



Recognition, Management, and Reporting of Pesticide Illness

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Pesticide Usage in California

- California (58 counties)
 - 208,972,917 lbs. of pesticide active ingredients applied (agricultural use) in 2016
 - Fresno, Kern, and Tulare counties applied the most per county
 - 1,051,840,240 lbs. of pesticide active ingredients sold in 2017
 - This includes agricultural use pesticides as well as sanitizers and anti-microbials

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Classification of Pesticides

Target

- Insecticides: insects
- Herbicides: weeds
- Fungicides: fungi
- Rodenticides: rodents
- Acaricides: mites
- Nematicides: worms
- Antimicrobials: microbes

Chemical Groups

- Organophosphates
- N-methyl carbamates
- Pyrethroids
- Neonicotinoids
- Organochlorines

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Reporting Pesticide Illnesses

Physicians must report pesticide illnesses using one of these methods:

1. Call or fax local health officer. Numbers to call for each county found at: www.cdph.ca.gov/Programs/CCLHO/Pages/CCLHOHealthOfficerDirectory.aspx
2. Call the California Poison Control Center at:
 - a. For general public: (800) 222-1222
 - b. For health care providers: (800) 411-8080
3. Report it on the Confidential Morbidity Report, available at: www.cdph.ca.gov/CDPH%20Document%20Library/ControlledForms/cdph110b.pdf
4. In many counties physicians can report electronically using CalREDIE, the California Reportable Disease Information Exchange at: www.cdph.ca.gov/Programs/CID/DCDC/Pages/CalREDIE-HELP.aspx

There are additional requirements for reporting work related cases of pesticide illnesses

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Reporting a Work Related Case of Pesticide Illness

For a work related case of pesticide illness:

- A. Report the case using a method listed on the previous slide
- B. Send the Doctor's First Report to:
 - i. Employer or insurer within 5 days of first visit
 - ii. Local health officer within 7 days
 - iii. Div. of Labor Statistics and Research of Dept. of Industrial Relations

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Why Report Pesticide Illness

- Can serve as an early warning system
- Can get state agencies to assist
- Establishes history and trends in poisonings
- Helps identify problem pesticides
- Assists State in writing pesticide regulations
- Assists State in health based investigations
- Requirement of California Code of Law Health and Safety Code

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Pesticide Illness Surveillance Program

(Department of Pesticide Regulation)

Number of Pesticide Illness Reports

Year	Total	Associated with Pesticide		
		Total	Agric	Non-Agric
2011	1473	1067	239	816
2012	1418	992	245	741
2013	1718	1128	435	685
2014	1685	1073	265	798
2015	1757	1187	397	787

Which pesticide usually generates the most reports?

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Pesticide Illness Surveillance Program of 2015

- Occupational cases
 - 573 of 1187 reports associated with pesticides
 - Fieldworkers (206), applicators (110), mixer/loaders (38), routine indoor (24)
- Non-occupational cases
 - 609 of 1187 reports associated with pesticides
- 5 cases could not be determined

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Pesticide Illness Surveillance Program of 2015

- 26 hospitalizations
 - 12 for ingestions
 - 9 intentional ingestions to cause “self harm”
- 2 deaths reported definitely associated with pesticides
 - A pool maintenance tech consolidating containers of muriatic acid in an unventilated garage
 - A 34 y.o. male with hx. of mental illness reentered a tented house being fumigated with sulfuryl fluoride

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Pesticide Illness Surveillance Program of 2015

- 225 cases associated with pesticides involved children (less than 18 y. o.). Almost all were non-occupational. 4 cases due to agricultural use pesticides.
 - 5 involved hospitalizations
 - None resulted from “self harm” attempts
 - 44 (20%) exposed by ingestion
 - 33 less than 6 years old
 - 28 probably due to improperly stored pesticides
- 53 cases were exposed at school when a pesticide drifted from a nearby service establishment

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Diagnosing Pesticide Related Illness

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What Is Most Important to Make a Diagnose of a Pesticide Related Illness?

- A) History
- B) History
- C) History
- D) All of the above

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Diagnosis

- Most important in the HISTORY is:
 - **Exposure History**. Ask in detail.
 - What was the toxin?
 - What was the exposure?
 - Enough to cause the signs & symptoms?
 - » Consistent with the known effects?
 - Was the protective equipment working?
 - When was the exposure and when did the signs and symptoms occur?

