# Carbon Monoxide & Cyanide Poisoning

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# Carbon Monoxide Poisoning

*Carbon Monoxide with Cyanide Intoxication*

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## Carbon Monoxide Poisoning
*…Objectives*

- Brief overview – clinical and physiological
- Brief review of the literature
- Provide a clinical focus for the clinician

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## Carbon Monoxide Poisoning
*…Background*

- One of the most common poisonings worldwide
- 50,000 annual exposures each year in the USA
- On average 3800 die/yr of CO poisoning
- Up to 2000 accidental deaths each year
- Up to 30% of the victims undiagnosed

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## Carbon Monoxide Poisoning
*…Physical Properties*

- See no evil
- Smell no evil
- Taste no evil
- Non-irritating
- Almost everywhere
- CO is produced by incomplete combustion of carbon containing compounds

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## Carbon Monoxide Poisoning
*…Sources of CO*

- Fires
- Automotive exhausts
- Faulty home heating and cooking systems
- Portable generators
- Propane engines
- Charcoal burners and camp stoves
- Paint strippers/solvents containing Methylene chloride

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## Carbon Monoxide Poisoning
*…Endogenous Sources of CO*

- Hemoglobin metabolism 1% or less
- Rapid red cell turn over may increase levels
- Hepatic microsomal enzyme induction may increase levels

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## Carbon Monoxide Poisoning
*…Cigarette Smoking*

- CO levels 4-5% per pack per day
- Second hand smoke increases CO levels.
- CO levels >10% in smokers is likely secondary to exogenous CO exposure.

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## Carbon Monoxide Poisoning
*…Carboxyhemoglobin Levels*

- Elevated COHgb is only an indicator of poisoning
- COHgb does not correlate well with SYMPTOMS or OUTCOMES
- Great mimicker with diverse presentations

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## Carbon Monoxide Poisoning
*…Clinical Symptoms*

- Carboxyhemoglobin Level
- Less than 10% — minimal to no symptoms
- 10 - 19% — headache, tightness across forehead
- 20 - 29% — throbbing temporal headache

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## Carbon Monoxide Poisoning
*…Clinical Signs and Symptoms*

- Carboxyhemoglobin Level
- 30 - 39% — vomiting, weakness, dizziness, visual changes
- 40 - 49% — syncope, tachycardia
- 50 - 59% — coma, convulsions
- > 60% — cardio-respiratory failure

*- Sayers and Davenport, 1930*

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## Carbon Monoxide Poisoning
*…Comorbidities*

- Preexisting coronary artery disease
- Low levels of COHgb increases myocardial ischemia during graded exercise testing
- At COHgb levels of 15% risk of MI is increased
- At COHgb levels of 9% lower the threshold for ventricular fibrillation
- Increased incidence of intermittent claudication

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## Carbon Monoxide Poisoning
*…Comorbidities*

- Preexisting obstructive lung disease
- Low levels of COHgb may cause decompensation of chronic obstructive pulmonary disease

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## Carbon Monoxide Poisoning
*…Physical Findings*

- Tachycardia and tachypnea are most common
- Hypotension or hypertension
- Arrhythmias

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## Carbon Monoxide Poisoning
*…Physical Findings*

- Dermatologic Findings
  - Extremely rare to find cherry red color
  - This represents true soaking of tissues
  - Bullous and vesicular skin lesions

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## Carbon Monoxide Poisoning
*…Physical Findings / Symptoms*

- Ophthalmologic findings
  - Retinal hemorrhages
  - Papilledema
  - Blindness
  - Hemianopsia
  - Scotomata

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## Carbon Monoxide Poisoning
*…Physical Findings / Symptoms*

- Neurological findings secondary to labyrinth, eight nerve, and brainstem nuclei lesions
  - Loss of balance
  - Ataxia
  - Nystagmus
  - Hearing loss
  - Tinnitus

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## Carbon Monoxide Poisoning
*…Multisystem Organ Failure*

- Other findings
  - Rhabdomyolysis
  - Renal failure

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## Carbon Monoxide Poisoning
*…Neuropsychiatric Presentation*

- Non focal changes in mental status
- Seizures
- Incontinence
- Amnesia
- Apraxia
- Parkinsonism
- Cortical blindness
- Agnosia
- Peripheral neuropathy

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## Carbon Monoxide Poisoning
*…Neuropsychiatric Presentation*

