Hot Talks  Cool Tox
The Toxicology Roadshow
The Toxicology Roadshow

Integrative modules:

☠ Visual Stimuli
☠ “One pill can kill”
☠ Antidote quiz
☠ Tox ACLS
☠ Mystery case
☠ Coma case
ONE PILL CAN KILL

Gopalan
One Pill Can Kill

Objectives:

• Identify 10 drugs where 1 pill or 1 sip can kill a toddler
Epidemiology

- US Poison Centre reports 2.5 million calls/year
- 50% occurred in children < 6 years old
- Peak Incidence is between 1-3 years old
- Less than 50 deaths annually
Terrible Twos

- Toddlers explore their world
- Much ends up in their mouth
- Child-resistant containers a major difference

Gopalan
Common Presentations

• Unwitnessed: Suspected ingestion or exposure
• Found playing with pills or with pills in mouth
• Empty bottles
• Missing medications
• Unexplained signs or symptoms
One Pill Can Kill

• Life threatening toxicity or death despite ingestion of only one or two tabs or sips!!!
Alpha-2 Adrenergic Agonists

• Clonidine, naphazoline, oxymetazoline, tetrahydrozoline
Alpha-2 Adrenergic Agonist

• Opioid toxidrome

• Management:
  – ABCs
  – Atropine
  – Fluids, pressors

• Naloxone
Sulfonylureas

- Chlorpropamide, glyburide, glipizide, glimepiride
- Loss of appetite, weakness, lethargy, seizure or coma
- Clinically significant hypoglycemia can occur after 18-24 hours
Sulfonylureas

Oral Hypoglycemics

Pancreas

Octreotide "Antidote"

Insulin

Glucose

D5 1/2 NS

Supplemental dextrose exacerbates overstimulation of pancreas

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Calcium Channel Blockers

- Present with cardiovascular collapse
- Delayed symptoms for sustained release preparations
Calcium Channel Blockers

CCB

Block L-type channels

Pancreas

L-type channel blockage

Decrease inotropy

Glucose

Fuel

Increase inotropy

1 mg/kg insulin

Insulin

Glucose

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Beta-Blockers

- β-blocker
- G protein
- Adenyl cyclase
- Glucagon receptor
- Glucagon
- Increased Ca++
- Increased contractility
- Cardiac myocyte

ATP → cAMP → Increased Ca++ → Increased contractility
TCAs

- Imipramine, desipramine, amitriptylline
- Mortality due seizures, conduction abnormalities, hypotension
- Management
  - Aggressive ABCs
  - Activated Charcoal
  - Sodium Bicarbonate 1-2 mEq/kg IV bolus
  - Benzos
Opioids

• Opioid toxidrome
• Death due to apnea, hypoxia
• Management:
  – Supportive care
  – Naloxone
Lomotil

- Antidiarrheal agent
- Contains 2.5mg of diphenoxylate (opioid) plus 0.025mg atropine
- Biphasic toxicity
  - Initially anticholinergic then delayed long-lasting opioid
- Management
  - Similar to opioids
Salicylates
Salicylates

- Toxic dose in children is 150 mg/kg
- 1 tsp 98% methyl salicylate = 7000 mg salicylate = 90 Baby Asprins = > 4x toxic dose for a 10 kg child
Salicylates

- Present with nausea & vomiting, diaphoresis, tinnitus, CNS, hyperventilation
- Primarily metabolic acidosis
- Management
  - Supportive
  - Activated Charcoal
  - Urine alkalinization
  - Dialysis
Antipsychotics

- Seizures, anticholinergic toxidrome, prolonged QT, wide QRS
- Management
  - Supportive
  - Benzos
  - Sodium bicarbonate
Antimalarials

• Chloroquine, hydroxychloroquine, quinine
• Seizures, prolonged QT, wide QRS
• Cinchonism
  – Quinine toxidrome
  – Hearing impairment and blurred vision

• Management
  – Supportive
  – Benzos
  – Sodium bicarbonate
Anti-arrhythmics

- Qunidine, disopramide, procainamide, flecainide
- Cardiovascular abnormalities
- Management
  - ABCs
  - Sodium bicarbonate
Camphor