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Other Questions in Occupational Medicine History

- Does occupation involve an exposure to a toxin?
- Exactly, what are the job duties (not just the job title)?
- Do other co-workers have the same symptoms?
 - Are the symptoms related to anything at work?
 - Are the symptoms worse at work and improved when off work or on vacation?
- Are there exposure to other chemicals (at work or home with hobbies)?
- List past occupations and exposures
- (Consider a visit to the worksite)

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“The Quick Survey”

1. What kind of work do you do?
2. Do you think your health problems are related to your work?
3. Are your symptoms better or worse when you're at home or at work?
4. Are you now or have you previously been exposed to dusts, fumes, chemicals, radiation, or loud noise?

(Newman LS)

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Important Points for Diagnosis

- The **EXPOSURE HISTORY** is the most important information the physician needs to make the correct diagnosis
- Pesticide related illnesses can commonly resemble other illnesses
- Pesticide illnesses can be difficult to diagnose
- **For most pesticide poisonings, there are no diagnostic tests or specific therapies**

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Accuracy of Diagnosis

- Lamminpaa and Riihimaki
 - Study of a case series of serious acute pesticide poisonings (79)
 - Less than 50% of the cases were correctly diagnosed
- Zweiner et al
 - 37 children, 1 month to 11 years old, with serious organophosphate poisonings
 - Only 20% of cases (4 of 20) transferred from other ER's were correctly diagnosed

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Sources of Information

- Safety Data Sheet (formerly MSDS)
- Pesticide label
- Employers
- Agricultural commissioner
- California Poison Control Center
 - a. General public: (800) 222-1222
 - b. Health care providers: (800) 411-8080

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Sources of Information

- California Department of Public Health
 - Occupational Health Branch (800) 970-6680
 - <http://www.dhs.ca.gov/ohb/contact.htm>
- California Department of Pesticide Regulation
 - Worker Health and Safety Branch (916) 445-4222
 - www.cdpr.ca.gov
- California Office of Environmental Health Hazard Assessment
 - (510) 622-3170
 - www.oehha.ca.gov

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Sources of Information

- Recognition and Management of Pesticide Poisonings, 6th Edition by the U.S. EPA
 - <http://www2.epa.gov/pesticide-worker-safety/recognition-and-management-pesticide-poisonings>
 - 5th Edition available in Spanish
- U.S. Environmental Protection Agency, Region 9 Office
 - Phone: (415) 744-1500
 - www.epa.gov/pesticides
- National Pesticide Information Center
 - Phone: (800) 858-7378
 - <http://npic.orst.edu/>

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Organophosphate and N-methyl Carbamate Pesticide Related Illness

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Cholinesterase Inhibiting Pesticides

- Cause more serious illness than any other class of pesticides
 - Historically, were 30-36% of all reports
 - 26% in 2008
- A test that can aid the diagnosis and an antidote are available for poisonings due to them

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Case Example 1

23 farm workers went into a cauliflower field in Salinas Valley 6 hrs. after mevinphos and phosphamidon were applied. A 72 hr. restricted entry interval was required. 2 hrs. later, a few noticed onset of blurred vision and eye irritation. Shortly, some developed dizziness, weakness, disorientation, headache, nausea, and vomiting. Several had cramps of arms, legs, and stomach. 2 collapsed with bradycardia, increased salivation, miosis, and muscle fasciculations. 16 sought treatment and 5 were hospitalized. The sickest ones were treated with IV atropine and pralidoxime.

15 received weekly followup of their RBC cholinesterase. (Midtling et al.)

