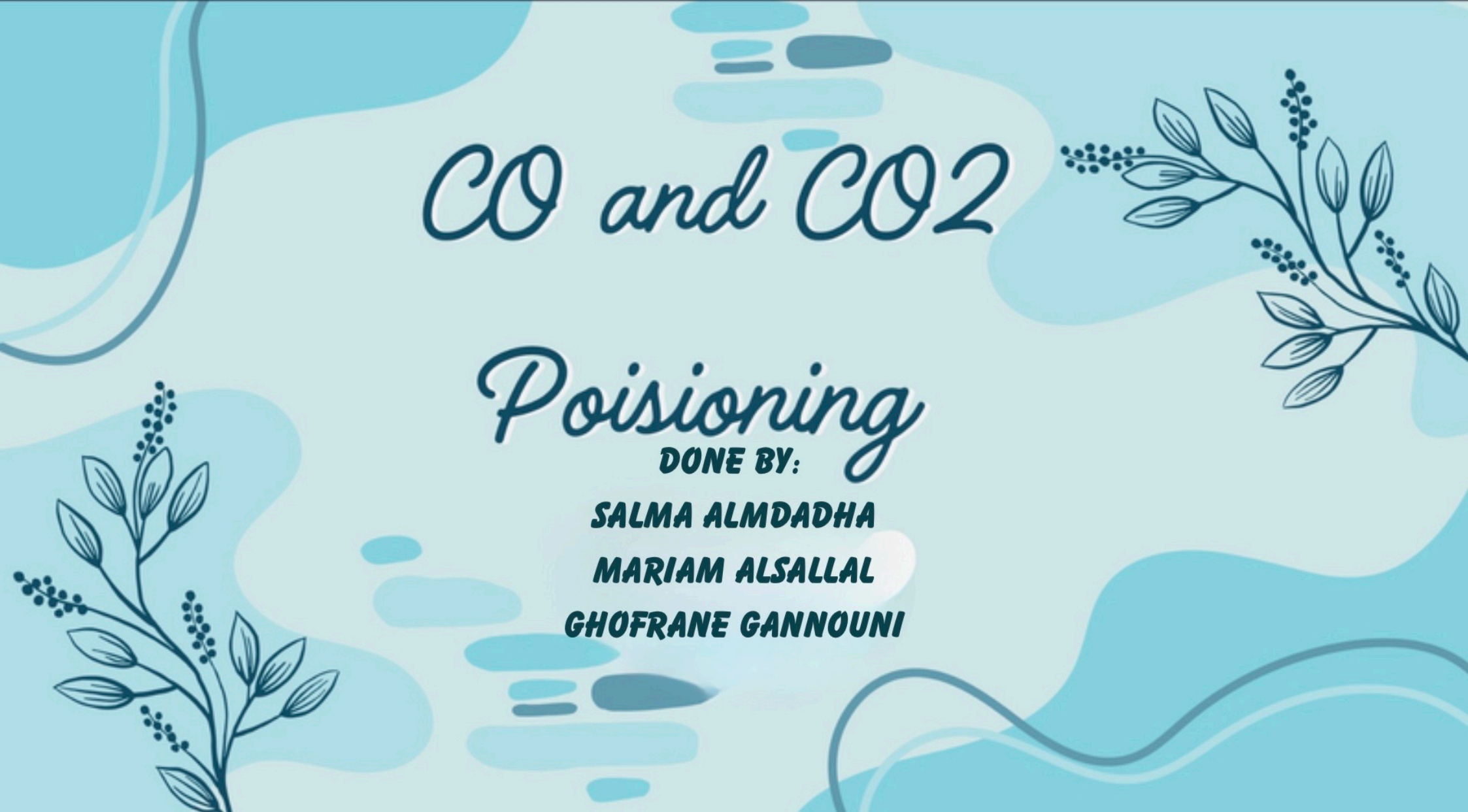




CO and CO2

Poisoning

DONE BY:

SALMA ALMDADHA

MARIAM ALSALLAL

GHOFRANE GANNOUNI

CO POISONING

- INTRODUCTION MECHANISM OF ACTION
- TOXICOLOGY INTERPRETATION
- ACUTE & CHRONIC POISONING CO POISONING IN PREGNANCY
- DIAGNOSIS
- TREATMENT POISONING ATOPSY FINDINGS