Gopalan
Camphor

• Death due to respiratory depression and status epilepticus

• Management:
  – Supportive
  – Benzos
Podophyllin and Colchicine

- GI symptoms, CNS changes
- Pancytopenia
- Management
  - Supportive
<table>
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<tr>
<th>Drug</th>
<th>Minimal fatal dose (per kg weight)</th>
<th>Maximal available</th>
<th>Unit-dose</th>
<th>Number of tablets that can cause fatality</th>
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<td>100 mg</td>
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<td>Imipramine</td>
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<td>Desipramine</td>
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<td>Disopyramid</td>
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<td>Procainamide</td>
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<td>100 mg/kg</td>
<td>1 gr/5 ml</td>
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<td>Methyl Salicylate</td>
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<td>1.4 gr/ml</td>
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<td>Morphine</td>
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<td><strong>Oral hypoglycemic</strong></td>
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<td>Chlorpropamide</td>
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<td>25 mg</td>
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<td>Podophyllin 25%</td>
<td>15-20 mg/kg</td>
<td>1.25 gr/5 ml</td>
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One Pill Can Kill

- Alpha-2 Adrenergic Agonists
- Opioids
- Sulfonylureas
- Lomotil
- Calcium Channel Blockers
- Salicylates
- Beta-Blockers
- Antipsychotics
- TCAs
- Antimalarials
- Anti-arrhythmics
- Camphor
- Podophyllin
- Colchicine
Poisoned Patient

• **Treatment**
  – ABCs
  – Dextrose, naloxone, thiamine
  – Decontamination
  – Enhanced elimination
  – Focused therapy
  – Antidotes
  – Get Tox help

• **Diagnosis**
  – History
  – Physical exam
  – Toxidrome recognition
  – Diagnostic tests

Questions?
Let me pick your brain a little ...

ANTIDOTES QUIZ!
Objectives

• Use vignettes to elicit clinical associations where antidotes may be useful
• Describe specific antidotes and their uses / pitfalls
• While an antidote may be indicated for a specific poison, never forget good old supportive care!
Case 1 - Sleeping Beauty

25M
Found unconscious in the park.
BP 110/70
HR 40 sinus
RR 6
T 35 C
O2 sat N
glc 6
Case 1 - Sleeping Beauty

- What’s the drug?
- What’s the antidote?
- What are the pitfalls?
Case 1 - Sleeping Beauty

- Opioids: 3 major receptors (μ, κ, δ)
- Naloxone: pure competitive opioid antagonist at μ, κ, δ
- Pitfalls?
Case 1 - Sleeping Beauty

• Pitfalls?
  – Not enough naloxone, can start with 0.04 mg; by 2 mg it’s clear if it’s an opioid...
  – Long-acting opioids / methadone
  – Naloxone duration of action is 20-90 min, depending on route of administration and opioid being reversed
  – Can precipitate withdrawal in chronic users
  – Naloxone drip, reassessments.
Case 2 - Intractable Seizures

- 25 year old male from Vietnam
- Being treated for a “lung infection”
- Took “many pills” this morning
- Presents with intractable seizures, AG metabolic acidosis, coma
- CT head -ve
- CXR...
Case 2 - Intractable Seizures
Case 2 - Intractable Seizures

- What’s a potential culprit?
- Antidote?
Case 2 - INH and B6
Case 2 - INH Toxicity

• INH creates functional deficiency of pyridoxine via 2 mechanisms

• Less B6:
  – Impaired activity of B6-dependent systems
  – Less GABA formation --> seizures?
  – Seizures at > 20 mg/kg of INH
Case 2 - INH Toxicity and B6

- For acute toxicity: 1:1 ratio of ingested INH to B6
- First dose 5 g IV in adults as empiric treatment, 1 g every 2-3 minutes
- Most pharmacies won’t have enough
- Can use crushed tabs via NG

- Chronic toxicity: B6 doesn’t reverse hepatic injury, does prevent neurologic toxicity at low dose
Case 3 - My Joints HURT!

75F
Mistakenly took 25 mg of MTX die instead of q week, using NSAIDS+++ New ACEI and HCTZ

Nausea, vomiting, pancytopenia, increased LFT’s ARF
Case 3 - My Joints HURT!

- Methotrexate toxicity?
- What’s the antidote?
Case 3 - My Joints HURT!
Methotrexate = $ slide

FH4 = cofactor in purine synthesis
Methyl donor for TS -> DNA synthesis
Case 3 - My Joints HURT!

• Methotrexate toxicity-  
  – Acute vs. chronic  
  – Small doses vs. errors in dosing of onco meds

• Antidote = Leucovorin = DNA and RNA precursor.