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Pathophysiology of Cholinesterase Inhibition

- Acetylcholine is a neurotransmitter
 - Enzyme, **acetylcholinesterase**, hydrolyzes acetylcholine to choline and acetic acid
- OP and carbamates covalently bind to acetylcholinesterase at the binding site for acetylcholine rendering the enzyme inactive
 - Leads to accumulation of acetylcholine at the synapses and continuous or overstimulation of the receptors
 - Signs and symptoms depend on which sites are stimulated

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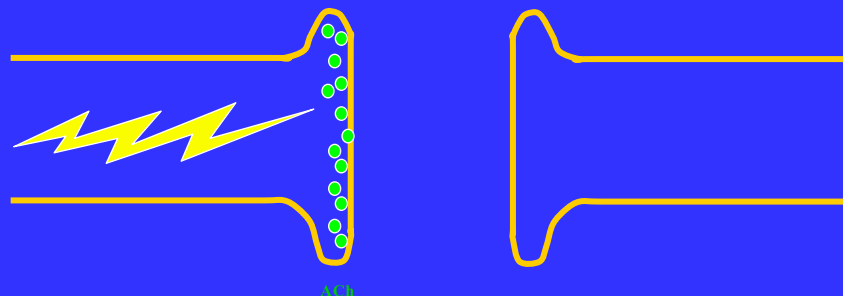
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Nerve Transmission: Nerve to Nerve



(Slide from US Army Medical Research Institute of Chemical Defense)

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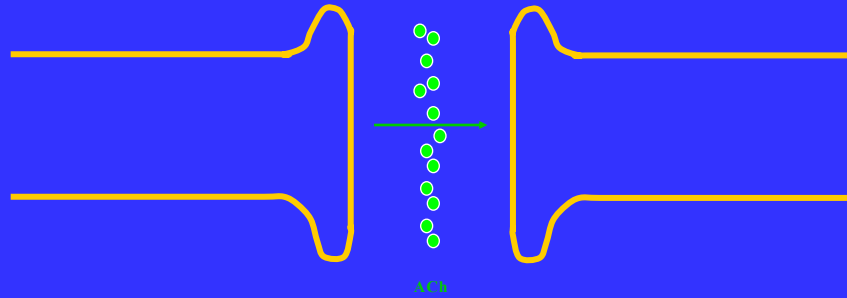
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Nerve Transmission: Nerve to Nerve



(Slide from US Army Medical Research Institute of Chemical Defense)

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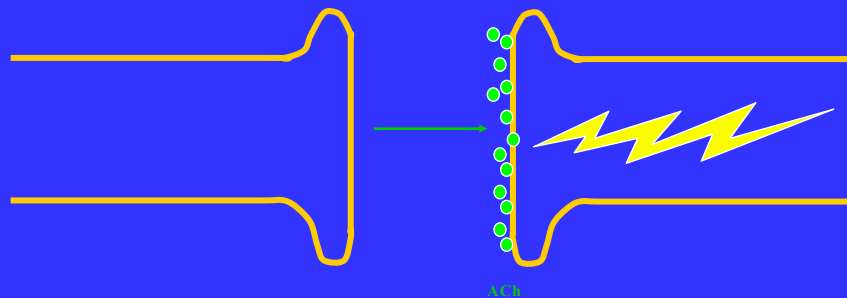
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Nerve Transmission: Nerve to Nerve



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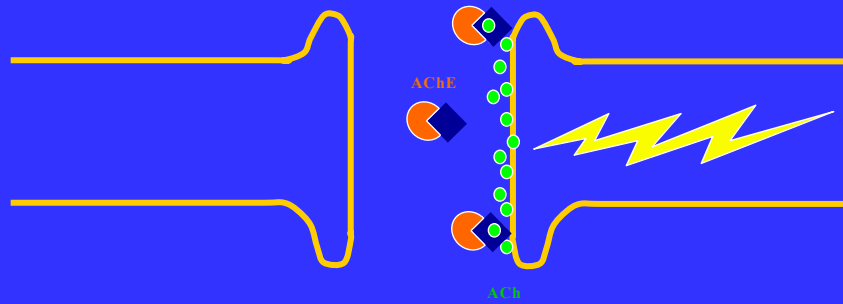
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Impulse Termination: The Role of AChE



(Slide from US Army Medical Research Institute of Chemical Defense)

