

		toxicity category	toxicity mechanism	clinical presentation	treatment	treatment category
carbon monoxide	block oxygen transport / utilization	CO binds Hgb w/ 200x affinity of O ₂ , decreases cooperativity	N/V, abd cramping, bright red skin	O ₂ at high partial pressure	block or compete for essential receptors	
		methemoglobinemia: Fe ²⁺ --> Fe ³⁺	venous blood chocolate brown color, gray cyanosis, dyspnea, tachycardia, fatigability	methylene blue: electron donor	bypass effect of poison	
cyanide	alter enzyme activity	reacts w/ cytochrome oxidase and uncouples ATP production	heat, CNS and cardiac dysfunction	1. amylnitrate 2. sodium thiosulfate	acceleration of bioconversion to nontoxic metabolite	
		phosphorylates AChase	cholinergic crisis: SLUD	1. atropine 2. pralidoxine (PAM)	bypass effect of poison	
gasoline			CNS depression, cardiac arrest	supportive therapy, emesis?, cathartics		
heavy metals	iron	alter selective permeability of membranes	react w/ essential functional groups (esp. sulfhydryl) to disrupt transport	GI, hemorrhage, hepatotox, period of apparent recovery	chelation: deferoxamine, EDTA	complexation w/ poison + accelerate excretion
	mercury			erethism: CNS, anger, memory loss, drowsy	chelation: BAL	
	lead			developmental disabilities	chelation: EDTA	
	cadmium			osteomalacia, testicular damage, immunosuppress		
corrosives		tissue destruction		ingestion, inhalation, dermal, ocular	flush, supportive therapy	
carbon tetrachloride		free radical formation and lipid peroxidation	CCl ₄ --> CCl ₃ (free radical), membrane disruption	CNS depression, N/V, hepatotox, nephrotox	supportive, antioxidant?	
paraquat		formation of reactive oxygen species		pulmonary toxicity		
acetaminophen		binding to critical macromolecules	depletion of glutathione leads to toxic metabolites	hepatotoxicity	acetylcysteine	block formation of a more toxic metabolite
drug allergies		hapten and antigen formation				