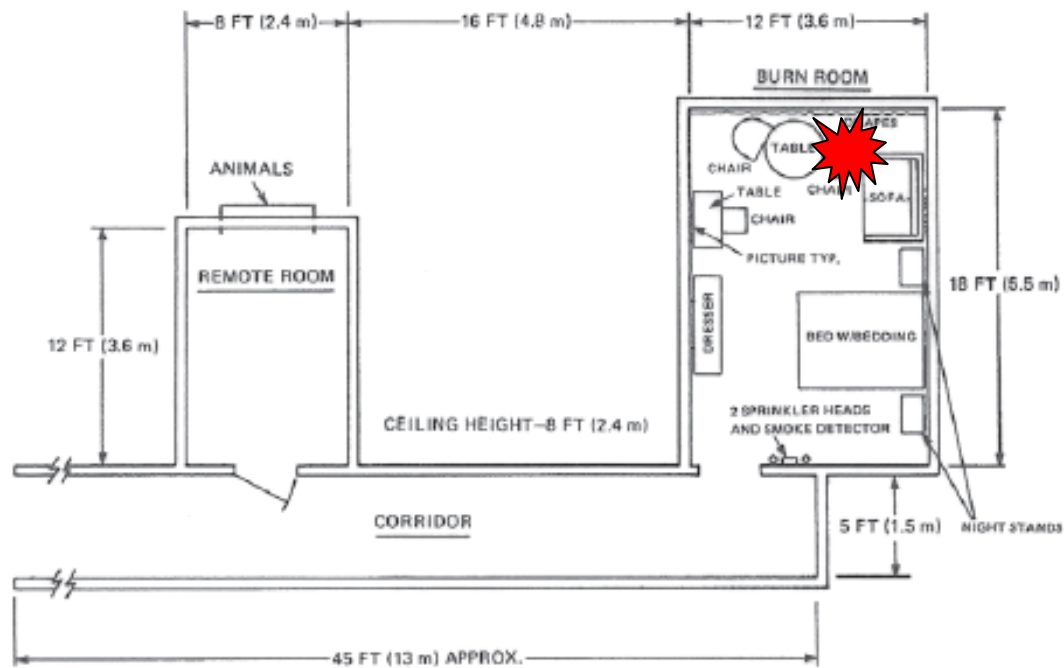


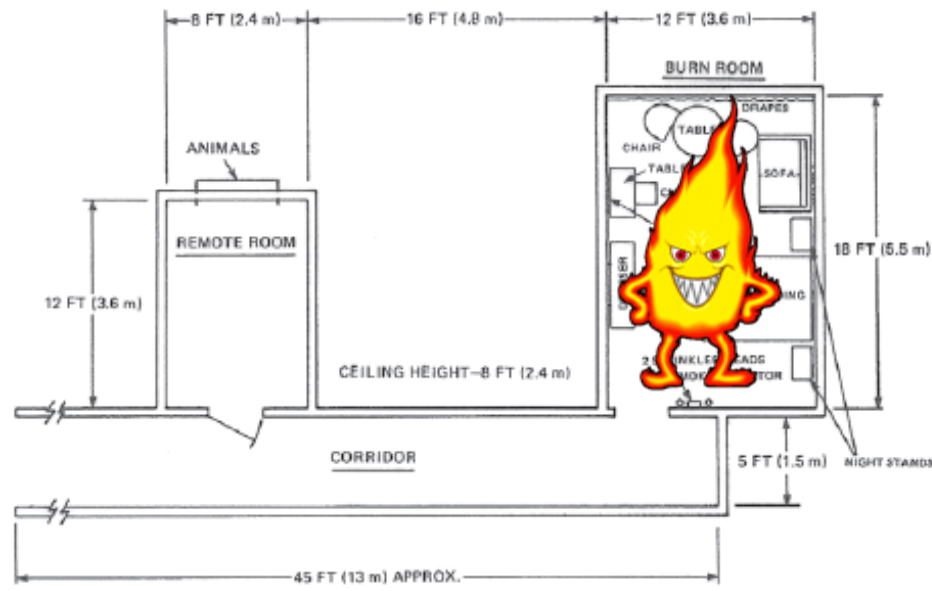
Conflict of interest

- No conflict of interest to declare

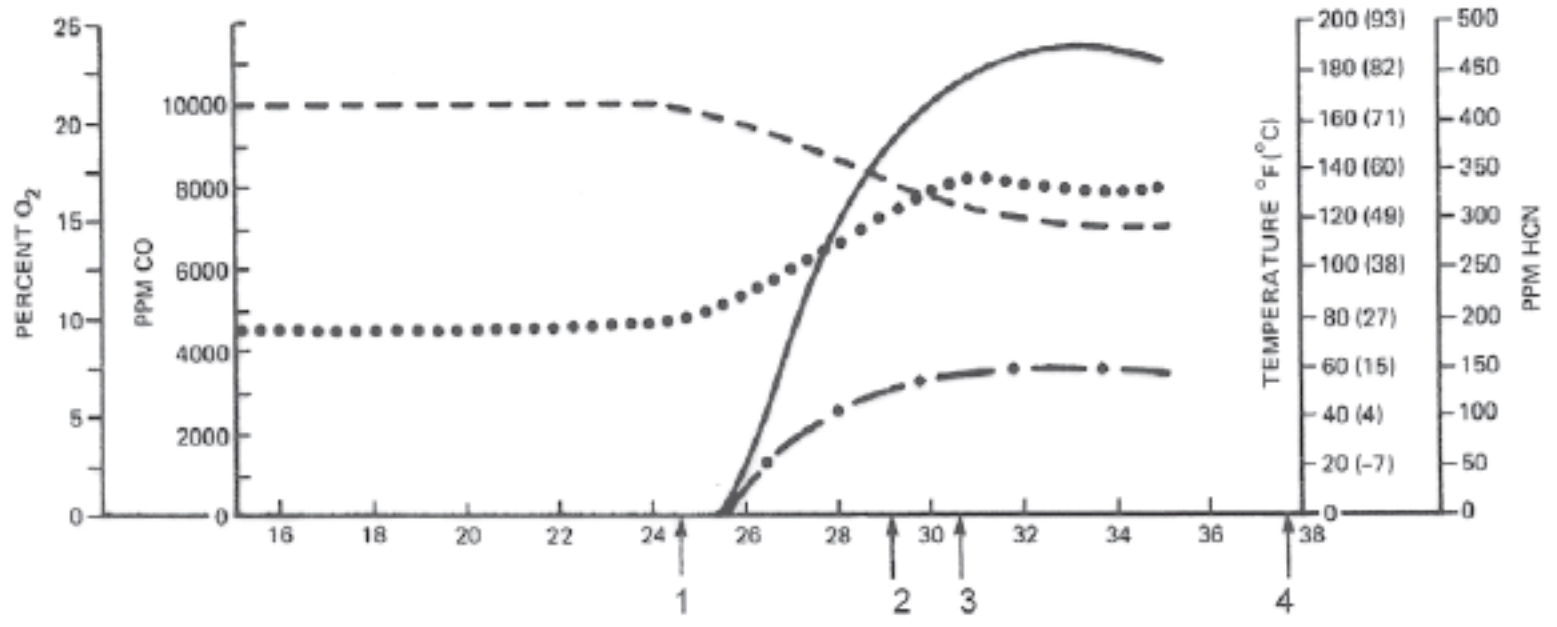
Objectives

- Understand different mechanisms of injury
- Anticipate types of injury based on clinical presentation
- Understand criteria for airway intervention
- Treat potential toxic exposures





- PPM CO
- PERCENT O₂
- TEMPERATURE °F(°C)
- PPM HCN



Mechanisms of Injury

- Heat (Thermal)
- Hypoxia
- Direct toxins
- Systemic toxins



Thermal Injury

- Smoke is hot, but dry
- Low specific heat capacity
 - Rare to have subglottic burns from smoke alone
- Can burn if moist heat, or with general burn injury (ambient heat)

Hypoxia

- Fire consumes oxygen
- F_iO_2 of inhaled air decreases
 - Causes neural damage
 - Potentiates other toxins
 - Increases minute ventilation
 - More smoke inhaled



