Poisoning In Children

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Objectives

- Review the initial assessment of the child with a possible ingestion
- Describe the general management principles for ingestions and toxic exposures
- Describe likely presentations for common and/or potentially fatal pediatric ingestions

Introduction

- Since 1960, there has been a *95%* decline in the number of pediatric poisoning deaths
  - child resistant packaging
  - heightened parental awareness
  - more sophisticated interventions

Introduction

- 60% of poison control center calls are for patients under the age of 17
- Most pediatric ingestions are accidental and minimally toxic
- Higher morbidity in adolescent ingestions
- Many pediatric patients present with unexplained signs and symptoms

Initial Assessment: Overview

- Treat the patient, not the poison
- Assessment triangle
  - General appearance
  - Work of breathing
  - Circulation
- ABCDs
- IV access and monitors
- High Suspicion

Initial Assessment: Physical Examination

- Directed exam (after ABCs)
  - mental status
  - vital signs
  - pupillary size
  - skin signs

Initial Assessment: Diagnostics

- Cardiac monitoring or 12-lead EKG
- Chest and abdominal radiographs
- Electrolytes (anion and osmolar gaps)
- Toxin screening rarely helpful
- Specific drug levels

Secondary Assessment

- AMPLE

- A- Allergies
- M- Medications
- P- Past Medical History
- L- Last Po Intake
- E- Events Prior To Presentation

Secondary Assessment

- Obtain detailed history of the amount and time of ingestion
- Use family or friends as historians
- May need to search the home

Prevention or Minimization of Absorption

- Ipecac
  - No longer recommended
- Gastric lavage (also almost never used)
  - massive ingestions
  - arrival within one hour of ingestion

Activated Charcoal

- Ineffective in some ingestions
  - pesticides
  - hydrocarbons
  - acids, alkalis, and alcohols
  - iron
  - lithium

Activated Charcoal

- Recommended dose
  - child under 6 years: 1 - 2 grams/kg
  - 6 years and older: 50 - 100 grams
  - Sorbitol?
    - Hypernatremia
    - Dehydration

Cathartics

- Studies of the effectiveness of cathartics are inconclusive

- Complications related to systemic absorption
  - electrolyte disturbance and severe dehydration
  - neuromuscular impairment and coma

Whole Bowel Irrigation

- Golytely® (PEG-ELS)
  - combination of electrolytes and polyethylene glycol (PEG)
  - 0.5 L/hr for small children and 2 L/hr for adolescents and adults
  - administer for 4 - 6 hours or until effluent is clear
  - useful for ingestions of iron, lithium, and sustained release preparations

Enhancement of Excretion

- Ion trapping
  - Traps weak acids in renal tubular fluid
  - Dose 1-2 mEq/kg every 3-4 hours
  - alkalinization of the urine (goal pH 7-8)
    - salicylates, phenobarbital, TCA

Enhancement of Excretion

- Multiple dose charcoal
  - May cause bowel obstruction
  - phenobarbital, theophylline

- Hemodialysis
  - Alcohols
  - Salicylates
  - Lithium

WHO INGESTS???

High School Social Outcasts

What is ingested?

- Toddler/Preschoolers
  - Most common ingestion: *Acetaminophen*
  - Most common **fatal** ingestion: *Iron*

- Adolescents
  - Most common ingestion: *Acetaminophen*
  - Most common **fatal** ingestion: *Cyclic antidepressants*


TYLENOL®
Acetaminophen

Case #1

- You are called to transport a 16 year old girl after she tells her boyfriend “I took as much Tylenol® as I could”
- Denies other ingestions or medication use
- Ingestion occurred three hours prior

Case Progression

- Patient is anxious, diaphoretic nauseated
- PE reveals a mildly tender abdomen
- HR- 120 RR-20 BP 100/70

DO YOU TRANSPORT???

YES

Case Discussion: Acetaminophen

- Most widely used pediatric analgesic on the market
- Most common ingestion in toddlers, preschoolers and adolescents
- Normal cytochrome P-450 metabolism yields small amounts of free oxidants that are hepatotoxic
  - Glutathione depletion

Case Discussion: Stages

- stage 1 (4 - 12 hours)
  - malaise, nausea, vomiting
- stage 2 (24 - 72 hours)
  - asymptomatic, increasing LFTs
- stage 3 (48 - 96 hours)
  - liver failure, elevated prothrombin time
- stage 4 (7 - 8 days)
  - resolution of liver injury

Stage	Time Following Ingestion	Characteristics
I	½ -24hr	anorexia, nausea, vomiting, malaise, pallor, diaphoresis
II	24-48hr	resolution of above symptoms. RUQ pain and tenderness. Elevated LFT's, INR,PT, oliguria
III	72-96 hr (fourth day)	Peak LFT abnormalities Anorexia, nausea, vomiting, malaise may return.
III	4d-2wk	Death in patients with fulminant liver failure. Resolution of hepatic failure in survivors.
IV	>1 month	Mild liver function abnormalities may persist

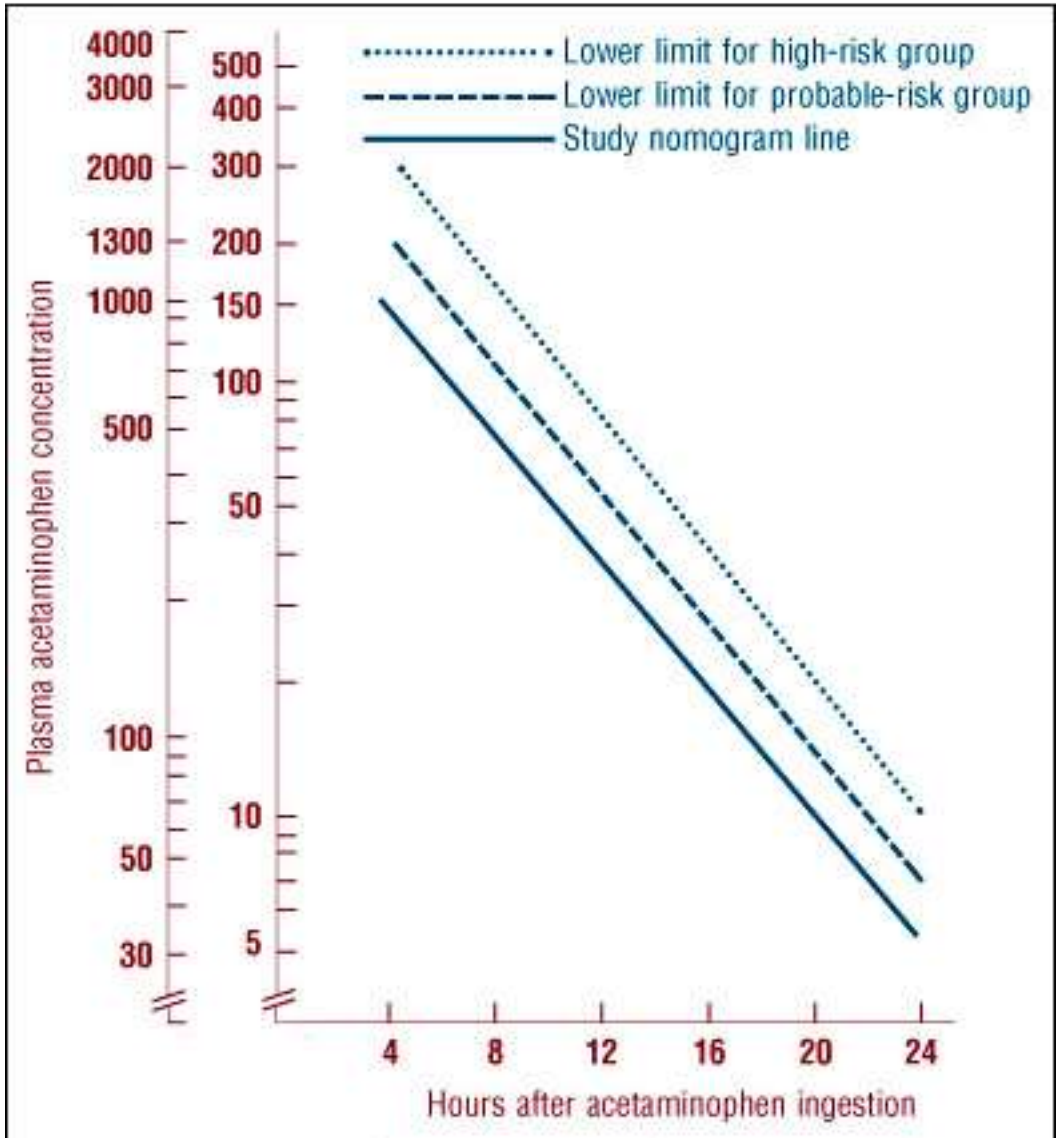
Reference:

Behrman RE, Kliegman RM, Jenson HB (eds): Nelson Textbook of Pediatrics, 17th ed. WB Saunders Company, Philadelphia, 2004

Case Discussion: Diagnosis

- Kinetics dictate that a serum level be checked 4 hours after ingestion
- Toxic dose: 150 mg/kg
- 4 hour toxic blood level 150mg/dl
- Apply the level to the management nomogram

Rumack-Matthew nomogram for predicting prognosis of hepatotoxicity in acetaminophen overdoses.



Our Patient

- Charcoal 50mg
- 4 hour level is 215 μg/ml
- Now What?????

Case Discussion:
N-acetylcysteine (NAC) Therapy

- Proven to be 100% effective when given within 8 - 16 hours of ingestion
- Load with 140 mg/kg orally
- Complete regimen with 17 subsequent doses of 70 mg/kg every four hours

Case Discussion: *N*-acetylcysteine (NAC) Therapy

- IV NAC (Acetadote)
- Load with 50 mg/kg over 4 hours
- Maintenance 100mg/kg over 16 hours

Q

A 16-year-old girl presents after telling her ex-boyfriend that she took 40 Tylenol tablets. She is angry about having been brought to a medical facility and is tearful. She is otherwise asymptomatic and "feels fine." Which of the following statements about this clinical scenario is NOT TRUE?

a) Acetaminophen level should be checked 4 hours after ingestion
b) If the patient is asymptomatic at this time, she is unlikely to have a significant ingestion
c) A psychiatric consult is indicated after the patient is medically cleared
d) Teenagers are more likely to talk about an act rather than actually doing it
e) If acetaminophen overdose is present, N-acetylcysteine is the antidote of choice and may be given IV or PO
f) The liver is the primary organ affected by acetaminophen overdose

Case #2

Case #2

- 12 year old boy was dared by his friends to drink from a bottle filled with antifreeze
- Swallowed a few gulps, and then yelled and dropped the bottle
- His father, utters a few choice words and calls an ambulance

Case Progression

- Upon arrival, the child has clumsy movements with a decreased level of consciousness

- Vital signs: HR 120, RR 20,
  BP 80/50, T 37.4º C, weight 45 kg

- What class of toxin has this child ingested?

Alcohol

- Why can’t we let him ‘sleep it off’?

Case Discussion: Alcohols

- Ethanol
  - hypoglycemia, osmolar gap, ketoacidosis
- Methanol
  - blindness, large osmolar gap, metabolic acidosis
- Ethylene glycol
  - renal failure (calcium oxalate crystals), osmolar gap, metabolic acidosis

Alcohol metabolism

- Ethylene glycol
  - Broken down by ADH to oxalic acid
  - Results in renal failure
- Methanol
  - Broken down by ADH to formic acid
  - Results in blindness

Alcohol metabolism

- Ethanol
  - Broken down by ADH to CO_2 and H_2O
  - Results in DRUNK
- Isopropanol
  - Broken down by ADH to CO_2 and H_2O
  - Results in REALLY DRUNK

Osmolar Gap

- osmolar gap = measured – calculated
- calculated = (2 x Na) + (glucose/18) +(BUN/2.8)
- normal = 10 – 15 mOsm/kg H_2O
- all alcohols cause an elevated osmolar gap

Anion Gap

- [Na + K] – [HCO3 + Cl] > 12
  - M- Methanol
  - U- uremia
  - D- DKA
  - P- Paraldehyde
  - I- Iron
  - L- Lactic Acidosis
  - E- Ethylene Glycol
  - S- Salicylates

Case Progression

- Patient has an osmolar gap and metabolic acidosis consistent with ingestion of ethylene glycol

- Now what?????