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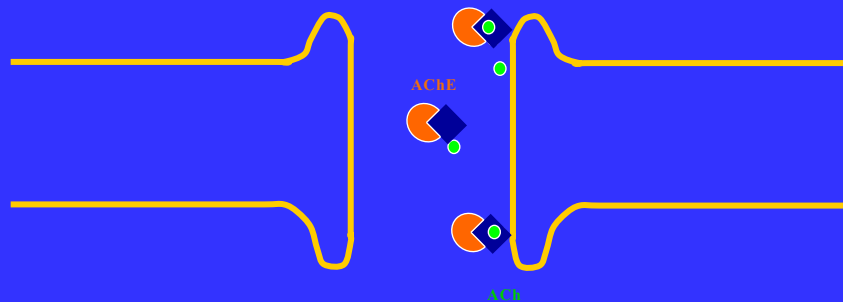
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Impulse Termination: The Role of AChE



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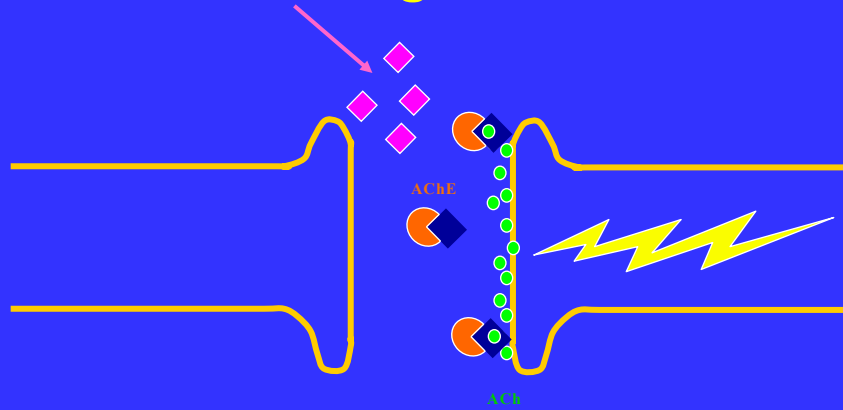
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Exposure to a Cholinesterase Inhibiting Pesticide



(Slide from US Army Medical Research Institute of Chemical Defense)

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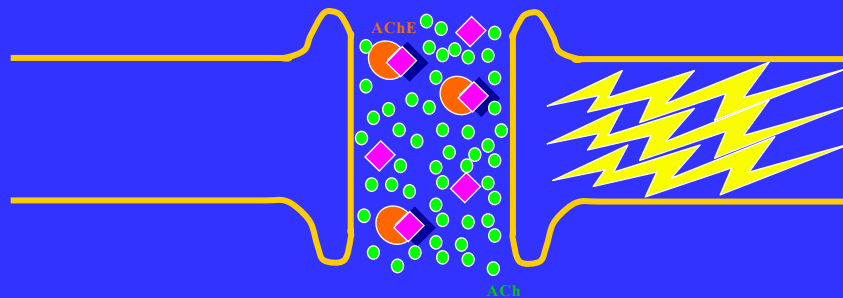
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Exposure to a Cholinesterase Inhibiting Pesticide



(Slide from US Army Medical Research Institute of Chemical Defense)

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Organophosphates

- Binding with acetylcholinesterase is a two step process
 - Initial binding is reversible
 - After “aging”, bond becomes irreversible. Time to “aging” varies with the OP but usually begins after 6 hours
- Some organophosphates: Chlorpyrifos (Dursban, Lorsban), malathion, parathion, diazinon

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N-methyl Carbamates

- Binding with acetylcholinesterase is reversible
 - Do not undergo “aging”
- Do not cross as well into the central nervous system
- Some carbamates: Aldicarb (Temik), carbaryl (Sevin), carbofuran, methomyl

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Cholinergic Syndrome

- **Muscarinic Receptors**

- Diaphoresis
- Eyes - miosis, lacrimation, blurred vision, discomfort
- Resp - Wheezing, cough, SOB, bronchorrhea
- Cardiac - Bradycardia, hypotension
- GI - Salivation, nausea, vomiting, defecation, incontinence, diarrhea, abdominal pain
- GU - Urinary incontinence, frequency

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Cholinergic Syndrome

- **Nicotinic Receptors**

- Cardiovascular – Tachycardia, hypertension
- Musculoskeletal – Fasciculations, muscle weakness, paralysis, cramps, weakness of muscles of respiration

- **CNS Receptors**

- Headache, anxiety, confusion, psychosis, ataxia, dysarthria, tremor, seizures, stupor, coma, respiratory depression, Cheynes-Stokes respiration

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Which Signs and Symptoms Can Help Make the Diagnosis

- Signs and symptoms are variable. Many are non-specific. Certain combinations, esp. if the MD has a high of index of suspicion, can lead to the diagnosis
- Some signs are reasonably specific and are good clues
 - Excessive salivation
 - Lacrimation
 - Muscle fasciculations and weakness
 - Pupil constriction
 - Urinary incontinence
 - Fecal incontinence

(Bardin et al.)