Direct Toxins

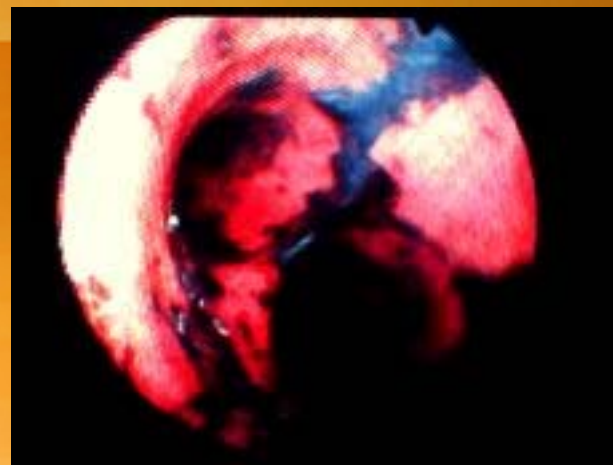
- Substances toxic to respiratory epithelium
 - SO₂, NO₂, N₂O, Chlorine, multiple organic compounds
 - HCl, HBr, HF, other inorganic compounds
- Effects usually appear 12-48h after exposure
 - Neutrophilic invasion
 - Mucociliary sloughing
 - Increased epithelial permeability

Airway evaluation

- Should be carefully performed in any potential inhalational injury victim
- Indications for early intubation
 - Hoarse voice
 - Stridor
 - Burns above clavicles
 - Soot in nares or mouth, carbonaceous sputum
 - Singeing of facial hair
 - Significant (>30% BSA) burns
 - Altered mental status

Bronchoscopy

- Controversial
- Demonstrates
 - depth of burn
 - carbonaceous sputum
- May help with unclear airway status
- No clear correlation with long-term prognosis



ARDS

- Common after inhalational injury
- Due to toxin inhalation
 - Edema, inflammation, bronchospasm, mucociliary sloughing
 - Microatelectasis, loss of surfactant, capillary leak
 - Can form bronchial casts
- Secretions build up, can obstruct ETT
- Peaks at ~72h post injury

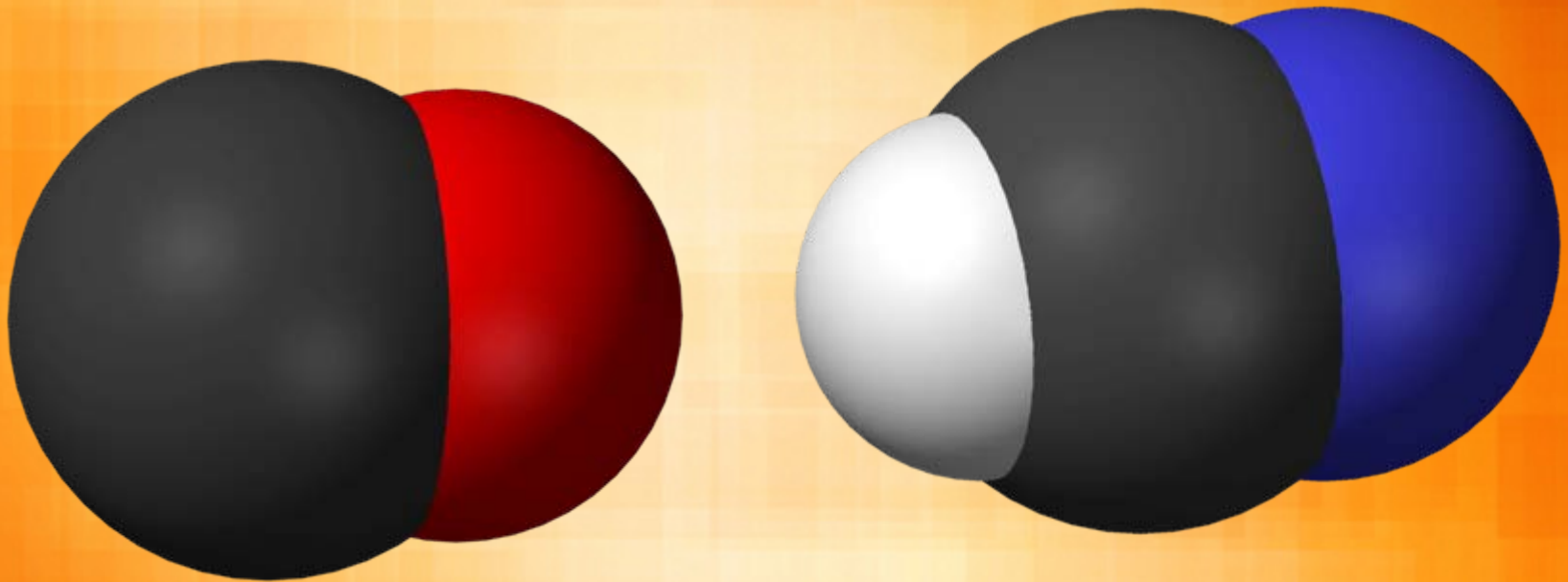
ARDS

- PEEP
- Permissive hypercapnia
- Frequently complicated by pneumonia (30-70%)