Therapeutic Intervention

- IV ethanol (old)
  - competes for alcohol dehydrogenase (ADH) to prevent build up of toxic metabolites
- Fomepizole (4-methyl pyrazole)
  - Blocks alcohol dehydrogenase (ADH)
- Requires ICU admission

Q

The osmolal gap is calculated by subtracting the calculated osmolarity, (2 * Na) + (BUN/2.8) + (glucose/18), from the measured serum osmolarity. An osmolal gap >10 mmol/L suggests the presence of large amounts of osmotically active substances of low molecular weight, including all of the following EXCEPT:

a) glycerol.
b) mannitol.
c) ethylene glycol.
d) ethanol.
e) gasoline or other petroleum distillates.
f) isopropyl alcohol.

Q

Clinical and laboratory findings with isopropanol poisoning include all of the following EXCEPT:

a) fruity breath odor is common.
b) CNS depression.
c) ketosis without metabolic acidosis.
d) hemorrhagic gastritis with nausea and bloody emesis may occur.
e) Hyperglycemia is a common complication.

Case #3

Case #3

- You arrive at a home where a parent has called 911. You find a 5 year old who is crying and rubbing at his arms yelling “get the bugs off me.”
- T-102, HR- 150, RR-23, BP- 100/60
- Skin is flushed, pupils are dilated and extremities are warm and dry.
- His neuro exam is nonfocal
- What toxidrome?

ANTI-CHOLINERGIC

You decide to????

Case #3

a. Transport to the nearest ED with lights and sirens
b. Tell the mom her child is hallucinating and call psychiatry
c. Run away- you are deathly afraid of insects
d. Transport to a medical facility after astutely recognize that this child likely took a large dose of benadryl

Toxidrome: Anticholinergics/antihistamines

- Mad as a hatter
- Red as a beet
- Dry as a bone
- Hot as a hare
- Blind as a bat

Anticholinergic Toxidrome

- CNS
  - agitation, hallucinations, coma
- Respiratory
- Circulation
  - tachycardia, arrhythmias, hypertension
- Skin
  - warm, flushed, dry
- Eyes
  - mydriasis

Case Progression

- gastric decontamination
  - charcoal, 50 grams
- supportive care
- antidote: physostigmine
  - indications: coma, unstable vital signs
  - 0.5 mg IV (child) or 1 - 2 mg IV (teen)
  - Contraindicated if wide QRS

Case # 4

Case #4

- You are dispatched to a home after a call by a parent whose 2 year old was found with a container of dishwasher detergent in his hands and some around the mouth
- patient is asymptomatic
- physical exam is normal, including oropharynx

Case #4

- What are you going to do?
- Reassure parents and leave them to follow-up with the pediatrician as needed?
- Offer transport to the local ED?

Case Discussion: Caustics

- drain cleaners, oven cleaners, automatic dishwasher detergents
- If pH <3 or >12 = BAD
- *DO NOT LAVAGE, GIVE ACTIVATED CHARCOAL, GIVE CATHARTICS OR GIVE IPECAC*

Caustics

- Acids
  - Coagulation necrosis
  - Stomach injury
- Alkali
  - Liquefaction necrosis
  - Oropharyngeal and esophageal injury

Caustics

- Dilution
  - Water
  - Milk
  - Saline
  - Give within 30 minutes

Caustics

- Can your PE predict injury?

NO!!!!!

Q

- Management of the patient with caustic ingestion includes all of the following EXCEPT:

a) Respiratory distress is usually due to upper airway edema. In this case, intubation should be performed early under direct visualization.
b) Activated charcoal should be given upon arrival to the ED.
c) Immediately give water or milk to drink.
d) Give steroids (prednisone 2 mg/kg per day) for deep discrete burns and circumferential esophageal burns.

Q

- The most frequent delayed complication of alkali (e.g., lye) ingestion is:

- upper airway obstruction
- esophageal stricture
- esophageal or gastric perforation
- duodenal atresia

Q

All of the following are true statements regarding the effects of a significant caustic ingestion EXCEPT:

a) Dyspnea or hoarseness may result from upper airway edema.
b) Caustic burns may cause dysphagia, odynophagia, and drooling.
c) Absence of oral lesions rules out significant esophageal injury and the need for endoscopy.
d) Dyspnea, hematemesis, metabolic acidosis and shock can occur.
e) All choices listed are true.

Case #5

- Grandma says her 18 month old grandson “isn’t acting right”

- Grandmother is concerned that child may have ingested some of her medication
  - Digoxin
  - Furosemide
  - “some kind of” antihypertensive medication

Case Progression

- Examination reveals lethargic child with 1 - 2 mm pupils

- vital signs: HR 70, RR 12, BP 80/45, T 37º C, weight 13 kg

Case Progression

- 1 - 2 mm pupils- miosis
- HR- 70- bradycardia
- RR- 12- bradypnea

Which medication?

- Digoxin?
- Furosemide?
- Other Antihypertensive?
- Opiate?

Case Discussion: Clonidine

- central acting antihypertensive; also used to treat narcotic withdrawal
- comes in small tablets and in patch form
- low blood pressure (after transient hypertension), miosis, coma
- naloxone may work to reverse respiratory depression

Clonidine

- Always be ready to support breathing

- Rapid decline

Opiate/Clonidine Toxidrome

- CNS
  - lethargy, seizures, coma
- respiratory
  - slow respirations, pulmonary edema
- circulation
  - hypotension, bradycardia
- eyes
  - miosis


Casoline

Case #6

- 3 year old boy who drank from a soda bottle containing gasoline
- Cried immediately, gagged and coughed, and then vomited
- Alert and crying. HR- 122, RR-24, BP-90/60
- You arrive on the scene…do you transport?

Case Discussion: Hydrocarbons

- Degreasers, solvents, fuels, pesticides, and additives in household cleaners and polishes
- Low surface tension allows for rapid movement through pulmonary system
- Toxic effects
  - pulmonary, cardiovascular, or systemic

Case Discussion: Management Issues

- Admit all symptomatic patients and obtain ABG, EKG, and CXR
- Absence of symptoms for 4-6 hours after ingestion makes chemical pneumonia unlikely
- Ipecac? **NO!!**
- Steroids? **NO!!**
- Prophylactic antibiotics? **NO!!**

Case #7

- A 5 year old girl was at school, when she developed
  - Nausea
  - Vomiting
  - bloody diarrhea

Case #7

- Patient reports that she ate some of her mother's prenatal vitamins at breakfast

- The bottle had contained 30 pills of ferrous sulfate, and is now empty

Case Discussion: Iron

- Toxic exposure is based on elemental iron load

- Most children's preparations contain less iron than adult preparations
  - children's: 3 - 25 mg per pill
  - adult: 37 - 65 mg per pill

Case Discussion: Iron

- Toxic dose: 40-69 mg/kg elemental iron

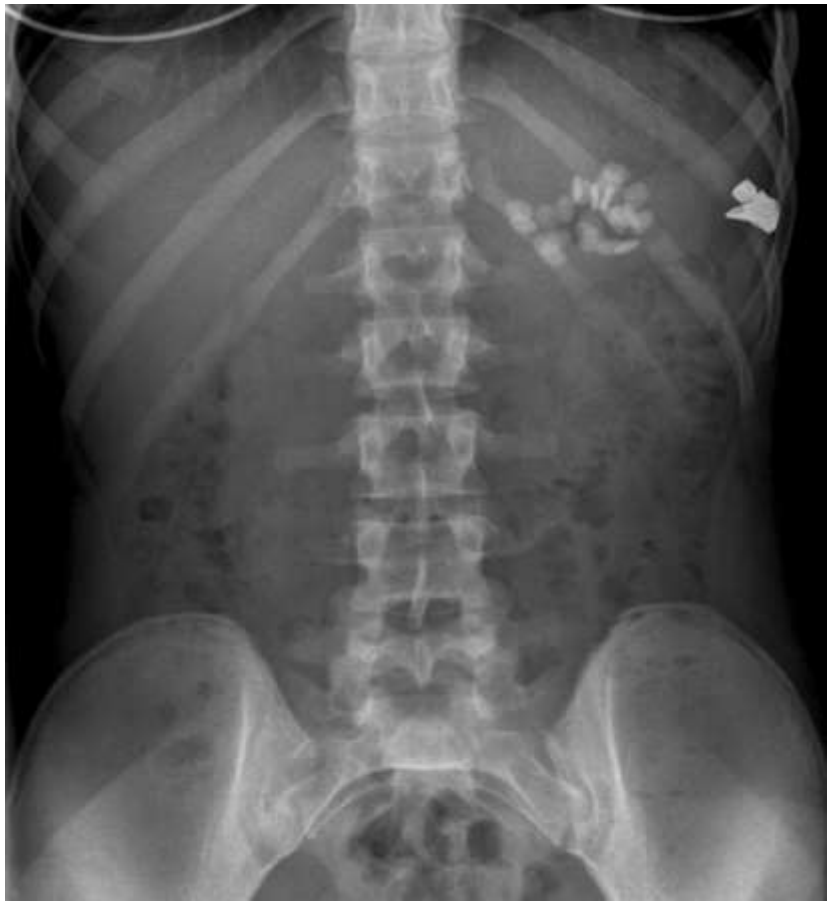
- Lethal Dose: 180 mg/kg elemental iron

Case Discussion: Clinical Presentation

- Gastrointestinal stage (30min-6h)
  - nausea, vomiting, and bloody diarrhea
- Relative stability (6-24h)
  - apparent clinical improvement
- Shock stage (12-48h)
  - coma, shock, seizures, coagulopathy
- Hepatotoxicity stage (within 48 hours)
- GI scarring (4-6 weeks)

Case Discussion: Management

- AXR- iron tablets are radio-opague



Case Discussion: Management

- Whole bowel irrigation
  - 500cc/hour (children) 1-2L/hr (adults)
  - Effluent=Influent
- Deferoxamine
  - Serum fe >500mcg/dl
  - Significant clinical toxicity
  - Persistent XR findings despite GI decontamination

Q

Serum iron levels usually peak 2-6 hrs after ingestion. Which of the following statements regarding the treatment of iron poisoning is incorrect?

a) Administer activated charcoal within one hour of ingestion.
b) With toxic ingestions, iron pills may be visible on x-ray.
c) Patients remaining asymptomatic for 6 hours after ingestion may be discharged with appropriate follow-up for psychiatric evaluation.
d) Treat hypotension caused by hemorrhagic gastroenteritis with crystalloid fluid and blood transfusions.

Q

All of the following are true about the use of deferoxamine EXCEPT:

- Indicated for serum iron >500-600 microgram/dL, and all patients showing signs of serious toxicity such as shock and acidosis.
- Rapid infusion is indicated in the sickest of patients.
- Dosages of 10-15 mg/kg/h by constant infusion are well tolerated in most cases.
- Deferoxamine therapy will turn the urine orange or pink, termed "vin rose urine." Therapy can be discontinued when the patient's urine returns to a normal color, and the patient's serum iron falls into the normal range.

Case #8

- 6 year old boy who was playing outside and returned to his house with respiratory distress

- You arrive on the seen and you note him to be lethargic, diaphoretic, and in moderate respiratory distress

Case Progression

- Physical exam reveals rales and wheezing in all lung fields with copious oral secretions
- Lethargic with 1 mm pupils
- Vital signs: HR 50, RR 70, BP 90/palp, T 37.8º C, weight 25 kg

Cholinergic (Organophosphate) Toxidrome

- clinical presentation
  - D diarrhea
  - U urination
  - M miosis
  - B bradycardia
  - B bronchosecretions
  - E emesis
  - L lacrimation
  - S salivation

Cholinergic toxidrome- organophosphate poisoning

- ATIONS
  - Salivation
  - Lacrimation
  - Urination
  - Fasciculation
- HEAS
  - Diarrhea
  - Bronchorrhea
  - Rhinorrhea
  - Bradycardia

Cholinergic agents

- Inhibit

ACETYLCHOLINESTERASE

Case Discussion: Management

- REMOVE CLOTHING- Skin decontamination

- Atropine (vagal block)
  - Dries secretions, decreases bronchoconstriction and increases heart rate
  - large doses (0.5 - 10 mg IV) may be needed

- Pralidoxime (Protopam, 2-PAM)
  - Regenerates acetylcholinesterase
  - 20 - 50 mg/kg/dose (IM or IV)


NATURE'S
ALCHEMY
Wintergreen
Gaultheria procumbens
100% Pure
ESSENTIAL OIL
.5 oz 15 ml

Case #9

- 3 year old has fever, progressive sleepiness, and respiratory distress 2 hours after drinking some oil of wintergreen from the kitchen cabinet

- Patient noted to be lethargic and tachypneic, with adequate circulation

Case Progression

- Patient responds to mother's voice, and there are no focal findings on neurologic exam
- Vital signs: HR 140, RR 60 and deep, BP 90/70, T 40º C, weight 12 kg
- I stat shows 7.25/25 HCO3-10

What did this patient ingest????

