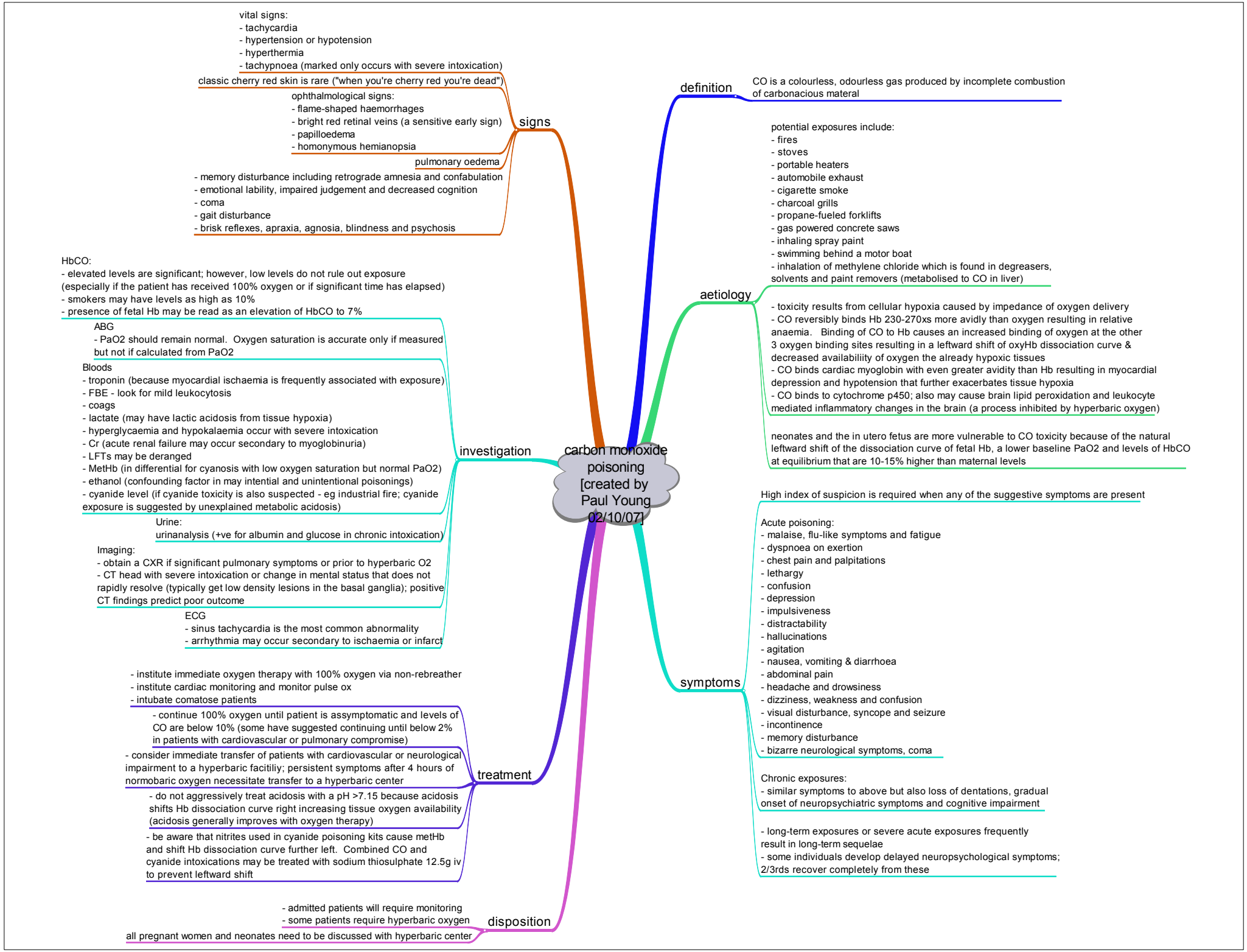
carbon monoxide poisoning [created by Paul Young 02/10/07]
definition
CO is a colourless, odourless gas produced by incomplete combustion of carbonacious materal
aetiology
potential exposures include:
- fires
- stoves
- portable heaters
- automobile exhaust
- cigarette smoke
- charcoal grills
- propane-fueled forklifts
- gas powered concrete saws
- inhaling spray paint
- swimming behind a motor boat
- inhalation of methylene chloride which is found in degreasers, solvents and paint removers (metabolised to CO in liver)
- toxicity results from cellular hypoxia caused by impedance of oxygen delivery
- CO reversibly binds Hb 230-270xs more avidly than oxygen resulting in relative anaemia. Binding of CO to Hb causes an increased binding of oxygen at the other 3 oxygen binding sites resulting in a leftward shift of oxyHb dissociation curve & decreased availabiliity of oxygen the already hypoxic tissues
- CO binds cardiac myoglobin with even greater avidity than Hb resulting in myocardial depression and hypotension that further exacerbates tissue hypoxia
- CO binds to cytochrome p450; also may cause brain lipid peroxidation and leukocyte mediated inflammatory changes in the brain (a process inhibited by hyperbaric oxygen)
neonates and the in utero fetus are more vulnerable to CO toxicity because of the natural leftward shift of the dissociation curve of fetal Hb, a lower baseline PaO2 and levels of HbCO at equilibrium that are 10-15% higher than maternal levels
symptoms
High index of suspicion is required when any of the suggestive symptoms are present
Acute poisoning:
- malaise, flu-like symptoms and fatigue
- dyspnoea on exertion
- chest pain and palpitations
- lethargy
- confusion
- depression
- impulsiveness
- distractability
- hallucinations
- agitation
- nausea, vomiting & diarrhoea
- abdominal pain
- headache and drowsiness
- dizziness, weakness and confusion
- visual disturbance, syncope and seizure
- incontinence
- memory disturbance
- bizarre neurological symptoms, coma
Chronic exposures:
- similar symptoms to above but also loss of dentations, gradual onset of neuropsychiatric symptoms and cognitive impairment
- long-term exposures or severe acute exposures frequently result in long-term sequelae
- some individuals develop delayed neuropsychological symptoms; 2/3rds recover completely from these
investigation
HbCO:
- elevated levels are significant; however, low levels do not rule out exposure (especially if the patient has received 100% oxygen or if significant time has elapsed)
- smokers may have levels as high as 10%
- presence of fetal Hb may be read as an elevation of HbCO to 7%
ABG
- PaO2 should remain normal. Oxygen saturation is accurate only if measured but not if calculated from PaO2
Bloods