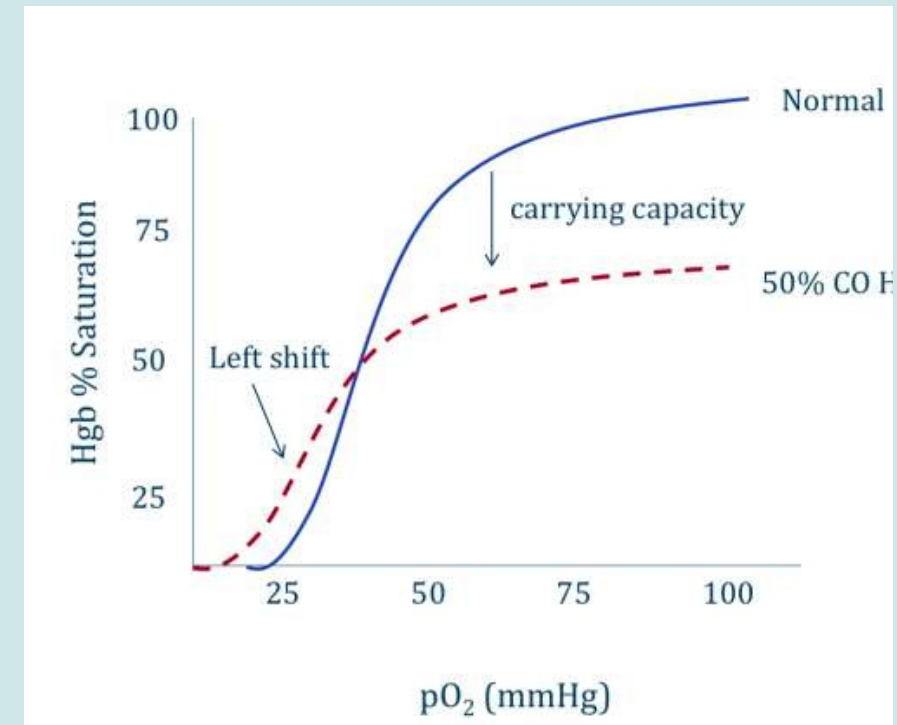
- CO is a systemic asphyxiant that is an important cause of accidental and suicidal death. Carbon monoxide (CO) is an odorless, tasteless, colorless, nonirritating gas formed by hydrocarbon combustion
- CO is short-lived in the atmosphere, being rapidly oxidized to CO₂; thus elevated levels in ambient air are transient and occur only in close proximity to sources of CO
- The atmospheric concentration of CO is generally below 0.001 percent, but it may be higher in urban areas or enclosed environments
- CO poisoning is also known as the silent killer
- CO is produced from incomplete combustion of fuels
- Potential sources of CO include :
 1. Smoke inhalation from fires
 2. Poorly functioning heating systems.
 3. Improperly vented fuel-burning devices (kerosene heaters, charcoal grills, camping stoves)
 4. Motor vehicles operating in poorly ventilated areas. (ice rinks, warehouses, parking garages, keyless cars left running, tailpipe blocked by snow drift).
 5. Inhaled or ingested methylene chloride (dichloromethane), an industrial solvent and a component of paint remover, is hepatically metabolized to CO and can cause CO toxicity in the absence of ambient CO exposure to burning materials (gasoline, wood, natural gas) and by entrapment in fires and smoke inhalation

MECHANISM OF ACTION

THE HEMOGLOBIN

- is a tetramer with four oxygen binding sites. Carbon monoxide binds to one of these sites with approximately 230–270 times the affinity of oxygen forming carboxyhemoglobin (COHb).
 - The binding of carbon monoxide at one of these sites increases the oxygen affinity of the remaining three sites, which causes the hemoglobin molecule to retain oxygen that would otherwise be delivered to the tissue.
 - This situation is described as carbon monoxide shifting the oxygen dissociation curve to the left. Because of the increased affinity hemoglobin and oxygen during carbon monoxide poisoning, the blood oxygen content is increased. But because all the oxygen stays in the hemoglobin, none is delivered to the tissues. This causes hypoxic tissue injury.
 - Carboxyhemoglobin causes cellular hypoxia by binding reversibly to hemoglobin, resulting in relative functional anemia, even small concentrations
- As Summary :