- Hint: Remember your blood gas

- PH: 7.25
- CO2: 25
- HCO3: 10

Salicylates

- Metabolic acidosis with respiratory alkalosis=

- SALICYLATE toxicity until proven otherwise

Case Discussion: Salicylates

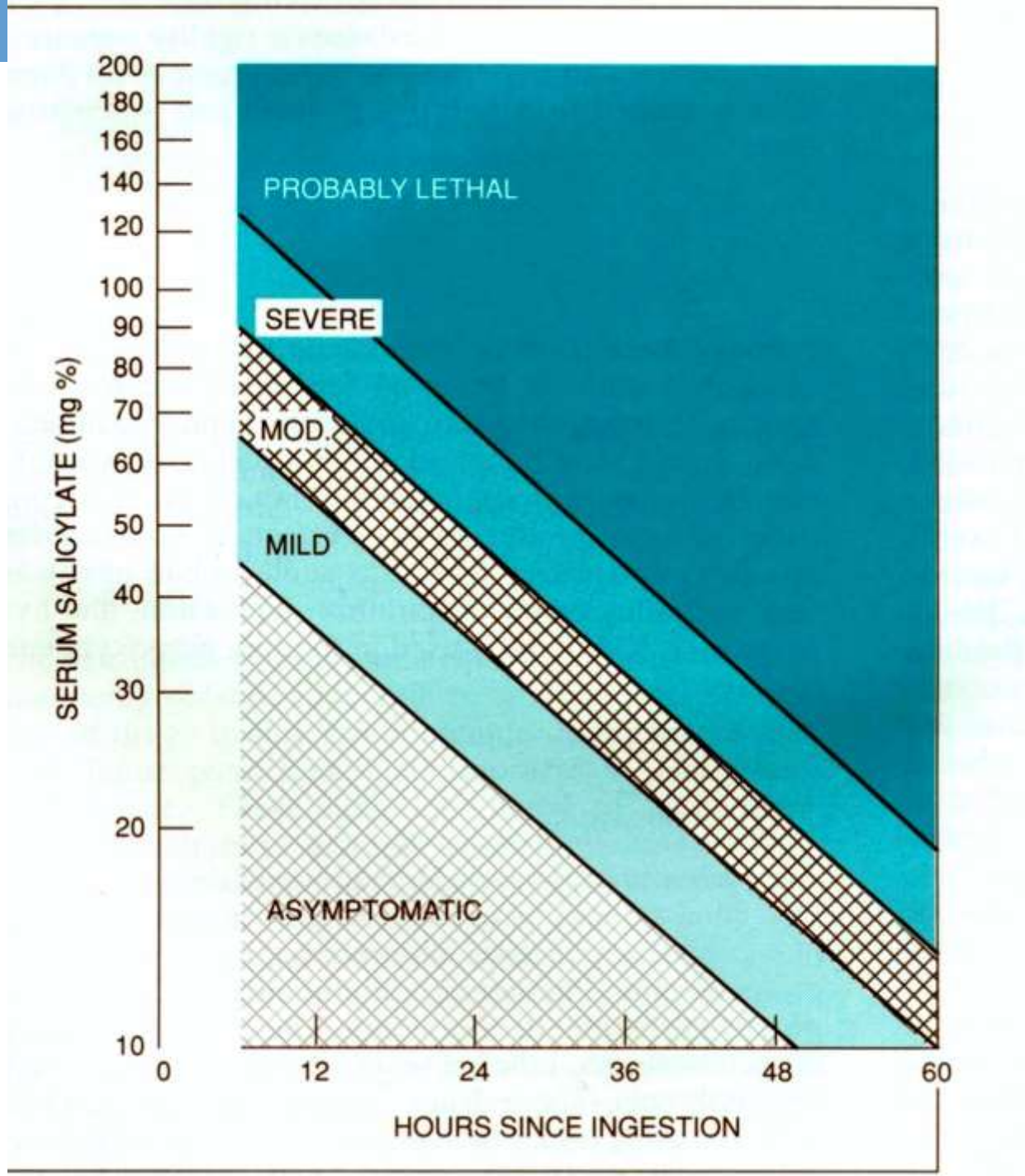
- Respiratory alkalosis
- Increased Temp, HR, RR
- Alters platelet function and bleeding time
- May develop cerebral edema secondary to vasoactive effects
- Tinnitus

Case Discussion: Clinical Manifestations

- Vomiting, hyperpnea, tinnitus, and lethargy
- Severe intoxication: coma, seizures, hypoglycemia, hyperthermia, and pulmonary edema
- Death from cardiovascular collapse

Case Discussion:
Toxic Dose

- Therapeutic dose is 10 - 15 mg/kg
- Toxic dose is over 150 mg/kg
- Done nomogram ONLY useful in acute toxicity


200
180
160
140
120
100
90
80
70
60
50
40
30
20
10
SERUM SALICYLATE (mg %)
PROBABLY LETHAL
SEVERE
MOD.
MILD
ASYMPTOMATIC
0
12
24
36
48
60
HOURS SINCE INGESTION

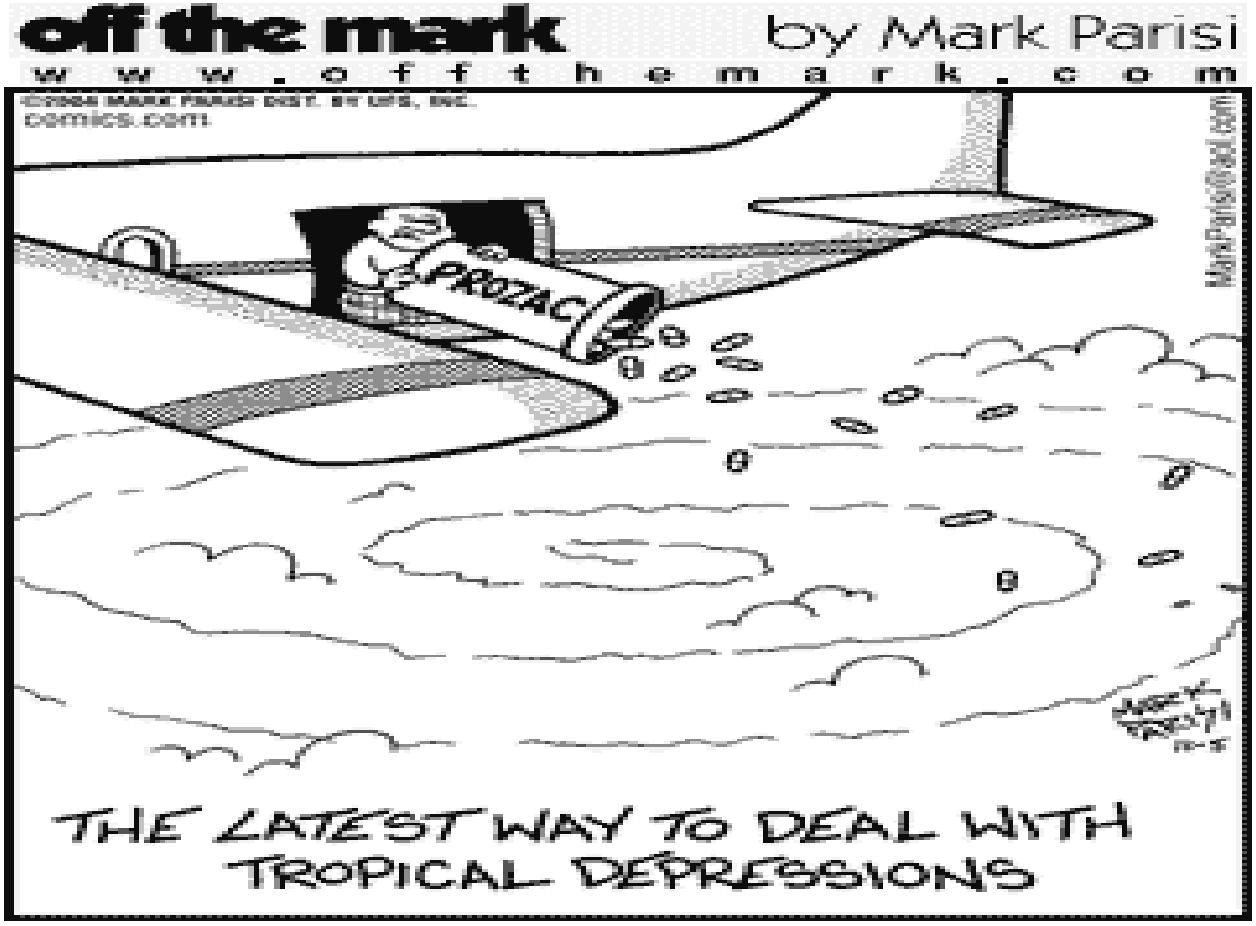
Salicylate toxicity management

- Urinary alkalinization with sodium bicarbonate to maintain urine pH > 7
  - Keeps ASA in renal tubules

Salicylate toxicity management

- Hemodialysis is very effective for drug removal and to control acid-base imbalance
  - Acute ingestions > 100mg/dl
  - Chronic ingestions > 60 mg/dl
  - Persistent rise in ASA
  - Renal insufficiency
  - Refractory metabolic acidosis
  - Altered mental status

There's not enough serenity candles in the WORLD to create silence in MY head!!


off the mark
by Mark Parisi
w w w . o f f t h e m a r k . c o m
comics.com
PROZAC
THE LATEST WAY TO DEAL WITH TROPICAL DEPRESSIONS

Case #10

- Called to transport a 13 year old after her parents arrived home from work to find the patient unresponsive

- Long history of psychiatric problems in the family, including the patient

Case Progression

- VS: T 38°C, HR 120s with widened QRS on the monitor, RR 24, BP 90/50

- Pupils are dilated and reactive, skin is dry and flushed, and patient is responding to deep pain only

Case Discussion: Tricyclic Antidepressants

- Clinical picture is….. anticholinergic intoxication, CNS depression, and cardiovascular instability

- Mainstay of therapy is sodium bicarbonate in addition to supportive measures

Case Progression: Management

- Charcoal, 50 grams after airway secured
- Fluid bolus
- Alkalinization
  - 100 meq/L of $NaHCO_3$
- EKG
  - QRS duration, PR interval, QTc
    - R wave height of > 3 mm in aVR
    - QRS duration of > 120 ms

QRS duration

- QRS > 100ms associated with seizures
- QRS > 160ms associated with cardiac arrhythmia

Case #11

- 2 year old who was found unconscious with empty bottle of grandma's calcium channel blockers at his side

- multiple episodes of vomiting on transport to the hospital, producing pill fragments

Case Progression

- VS: T 37.5°C, HR 45 with third degree heart block, RR10, BP 70/25

- Patient responsive to deep pain only, extremities cool with decreased pulses

Case Discussion: Calcium Channel Blockers

- Morbidity and mortality after toxic exposures result from cardiovascular collapse
- Therapy
  - gastric decontamination (charcoal, WBI)
  - blood pressure support
  - calcium
  - glucagon

Q

Patients with serious calcium channel blocker toxicity generally have ingested doses of at least 5-10 times the normal therapeutic dose. Findings with calcium channel blocker overdose may include all of the following EXCEPT:

a) hypotension
b) atrioventricular block and bundle branch blocks
c) pulmonary edema
d) hepatic failure
e) metabolic acidosis with hyperglycemia

Case # 12

- 15 yo twins are brought to the ED by mom.
- She found them both unconscious in the hallway at home and dragged them out of the house where they both woke up.
- She is now in the ED and they both are alert and appropriate.