• 1:1 ratio if ingested dose known, 100 mg/m² if unknown dose

• IV administration: leucovorin calcium < 160 mg/min to avoid hypercalcemia
Case 4 - Weak and Dizzy

• 45 year old female with a personality disorder, bipolar, also a history of seizures
• Brought in unconscious post overdose
• Increased liver enzymes, increased ammonia, AG metabolic acidosis, mild hypocalcemia, APAP neg, CT head neg
• What’s the offending drug?
• Antidote?
Case 4 - Weak and Dizzy

• Valproic acid!
• Toxicity: liver, brain edema, high ammonia, AG metabolic acidosis.
• Metabolized in the liver using carnitine for oxidation
• Carnitine depletion = BAD

• **Carnitine for VA is like glutatione for APAP**

• Antidote: L-CARNITINE
Case 4 - Weak and Dizzy

• L-carnitine indications:
  – serum VA concentration > 3125 umol/L
  – hepatotoxicity
  – high ammonia

• 25 mg/kg IV over 30 min with high serum concentration Q6H (max 3g/day)
• 100 mg/kg IV with hepatotoxicity
• Maintenance 15 mg/kg Q4H (max 6 g/day)
• Stop when improvement
Case 5 - Difficult one ... 

- Unknown ingestion of white powder
Case 5 – Here’s a tip ...
Case 5 - Here’s another tip ...

- Contaminated soil, water are the most common sources
- Think of contaminated wells in Bangladesh ... thousands of people got sick ...
OK ... final tip for lovers of French literature!

What did she take?

What is the antidote?
Case 5 - Arsenic Poisoning!

• Metalloid, exists in many forms
• Elemental, gas, organic, inorganic
• Essentially a cellular toxin = make less ATP, less glucose production
• CELL STARVATION --> Multiorgan involvement
• Diagnosis: urine arsenic level
• Pitfalls???
Case 5- Arsenic Pitfalls!

- Beware the lobster! Arsenobetaine and arsenosugars have low toxicity
Case 5 - Arsenic

• Antidote:
  – Chelation therapy:
    • DIMERCAPTOL (BAL)
    • SUCCIMER = oral hydrophilic version of BAL for chronic and subacute toxicity.
  
• Bind arsenic
• Call Poison Control for dosing regimens
Antidote quiz! Summary

- Antidotes are cool, but remember supportive management

- Opioids --> naloxone
- INH --> B6
- Methotrexate --> leucovorin
- Valproic acid --> L-carnitine
- Arsenic --> chelators (BAL, Succimer)
Questions?
TOX RESUSCITATION
Objectives

• Discuss the ACLS guidelines for poisoned patients

• Illustrate resuscitation situations not defined by the ACLS guidelines
TOX-ACLS: Toxicologic-Oriented Advanced Cardiac Life Support

• 2001 ACLS 1st resuscitation guidelines.

• 2005


• 2010.
Other pearls not addressed in ACLS-guidelines

HOW DO PUT ALL THESE RECOMMENDATIONS TOGETHER?
Important principles

• Goal of resuscitation
  – Keep alive until the toxic agent is eliminated from the body

• Primary cellular toxin
  – Special antidote ASAP
Acute intoxication

- Detection and correction of vital functions failures
  - Airways (Intubate)
  - Respiratory (hyperventilate)
  - Cardio-circulatory (fluids; then assess what’s the problem)
  - Neurological (seizures)
  - Thermal (external cooling, rewarming)
  - Metabolic (glucose, electrolytes, pH)

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Tox Threats to
Airways- Laryngeal Obstruction

• Ingestion of corrosives substances
• Inhalation of toxic substances
  – Intubate early if symptoms

• Coma is a threat to airway protection
  – Intubate to decontaminate or wait for loss of airway protection then decontaminate?
  – Neurovitals
Tox Threats to
Ventilation - Metabolic acidosis

• Hyperventilation MUST be maintained
• Before, during & after intubation
• Avoid fatal exacerbation of metabolic acidosis
  – Cyanide
  – Salicylates
  – Toxic alcohols
  – Metformin
# Ventilation

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<th>CNS &amp; Respiratory</th>
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<td><strong>β- blockers</strong></td>
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<td>Calcium channel blockers</td>
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<td>Methanol</td>
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<td>Antipsychotics</td>
<td>Benzodiazepine / GHB</td>
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<tr>
<td>PCP</td>
<td>Carbamazepine, valproate, barbiturates</td>
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Tox Threats to

