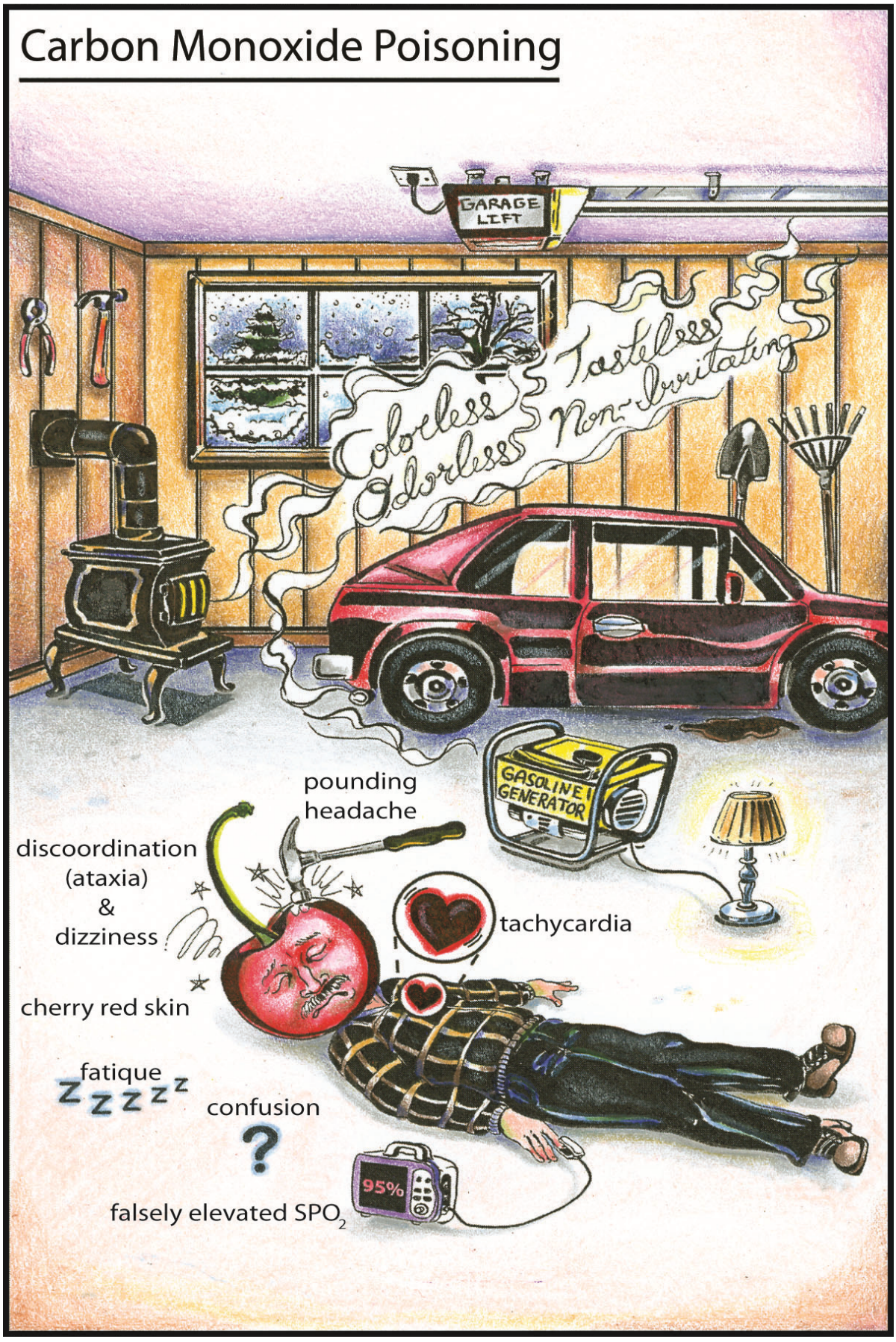
TOXICOLOGY

CARBON MONOXIDE POISONING



Pathophysiology

Carbon monoxide's affinity for haemoglobin is much greater than oxygen and forms carboxyhaemoglobin (COHb). It reduces the oxygen carrying capacity of blood causing hypoxia and ischaemic injury. It also causes an inflammatory cascade which leads to CNS injury with delayed neurological sequelae.

Carbon monoxide is produced during incomplete combustion of carbonaceous substances. Exposure may be from motor car fumes (deliberately filling the car with exhaust fumes), faulty boilers, heaters and fires. The gas itself is colourless and odourless.

Clinical Features

Features of carbon monoxide poisoning correlate poorly with COHb level. The normal level is up to 3% but smokers may have levels of 10-15%. Remember that SpO2 may be normal as pulse oximetry does not differentiate between carboxyhaemoglobin and oxyhaemoglobin.

- CNS: headache, confusion, ataxia, seizures, coma.
- Cardiovascular: tachycardia, arrhythmias, myocardial ischaemia.
- Gastrointestinal: abdominal pain, N&V, diarrhoea.
- Respiratory: SOB, tachypnoea, non-cardiogenic pulmonary oedema.
- Other: rhabdomyolysis, lactic acidosis, DIC.

A delayed-onset neuropsychiatric syndrome can arise 3 to 240 days after recovery. There may be cognitive deficits, focal neurology and movement disorders. Symptoms may last for a year or longer.

Treatment

- Intubate if unconscious
- Provide high flow oxygen
- Cardiac monitoring
- Manage complications.

Carbon Monoxide is eliminated by the lungs and its half-life is 4-5 hours. Oxygen reduces the half life by competitively binding haemoglobin. Hyperbaric oxygen involves exposing the patient to pressures of oxygen greater than atmospheric pressure. This leads to more oxygen dissolved in the blood, increasing provision of non-haemoglobin-bound oxygen to the tissues and decreases the half-life further. Discuss with your senior regarding its use.