1. It blocks O₂ binding sites [less O₂ can be absorbed]
2. Modifying the structure of Hb [shifts dissociation curve left]



-WITH LEFT SHIFT , THE TISSUE IS LEFT BEHIND

MECHANISM OF ACTION

THE MYOGLOBIN

- Carbon monoxide, after being released from hemoglobin, could also bind to myoglobin, forming carboxymyoglobin, thereby reducing the amount of oxygen transported by myoglobin, diminishing the amount of ATP synthesized, and decreasing the ability of muscle cells to contract about 60 times greater than that of oxygen.
- CO binds myoglobin, preventing its normal function, and this probably explains the development of

atraumatic rhabdomyolysis .

THE.CYTOCHROME OXIDASE

- This binding interferes with aerobic metabolism and efficient adenosine triphosphate synthesis.
- Cells respond by switching to anaerobic metabolism causing anoxia lactic acidosis and eventual cell death.
- The rate of dissociation between carbon monoxide and cytochrome oxidase is slow, causing a relatively prolonged impairment of oxidative metabolism.



MECHANISM OF ACTION

THE CNS EFFECT

- Carbon monoxide inhibits the mitochondrial electron transport system and activates polymorphonuclear leucocytes that cause brain lipid peroxidation. This process may explain the delayed outcomes of carbon monoxide intoxication such as late encephalopathy. Acute brain injury in patients exposed as to carbon monoxide is usually caused by hypoxia.
- Reactive oxygen species: CO results in superoxide generation and oxidative stress, which likely contributes to lipid peroxidation and neurologic injury.



TOXOCLOGY INTERPRETATION

- % SAT. OF CO = PERCENTAGE OF HEMOGLOBIN COMBINED WITH CARBON MONOXIDE IN THE FORM OF CARBOXYHEMOGLOBIN
- A CONCENTRATION OF 0.5 TO 1% IN THE ATMOSPHERE CAN PRODUCE CARBOXYHEMOGLOBIN SATURATION LEVELS OF 75% IN 2 TO 15 MIN.

NORMAL LEVELS OF CARBOXYHEMOGLOBIN :

- NON-SMOKERS CARBOXYHEMOGLOBIN LEVELS OF 1–3%.
- SMOKERS OFTEN NORMAL LEVELS OF CARBOXYHEMOGLOBIN OF 5–6%, MAY REACH 10% AND MAY EVEN EXCEED 15%.

FATAL LEVELS OF CARBON MONOXIDE:

- The carboxyhemoglobin levels of individuals who die of CO poisoning can vary greatly, depending on the source of the CO, the circumstances surrounding the death, and the health of the individual.
- In elderly individuals, and those suffering severe diseases, such as coronary artery disease or chronic obstructive pulmonary disease, saturations as low as 20–30% can be fatal.

ACUTE & CHRONIC POISONING

- The clinical findings of CO poisoning are highly variable and largely nonspecific.
- The link between levels of CO and effects is not direct, the amount of uptake is governed by a number of Variables, all of which are interrelated and include :

1. Relative Concentration of CO and oxygen
2. Alveolar ventilation
3. Duration and intensity of exposure

Severity of poisoning may not correlate with COHb levels because prolonged exposure to low levels can be fatal with a low measured COHb, but a brief, high- concentration exposure can produce a high COHb level with minimal symptoms.

MILD TO MODERATE CO POISONING:

- is a difficult diagnosis to establish clinically, and patients are easily misdiagnosed as having a benign headache syndrome or viral illness, patients often present with constitutional symptoms
- CO poisoning should be suspected in patients with persistent or recurrent headache, especially if a group of people have similar symptoms or if the headache improves soon after the person leaves an exposure site.