- Long term personality deterioration and memory impairment
- Directly related to the level of consciousness on admission

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## Carbon Monoxide Poisoning
*…Neuropsychiatric Testing*

- Why do it?
  - CNS morbidity is often subtle
  - May help in in initial stratification
  - Used pre- and post-HBO to document improvement

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## Carbon Monoxide Poisoning
*…MIEMSS Neuropsychometric Testing*

- Timed test (14 minutes)
- General orientation
- Trail making
- Digit symbol
- Aphasia screening
- Block design

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## Carbon Monoxide Poisoning
*…Delayed Neurologic Syndrome (DNS)*

- Clinical decline 1-21 days after CO poisoning
- Incidence 3-47%
- Personality changes may be subtle and may be missed on routine clinical exam

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## Carbon Monoxide Poisoning
*…Common Manifestations of DNS*

- Intellectual deterioration
- Personality changes
- Incontinence
- Tremor
- Cerebellar signs
- Speech impairment

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## Carbon Monoxide Poisoning
*…Poisoning in Children*

- Lethargy and syncope are the most common symptoms
- Gastrointestinal disturbances may occur early
- Toxicity may occur at lower COHgb levels

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## Carbon Monoxide Poisoning
*…Fetal Toxicity*

- Fetal Hgb has a higher affinity for CO
- Fetal COHgb levels may be 10-15% higher than maternal levels
- Diffusion increases with placental size
- At any given level of elevated COHgb, the fetus is at higher risk of toxicity than mother

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## Carbon Monoxide Poisoning
*…Mechanisms of Toxicity*

- CO binds readily with hemoglobin to form carboxyhemoglobin (COHgb)
- Affinity for hemoglobin 200-250 times greater than oxygen

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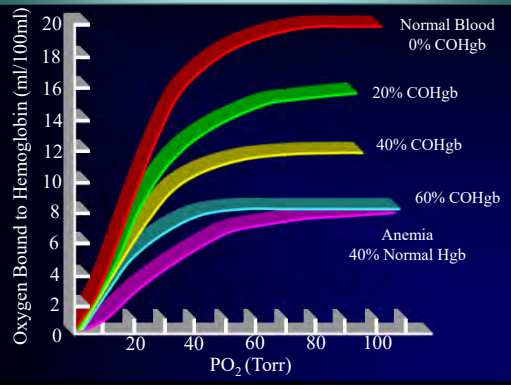
## Hemoglobin Binding

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## Carbon Monoxide Poisoning
*…Mechanisms of Toxicity*

- CO increases the affinity for oxygen of the unoccupied sites on hemoglobin
- Shifts oxygen dissociation curve to the left

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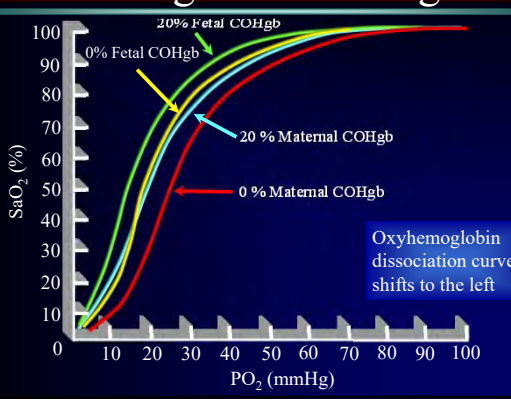
## Hemoglobin Binding

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## Carbon Monoxide Poisoning
*…Mechanisms of Toxicity*

- CO binding to intracellular proteins
  - Compounds containing heme or copper
  - Hemoproteins such as MYOGLOBIN, GUANYLYL CYCLASE, CYTOCHROME P-450 and CYTOCHROME C oxidase
  - 15% of total body CO is found in extravascular tissue

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## Carbon Monoxide Poisoning
*…Mechanisms of Toxicity*

- Neuropathological features
  - Appear to relate to oxidative stress and mitochondrial dysfunction
  - Vascular insults coincide or precede neurological deficits

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## Carbon Monoxide Poisoning
*…Mechanisms of Toxicity*

- Neuropathological features continued…
  - CO enhances release of nitric oxide
  - NO interacts with superoxide to form peroxynitrite
  - Peroxynitrite is a potent oxidizing and nitrating agent

