

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