Case Progression

- On arrival in the ER, the boys are afebrile with normal vital signs
- O_2 sats of 98%
- CBC, EKG, and CXR are normal

- You are bothered by the fact that both boys had LOC.
- You decide to order a…….
- Carboxy hemoglobin level

Case Discussion:
Carbon Monoxide Poisoning

- CO-hgb affinity is *250 times* O_2-hgb affinity; results in decreased oxygen delivery to the tissues
- Non-irritating, tasteless, odorless, and colorless gas
- Sources: smoke inhalation, auto exhaust, poorly ventilated charcoal, kerosene or gas heaters, and cigarette smoke

Case Discussion: Carbon Monoxide

- Toxic effects are the result of cellular hypoxia
- Concentrations of 20% produce neurologic symptoms, and death can occur with concentrations over 60%
- Pulse oximetry may be normal
- Peak level may occur in the field prior to O_2 delivery

Case Discussion: Therapy

- Administering oxygen at high concentrations reduces half life of CO from 6 hours to 1 hour
- Hyperbaric therapy
  - neurologic dysfunction
  - pregnant women
  - Unstable
  - children with levels over 25%

Q

Treatment for CO poisoning includes all of the following EXCEPT:

a) Administration of oxygen by nasal prongs.
b) Hyperbaric oxygen therapy (100% O_2 at 3 ATA) for patients experiencing confusion, loss of consciousness, seizures, and coma.
c) In cases of carbon monoxide poisoning as a result of smoke inhalation, consider the possibility of cyanide poisoning or methemoglobinemia.
d) Sedation, intubation and hyperventilation with room air.

Q

Which of the following statements are false about the pathophysiology of carbon monoxide poisoning:

a) Carbon monoxide binds tightly to heme proteins, resulting in a decrease the oxygen carrying capacity of the blood.
b) Carbon monoxide inhibits cytochrome oxidase, interfering with the cell's ability to utilize oxygen
c) Carbon monoxide binds to myoglobin, which may result in impaired contractility of the heart
d) Carbon monoxide shifts the oxyhemoglobin dissociation curve to the right, resulting in disrupted oxygen delivery to the tissues

Q

Which statement is true about the interpretation of monitoring in patients poisoned by carbon monoxide?

a) Arterial blood gases show a low P(O_2)
b) Pulse oximetry reading shows desaturation
c) Pulse oximetry shows false high reading
d) Arterial blood gases show an extremely high P(C02) and are diagnostic

Summary

- Most pediatric ingestions are non-life threatening
- Recognition of toxidromes and knowledge of available antidotes MAY assist in the initial management of the poisoned patient, but supportive measures are more likely to be life saving


Have a poisoning Question?
POISON
Help
1-800-222-1222
Call Now!
1-800-222-1222

Initial Assessment: Pupillary Size

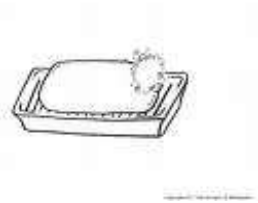
- Miosis
  - C cholinergics, clonidine
  - O opiates, organophosphates
  - P phenothiazines, phenobarbital, pilocarpine
  - S sedative-hypnotics

Initial Assessment: Pupillary Size

- Mydriasis
  - A antihistamines
  - A antidepressants
  - A anticholinergics, atropine
  - S sympathomimetics

Initial Assessment: Skin Signs

- Diaphoresis
  - S sympathomimetics
  - O organophosphates
  - A ASA (salicylates)
  - P PCP (phencyclidine)



Antidotes

- opiates ⇨ naloxone
- acetaminophen ⇨ NAC
- iron ⇨ deferoxamine
- digoxin ⇨ Fab fragments (Digibind®)
- phenothiazines ⇨ diphenhydramine
  cogentin
- organophosphates ⇨ atropine
  pralidoxime

Antidotes

- ethylene glycol, methanol ⇨ ethanol
  fomepizole
- nitrates, dapsone ⇨ methylene blue
- ß and Ca^{+} channel blockers ⇨ glucagon
- carbon monoxide ⇨ oxygen
- isoniazid ⇨ pyridoxine
- cyanide ⇨ amyl or sodium nitrite
  sodium thiosulfate

Antidotes

- sulfonylureas ⇨ glucose
  octreotide
- tricyclic antidepressants ⇨ Na^+ HCO_3^-
- crotalid snakebite ⇨ antivenom
- midazolam ⇨ flumazenil (WITH CAUTION)
- methemoglobinemia ⇨ methylene blue

Clinical Clues: Odor

- Bitter almond
  - cyanide
- Acetone
  - isopropyl alcohol, methanol, ASA
- Oil of wintergreen
  - salicylate
- Garlic
  - arsenic, phosphorus, thallium, organophosphates

Clinical Clues: Skin

- Cyanosis
  - methemoglobinemia secondary to nitrites, nitrates, phenacetin, benzocaine
- Red flush
  - carbon monoxide, cyanide, boric acid, anticholinergics

Clinical Clues: Skin

- Sweating
  - amphetamines, LSD, organophosphates, cocaine, barbiturates
- Dry
  - anticholinergics

Clinical Clues: Mucous Membranes

- Dry
  - anticholinergics
- Salivation
  - organophosphates, carbamates
- Oral lesions
  - corrosives, paraquat
- Lacrimation
  - caustics, organophosphates, irritant gases

Clinical Clues: Temperature

- Hypothermia
  - sedative hypnotics, ethanol, carbon monoxide, clonidine, phenothiazines, TCAs
- Hyperthermia
  - anticholinergics, salicylates, phenothiazines, cocaine, TCAs, amphetamines, theophylline

Clinical Clues: Blood Pressure

- Hypertension
  - sympathomimetics (including phenylpropanolamine in OTC cold meds), organophosphates, amphetamines, phencyclidine, cocaine
- Hypotension
  - antihypertensives (including beta and Ca channel blockers, clonidine), barbiturates, benzodiazepines, TCAs

Clinical Clues: Heart Rate

- Bradycardia
  - digitalis, sedative hypnotics, beta blockers, opioids
- Tachycardia
  - anticholinergics, sympathomimetics, amphetamines, alcohol, aspirin, theophylline, cocaine, TCAs
- Arrythmias
  - anticholinergics, TCAs, organophosphates, digoxin, phenothiazines, beta blockers, carbon monoxide, cyanide

Cinical Clues: Respirations

- Depressed
  - alcohol, opioids, barbiturates, sedative-hypnotics, TCAs, paralytic shellfish poison
- Tachypnea
  - salicylates, amphetamines, carbon monoxide
- Kussmauls
  - methanol, ethylene glycol, salicylates

Clinical Clues: CNS

- Seizures
  - carbon monoxide, cocaine, amphetamines and sympathomimetics, anticholinergics, aspirin, pesticides, organophosphates, lead, PCP, phenothiazines, INH, lithium, theophylline, TCAs
- Miosis
  - opioids, phenothiazines, organophosphates, benzodiazepines, barbiturates, mushrooms, PCP

Clinical Clues: CNS

- Mydriasis
  - anticholinergics, sympathomimetics, TCAs, methanol
- Blindness
  - methanol
- Fasciculations
  - organophosphates

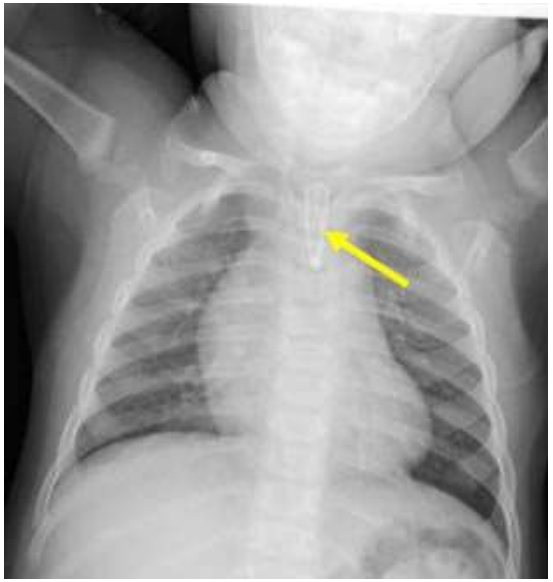
Clinical Clues: CNS

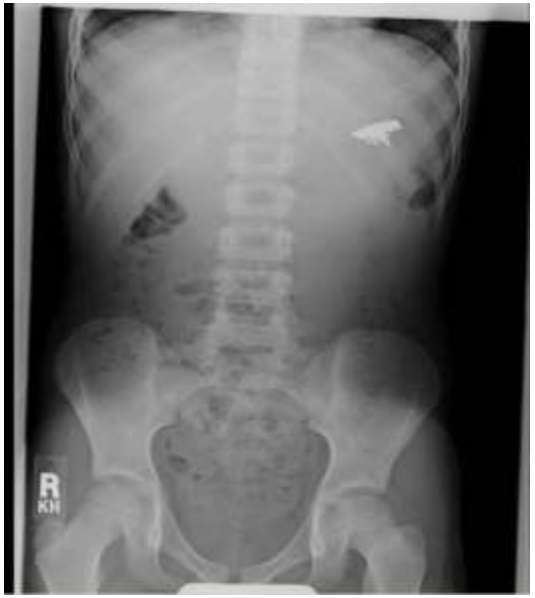
- Nystagmus
  - barbiturates, carbamazepine, PCP, carbon monoxide, ethanol
- Hypertonia
  - antocholinergics, phenothiazines
- Myoclonus/rigidity
  - anticholinergics, phenothiazines, haloperidol

Clinical Clues: CNS

- Delirium/psychosis
  - anticholinergics, sympathomimetics, alcohol, phenothiazines, PCP, LSD, marijuana, cocaine, heroin, heavy metals
- Coma
  - alcohols, anticholinergics, sedative hypnotics, opioids, carbon monoxide, TCAs, salicylates, organophosphates
- Weakness/paralysis
  - organophosphates, carbamates, heavy metals

Pediatric Foreign Body Ingestions

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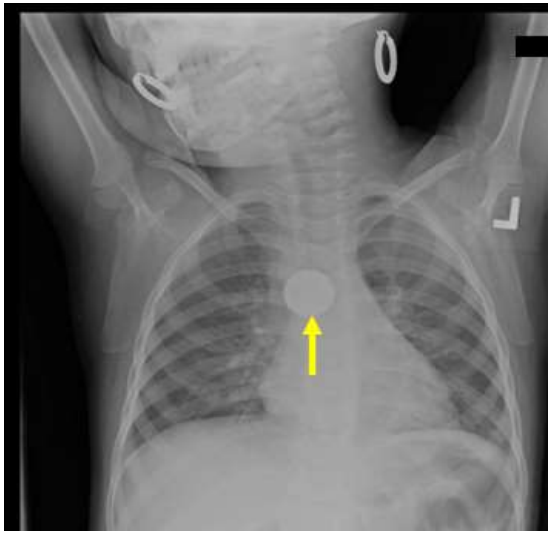
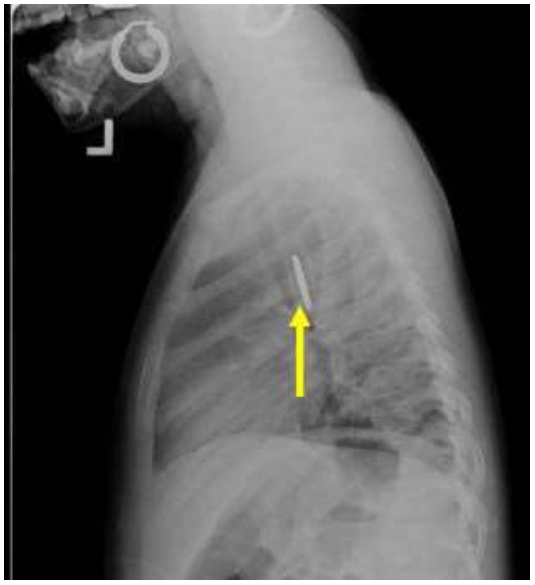
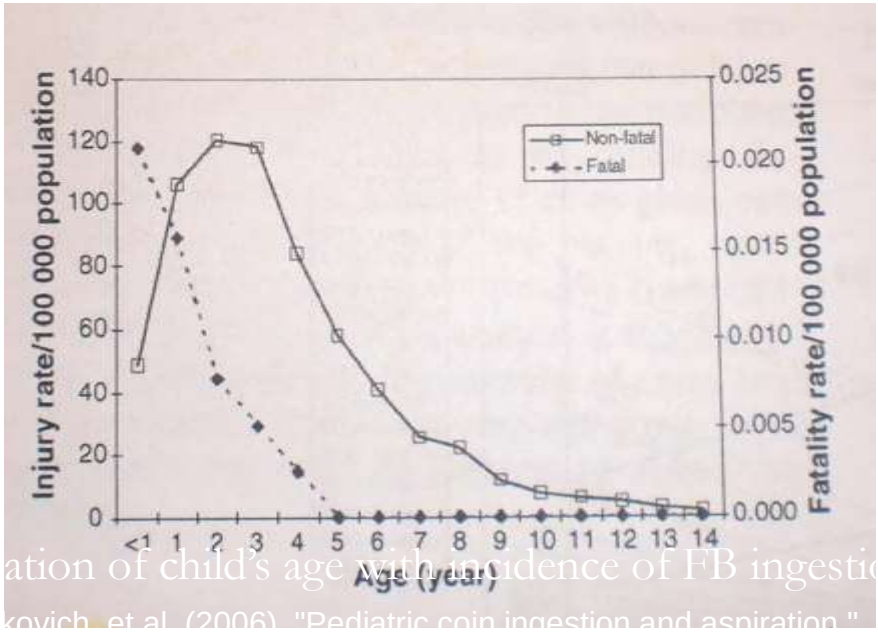