SEVERE CO POISONING :

- Patients present with coma or cardiovascular collapse
 - More serious effects include loss of consciousness, myocardial ischaemia, hypotension, congestive cardiac failure, arrhythmias, mental confusion, and mood variation
-

ACUTE SYMPTOMS

PULMONARY	CARDIAC	EARLY NEUROLOGIC	LATE NEUROLOGIC
• Dyspnea	• Chest pain • Arrhythmia • Hypotension	• Headache • Nausea • Blurry Vision • Dizziness	• Confusion • Seizures • Impaired Judgment • Syncope

- Sometimes these may occur in clusters and sometimes in isolation
- One of the major concerns following acute CO poisoning is the severe delayed neurological manifestations that may occur
- Problems may include difficulty with higher intellectual functions, short-term memory loss, dementia, amnesia, psychosis, irritability, a strange gait, speech disturbances, Parkinson's disease like syndromes, cortical blindness, and a depressed mood.
- Risk factors that predict the development of delayed neurologic sequelae include extremes of age and loss of consciousness

CHRONIC SYMPTOMS

- CHRONIC EXPOSURE TO RELATIVELY LOW LEVELS OF CO MAY CAUSE PERSISTENT HEADACHES, LIGHTEADEDNESS, DEPRESSION, CONFUSION, MEMORY LOSS, NAUSEA

AND VOMITING.

- CHRONIC EXPOSURE MAY WORSEN CARDIOVASCULAR SYMPTOMS IN SOME PEOPLE.
- CHRONIC CO EXPOSURE MIGHT INCREASE THE RISK OF DEVELOPING ATHEROSCLEROSIS .
- TYPICALLY, UPON REMOVAL FROM EXPOSURE TO CO, SYMPTOMS USUALLY RESOLVE THEMSELVES, UNLESS THERE HAS BEEN AN EPISODE OF SEVERE ACUTE POISONING



CONTINUE...

THE SYMPTOMS, WHEN THEY APPEAR, ARE PROGRESSIVE, ROUGHLY PARALLELING THE BLOOD LEVELS OF CO:

LEVELS OF CARBOXYHEMOGLOBIN	SYMPTOMS
At levels of 0–10% carboxyhemoglobin saturation	no symptoms
In individuals at rest, CO levels from 10 to 20	often symptomless, except for a headache
At level of 30 and 35%	headache with throbbing and fullness of the head.
Between 30–40% CO	throbbing headache, nausea, vomiting, faintness and drowsiness even at rest.
as levels approach 40%	slightest exertion caused faintness. Pulse and respiration are rapid .blood pressure falls
Between 40–60% CO	mental confusion, weakness and loss of coordination
At 60% CO and beyond	individuals lose consciousness, develop Cheyne–Stokes respiration ,have intermittent convulsions, depressed heart action and respiratory failure, and die , elevated body temperature may be present [seizures, coma, death]

CO POISONING IN PREGNANCY

- Carbon monoxide poisoning in pregnant women may cause severe adverse fetal effects.
- Carbon monoxide also crosses the placenta and combines with fetal hemoglobin, causing more direct fetal tissue hypoxia.
- Poisoning causes fetal tissue hypoxia by decreasing the release of maternal oxygen to the fetus.
- Additionally, fetal hemoglobin has a 10 to 15% higher affinity for carbon monoxide than adult hemoglobin, causing more severe poisoning in the fetus than in the adult.
- Elimination of carbon monoxide is slower in the fetus, leading to an accumulation of the toxic chemical.
- The level of fetal morbidity and mortality in acute carbon monoxide poisoning is significant, so despite mild maternal poisoning or following maternal recovery, severe fetal poisoning or death may still occur.



Don't Let Carbon Monoxide
Cause Damage To Your Pregnancy

Long-term Exposure To CO Can Harm Your Baby

- ➔ Pre-Natal Death
- ➔ Fetal Brain Damage
- ➔ Behavioral Problems
- ➔ Low Birth Weight
- ➔ Limb Abnormalities
- ➔ Cerebral Palsy

247homerescue.co.uk



HISTORY & PHYSICAL EXAM

CH2OPD2 MNEMONIC

- If exposure is suspected, it is appropriate to use this system
- It explore the source of environmental exposure

enquiring about :

- Community
- Home
- Hobbies
- Occupation
- Personal issues
- Diet
- Drug

PHYSICAL EXAM

- Classic but rare sign: cherry red lips
- Tachypnea
- Tachycardia
- hypotension
- Hyperthermia
- Noncardiogenicpulmonary edema
- Vestibular signs common
- retinal hemorrhages uncommon but more specific

CO-OXIMETRY

- an inexpensive method
- can distinguish between normal hemoglobin and COHb (and MetHb)
- confirms exposure to CO [by carboxyhemoglobin level]
- Normal < 3%
- > 15% suggest poisoning