- troponin (because myocardial ischaemia is frequently associated with exposure)
- FBE - look for mild leukocytosis
- coags
- lactate (may have lactic acidosis from tissue hypoxia)
- hyperglycaemia and hypokalaemia occur with severe intoxication
- Cr (acute renal failure may occur secondary to myoglobinuria)
- LFTs may be deranged
- MetHb (in differential for cyanosis with low oxygen saturation but normal PaO2)
- ethanol (confounding factor in may intential and unintentional poisonings)
- cyanide level (if cyanide toxicity is also suspected - eg industrial fire; cyanide exposure is suggested by unexplained metabolic acidosis)
Urine:
urinanalysis (+ve for albumin and glucose in chronic intoxication)
Imaging:
- obtain a CXR if significant pulmonary symptoms or prior to hyperbaric O2
- CT head with severe intoxication or change in mental status that does not rapidly resolve (typically get low density lesions in the basal ganglia); positive CT findings predict poor outcome
ECG
- sinus tachycardia is the most common abnormality
- arrhythmia may occur secondary to ischaemia or infarct
signs
vital signs:
- tachycardia
- hypertension or hypotension
- hyperthermia
- tachypnoea (marked only occurs with severe intoxication)
classic cherry red skin is rare ("when you're cherry red you're dead")
ophthalmological signs:
- flame-shaped haemorrhages
- bright red retinal veins (a sensitive early sign)
- papilloedema
- homonymous hemianopsia
pulmonary oedema
- memory disturbance including retrograde amnesia and confabulation
- emotional lability, impaired judgement and decreased cognition
- coma
- gait disturbance
- brisk reflexes, apraxia, agnosia, blindness and psychosis
treatment
- institute immediate oxygen therapy with 100% oxygen via non-rebreather
- institute cardiac monitoring and monitor pulse ox
- intubate comatose patients
- continue 100% oxygen until patient is assymptomatic and levels of CO are below 10% (some have suggested continuing until below 2% in patients with cardiovascular or pulmonary compromise)
- consider immediate transfer of patients with cardiovascular or neurological impairment to a hyperbaric facitiliy; persistent symptoms after 4 hours of normobaric oxygen necessitate transfer to a hyperbaric center
- do not aggressively treat acidosis with a pH >7.15 because acidosis shifts Hb dissociation curve right increasing tissue oxygen availability (acidosis generally improves with oxygen therapy)
- be aware that nitrites used in cyanide poisoning kits cause metHb and shift Hb dissociation curve further left. Combined CO and cyanide intoxications may be treated with sodium thiosulphate 12.5g iv to prevent leftward shift
disposition
- admitted patients will require monitoring
- some patients require hyperbaric oxygen
all pregnant women and neonates need to be discussed with hyperbaric center