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Mnemonics

- | | | |
|-----------------------------------|------------------|------------------|
| • <u>SLUD</u> or
<u>SLUDGE</u> | • <u>DUMBELS</u> | • <u>MUDDLES</u> |
| S alivation | D iarrhea | M iosis |
| L acrimation | U rination | U rination |
| U rination | M iosis | D iarrhea |
| D efecation | B roncorrhea | D efecation |
| | B roncospasm | D iaphoresis |
| | E mesis | L acrimation |
| G astrointestinal
cramping | L acrimation | E mesis |
| E mesis | S alivation | S alivation |

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Which is the “critical” organ system in an organophosphate or carbamate poisoning?

(All organ systems are important!)

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Respiratory System

- Primary cause of death is respiratory failure
 - Tsao et al. 43 of 107 with OP or carbamate poisoning developed respiratory failure.
 - Of these forty-three, 51.2% died.
 - 100% of pts. without respiratory failure survived.
- Bronchospasm and bronchorhea in combination with weakness of the muscles of respiration and depression of medullary respiratory centers can cause respiratory arrest (can be sudden)
- Pneumonia can develop

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Lab Tests

- **Plasma Cholinesterase Activity**

- (aka pseudo, serum, or butyryl cholinesterase)
- More labile
- More actively inactivated
- More rapidly regenerated
- Preferentially lowered by some pesticides (eg. chlorpyrifos, mevinphos)

- **RBC Cholinesterase Activity**

- (aka true or acetyl cholinesterase)
- Same as enzyme in nervous system
- Depressed slower
- Thought to reflect inactivation at neuroeffector site more accurately
- Regenerates slower
- Preferentially lowered by some pesticides (eg. phosmet, dimethoate)

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Factors Affecting Cholinesterase Activity Levels

- **Plasma cholinesterase activity level**

- 3% have genetically determined lower level and are susceptible to a muscle paralyzer, succinylcholine, but not to organophosphates. They have normal RBC ChE.
- Can be lowered by liver disease, malnutrition, alcoholism, nephrotic syndrome, early pregnancy, cocaine, carbon disulfide, organic mercury, birth control pills, and metoclopramide.

- **RBC cholinesterase activity level**

- can be affected by hemolytic anemia, pernicious anemia, recovery from hemorrhage, and conditions associated with reticulocytosis.

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Lab Tests

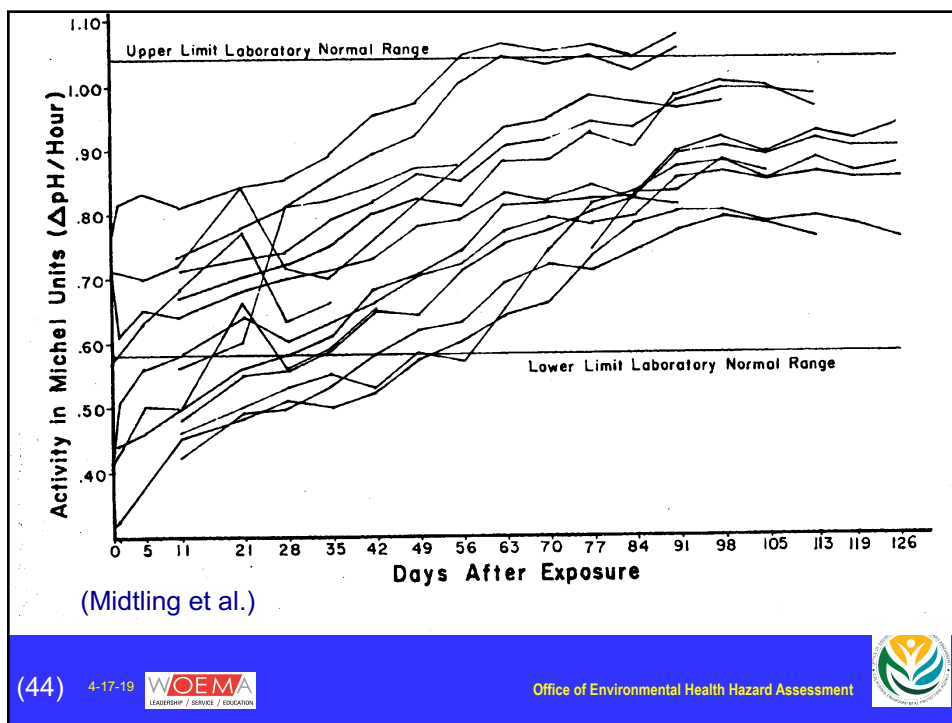
- **Cholinesterase activity level (for OPP)**
 - Association of symptoms and levels is variable
 - Rapidity of decrease of levels is important
 - Can help make diagnosis if very low
 - **Be careful how to interpret if levels normal**
 - Can confirm diagnosis retrospectively by obtaining levels acutely and repeating over next few weeks and months