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## Carbon Monoxide Poisoning
*…Mechanisms of Toxicity*

- Neuropathological features continued…
  - Nitric oxide causes changes in the endothelium
  - If there is a decrease in blood flow platelets adhere to the endothelium
  - $\beta_2$ integrin molecules activate these platelets

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## Carbon Monoxide Poisoning
*…Mechanisms of Toxicity*

- Neuropathological features continued…
  - Activated platelets liberate proteases and convert xanthine dehydrogenase to xanthine oxidase
  - Xanthine oxidase is involved in subsequent brain lipid peroxidation

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## Carbon Monoxide Poisoning
*…Mechanisms of Toxicity*

- Neuropathological features
  - Reactive oxygen species are formed by activated leukocytes
  - CO also elevates monoamine neurotransmitters. Their breakdown forms more reactive oxygen species

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## Carbon Monoxide Poisoning
*…Mechanisms of Toxicity*

- Neuropathological features continued…
  - Brain injury in CO poisoning and post anoxic encephalopathy is very similar
  - Fatal CO poisoning produces lesions in basal ganglia in 70% of cases

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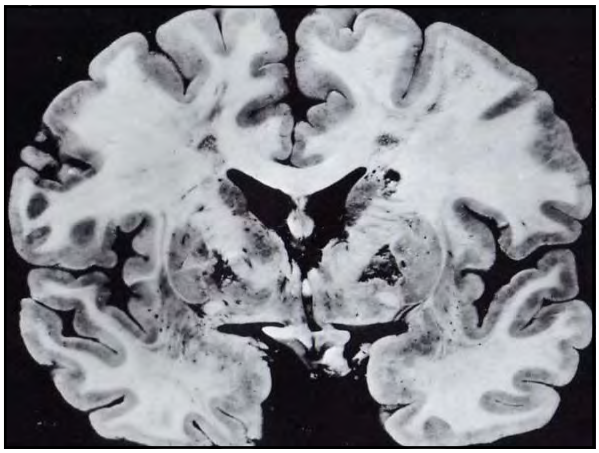

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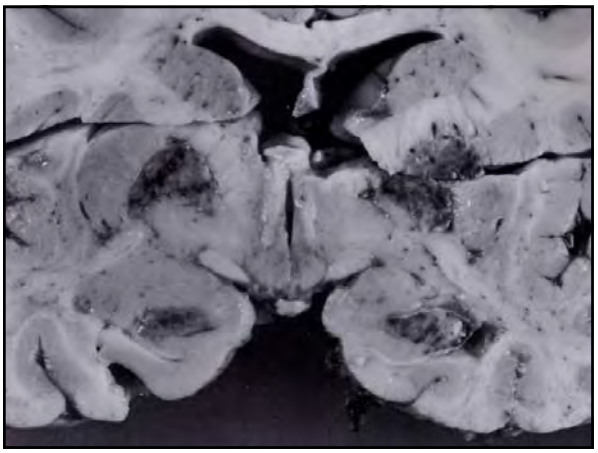

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## Carbon Monoxide Poisoning

*…Diagnosis*

- Maintain a high index of suspicion especially with similar simultaneous presentation of flu like illness in family members

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## Carbon Monoxide Poisoning

*…Diagnosis*

- Carboxy Hemoglobin Levels
  - Best used as an indicator of CO exposure
  - Arterial or venous blood levels are similar
  - Use of oxygen prior to blood draw will lower the COHgb level

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## Carbon Monoxide Poisoning

*…Diagnosis*

- Once CO poisoning has been detected, determine its effect on the patient
  - CXR, EKG, CBC, glucose, electrolytes, BUN, Creatinine
  - Cardiac enzymes
  - ABG, lactate levels, drug screen, alcohol levels
  - Serum, urine HCG
  - Cyanide and thiocyanate level in suspected smoke inhalation

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## Carbon Monoxide Poisoning

*…Diagnosis*

- Hyperglycemia may indicate severe multisystem organ and muscles involvement
- Lactate levels of 10mmol/l or more may indicate co-existent cyanide poisoning

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## Carbon Monoxide Poisoning
*…Patient Management*

- A B C's
- Stabilize hemodynamics
- Neurological assessment
- Stratify patients by severity of poisoning and identify high risk patients
- Administer oxygen (100% via NRB)

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## Carbon Monoxide Poisoning
*…Risk Stratification*

