



9. Clinical improvement should be based upon the drying of secretions and easing of respiratory effort rather than heart rate or pupillary response
10. Continuous and ongoing patient reassessment is critical

Assessment

1. Acetylcholinesterase inhibitor agents are highly toxic chemical agents and can rapidly be fatal
2. Patients with low-dose chronic exposures may have a more delayed presentation of symptoms
3. Antidotes (atropine and pralidoxime) are effective if administered before circulation fails
4. The patient may develop:
 - a. Miosis (pinpoint pupils)
 - b. Bronchospasm
 - c. Bradycardia
 - d. Vomiting
 - e. Excessive secretions in the form of:
 - i. Tearing
 - ii. Salivation
 - iii. Rhinorrhea
 - iv. Diarrhea
 - v. Urination
 - vi. Bronchorrhea
5. Penetration of an acetylcholinesterase inhibitor agent into the central nervous system (CNS) will cause:
 - a. Headache
 - b. Confusion
 - c. Generalized muscle weakness
 - d. Seizures
 - e. Lethargy or unresponsiveness
6. Estimated level of exposure based upon signs and symptoms
 - a. Mild
 - i. Miosis alone (while this is a primary sign in vapor exposure, it may not be present in all exposures)
 - ii. Miosis and severe rhinorrhea
 - b. Mild to moderate (in addition to symptoms of mild exposure)
 - i. Localized swelling
 - ii. Muscle fasciculations
 - iii. Nausea and vomiting
 - iv. Weakness
 - v. Shortness of breath
 - c. Severe (in addition to symptoms of mild to moderate exposure)
 - i. Unconsciousness
 - ii. Convulsions
 - iii. Apnea or severe respiratory distress requiring assisted ventilation
 - iv. Flaccid paralysis
7. Onset of symptoms can be immediate with an exposure to a large amount of the acetylcholinesterase inhibitor