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Tests

- Cholinesterase activity level
 - Not usually useful for carbamate poisoning
 - Binding with cholinesterase does not “age”
- Metabolites. May not be readily available.
- Chest x-ray, EKG and cardiac monitoring if indicated
- “Test dose” of atropine, 0.5–1.0 mg. IV
 - If OPP, then no response
 - If not OPP, then should get atropinization with dry mouth, mydriasis, increased heart rate, flushing

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Differential Diagnosis

- Mild cases
 - Acute viral syndromes (flu-like illness)
 - Gastroenteritis
 - Respiratory infections
 - Asthma
 - Psychological dysfunction
 - Allergic dermatitis
- Severe cases
 - Acute cerebrovascular accident
 - Heat stroke
 - Heat exhaustion
 - Epilepsy
 - Infections
 - Meningitis
 - Encephalitis
 - Pneumonia
 - Psychosis

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Routes of Absorption

- **Inhalation**
 - Fastest
- **Ingestion**
 - Used in suicides
 - Children
- **Dermal absorption**
 - Slower
 - Symptoms may not appear for 6-12 hours after exposure
 - Most occupational exposures
- **Need to observe patient with significant exposures**

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Children

- Study done at Children's Medical Center in Dallas
- 20 patients were transferred from other ER's
 - 16 of these 20 had incorrect diagnoses
 - Difficulties in diagnosis
 - 75% were < 3 years old
 - Developmentally incontinent
 - Lacrimation secondary to anxiety and pain
 - » SLUD difficult to use
 - Almost 25% had tonic-clonic seizures

(Zweiner et al)

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Children

- 37 patients, 1 month to 11 years old (median age 22 months)
 - 36 (97%) occurred in the home
 - 28 (76%) oral ingestion
 - 25 (70%) drank liquid improperly stored organophosphate
 - 6 (16%) dermal absorption
 - 3 (8%) unknown

(Zweiner et al.)

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Diagnosis

- History
- Physical findings
- Cholinesterase activity levels
 - Acutely
 - Retrospectively
- Response to therapy

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Management

- Supportive care
 - Endotracheal intubation and mechanical ventilation for oxygenation and to prevent aspiration
- Decontamination
- Anticholinergic med is mainstay of treatment
 - Atropine
- Cholinesterase reactivator
 - Pralidoxime (2-PAM, Protopam)
- Other measures

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Atropine

- Atropine competitively blocks the action of acetylcholine at the muscarinic receptor sites
- Correct hypoxia before administering to avoid arrhythmias
- First dose is “test dose”
- Standard dose is 2.0-4.0 mg. IV Q 15 minutes
 - End points
 - Lung rales clear
 - “Dry out” lungs to prevent pneumonia and respiratory failure
 - Reversal of muscarinic signs and symptoms
 - Full atropinization (flushing, dry mouth, mydriasis, tachycardia)

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Atropine

- Pediatric dose (<12 y.o.) is 0.05-0.1 mg/kg body weight
- Atropine toxicity can cause fever, muscle fibrillation, delirium
- **May need large quantity for treatment (up to 50 mg. in 24 hrs.)**
- IV continuous infusion 30 mg. in 200 ml. of saline and titrate against symptoms
- No effect on nicotinic receptor sites (including muscles of respiration)

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Case Example 2

68 y.o. male, hx. of depression, attempted suicide. Drank 3 oz. of concentrated solution of dimethoate. Initially responded to treatment with ipecac, charcoal, atropine, and pralidoxime. Relapsed and treated for the next 5 weeks with supportive care and large quantities of atropine. Some days required over 3000 mg. of atropine to control the hypersecretions. **The total amount of atropine used for this patient was over 30,000 mg.** Discharged with a slight hearing loss and a nonspecific personality change.