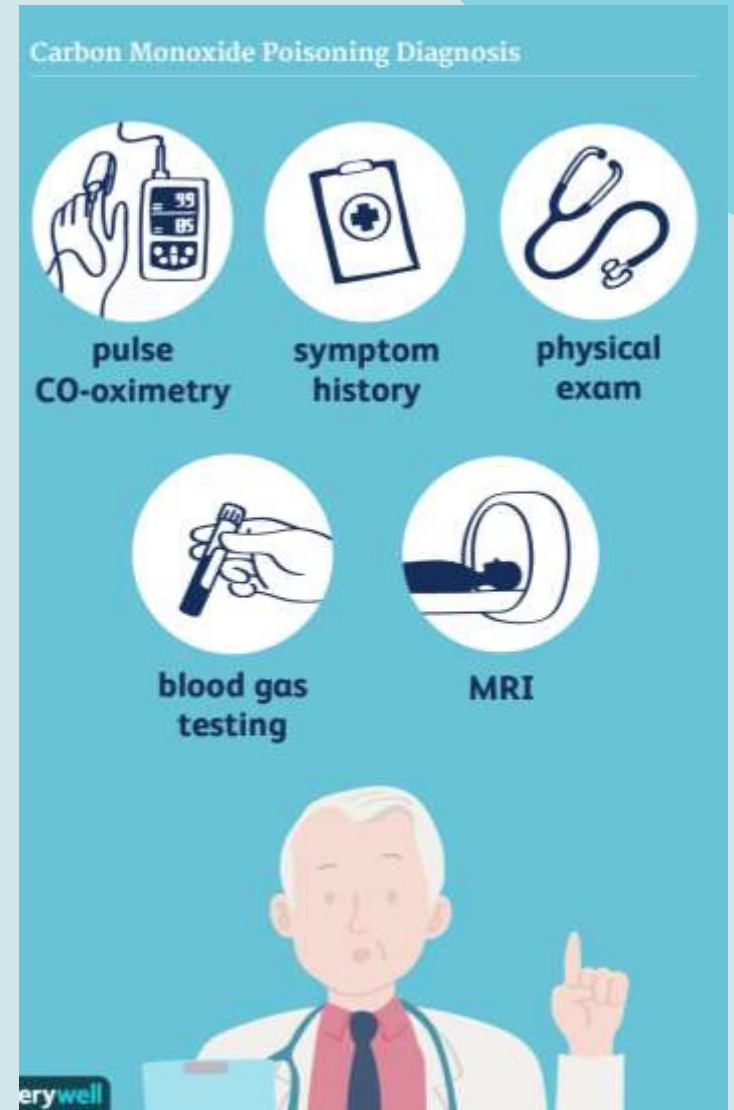
Smokers 10–15% [NORMAL]

STANDARD PULSE OXIMETRY NORMAL

- Cannot differentiate carboxyhemoglobin/oxyhemoglobin
- Often reads normal saturation despite low oxyhemoglobin

THE STANDARD BLOOD GAS (ABG OR VENOUS BLOOD GAS [VBG]) ANALYSIS

- is a poor screening test for CO poisoning other than to identify the presence of a metabolic acidosis and a normal partial pressure of oxygen (Po₂)



TREATMENT:

- FIRST STEP IS TO REMOVE VICTIM FROM THE SOURCE OF CARBON MONOXIDE
- THOSE WHO ARE UNCONSCIOUS MAY REQUIRE CPR ON SITE.
- HIGH-FLOW 100% FIO2 OR HYPERBARIC OXYGEN CONTINUED UNTIL HBCO LEVELS HAVE NORMALIZED
- ADMISSION FOR UNDERLYING MEDICAL CONDITIONS (CARDIAC) AND ASSOCIATED INJURIES
- SCREENING OF OTHER POSSIBLE VICTIMS AT SCENE



TREATMENT:

- ELIMINATION OF CARBON MONOXIDE IS RELATED TO MINUTE VENTILATION, DURATION OF EXPOSURE TO O₂, AND FIO₂

- HALF-LIFE OF HBCO:

4 TO 6 HOURS BREATHING ROOM AIR

40 TO 80 MINUTES BREATHING 100% O₂

15 TO 30 MINUTES BREATHING HYPERBARIC O₂

AUTOPSY FINDINGS



DECOMPOSITION AND CARBON MONOXIDE:

- CARBON MONOXIDE LEVELS IN BLOOD AND BODY CAVITY FLUIDS OF DECOMPOSED BODIES ARE DEPENDENT ON THE CARBON MONOXIDE LEVEL OF THE BLOOD PRIOR TO THE DEATH.
- THEY ARE NOT PRODUCED BY POSTMORTEM CARBON MONOXIDE FORMATION THROUGH THE DECOMPOSITION OF HEMOGLOBIN, MYOGLOBIN, AND OTHER SUBSTANCES.