- High risk characteristics
  - COHgb levels 25% or more
  - COHgb 10% or more in pregnant women
  - COHgb levels 15% or more with H/O CAD
  - Angina or ischemia on EKG
  - Acidosis
  - Abnormal psychometric testing
  - Loss of Consciousness
  - Symptomatic after 4 hours of oxygen (1 ATA)
  - CO poisoning in a facility that lacks CO testing

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## Carbon Monoxide Poisoning
*…Treatment*

- Oxygen supplementation
  - Cornerstone of treatment
  - Provides greater tissue oxygenation
  - Enhances dissociation of CO from tissues
  - Enhances dissociation from Hgb

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## Carbon Monoxide Poisoning
*…Treatment*

- Carboxyhemoglobin Half-life
  - Air 300 min
  - 100% O2 60 min
  - HBO (3.0 ATA) 23 min

∗ *Fetal COHgb $T^{1/2}$ is 1.5 times that of adult*

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## Carbon Monoxide Poisoning
*…Exclusive Effects of HBO*

- Additional beneficial effects of HBO over normobaric oxygen have been noted in animal models
  - Improvement in mitochondrial oxidative processes
  - Inhibition of lipid peroxidation
  - Inhibition of leukocyte adherence to endothelium
  - Lower incidence of neurologic sequelae
  - Lower mortality

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## Carbon Monoxide Poisoning
*…Treatment*

- Patients treated with the shortest delay have a better outcome
- More than one hyperbaric treatment may be better in select patients
- There is no consensus as to the length of delay from poisoning beyond which therapy is not beneficial

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## Carbon Monoxide Poisoning
*…Clinical Trials – Evolution of the Evidence*

- Raphael (1989) — No benefit
- Ducasse' (1995) — Positive effect
- Thom (1995) — Positive effect
- Scheinkestel (1999) — No benefit
- Weaver (2002) — Positive effect
- Huang (2017) — Positive effect

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## Carbon Monoxide Poisoning
*…Clinical Trials – Raphael (1989)*

- 343 patients receiving either 6 hours of normobaric oxygen or 2 hours of HBO at 2.0 ATA plus 4 hours of normobaric oxygen
- Second arm - 286 patients receiving either one or two sessions of HBO at 2.0 ATA

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## Carbon Monoxide Poisoning
*…Clinical Trials – Raphael (1989)*

- Found no difference between normobaric oxygen and HBO
- Excluded patients with LOC
- Used lower pressure HBO (2.0 ATA)
- Over half of pts were treated more than 6 hrs after they were discovered
- No formal psychometric testing
- Served as a pivotal point for further testing

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## Carbon Monoxide Poisoning
*…Clinical Trials – Ducasse'* (1995)

- Non-comatose patients with acute CO poisoning
- 26 patients receiving either 100% oxygen for 6 hours followed by 50% oxygen or HBO at 2.5 ATA for 2 hours followed by normobaric oxygen for 4 hours

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## Carbon Monoxide Poisoning
*…Clinical Trials – Ducasse'* (1995)

- Positive HBO effect (mild to moderate poisoning)
- Not blinded
- Small sample size

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## Carbon Monoxide Poisoning
*…Clinical Trials – Thom* (1995)

- Study of delayed neuropsychological sequelae after CO poisoning
- 60 patients randomized to either 30 mins of HBO (2.8 ATA) followed by 90 mins of HBO (2.0 ATA) vs. normobaric oxygen until resolution of symptoms

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## Carbon Monoxide Poisoning
*…Clinical Trials – Thom* (1995)

- Positive HBO effect (mild to moderate poisoning)
- 7/30 (23%) of patients treated with 100% O2 developed delayed neurological sequelae (DNS)
- No patients treated with HBO developed DNS

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## Carbon Monoxide Poisoning
*…Clinical Trials – Scheinkestel* (1999)

- Randomised controlled trial of HBO vs. normobaric oxygen for acute CO poisoning
- 191 patients randomized to daily HBO (2.8 ATA) for 60 mins plus high flow oxygen therapy between HBO treatments vs. high flow normobaric oxygen (100%) for 3-6 days

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## Carbon Monoxide Poisoning
*…Clinical Trials – Scheinkestel* (1999)

- No benefit of HBO
- Poor one month follow-up (46%)
- 69% of patients were suicide attempts, half the patients having ingested alcohol and other drugs
- Cluster randomization
- Differences in CO exposure and types