Epidemiology

- Over 100,000 cases of foreign body ingestion reported per year in US. Many go un-reported or un-discovered.
- 80% of cases occur in children and infants, who are prone to sticking objects in their mouth and less able to control their oropharnxy and airways.
- Fatalities have been reported for children under age 4.

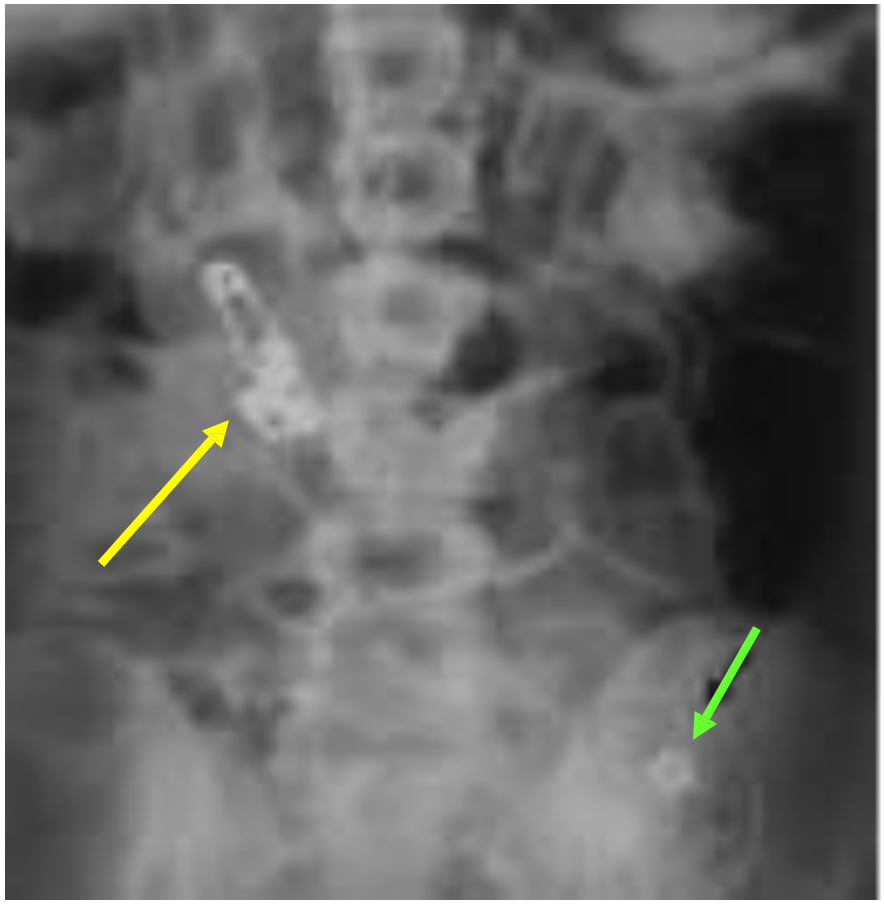


ation of child's age with incidence of FB ingestio

kovich, et al. (2006). "Pediatric coin ingestion and aspiration."

Menu of FB Ingestions

- Frequently found objects include coins (most common), safety pins, batteries, toy parts, magnets, bones.
- Anything a child can possibly grab and swallow is fair game!



FB ingestions by the numbers

- At diagnosis, 60% located in stomach, 20% located in esophagus.
- Older children and male children more likely to spontaneously pass FB.
- 60-90% spontaneously pass when located in distal esophagus or below GE junction.
- Only 10-20% require endoscopic removal.
- 66% of spontaneously passed FB's are never found in stool by parents.
- Previous surgery or congenital malformations (TEF's) increase risk of obstruction and complications.

Symptoms of FB ingestion

- Most are asymptomatic! History is most important clue.
- Symptoms most often associated with location in upper esophagus.
- Acute Esophageal: retrosternal pain, cyanosis, dysphagia, drooling, wheezing, stridor, choking, vomiting, hemoptysis, decreased PO intake, gagging.
- Chronic Esophageal: weight loss, recurrent aspiration.
- Stomach or Bowel: Abdominal pain, bloody stool.

Complications of FB Ingestion

- Aspiration and airway obstruction
- Stricture or fistula formation
- GI obstruction, perforation, or bleeding
- Erosion into esophagus, aorta, or other structures
- Death

Indications for imaging

- Previous recommendations: asymptomatic children tolerating PO intake do not need radiographs.
- However, 20% of asymptomatic patients had an esophageal FB.
- 28% of esophageal coins pass spontaneously within 24 hours.
- Risk of complications increases with esophageal FB.
- Current recommendations: ALL suspected foreign body ingestion patients need radiographs.
- Frontal radiograph of chest, KUB, and lateral radiograph of neck needed to image entire length of GI tract.

Diagnosing Foreign Bodies

- Opaque: glass, most metal except aluminum, animal bones, food, soil.
- Nonopaque: Fish bones, wood, plastics, aluminum.
- Consider CT, US, or oral contrast for non- opaque objects.

Indication for removal of FB

- Patient Symptomatic
- Sharp or long (>5cm)
- Magnet
- Disk battery
  in esophagus
- In esophagus >24 hours
- In stomach >4-6 wks

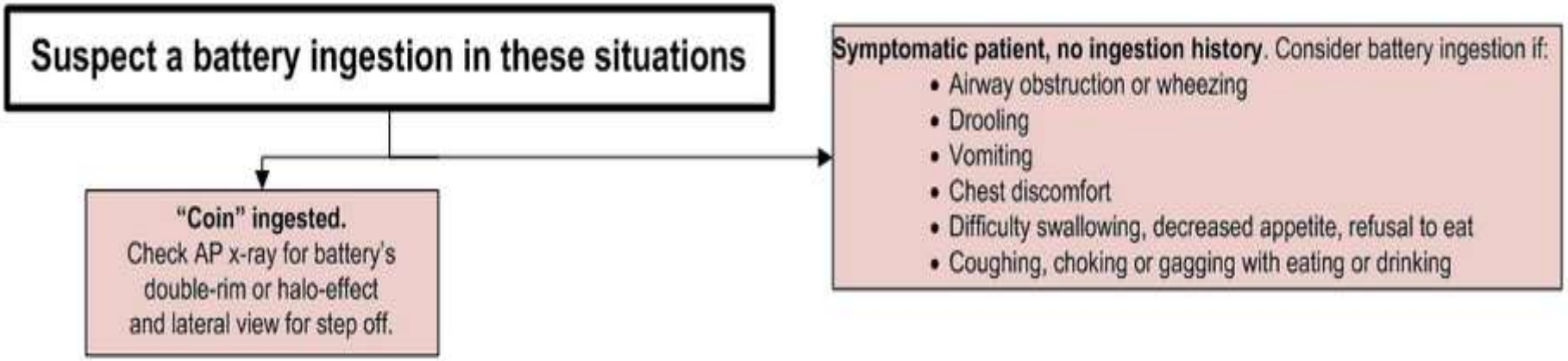
Observation

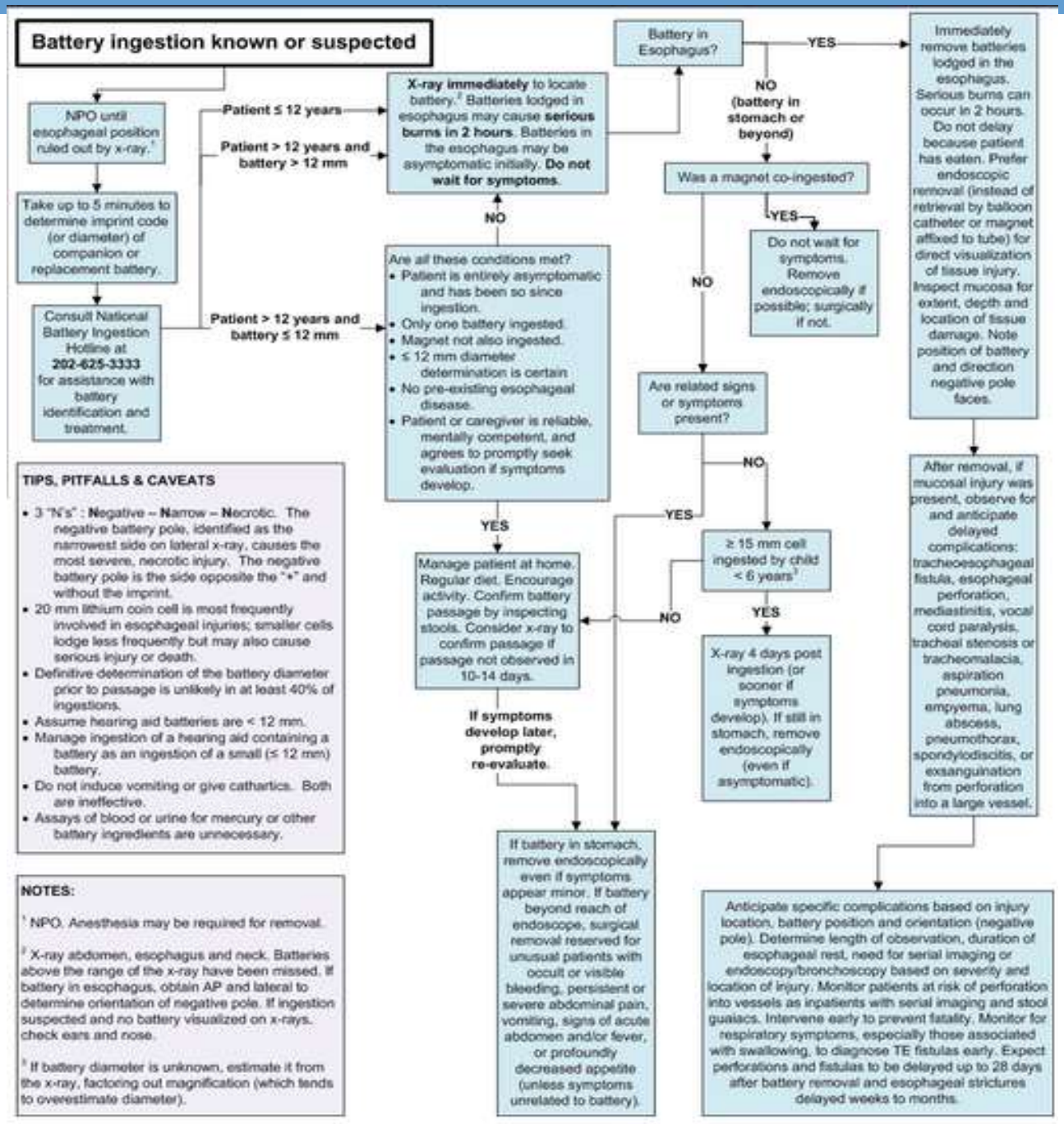
- Acceptable if patient asymptomatic, FB not sharp or long (>5cm), not magnet, not esophageal battery.
- 20-30% of esophageal FB's pass spontaneously.
- Most FB's pass spontaneously after passing the narrow esophagus, pylorus and duodenal sweep.
- Repeat radiograph in 8-16 hours for esophageal FB. Serial radiographs weekly for distal FB until it passes.
- Endoscopic removal of FB if retained in esophagus
  - >16 hours or retained in stomach >4 weeks, or if patient becomes symptomatic.