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Bradycardia

• Atropine
  – Organophosphates, carbamates, nerve gas
  – Doubling doses 2-4 mg

• Digoxin specific antibodies
  – Digoxin related bradycardia or arrest
  – Chinese herbs with digitalis glycosides
Bradycardia

- **Isoproterenol**
  - β- blocker toxicity (OK)
  - Acetylcholinesterase toxicity (NO)

- **Transcutaneous pacing**
  - Mild to moderate bradycardia (NO)

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Drug-induced Hypertensive Emergencies

• 1st choice = benzodiazepine
  – decrease effect of endogenous catecholamine release

• 2nd choice = nitroprusside (short-acting)

• 3rd choice = cautiously labetalol (mix α et β antagonist)

• β-blocker can worsen hypertension
Drug-induced ACS

- Cocaine or other sympathomimetics
- Reversal of cocaine-induced vasoconstriction
  - 1\textsuperscript{st} agent = benzodiazepine and nitroglycerin
  - 2\textsuperscript{nd} agent = phentolamine

- Labetalol no effect, propanolol worse
- Fibrinolytics higher risk than benefit
Drug-induced ACS

• Instability and polymorphic VT
  – Unsynchronized high energy defibrillation

• Stability
  – Lidocaine 1st choice monomorphic VT after sodium bicarbonate - effectiveness not demonstrated.
    – ANY type 1A or 1C is contraindicated (antagonize Na channel)
    – Phenytoin for TCA not recommended
Drug-Induced VT and VFib

- Torsade de pointes
- Magnesium sulfate even if serum [Mg] is normal
  - Overdrive pacing at 100-120 bpm
  - Isoproterenol as pharmacological overdrive
Drug-Induced Shock

- Hypovolemic shock
  - FLUID CHALLENGE and verify preload
  - Vasopressors
- Distributive shock
  - Vasopressors; ? vasopressin
  - Avoid dobutamine and isoproterenol - worsen SVR
- Cardiogenic shock
  - Mix of agents: no specific recommendation
  - Amrinone, insulin, phenylephrine, sodium bicarbonate
Circulation – VFib after HF burn

• Severe hypocalcemia

• Defibrillation alone might not work

• Aggressive calcium repletion
  – 60 ml of calcium gluconate 10% Q 2 minutes until ROSC

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Circulation - Theophylline induced SVT

• Bad prognosis
  – Arrhythmias
  – Hypotension
  – Convulsions

• Only hope is early hemodialysis

• β-blockers can be useful as a bridge while organizing for dialysis (metoprolol, esmolol)
Circulation - Wide QRS

• Cocaine and other Na channel antagonist:
  – Sodium bicarbonate
  – Sodium bicarbonate
  – Sodium bicarbonate
  – Epinephrine ?
  – Lipid emulsion (a few case reports)
Digoxin

- **DIGOXIN and other cardiac glycosides**
  - There are no data to support the use of specific antidotes in digoxin-induced cardiac arrest.

- **Severe life-threatening toxicity**

- **K > 5.0 mEq/L**

- **Empiric dosage**
  - 10 vials for acute ingestion
    - (380 mg)
    - 5 is probably enough
  - 5 vials for chronic intoxication
    - 2-3 probably enough
  - 5 extra vials extra if no response after 20 minutes.
Cocaine

- Cardiac arrest

  - Standard ACLS

- Wide complex tachycardia

  - Sodium bicarbonate

  - 1 mEq/kg of 8.4% IV

  - REPEAT until QRS < 120 msec
Local Anesthetics

- Lipid emulsion
- Weinberg protocol
- 1.5 mL/kg of 20%
- Maximum 8-12mL/kg

- **BMV hyperventilation**
- **Epinephrine**
- (Have it in your crash cart)
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<th>Tachycardia &amp; HTN</th>
<th>Bradycardia</th>
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<td>Withdrawal states</td>
<td>Aconite</td>
<td>Any Na channel antagonist</td>
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Think Differently

• Standard ACLS originally for ISCHEMIC event
  – can CPR alone overcome pharmacological toxicity?
  