Systemic Toxins

- Carbon Monoxide and Cyanide



Carbon Monoxide Poisoning

- Has 240 times the affinity for Hb as O₂
- Also binds to porphyrins elsewhere
 - Mitochondrial cytochromes, NADPH reductase
- Half-life:
 - Room air: 300 minutes
 - 100% FiO₂: 90 minutes
 - 3 atmospheres 100% FiO₂: 30 minutes

Carbon Monoxide Toxicity - Diagnosis

- Cannot use SpO₂
- Only co-oximetry, or, where not available, high index of suspicion

Carbon Monoxide Poisoning - Treatment

- 100% O₂ for all potential victims
- Hyperbaric therapy considered if:
 - Unconscious, altered MS, neurologic deficit
 - ECG changes, CO-Hb >40% (20% if pregnant)

Carbon Monoxide - Treatment

- Hyperbaric therapy after acute injury
 - Controversial
 - 2 RCT's - opposite conclusions about neurocognitive sequelae
 - Both flawed studies
 - Poor randomization in one
 - Poor follow-up in the other



Carbon Monoxide - Treatment

- Isocapnic hyperpnea
 - Hyperventilating with a small amount of CO₂ in the circuit to maintain PaCO₂ at 40
 - Doubles rate of CO-Hb elimination

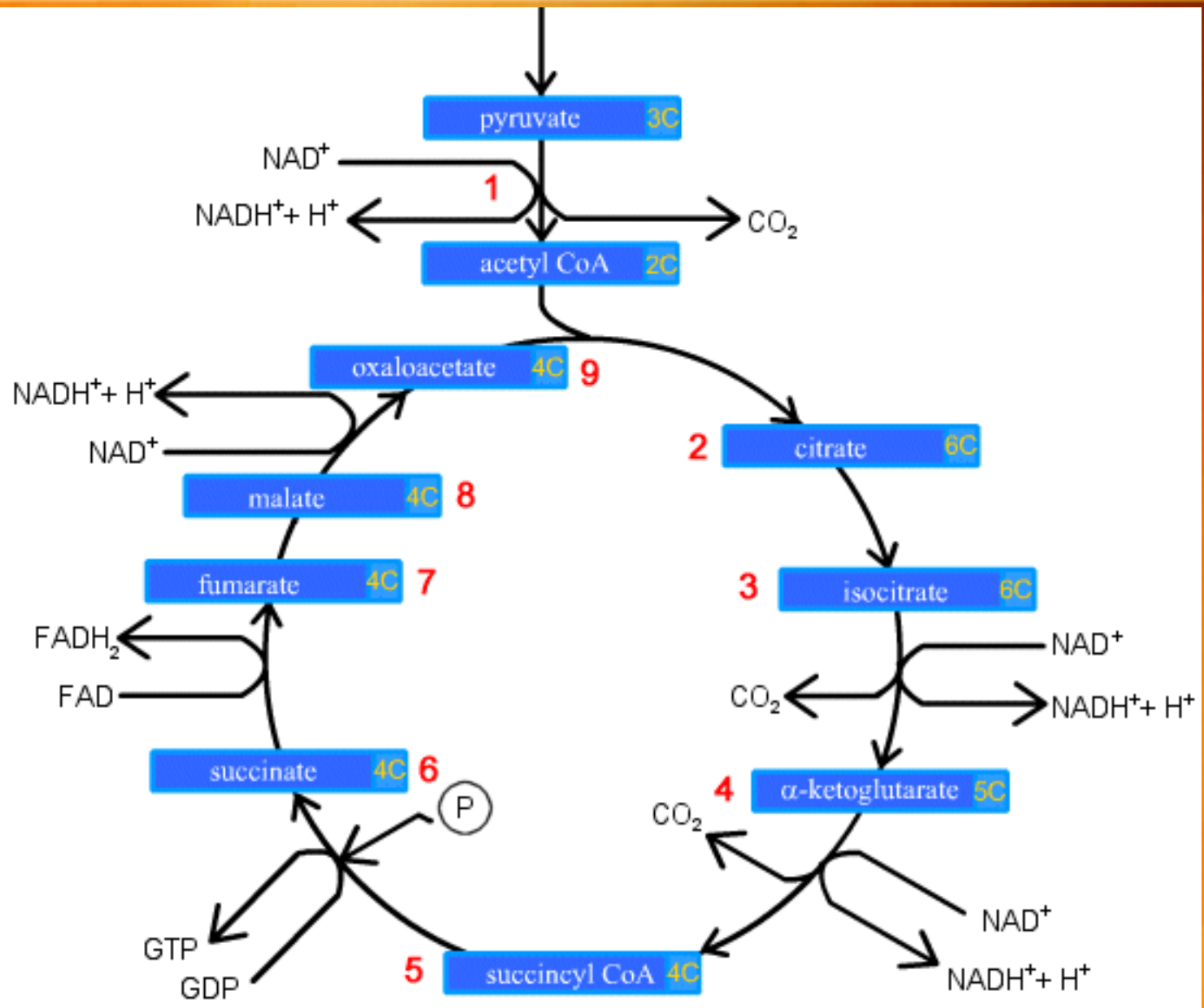
Hydrogen Cyanide (HCN)