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## Carbon Monoxide Poisoning
*…Clinical Trials – Weaver* (2002)

- Randomized controlled trial
- Best study published to date
- 76 patients in each arm
- Study stopped after third interim analysis

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## Carbon Monoxide Poisoning
*…Clinical Trials – Weaver* (2002)

- Randomized symptomatic CO poisoning patients
- Three chamber sessions within a 24 hour period
- Received either 3 hyperbaric treatment sessions or one normobaric oxygen session followed by 2 normobaric room air (sham) sessions

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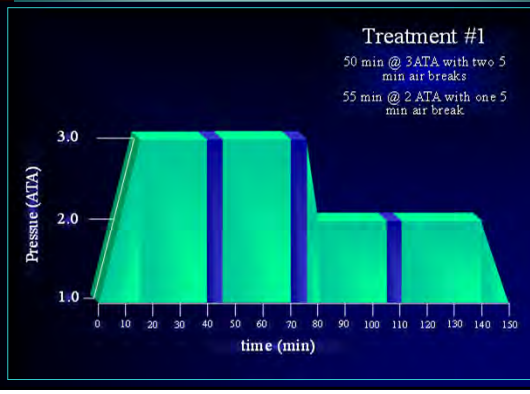
## Weaver Protocol

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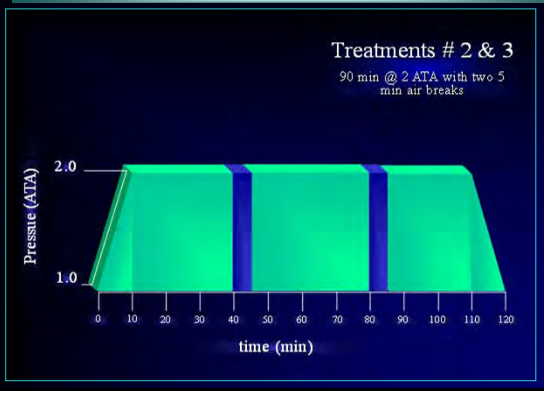
## Weaver Protocol

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## Carbon Monoxide Poisoning

*…Clinical Trials – Weaver* (2002)

- Double blind, randomized, controlled study
- Positive HBO effect
- Cognitive sequelae at 6 weeks
  - HBO group 25%
  - NBO group 46%
- The difference in improvement remained at 12 month follow up
- Presence of cerebellar dysfunction before

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## Carbon Monoxide Poisoning

*…Multivariable Analysis – Weaver* (2007)

- Age > 36 years
- Exposure intervals ≥ 24 hours

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## Carbon Monoxide Poisoning

*…Retrospectivve Population-based Cohort Study – Huang* (*Chest*, 2017)

- 25,737 CO patients
- 7,278 received HBO
- Stratified by:
  - Age
  - Comorbidities
  - Suicide attempt
  - Acute respiratory failure
  - Gender
  - Monthly income
  - Drug Poisoning
  - # of HBO treatments

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## Carbon Monoxide Poisoning

*…Findings – Huang* (*Chest*, 2017)

- Mortality highest in 1st month
- Mortality remained elevated for the 1st year
- Overall mortality decreased in HBO group
- Remained lower for a period of 4 years after HBO treatment.
- Two or more txs associated with better survival.

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## Carbon Monoxide Poisoning

*…Independent Mortality Predictors – Huang* (Chest, 2017)

- Age
- Male
- Lower income
- Diabetes Mellitus
- Malignancy
- Alcoholism
- Mental disorders
- Suicide attempts
- Acute respiratory failure

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## Carbon Monoxide Poisoning
*…Who Gets HBO*

- Patients with significant comorbidities
- COHgb levels of 25% or more
- Cerebellar findings on exam
- Loss of consciousness
- Long exposure times
- Older patients
- Respiratory failure
- ? All pregnant patients

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## Carbon Monoxide Poisoning
*…HBO in Children*

- Treatment can be more difficult due to age and anxiety
- An adult attendant may be necessary
- Wattel, F, etal. (2013) 412 infants evaluated on 8th day, 1st year, and 6th year. No differences in psychomotor development in HBO vs. non-HBO group.
- No contraindication to treating children with HBO

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## Carbon Monoxide Poisoning
*…Pregnancy and HBO – Case Presentation*