AUTOPSY FINDINGS:

- IN PATHOLOGICAL (AUTOPSY) EXAMINATION THE RUDDY APPEARANCE OF CARBON MONOXIDE POISONING IS NOTABLE BECAUSE DEAD PERSONS ARE NORMALLY BLUISH AND PALE.
- WHEREAS DEAD CARBON-MONOXIDE POISONED PERSONS MAY SIMPLY APPEAR UNUSUALLY LIFELIKE IN COLORATION.



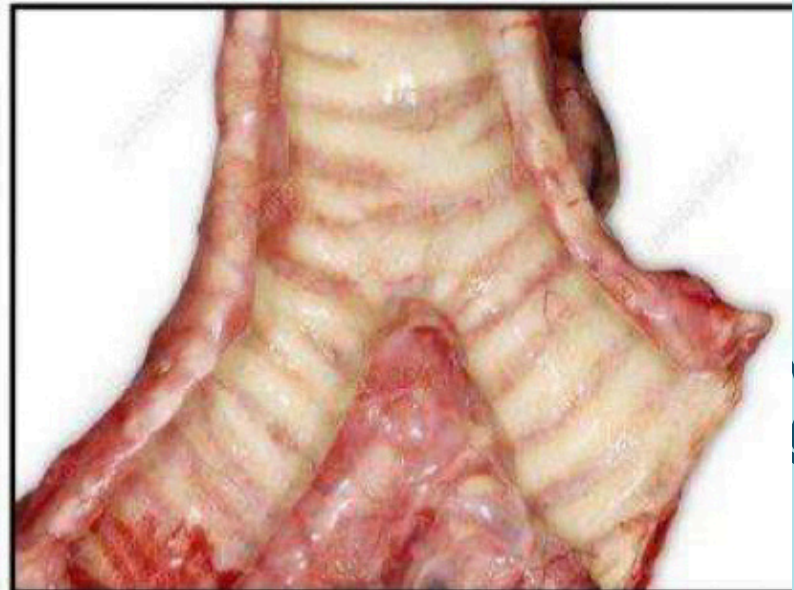


Fig. 2: Cherry red Post mortem seen on back

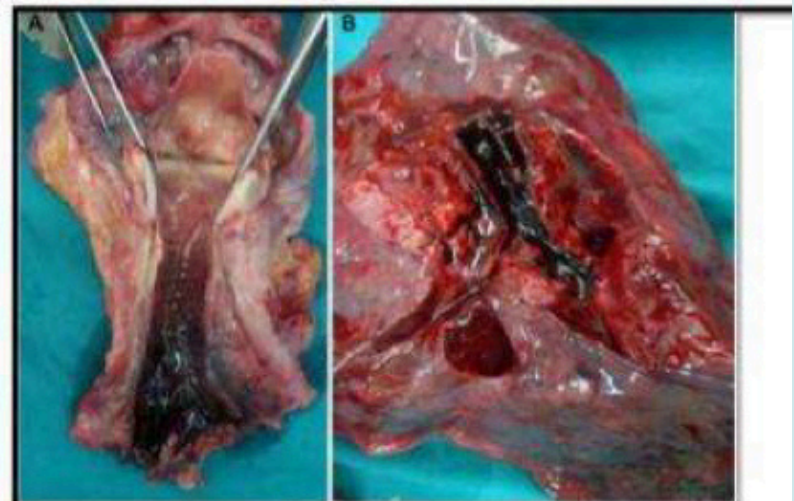


Figure 4: autopsy findings, A,B) black soot in the airways and cherry red coloration



Fig. 3: Cherry discolouration of tissues



Fig. 4: Cherry red discoloration of blood & viscera



AUTOPSY FINDINGS:

- THE CHERRY-RED COLORATION OF THE SKIN IS MOST CHARACTERISTIC APPEARANCE OF THE BODY SURFACE IN CO POISONING CASES.
- THIS IS USUALLY OBSERVED WITH COHB CONCENTRATIONS EXCEEDING 30 %
- HOWEVER, SINCE THIS "CHERRY- RED" APPEARANCE IS COMMON ONLY IN THE DECEASED, AND IS UNUSUAL IN LIVING PEOPLE, IT IS NOT CONSIDERED A USEFUL DIAGNOSTIC SIGN IN CLINICAL MEDICINE.
- CARBON MONOXIDE POISONING AS THE NAIL-BED, BLOOD AND SOME MUSCLES AND GENERALIZED ORGAN CONGESTION PULMONARY EDEMA WERE CHERRY-PINK IN COLOR, BLACK TRACHEA.

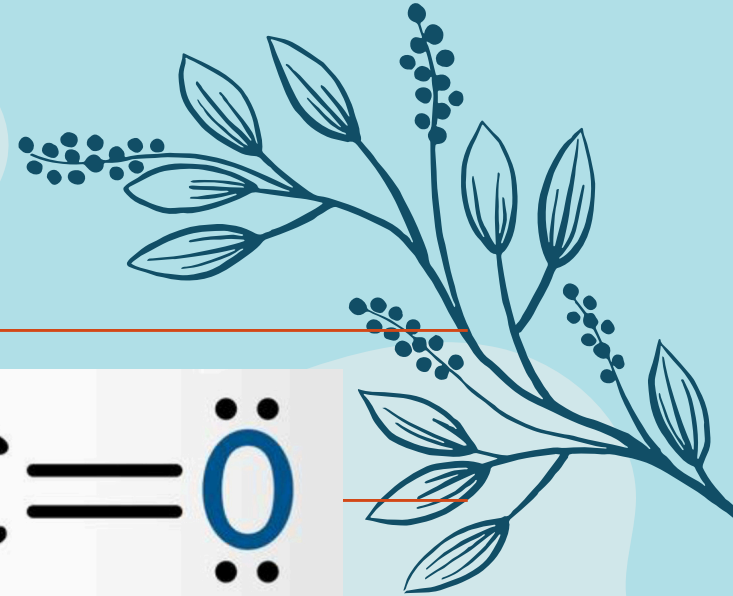
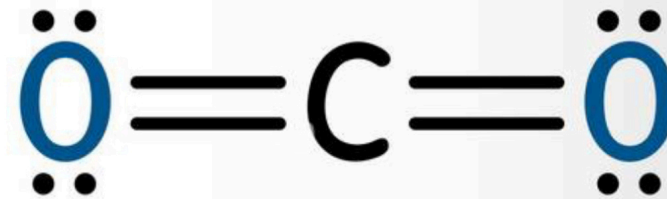
AUTOPSY FINDINGS:

- THE AUTOPSY FINDINGS IN EITHER CASE WERE SIMILAR TO THAT OF ACCIDENTAL AND HOMICIDAL CO ASSOCIATED DEATHS.
- THE EFFECT OF CARBON MONOXIDE ON THE FETUS IS OF GREAT CONSEQUENCE WHILE THE MOTHER SUFFERED MINOR DELETERIOUS EFFECT



CO2 POISONING

- INTRODUCTION
- MECHANISM OF ACTION
- TOXOCOLOGY INTERPRETATION
- SYMPTOMS AND SIGNS
- AUTOPSY FINDING



INTRODUCTION:

- CO₂ is a relatively inert gas and, unlike the monoxide, is not directly poisonous.
- It may also be caused by exposure to environments containing abnormally high concentrations of carbon dioxide (usually due to volcanic or geothermal causes).
- it can still kill because it is an irrespirable gas
- Hypercapnia is generally caused by hypoventilation, lung disease, or consciousness diminished