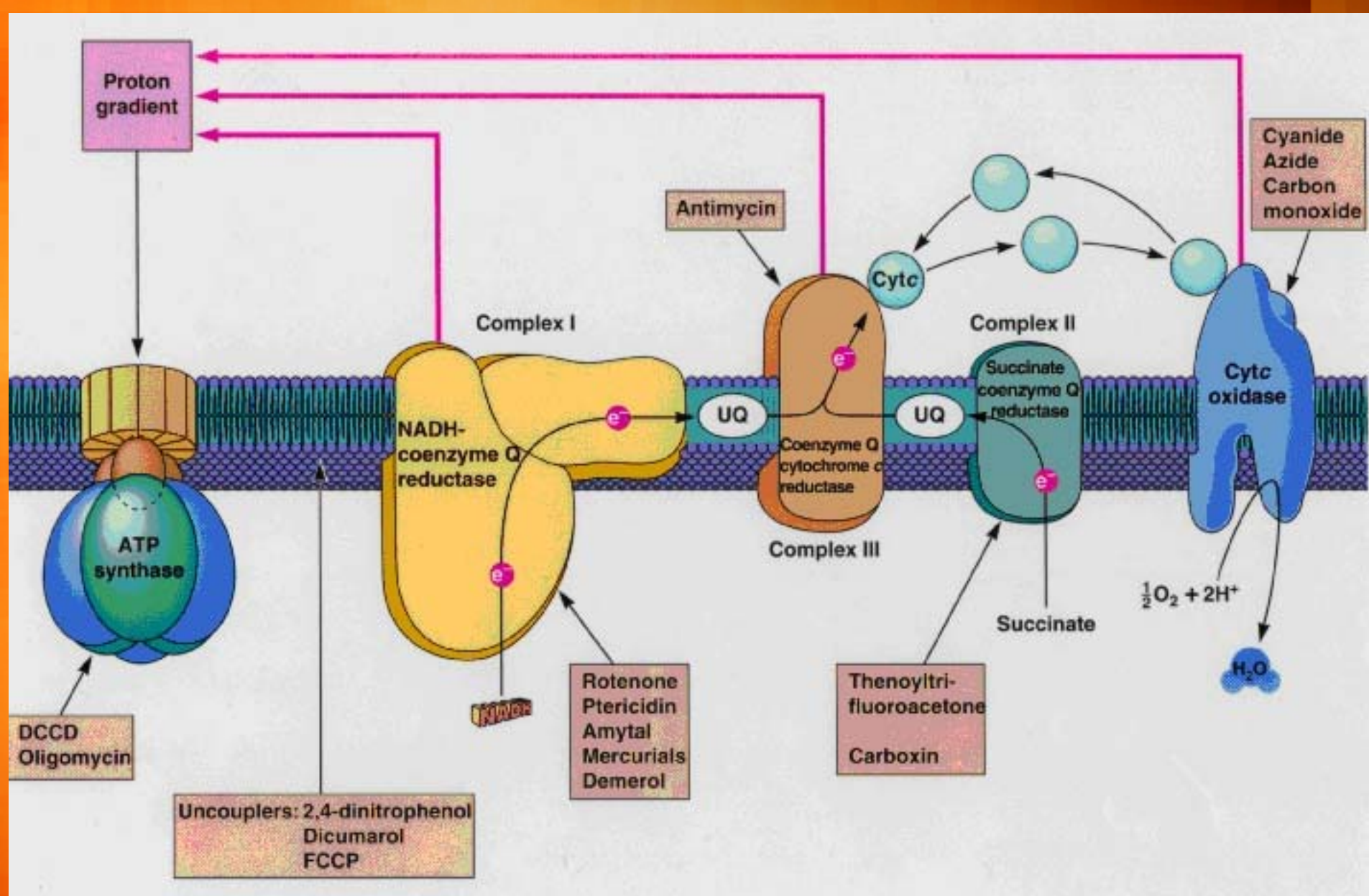
- Liberated from any burned substance that has carbon and nitrogen
 - Plastics, natural fibres, insulation, upholstery