- 17 y/o healthy female at 37 wks gestation
- Arrived at ER obtunded after 3 hr ride in back seat of a car
- Later became arousable with complaints of chest pain, HA, and nausea with vomiting

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## Carbon Monoxide Poisoning
*…Pregnancy and HBO – Case continued*

- Vital Signs:
  - Pulse: 110 regular
  - BP: 100/60
  - Resp: 24/min
- Physical Exam:
  - Unremarkable except neuro; orientation only to person and time

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## Carbon Monoxide Poisoning
*…Pregnancy and HBO – Case continued*

- Laboratory studies:
- Hgb 15.2  Hct 42
- ABG's:
  - pH 7.34
  - PaO2 229 (10 L)
  - PaCO2 29
  - SaO2 52% (by co-oximeter)
  - COHgb 47.2%

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## Carbon Monoxide Poisoning
*…Pregnancy and HBO – Case continued*

- Fetal heart tones present by doppler
- FHR monitor showed 170 BPM with decreased beat to beat variability and symptomatic uterine contractions every 6 min

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## Carbon Monoxide Poisoning
*…Pregnancy and HBO – Case continued*

- Treated with HBO due to neurological symptoms and fetal distress
- HBO @ 2.4 ATA for 90 minutes began approx 2 hrs after admission to ED
- 10 min after beginning tx:
  - FHR 130 BPM
  - Mother's HA, chest pain, and uterine contractions ceased
  - Maternal COHgb at end of Tx 2.4%

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## Carbon Monoxide Poisoning
*…Fetal Effects*

- Neurologic dysfunction
- Decreased birth weight
- Increased fetal death
- An accurate assessment of fetal risk at any elevated level of COHb is not possible

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## Carbon Monoxide Poisoning
*…Concerns: Fetal Effects of HBO*

- Teratogenicity
- Retinopathy of prematurity
- Alterations of fetal blood flow
- Premature closure of the ductus arteriosus

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## Carbon Monoxide Poisoning
*…Previous Studies and Case Reports*

| Author | Subject | $O_2$ Exposure | Outcome |
|---|---|---|---|
| Ferm | Hamster | 3.6 ATA x 3h<br>4.0 ATA x 2h | Increased fetal anomaly rate |
| Fujikura | Rabitt | 1.2 ATA x 15h | Increased RON |
| Telford | Rat | 2.0 ATA x 6h | Increased fetal resorption |
| Miller | Rat | 3.0 ATA x 6h | Increased cardiovascular defects |
| Ferm | Hamster | 3.0 ATA x 3h | No malformations |
| Cho, Yun | Rat | 3.0 ATA x 20min | Decreased fetal resorption |
| Gillman | Hamster | USN Table 6 | No differences from untreated controls |
| Hollander | Human | 3.0 ATA x 46min | Healthy Newborn |

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## Carbon Monoxide Poisoning
*…Concerns: Fetal Effects of HBO*

- Retrospective review of HBO for CO poisoning in pregnancy.
- 32 patients 8-35 (mean 23) weeks gestation
- Mean COHgb 6.9-40.2% (mean 24%)
- Followed up to 6 months post-partum

Clarke, D. *Am J Emerg Med (2021)*

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## Carbon Monoxide Poisoning
*…Concerns: Fetal Effects of HBO*

- No spontaneous abortions, congenital abnormalities, or developmental problems.
- 4 premature births
- 2 post-partum deaths not related to CO
  - Premature twin with NEC
  - Cyanotic heart disease dx prior to CO exposure

Clarke, D. *Am J Emerg Med (2021)*

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## Carbon Monoxide Poisoning
*…HBO Recommendations in Pregnancy*

- It is our practice to treat every pregnant patient with CO poisoning in a hyperbaric chamber provided no other contraindications exist
- One chamber session is suggested with the need for further therapy based upon patient assessment

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## Carbon Monoxide Poisoning
*…Summary of HBO Benefits*

- Enhances elimination of CO
- Inhibits lipid peroxidation
- Attenuates CNS inflammation by inhibiting CO adduct formation with myelin basic protein
- Impairs leukocyte adhesion to vascular endothelium
- Improves mitochondrial function

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## Carbon Monoxide Poisoning
*…Level of Evidence - Based on AHA classification*