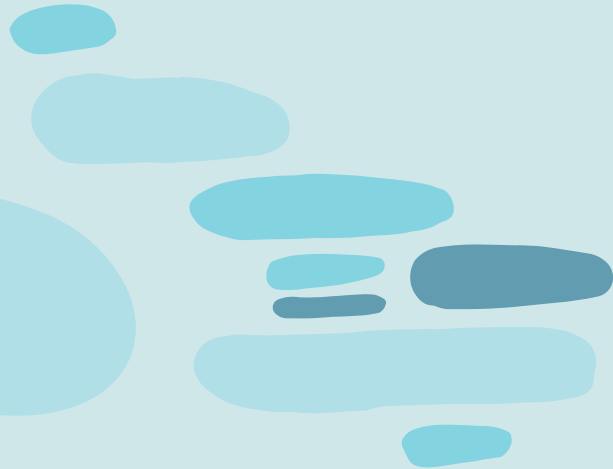


SOURCES :

SOURCES OF CO₂ ARE OFTEN INDUSTRIAL AS IT IS USED VERY WIDELY IN THE MANUFACTURE OF MANY PRODUCTS SUCH AS:

- PRECURSOR TO CHEMICALS
- FOODS
- BEVERAGES
- WINE MAKING
- FIRE EXTINGUISHER
- SUPER CRITICAL CO₂ AS SOLVENT
- AGRICULTURAL AND BIOLOGICAL APPLICATIONS
- OIL RECOVERY
- IN ANESTHESIA, DEATHS HAVE OCCURRED WHEN AN O₂ CYLINDER HAS BECOME EMPTY WHILST CONNECTED TO A FUNCTIONING CO₂ CYLINDER, SO THAT THE PATIENT HAS RECEIVED ONLY CO₂.

MECHANISM OF ACTION



CO₂ PHYSIOLOGY:

- CO₂ BOUND TO HEMOGLOBIN DOES NOT BIND TO THE SAME SITE AS OXYGEN. INSTEAD, IT COMBINES WITH THE N-TERMINAL GROUPS ON THE FOUR GLOBIN CHAINS.
- HOWEVER, BECAUSE OF ALLOSTERIC EFFECTS ON THE HEMOGLOBIN MOLECULE, THE BINDING OF CO₂ DECREASES THE AMOUNT OF OXYGEN THAT IS BOUND FOR A GIVEN PARTIAL PRESSURE OF OXYGEN.



CO2 PHYSIOLOGY:



CO2 IS CARRIED IN BLOOD IN THREE DIFFERENT WAYS:

MOST OF IT (ABOUT 70% TO 80%) IS CONVERTED TO BICARBONATE IONS

BY THE ENZYME CARBONIC ANHYDRASE IN THE RED BLOOD CELLS

5% -10% is dissolved in the plasma.

5% -10% is bound to hemoglobin as carbaminohemoglobin
COMPOUNDS

TOXICOLOGY INTERPRETATION



- AN UNEXPLAINED FEATURE OF CO₂ IS THE SPEED OF ITS TOXICITY ON MANY OCCASIONS. PURE ASPHYXIA SHOULD TAKE SEVERAL MINUTES AND BE ASSOCIATED WITH CYANOSIS AND CONGESTION, BUT CO₂ DEATHS CAN BE VERY RAPID AND THERE ARE SELDOM ANY ' SPHYXIAL SIGNS '. IT IS PRESUMED THAT THIS IS DUE TO SOME VASOVAGAL REFLEX CAUSING CARDI ARREST, TRIGGERED BY A CHEMORECEPTOR STIMULUS.
- THE MINIMUM FATAL CONCENTRATION OF CO₂ IS 25–30 PER CENT, THE HIGH CONCENTRATIONS CAUSING SUDDEN DEATH BEING 60–80 PER CENT.

SYMPTOMS AND SIGNS



SYMPTOMS AND SIGNS

Pulmonary

- Shortness of BREATH

Neurologic

- Headach
- DROWSINESS
- Mild Dizziness
- Narcosis
- Confusion
- SYNCOPE

Cardiac

- Hypertension
- TACHYCARDIA

Visual

- DIMMED SIGHT

Muscular

- Tremor

Auditory

- Reduced HEARING

TREATMENT



TREATMENT FOR HYPERCAPNIA DEPENDS UPON ITS SEVERITY ,AND STARTS WITH ADDRESSING THE UNDERLYING CAUSE. IF THIS IS INEFFECTIVE, ADDITIONAL TREATMENT MAY INCLUDE:

- TREAT THE UNDERLYING PROCESS.
- NON-INVASIVE POSITIVE PRESSURE VENTILATION (CPAP, BIPAP).
- MECHANICAL VENTILATION.





Thank You