Special considerations for button batteries

- Higher risk of perforation, erosion, fistula, stenosis if lodged in the esophagus.
- Electricity flow between both battery poles through contact of the tightly surrounding esophageal walls may cause liquefaction necrosis and perforation.
- Leakage of contents: acidic environment may erode seal of battery and release heavy metals and cause necrosis of membranes.

NBIH Button Battery Ingestion Triage and Treatment Guideline

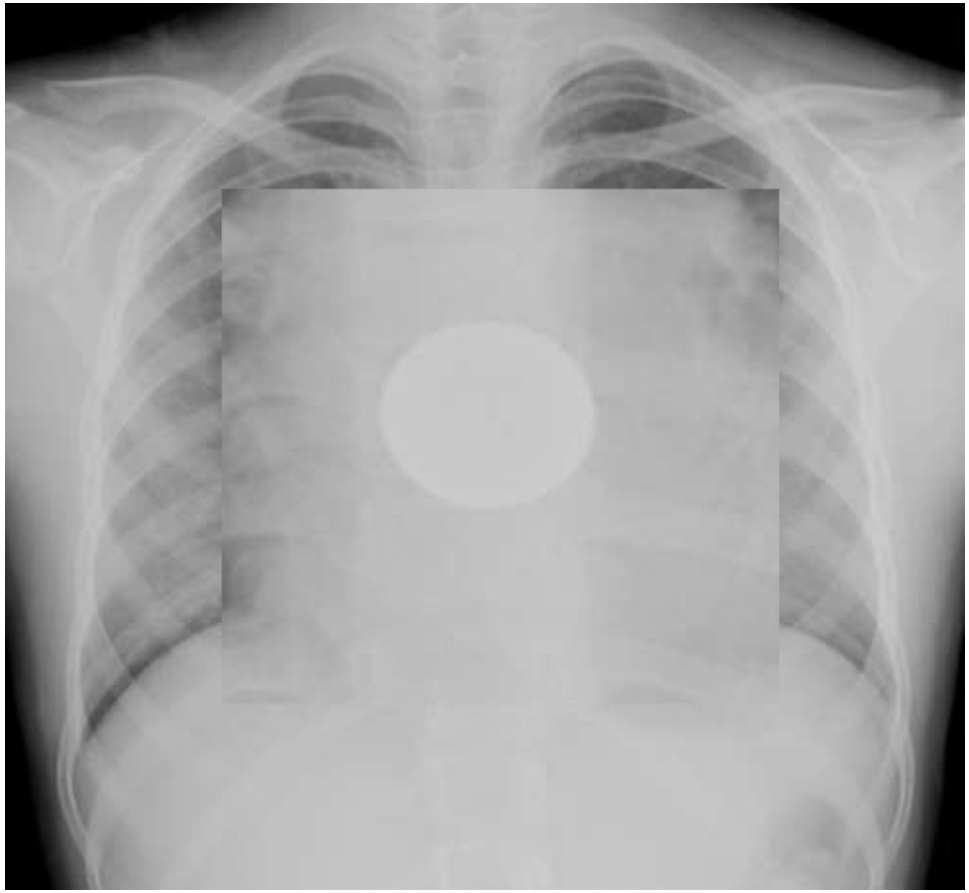


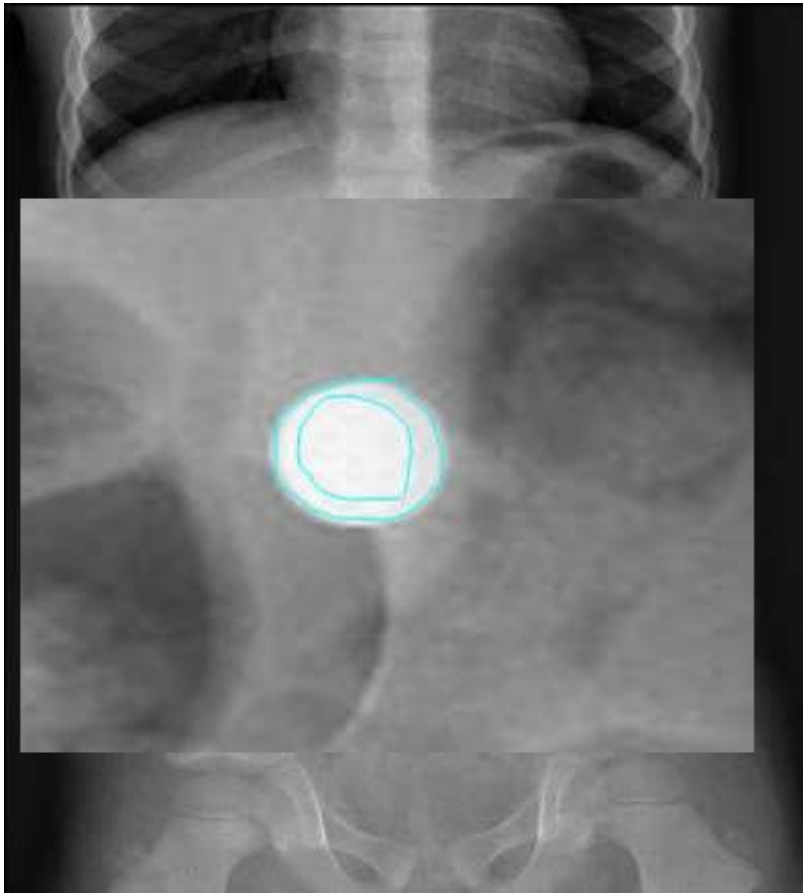

Battery ingestion known or suspected
NPO until esophageal position ruled out by x-ray.[1]
Take up to 5 minutes to determine imprint code (or diameter) of companion or replacement battery.
Consult National Battery Ingestion Hotline at 202-625-3333 for assistance with battery identification and treatment.
Patient ≤ 12 years
Patient > 12 years and battery > 12 mm
Patient > 12 years and battery ≤ 12 mm
X-ray immediately to locate battery.[2] Batteries lodged in esophagus may cause serious burns in 2 hours. Batteries in the esophagus may be asymptomatic initially. Do not wait for symptoms.
NO
Are all these conditions met?
• Patient is entirely asymptomatic and has been so since ingestion.
• Only one battery ingested.
• Magnet not also ingested.
• ≤ 12 mm diameter determination is certain
• No pre-existing esophageal disease.
• Patient or caregiver is reliable, mentally competent, and agrees to promptly seek evaluation if symptoms develop.
YES
Manage patient at home. Regular diet. Encourage activity. Confirm battery passage by inspecting stools. Consider x-ray to confirm passage if passage not observed in 10-14 days.
If symptoms develop later, promptly re-evaluate.
Battery in Esophagus?
YES
Immediately remove batteries lodged in the esophagus. Serious burns can occur in 2 hours. Do not delay because patient has eaten. Prefer endoscopic removal (instead of retrieval by balloon catheter or magnet affixed to tube) for direct visualization of tissue injury. Inspect mucosa for extent, depth and location of tissue damage. Note position of battery and direction negative pole faces.
NO (battery in stomach or beyond)
Was a magnet co-ingested?
YES
Do not wait for symptoms. Remove endoscopically if possible; surgically if not.
NO
Are related signs or symptoms present?
YES
NO
≥ 15 mm cell ingested by child < 6 years[3]
YES
NO
X-ray 4 days post ingestion (or sooner if symptoms develop). If still in stomach, remove endoscopically (even if asymptomatic).
If battery in stomach, remove endoscopically even if symptoms appear minor. If battery beyond reach of endoscope, surgical removal reserved for unusual patients with occult or visible bleeding, persistent or severe abdominal pain, vomiting, signs of acute abdomen and/or fever, or profoundly decreased appetite (unless symptoms unrelated to battery).
After removal, if mucosal injury was present, observe for and anticipate delayed complications: tracheoesophageal fistula, esophageal perforation, mediastinitis, vocal cord paralysis, tracheal stenosis or tracheomalacia, aspiration pneumonia, empyema, lung abscess, pneumothorax, spondylodiscitis, or exsanguination from perforation into a large vessel.
Anticipate specific complications based on injury location, battery position and orientation (negative pole). Determine length of observation, duration of esophageal rest, need for serial imaging or endoscopy/bronchoscopy based on severity and location of injury. Monitor patients at risk of perforation into vessels as inpatients with serial imaging and stool guaiacs. Intervene early to prevent fatality. Monitor for respiratory symptoms, especially those associated with swallowing, to diagnose TE fistulas early. Expect perforations and fistulas to be delayed up to 28 days after battery removal and esophageal strictures delayed weeks to months.
TIPS, PITFALLS & CAVEATS
• 3 "N's" : Negative – Narrow – Necrotic. The negative battery pole, identified as the narrowest side on lateral x-ray, causes the most severe, necrotic injury. The negative battery pole is the side opposite the "+" and without the imprint.
• 20 mm lithium coin cell is most frequently involved in esophageal injuries; smaller cells lodge less frequently but may also cause serious injury or death.
• Definitive determination of the battery diameter prior to passage is unlikely in at least 40% of ingestions.
• Assume hearing aid batteries are < 12 mm.
• Manage ingestion of a hearing aid containing a battery as an ingestion of a small (≤ 12 mm) battery.
• Do not induce vomiting or give cathartics. Both are ineffective.
• Assays of blood or urine for mercury or other battery ingredients are unnecessary.
NOTES:
[1] NPO. Anesthesia may be required for removal.
[2] X-ray abdomen, esophagus and neck. Batteries above the range of the x-ray have been missed. If battery in esophagus, obtain AP and lateral to determine orientation of negative pole. If ingestion suspected and no battery visualized on x-rays, check ears and nose.
[3] If battery diameter is unknown, estimate it from the x-ray, factoring out magnification (which tends to overestimate diameter).

http://www.poison.org/battery/guideline.asp

Button batteries: Beware the "coin fake out"