  – **Aggressive** alkalinization of serum
  – Digoxin-specific antibodies
  – ECMO if available in your centre.
  – Hydroxocobalamin +/- thiosulfate
  – Lipid emulsion
Drug-Induced Cardiac Arrest

- Prolonged efforts
- Good outcomes with CPR 3-5 hours
- ECMO has been used successfully
Post resuscitation care

• Decontaminate
  – more toxin might be lurking in the GI track
• IV fluids
  – check preload with US IVC filling
• Urinary output
• Serum levels (APAP, ASA, EtOH)
• Dialyse what you can
• **Call your Poison Control**
Questions?
Mystery Case

- 34 year old 70 kg male arrested at airport for “bizarre behaviour”
- Triage vitals: BP 238/124, HR 126, RR 34, T 41.8 C, SaO$_2$ 98%
- PERLA 7mm, diaphoretic, very agitated
- Too agitated to give any coherent history
- Supplemental oxygen, cardiac monitor
- Too agitated for intravenous access attempt!
First Treatment???
Case

- Still extremely agitated
- Dangerous to himself and staff
- Can’t get EKG or bloodwork
Case

- Successfully intubated with propofol 180mg and rocuronium 50mg (after midazolam 20 mg IV x 3)
- Propofol infusion at 75 ug/kg/min
- Generalized tonic-clonic seizure as rocuronium wears off
- Management options?
Seizing!

- (On Propofol 75ug/kg/min and Midazolam 5mg/hr)
- Midazolam boluses + increase infusion
- Propofol boluses + increase infusion
- NO Dilantin!

- Barbiturates
- Inhaled anesthetics
Case

- RN concerned – rectal temp now 42.3 C
- Management?
Case

• Propofol 75 ug/kg/min, Midazolam 5mg/hr
• Rocuronium infusion started

• Esophageal thermometer placed
• Cooling blanket + ice to groins, axillae and neck
Case

• BP 242/128, HR 136 sinus tachycardia
• ST depression leads V2-V4
• Beta blockade?
• Cardiology unlikely to be helpful at this point
Whole Bowel Irrigation

- PegLyte 500 cc/hr
- Increase to 2L/hr
Case

- Bolus 15mg Midazolam (no downside as airway protected), increase infusion to 15 mg/hr
- BP falls to 198/86 after several minutes
- ASA, clopidogrel, heparin given
- Hydralazine 10 mg X 2 → BP 142/68
Case

- BP 128/74, HR 68 sinus
- ST changes resolve
- PERLA 5 mm
- Esophageal temp 37.6 C

- Admit to ICU and provide advice to them!
Questions?
APPROACH TO THE COMA PATIENT
Case 1

- 3 year old male – ventilated with BVM
- Today - otitis media – amoxicillin
- First dose – vomit – promptly gave second dose
- Lethargic and unresponsive

- Healthy
- Meds - none
Case 1 continued ...

• Vital signs upon EMS arrival:
  – BP 80/55, HR 150, RR 6, SaO$_2$ 88% on RA
  – Note that SaO$_2$ becomes 99% with BVM

• There are no signs of trauma

• The child is flaccid and being bagged

• What is your initial management of this patient?
Poisoning Treatment Algorithm

1. Stabilize vital functions: Airway, Breathing, Circulation
   - Appropriate monitoring
2. If mental status depressed administer oxygen, glucose, thiamine and naloxone
3. Obtain history and perform physical examination
   - Identify agent(s) and/or toxidromes
Apply methods to decrease absorption of toxin

Obtain general labs and specific drug identification or levels as indicated

Use ancillary tests as needed

Continuous reevaluation

Administer symptomatic and supportive care

Perform enhanced elimination and institute antidotes

Disposition

Thurgur
Remember ...

- For the majority of patients with a toxicologic cause of coma, it is the clinical picture that guides treatment – NOT the identification of the particular causative substance.
The Toxicologic Physical Exam
Adjuncts to the Physical Exam

• IV for potential resuscitation
• Laboratory tests
• 12-lead ECG
• Odours or skin colour or track marks?
• Radiographic evaluation
JUST SAY NO TO DRUG SCREENS
Toxicologic Causes of Coma

- Anti-epileptics
- Barbiturates
- Benzodiazepines
- Beta blockers
- Calcium channel blockers
- Chloral hydrate
- Carbon monoxide
- Cyclic antidepressants
- Ethanol
- GHB
- Hypoglycemics
- Lithium
- Opioids
- Organophosphates
- Toxic alcohols
Toxicologic Coma Pearls

• Universal antidotes

• Key vital signs

• Say no to urine drug screens

• General approach to the Tox patient
Questions?