- Class I – Conditions for which there is evidence, general agreement, or both that a given procedure or treatment is useful and effective.
- Based on Level B-R evidence
  - One high-quality randomized trial
  - Two moderate quality randomized trials
  - Supportive meta-analysis
  - Significant animal research

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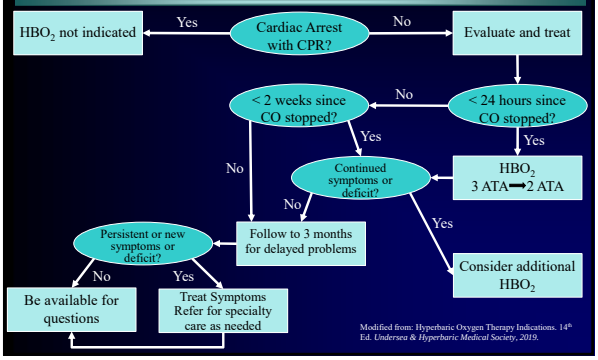
## Carbon Monoxide Poisoning
*…Treatment Flowchart*

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## Carbon Monoxide Poisoning
*…Utilization Review*

- Patients should receive at least one treatment of 2.5-3.0 ATA
- Further treatments for patients failing to improve
- Subsequent treatments are given 6-8 hours later up to 2 daily treatments
- Utilization review is mandatory after 5 sessions

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## Carbon Monoxide Poisoning
*…Questions Remain*

- Objective laboratory tests?
- Optimal number of treatments
- Interval after poisoning after which therapy is not beneficial
- Optimal pressure
- Risk of neurologic sequelae in patients not meeting high risk category - would HBO benefit them?

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## Carbon Monoxide Poisoning
*…Complicated by Cyanide Poisoning*

- Cyanide and CO poisoning frequently occur simultaneously
- Combination exhibits synergistic toxicity
- Inhaling extremely small amounts of cyanide gas can cause rapid death (100mg of gas or approximately 80ml)

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## Carbon Monoxide Poisoning
*…Complicated by Cyanide Poisoning*

- Cyanide causes intracellular hypoxia
- Affects the electron transport chain
- Cyanide ion binds to ferric ion of mitochondrial cytochrome oxidase
- Specifically attacks cytochrome $A_3$

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## Cyanide Poisoning
*…Sources*

- Residential and industrial fires
- Metal trades, mining, jewelry manufacture, electroplating, radiograph recovery
- Ship and warehouse fumigation
- Nitriles used as solvents and in manufacture of plastics. Nitriles may release HCN (hydrogen cyanide) during burning or metabolism

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## Cyanide Poisoning
*…Sources*

- A number of synthesized (polyurethane) and natural (wool, silk) compounds produce HCN when burned
- Cyanogens - compounds that release cyanide when metabolized (sodium nitroprusside)

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## Cyanide Poisoning
*…Sources*

- Furniture and bedding (polyurethane)
- Asphalt
- Paper
- Lighting, baths (acrylic)
- Carpeting (nylon)
- Synthetic rubber
- Moulding
- Insulation (melamine resins)
- Plastic household materials (polystyrene)
- PVC pipe

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## Cyanide Poisoning
*…Clinical Manifestations*

- Depends on amount of cyanide absorbed
- Rapidity of symptom onset depends upon type of exposure
  - Gas (most rapid)
  - Soluble salt
  - Insoluble salt
  - Cyanogens (least rapid)

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## Cyanide Poisoning
*…Clinical Symptoms*

- General weakness, malaise
- Giddiness, inebriation, confusion, headache, vertigo, coma, generalized seizures
- Shortness of breath, chest pain

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## Cyanide Poisoning
*…Physical Findings*

- Skin color may remain pink
- Tachycardia, bradycardia, and hypotension
- Tachypnea, apnea
- Bright red retinal veins and arteries
- Bitter almond breath

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## Cyanide Poisoning
*…Clinical Manifestations – Late Signs*

- Mydriasis
- Hypotension
- Apnea
- Non-cardiogenic pulmonary edema
- Cardiac dysrhythmias
- Asystole

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## Cyanide Poisoning
*…Laboratory Findings*

- Cyanide blood concentrations are generally not available in time
- Reduced A-V oxygen saturation difference (less than 10)
- Metabolic acidosis
- Pulse oximetry can be misleading

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## Cyanide Poisoning
*…Laboratory Findings -- continued*