- Look for "Halo Sign" of button battery

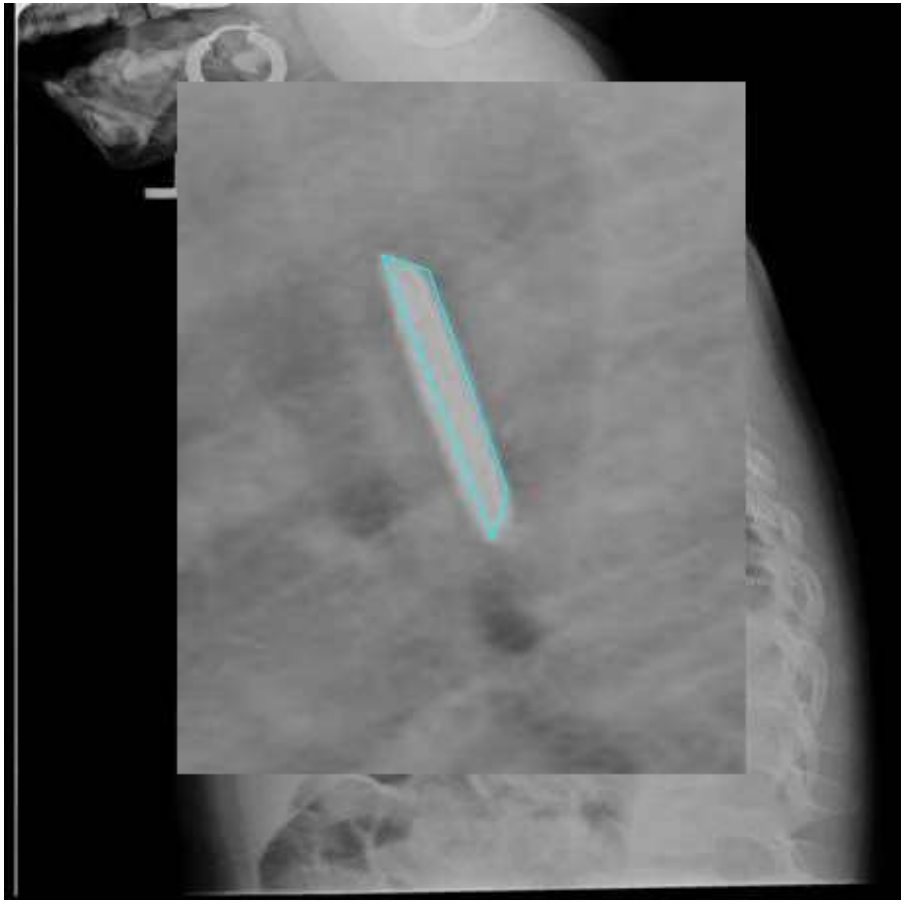




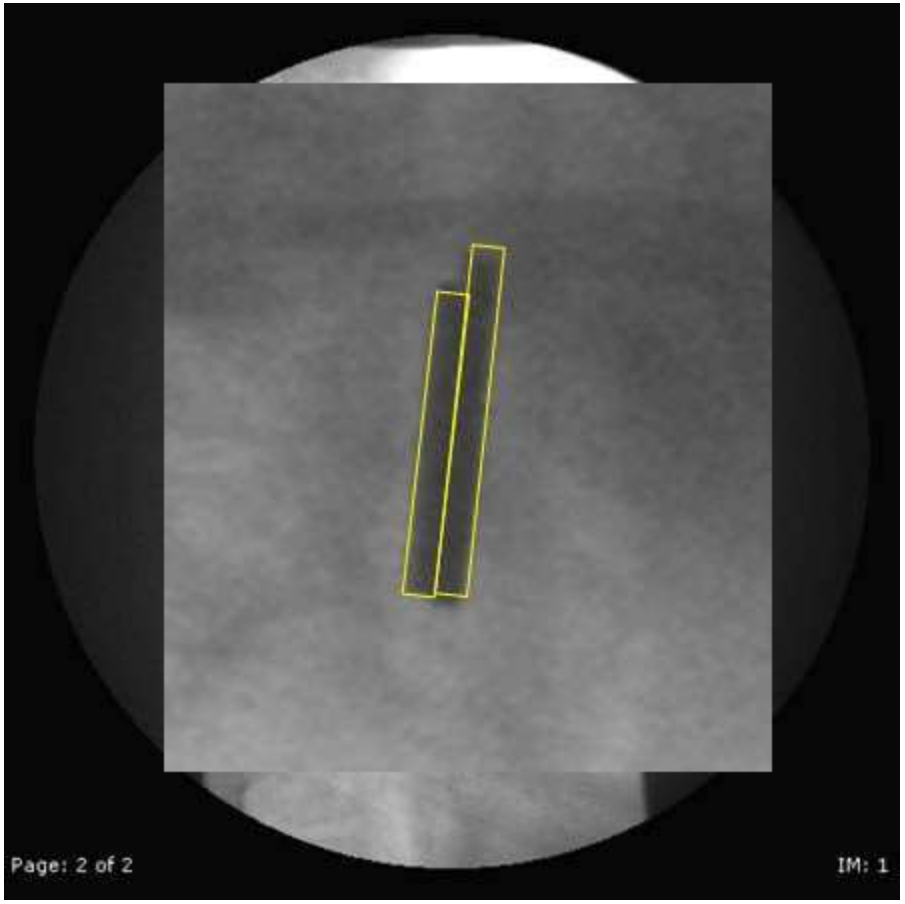
PA CXR of Coin ingestion (left) courtesy of Dr. Booya BIDMC and Upright KUB **Battery ingestion (right)** courtesy of Dr. Waltzman

Button batteries: Beware the "coin fake out"

- On lateral, battery shows "step off appearance of edges" while coin has sharp edges



Lateral CXR of Battery ingestion



Lateral neck fluoroscopy of two coin ingestion

Overview
of
Category A Bioterrorism Agents

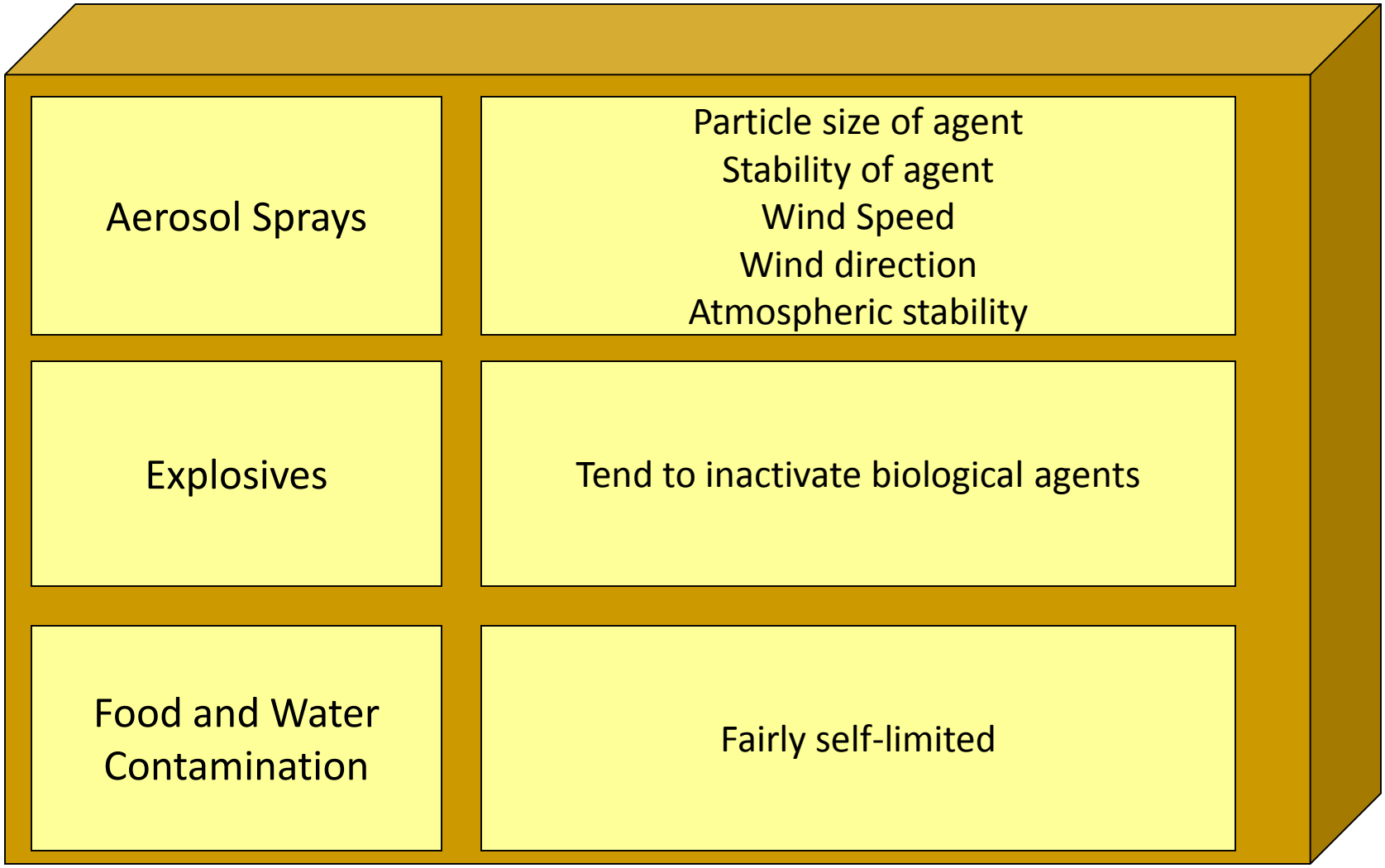
CHARACTERISTICS OF BIOWARFARE

- Potential for massive numbers of casualties
- Ability to produce lengthy illnesses requiring prolonged and intensive care
- Ability of certain agents to spread via contagion
- Paucity of adequate detection systems
- Diminished role for self-aid and buddy aid, thereby increasing sense of helplessness

CHARACTERISTICS OF BIOWARFARE

- Presence of an incubation period, enabling victims to disperse widely
- Ability to produce non-specific symptoms, complicating diagnosis
- Ability to mimic endemic infectious diseases, further complicating diagnosis

Bioterrorism: Modes of Spread



Epidemiologic Clues to Bioterrorism

- Multiple simultaneous patients with similar clinical syndrome
- Severe illness among healthy
- Predominantly respiratory symptoms
- Unusual (nonendemic) organsims
- Unusual antibiotic resistance patterns
- Atypical clinical presentation of disease
- Unusual patterns of disease such as geographic co-location of victims
- Intelligence information
- Reports of sick or dead animals or plants

“Agents Likely to be Used”

- Smallpox
- Plague
- Anthrax
- Botulism
- VEE
- Tularemia
- Q Fever
- Marburg
- Influenza
- Melioidosis
- Typhus

NEXT

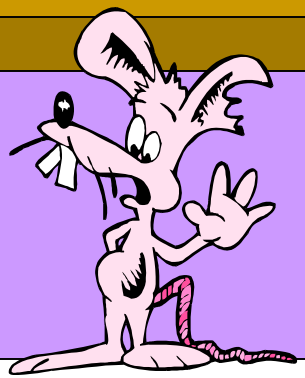
Category A: Highest Priority

- Can be easily disseminated or transmitted person-to-person
- Cause high mortality, with potential major public health impact
- Might cause public panic and social disruption
- Require special action for public health prepardeness

- Smallpox
- Anthrax
- Yersinia pestis
- Botulism
- Tularaemia
- Filoviruses (Ebola and Marburg)
- Arenaviruses (Lassa and Junin)

Category B: Second Highest Priority

- Moderately easy to disseminate
- cause moderate morbidity and low mortality
- Require specific enhancements of CDC's diagnostic capacity and enhanced disease surveillance



- Coxiella burnetti (Q fever)
- Brucella
- Burkholderia mallei (glanders)
- Alphaviruses (Venezuelan encephalomyelitis and Eastern and Western equine)
- Ricin toxin from Ricinus communis
- Epsilon toxin of C. perfringes
- Staph enterotoxin B
- Salmonella
- Shigella
- E. coli O157:H7
- Vibrio cholerae
- Cryptosporidium parvum

Category C: Third Highest Priority

- Pathogens that could be engineered for mass destruction because of availability, ease of production and dissemination and potential for high morbidity and mortality and major health impact

- Nipah virus
- Hantavirus
- Tickborne hemorrhagic fever viruses
- Tickborne encephalitis viruses
- Yellow fever
- MDR TB

Anthrax: Overview

- Gram positive bacillus that forms spores
- Spores found in soil worldwide
- Humans usually infected by contact with infected animals or contaminated animal products

- **<u>No person-to-person transmission of inhalation anthrax</u>**

Anthrax ~ Bacillus anthracis

- Spore-forming bacteria; found naturally in soil worldwide

3 Types of disease:

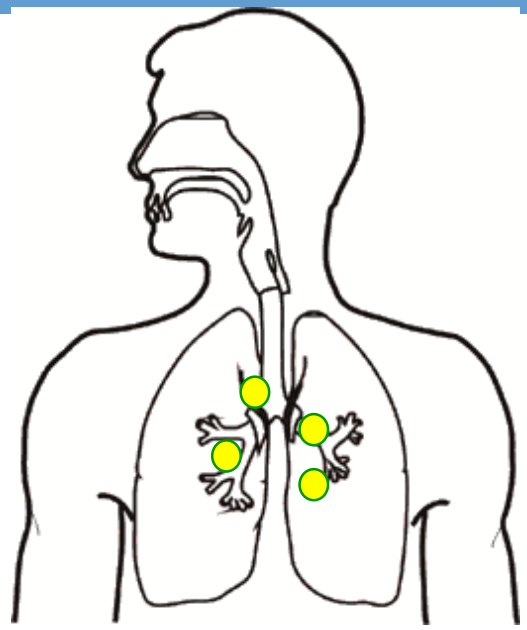
- Cutaneous
  - – most common naturally occurring form
  - skin inoculation with spores from infected animals, hides, wool, etc.
- Gastrointestinal
  - ingestion of undercooked, contaminated meat
- Inhalational
  - inhalation of spores in 1-5 micron particles
  - most deadly form and most likely in BT
  - odorless and invisible