STAND BACK



**I'M GOING TO TRY
SCIENCE**





Cyanide Toxicity

- Binds Fe^{3+} on Cytochrome Oxidase a3
- Stops oxidative phosphorylation
- Cells switch to anaerobic metabolism
 - Vomiting, abdominal pain
 - Confusion, headache, seizures
 - Hyper- and hypotension, tachycardia, CV collapse, AV block, Ventricular arrhythmias
 - Tachypnea
 - Flushing of skin, renal failure, hepatic necrosis

Cyanide Toxicity - Diagnosis

- Presumed present if significant burn or smoke inhalation
- Any base deficit, lactic acidosis, or anion gap in a burn/smoke inhalation victim
- VBG with abnormally high PO₂

Cyanide toxicity - Treatment

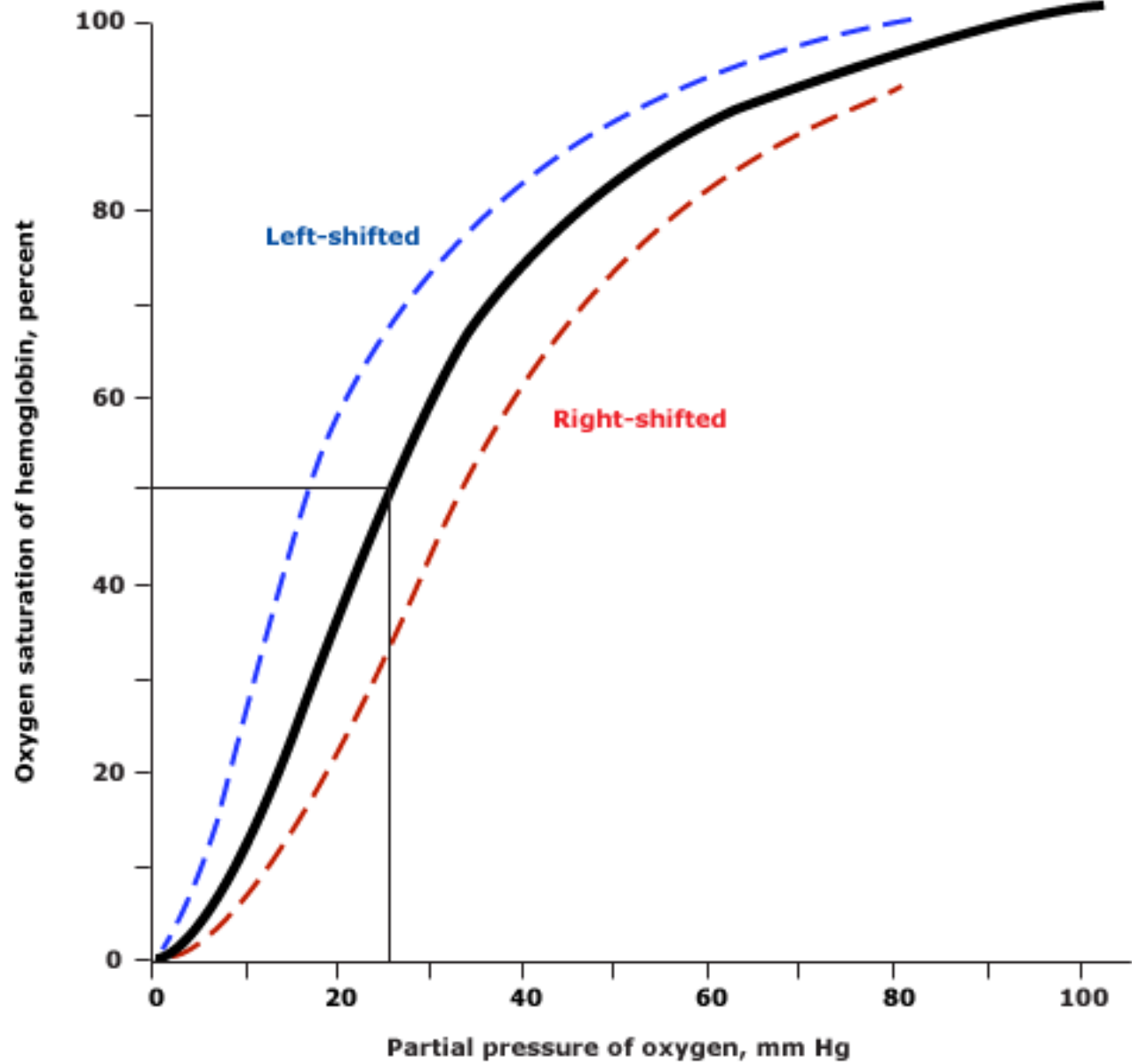
- Lilly Kit

→ Induction of methemoglobinemia

- Cyanide binds $\text{Fe}^{3+} \gg \text{Fe}^{2+}$
- Inducing methemoglobinemia creates a competitive site for the cytochrome binding site for CN^- - a cyanide “sink”
- CN-Hb is relatively nontoxic

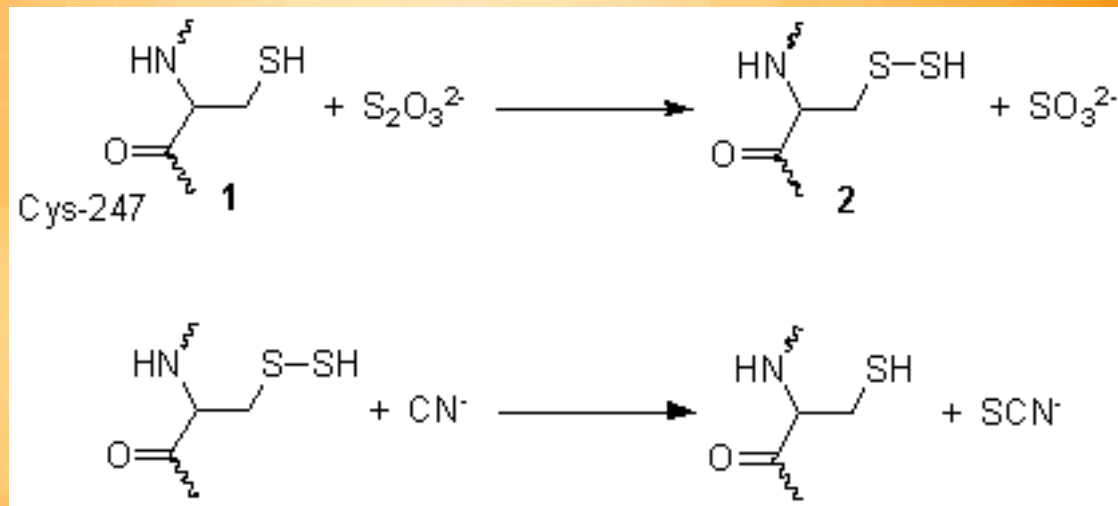
Cyanide Toxicity - MetHb

- Sodium nitrite
 - Given IV - dosed to achieve 10-30% MetHb
 - Causes left-shift of Oxygen-Hemoglobin dissociation curve
- Contraindicated in concomitant CO poisoning



