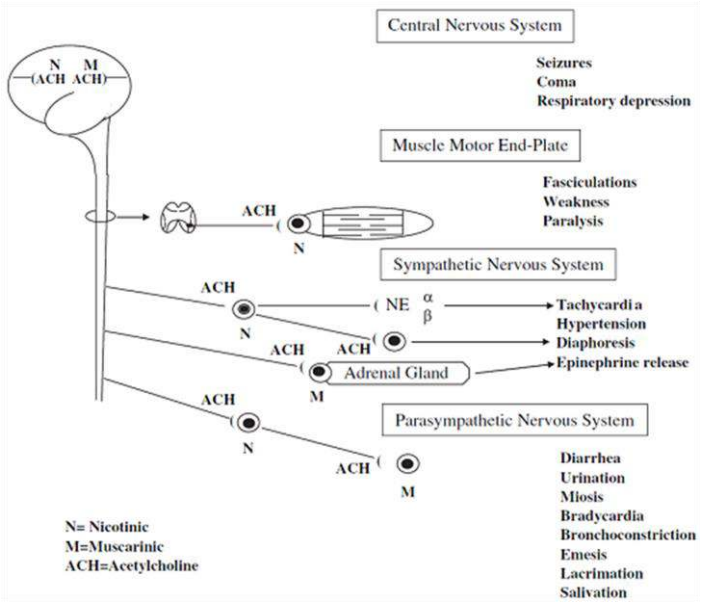
37. Organophosphate Poisoning Algorithm

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

DECONTAMINATION AND PERSONAL PROTECTION

- **WEAR PERSONAL PROTECTIVE EQUIPMENT (Gloves, Gowns and Masks)**
- **REMOVE ALL CLOTHING from and gently cleanse the patient with soap and water**. Consider clothing and PPEs as hazardous waste and discard accordingly

The action of acetylcholine released into a synaptic cleft or neuromuscular junction is normally terminated when the enzyme acetylcholinesterase cleaves acetylcholine into choline and acetic acid. Organophosphates bind to the active site of the cholinesterase enzymes causing an increase in the acetylcholine concentration and a marked hyper stimulation of the cholinergic system, which is responsible for the predominant signs of toxicity.



Muscarinic Manifestations

Ophthalmic: Conjunctival injection, lacrimation, miosis, blurred vision, diminished visual acuity, ocular pain

Respiratory: Rhinorrhea, stridor, wheezing, cough, excessive sputum, chest tightness, dyspnea, apnea

Cardiovascular: Bradydysrhythmias, hypotension

Dermal: Flushing, diaphoresis, cyanosis

Gastrointestinal: Nausea, vomiting, salivation, diarrhea, abdominal cramping, tenesmus, fecal incontinence

Genitourinary: Frequency, urgency, incontinence

Nicotinic Manifestations

Cardiovascular: Tachydysrhythmias, hypertension

Striated muscle: Fasciculations, twitching, cramping, weakness, paralysis

Central Nervous System Manifestations

Anxiety, restlessness, depression, confusion, ataxia, tremors, convulsions, coma, areflexia

- Monitor, support ABCs - The great majority of deaths due to nerve agents occur secondary to respiratory failure. This is due to bronchospasm, bronchorrhoea, paralysis of the muscles of respiration, and central apnoea. **Consider inserting an advanced airway or nursing in recovery position for airway protection. DO NOT USE SUCCINYLCHOLINE FOR RSI.**
- Check vital signs (BP, PR, RR, SPO_2, T° C, **RBS**). Start Oxygen **IF** SPO_2 < 94%. **If abnormal vital signs, START ATROPINE! (see indications below).**
- Send samples for **FBC, UEC, LFTs, VBG, toxicology.** Correct any electrolyte imbalances (**see 22: Electrolyte Abnormalities Algorithm**)
- Perform brief, targeted history, physical exam
- **DO NOT PERFORM GASTRIC LAVAGE.**
- **DO NOT GIVE ACTIVATED CHARCOAL** unless the patient has co-ingested other poisons (**see 36. Poisoning Algorithm** for indications and contraindications for activated charcoal)

Give IV Atropine

(2 mg IV for adults or **0.02 mg/kg IV for children repeated every 5 minutes)**

Indications for Atropine treatment (Miosis alone is **NOT** an indication for atropine administration)

Symptoms	Severity
Rhinorrhoea, lacrimation, or mild dyspnoea	Mild
Inability to ambulate, dyspnoea, vomiting, fasciculations, weakness	Moderate
Convulsions†, coma, respiratory insufficiency	Severe

* **Tachycardia** can occur in organophosphate poisoning due to stimulation of the sympathetic ganglia as well as respiratory distress and hypoxia. Tachycardia is **NOT** a contraindication to atropine administration.

Atropine doses should be **repeated every 5 minutes** until the therapeutic endpoint (**Atropinisation**) is reached i.e. **until pulmonary secretions are dried** [reflected by improved oxygenation] and ease of breathing [or ease of ventilation]), a **pulse rate > 80 beats per minute** and **systolic blood pressure > 80mm/Hg**.

Start atropine **infusion when atropinisation achieved – 0.05mg/kg/hour**. E.g. for a 70kg patient give 3.5 mg of atropine per hour as an infusion. Put 10mg of atropine in 200mLs of fluid run at 40 – 80mLs per hour (2-4mg/hr) depending on response.

Precautions - Excessive doses of atropine can result in deleterious effects including **delirium, agitation, and tachycardia and hypertension**. Atropine will likely **NOT improve miosis** or **skeletal muscle paralysis** (nicotinic receptors)**;** therefore, reversal of these effects is **not a therapeutic endpoint.** Attempting to reverse these findings with atropine can result in administration of excessive doses of atropine.

†Seizure control

(Midazolam 0.1mg/kg or Diazepam 0.1mg/kg)

Benzodiazepines are needed to prevent or treat nerve agent–induced seizures in **moderate to severe toxicity** because anticholinergic treatment is increasingly less effective from 5 – 40 minutes post exposure. Phenytoin does **NOT** affect GABA-A and has been found to be **ineffective** in controlling organophosphate –induced seizures. Benzodiazepines should be infused rapidly to unresponsive patients who have been exposed to organophosphates, because such patients may have non-convulsive seizures due to the onset of paralysis.

Pralidoxime (2-PAM)

WHO recommendation is **> 30-mg/kg IV/IM bolus** followed by **> 8-mg/kg/hour IV infusion**

(**Adults:** 2 g IM or slow IV infusion over 15 to 30 minutes followed by a 500-mg/hour infusion)

Neither atropine nor benzodiazepines will alleviate symptoms affecting the **nicotinic system** (CNS, NMJ, autonomic ganglia). 2-PAM should be given to any patient exposed to an organophosphate nerve agent who is showing any **systemic toxicity** especially **fasiculations** or **weakness**. The initial dose should be given as quickly as possible. **Caution:** Delivering 2-PAM more rapidly than recommended can result in **hypertension.** This is usually self-limited, but in extreme cases, phentolamine 5 mg IV may be effective. **Laryngospasm and rigidity** can also occur with rapid IV

Disposition

- **Consult a Physician**
- **Continue atropine infusion until** the therapeutic endpoint (**Atropinisation**) is reached i.e. **until pulmonary secretions are dried** [reflected by improved oxygenation] and ease of breathing [or ease of ventilation]).
- **Admit ALL symptomatic patients. Severe poising should be admitted to an ICU**