- Lactate level of 10 mmol/l or more in suspected cyanide inhalation strongly suggests high levels of blood cyanide
- Blood cyanide measurements taken after sodium nitrite administration may show an artificial increase

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## Cyanide Poisoning
*…Laboratory Findings -- continued*

- Blood Cyanide Levels
  - Normal level less than 0.1 mg/l
  - Level greater than 0.2 mg/l toxic
  - Lethal level usually considered to be 1 mg/l

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## Cyanide Poisoning
*…Treatment*

- Treatment is initiated based upon clinical suspicion and not cyanide level
- Oxygen
- Hemodynamic support
- Gastric lavage and charcoal for ingestion
- Antidote kit

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## Cyanide Antidote Kit


5 g vial of lyophilized hydroxocobalamin

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## Cyanide Poisoning
*…Treatment*

- Administer a compound that has more affinity for cyanide than the ferrous ion
- Hydroxocobalamin (Vit B12a) binds with CN to form cyanocobolamin (Vit B12) which is excreted in the urine.
- Cytochrome oxidase is freed to function

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## Cyanide Poisoning
*…Treatment – Hyperbaric Oxygen*

- Hyperbaric oxygen is known to be helpful adjunctive therapy - how?

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## Cyanide Poisoning
*…Treatment – Hyperbaric Oxygen*

- With use of HBO a constant electron flux can be maintained through a process known as “branching” or “cushioning”.
- Multiple cytochrome chains are able to transfer electrons to whatever few available cytochrome molecules may exist, despite the presence of an inhibitor

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## Cyanide Poisoning
*…Hyperbaric Oxygen – Animal Study (Skene 1966)*

- Room air — 96% mortality
- 100% oxygen — 50% mortality
- HBO @ 2 ATA — 20% mortality

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## Cyanide Poisoning
*…Treatment – Hyperbaric Oxygen*

- HBO may have a direct effect on cyanide toxicity reduction
- May augment the beneficial effect of antidote treatment

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## Cyanide Poisoning
*…Hyperbaric Oxygen – Animal Study (Takano 1980)*

- HBO @ 2 ATA will maintain mitochondrial electron transport despite a lethal dose of cyanide
- $O_2$ appeared to have an antidotal effect when CN is at the critical or just above the critical level

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## Cyanide Poisoning
*…UHMS Committee Report (2019)*

Evidence-Based Review of HBO for Cyanide Poisoning

"At this time, the evidence does not support HBO for pure cyanide poisoning, but HBO is indicated for acute CO poisoning including mixed poisoning. Clinically HBO has been widely applied for CO poisoning complicated by cyanide."

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## Cyanide Poisoning
*…Case Report – Davis and Ewer (1988)*

- 22 yo male observed to drink ½ of a wineglass of a mixture admitted to contain cyanide. Arrived by ambulance in ER 45 min later.
- On arrival deeply unconscious (GCS=5) with fixed dilated pupils, hypertonia, hyperreflexia, tachypnea, and tachycardia BP 160/80

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## Cyanide Poisoning
*…Case Report – Davis and Ewer (1988)*

- Treatment:
  - (2) 0.3 ml amyl nitrate ampoules given by inhalation
  - 100% O2 by anesthesia circuit
  - 300 mg dicobalt EDTA
  - 10 ml sodium nitrite 3%
  - 50 ml sodium thiosulfate 25%
  - 50 ml D50W
  - Gastric lavage and amorphous carbon instilled into stomach

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## Cyanide Poisoning

*…Case Report – Davis and Ewer (1988)*

- Treatment continued…
  - Little improvement over 1 hr
  - Transferred to HBO facility
  - Additional 300mg dicobalt EDTA given during transport
  - Arrived at HBO facility 2 hr 45 min after ingestion (remained unconscious, hypertonic, hyperreflexic, but had reactive pupils and spontaneous head and limb movements)

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## Cyanide Poisoning

*…Case Report – Davis and Ewer (1988)*

- Treatment continued…
  - Bilateral myringotomies performed
  - Compressed to 2.8 ATA on 100% O2 by Scott mask 3 hr 10 min post ingestion
  - Within 10 -15 minutes the pt became fully conscious but agitated complaining of eye and tooth pain
  - Oriented X 3 when he emerged from chamber after 70 min of HBO
  - Uneventful recovery

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