(LeBlanc et al.)

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Pralidoxime (2-PAM, Protopam)

- Reactivates acetylcholinesterase by removal of the phosphate group bound to the esteratic site
- **Draw blood sample for cholinesterase testing before administering pralidoxime**
- **Most effective before “aging” occurs.** Within the first 6 hours after exposure to OP but reported efficacious up to 24-48 hrs. later. Some initiate with muscle fasciculations
- Dose is 1g IV over 45-60 minutes (give slowly)
 - Can repeat after 2 hours and then Q 12h PRN
 - IV continuous infusion 250-500 mg./h. Titrate to sx's
- Pediatric dose (< 12 y.o.) is 20-50mg/kg of body weight

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Decontamination

- Clothes removed and bagged
 - Dispose of shoes
- Patient must be completely washed including skin, skin folds, scalp, and under the fingernails with water and soap or an alkaline detergent
- Flush eyes if indicated
- Protect personnel involved in treatment of the patient

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Other Measures

- GI measures
 - Activated charcoal
 - For the unconscious, intubate the airway for protection. Then gastric lavage with a large bore tube followed by use of charcoal
- Furosemide for persistent pulmonary edema after full atropinization
- Hydrocarbon aspiration. Treat as case of acute respiratory distress syndrome
- Monitor cardiac and pulmonary status
- Diazepam or lorazepam for seizure control

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Case Example 3

Three workers in a pesticide formulating plant.

Worker 1. 25 y.o. formulator spilled a 76% solution of parathion on his inguinal and scrotal areas and legs. He removed clothes, showered, changed overalls and boots. 2 days later, developed nausea and diarrhea. Went to ED and was treated with Compazine and sent home. **Did not tell the physician of his exposure.**

2 days later, returned to ED with weakness, nausea, and sweating. PE-appeared ill, pupils constricted, nystagmus.

History of parathion exposure was given on this visit.

Admitted and treated with atropine and pralidoxime. Did well and discharged the next day.

(Clifford and Nies)

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Worker 2. 23 y.o. formulator developed N & V 12 days after Worker 1's exposure. Worker 2 was sent to ED since his screening cholinesterase was suggestive of OPP, even though he had not worked with any OP's. In ED, had N, V, LOC, apnea, seizures, and fecal and urinary incontinence. Admitted, intubated, mechanically ventilated, treated with diazepam, phenobarb, atropine, and pralidoxime. He regained consciousness and improved rapidly and was discharged the next day.

Worker 3. 18 y.o. formulator developed nausea and vomiting 15 days after Worker 1's exposure. Worker 3 was sent to the ED where he was diaphoretic and had pinpoint pupils. Treated with pralidoxime 1 g. and did well and was discharged.

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The plant safety officer was very concerned since there were no poisonings over the previous few years and now there were 3 in a 2-week period. Safety measures were strictly enforced. Coveralls used and respirators and gloves were used when indicated. Clothing contaminated with spilled pesticide was bagged and burned.

What happened?

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What Happened?

- Worker 1 immediately changed out of his coveralls and bagged them to be burned
 - **Coveralls were laundered instead of burned**
- Worker 1 wore them subsequently
- After another washing, were worn by Worker 2
- After another washing, were worn by Worker 3
- Analysis of the coveralls showed a very high concentration of parathion. The wash water and other coveralls were found to be contaminated. All coveralls were then destroyed and replaced

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Important Points of Case 3

- Exposure history is most important for the diagnosis
- Pesticide illnesses can resemble other common illnesses
- Physical exam findings can be helpful
- Supportive therapy, especially of the respiratory system, is very important
- Dermal absorption is the most common route of occupational poisoning

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