Cutaneous Anthrax

- Incubation period 1-10 days; (usually 5 days)
- Small macule or papule forms ulcer – (day 2)
- Vesicle appears and ruptures – (5-7 days)
- Ulcer dries into black eschar – (1-2 weeks)
- Malaise, low grade fever, lymphadenopathy
- Complications: toxic shock and death within 36 hours in 20% of untreated patients

CDC Public Health Image Library

Inhalational Anthrax

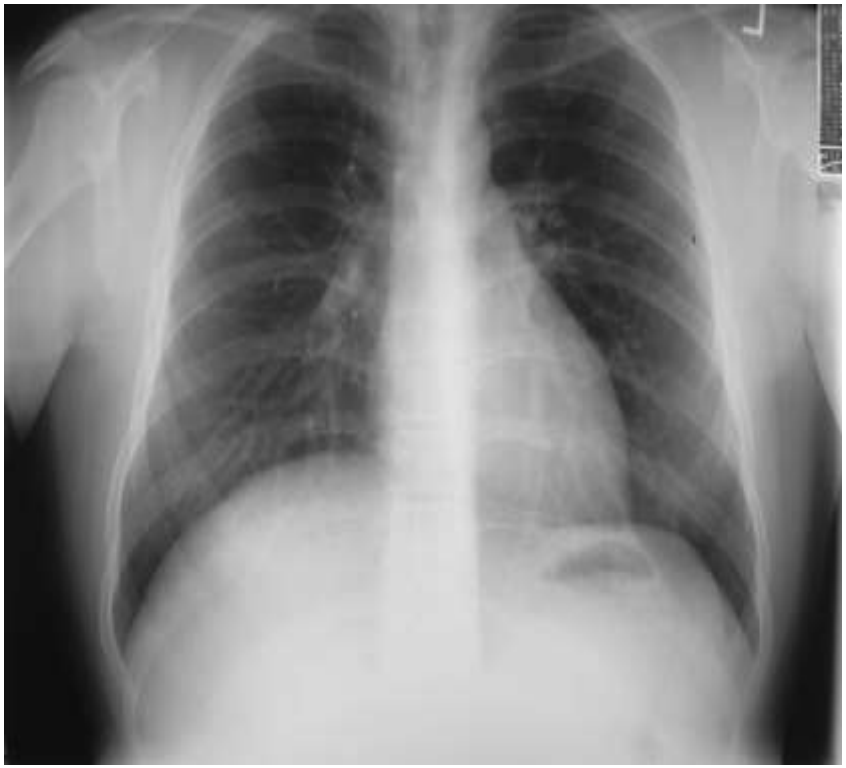


- Incubation period: 1-5 days (up to 60+)
- Inhalation of spores (1-5 microns)
- Infective dose may be quite low
- Fever, fatigue, cough, headache, and chest discomfort
- Severe dyspnea, chest pain, abdominal pain, nausea, vomiting, diaphoresis
- Hemorrhagic meningitis – 50%
- Toxic shock and death within 24-36 hrs

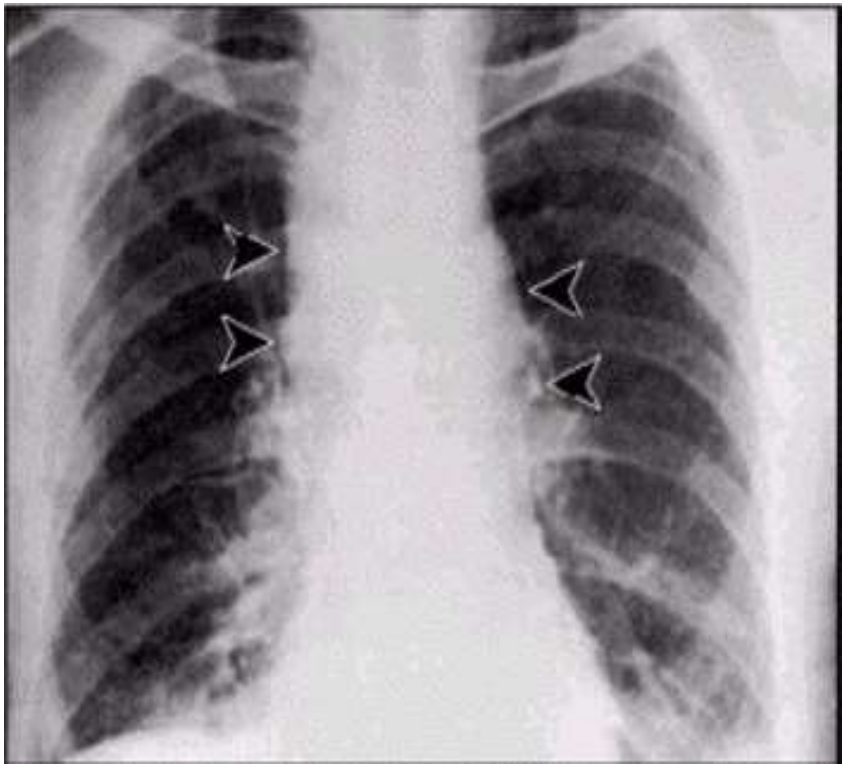
Pathophysiology of Inhalational Anthrax

- Spores are inhaled – taken up by alveolar macrophages which then move to lymph nodes
- Spores germinate, producing edema factor and lethal factor toxins
- Toxins produce local hemorrhagic lymphadenitis and necrosis in the chest (mediastinum)
- Septicemia can result, leading to sepsis and multi-organ failure
- Even with full ICU treatment, mortality is very high once symptoms develop

Inhalational Anthrax



Normal chest x-ray



Mediastinal widening with inhalation anthrax (*JAMA* 1999:281:1735-1745)

Diagnosing Inhalational Anthrax

- Possible history of exposure
- Differential diagnosis: tularemia, staph/strep
- Widened mediastinum/possible pleural effusion on chest xray
- Hemorrhagic mediastinal nodes on scan
- Gram positive bacteria (rods) on peripheral smear
- ELISA test – IgG for Protective Antigen -- rapid results
- Call your state epidemiologist for assistance with collection of specimens and diagnosis

Treatment for Inhalational Anthrax

- For symptomatic patients – IV therapy with two or more antibiotics, depending on sensitivity
- Supportive care (ICU – ventilator)
- Draw labs to confirm diagnosis and initiate therapy immediately – delayed treatment results in worse prognosis

Anthrax ~ Post-Exposure Prophylaxis (PEP)

CDC recommends combined therapy:

- 3 doses of vaccine - investigational new drug (IND)
- Oral antibiotics for 60 days:
  - ciprofloxacin
  - doxycycline
  - amoxicillin or penicillin (if susceptibility testing is supportive)
- Oral antibiotics – before symptom onset
- Vaccine alone is not protective for PEP
- **PEP may depend on numbers of people exposed**

Special Considerations - Anthrax

- Not spread person to person
  - No risk of spreading disease among clinic attendees
  - Minimal PEP needed to protect clinic staff
- Incubation period 1 to 60+ days
  - PEP must be started very early
- Inhaled spores may stay viable inside body for >60 days
  - PEP must be continued for at least 60 days

Special Considerations, Anthrax

- Inhalation anthrax is a DEADLY disease
  - If PEP isn't begun before symptoms arise, prognosis is grave ("worst-case scenario")
  - Need great risk communication to target pop.
- CDC recommendation: oral antibiotics x 60 days + series of 3 vaccinations
- Logistical challenges of delivering materiel on this scale

STEPS IN MANAGEMENT

1. Maintain an index of suspicion
2. Protect thyself
3. Assess the patient
4. Decontaminate as appropriate
5. Establish a diagnosis
6. Render prompt therapy
7. Practice good infection control

STEPS IN MANAGEMENT

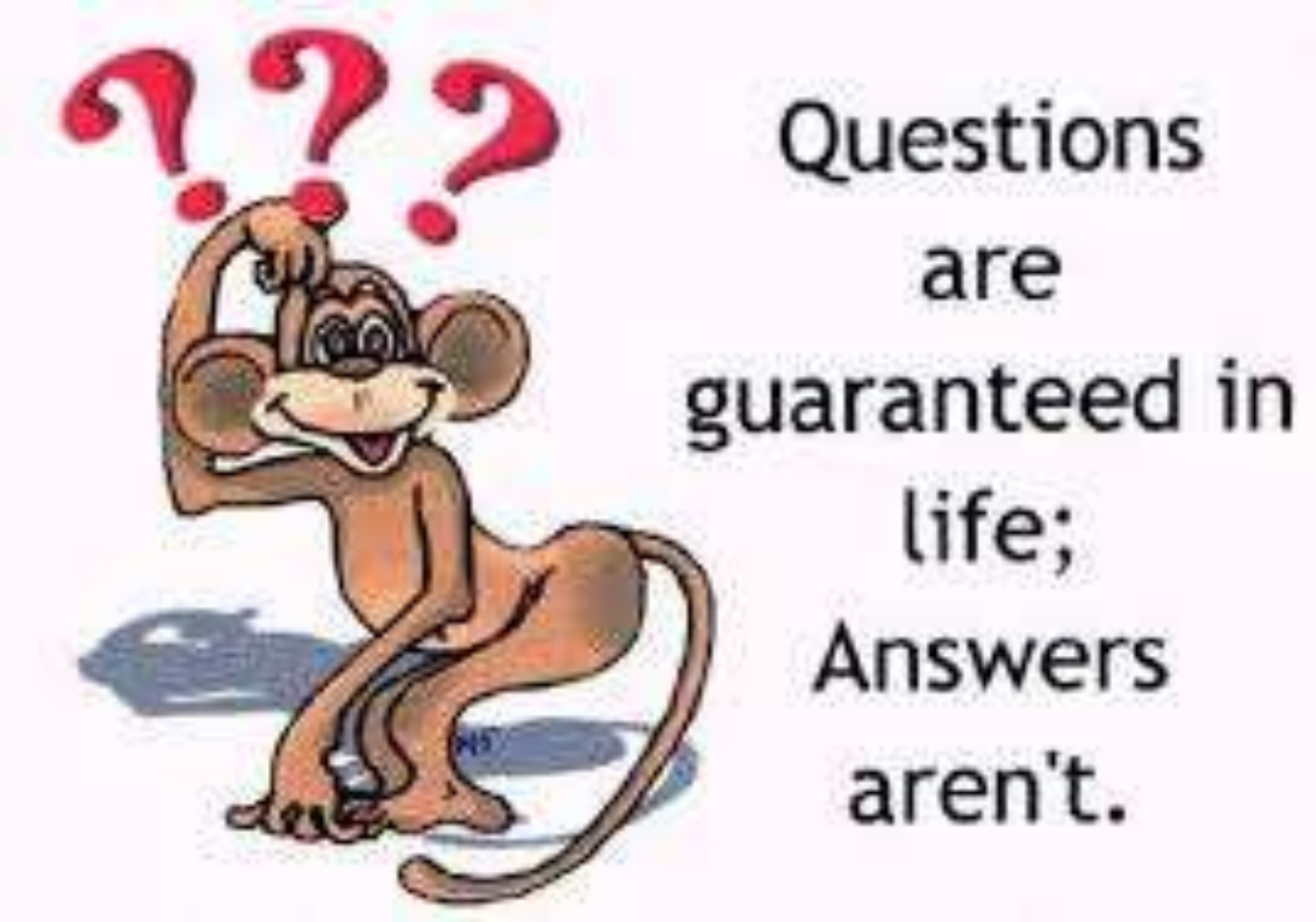
8. Alert the proper authorities
9. Assist in the epidemiologic investigation
10. Maintain proficiency and spread the gospel

US Army, Biologic Casualties Handbook, 2001


prevention


CHILDREN ACT FAST...
POISON
SO DO POISONS...


POISON PREVENTION
POISONS
KEEP
LOCKED
PAINT


Questions
are
guaranteed in
life;
Answers
aren't.