Cyanide Toxicity - Sulfur Donation

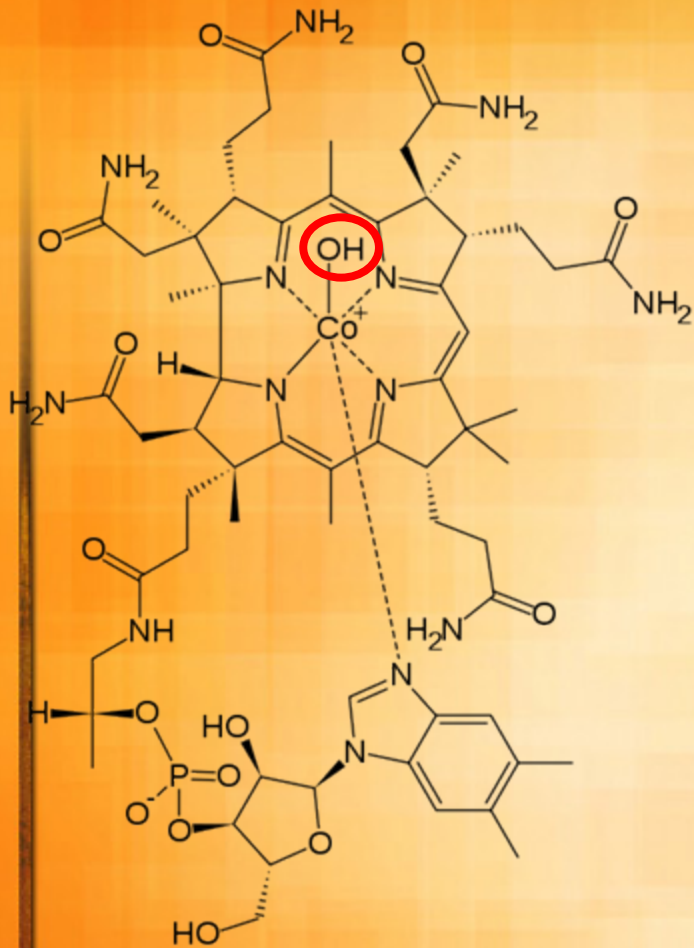
- Sulfur donor to allow natural elimination of cyanide by rhodanese



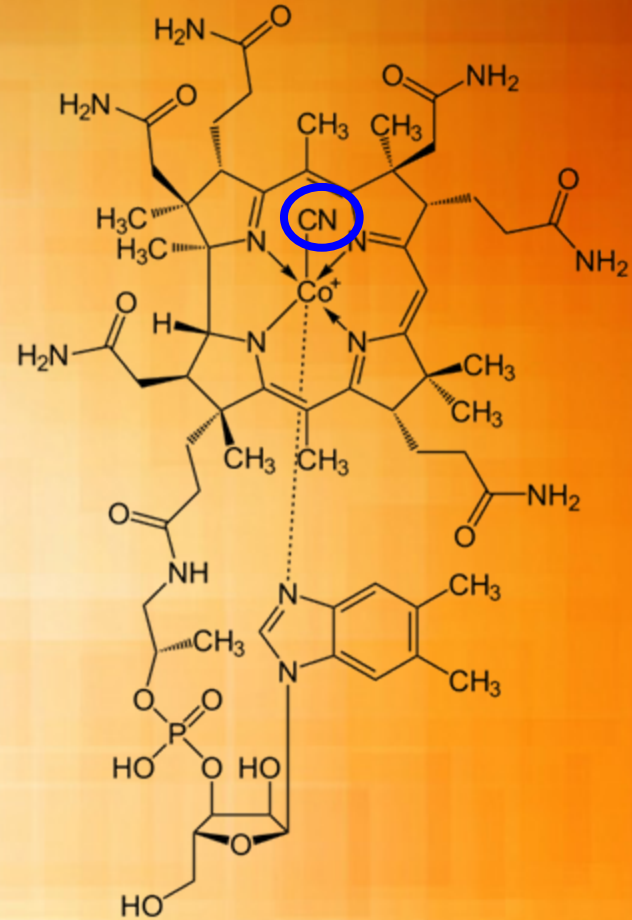
Cyanide Toxicity - Sulfur Donation

- Given via sodium thiosulfate
- Often in combination with inducing methemoglobinemia
 - Together increase lethal dose of cyanide 10-15x

Cyanide Toxicity - Treatment



Hydroxocobalamin



Cyanocobalamin

Cyanide Toxicity - Hydroxocobalamin

- Standard of care
- Rapid, safe, nontoxic
 - Side effects: Red skin, urine, mucous membranes
 - May interfere with co-oximetry
- May be used with thiosulfate

Conclusion

- Inhalational injury requires rapid diagnostics and interventions
- Must have high index of suspicion for toxic exposures
- Airway evaluation is critical
- Beware your sat probe!

References

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