“ACLS sucks for EMS/ED”
- Examples: amiodarone vs. bretylium
- No EMS providers and only two ED physicians involved with creating standards
- Then did “ACLS-EP” to keep advanced people from being bored with renewals
  - Added “enrichment” including environmental emergencies (by a bunch of cardiologists who know squat about EMS or emergency medicine.)
  - Luckily, the tox section was by emergency physician and toxicologist Thomas G. Martin, used to be in the Montefiore ED (but no prehospital experience)

Learning Objectives
- some important BAD drugs/toxins
- important toxidromes
- Tox for which to modify “standard ACLS”
- Best Rx for these ODs
- Prearrest Rx that prevent arrest

Case 1
- You are in ED; EMS arrives: “48-year-old man—suicidal OD—looks bad”
Friend: “OD on 2 kinds of pills, got weak and drowsy, called 911”

2 hours ago

VS in ED: HR=30 BP=50/palp, RR=10 (shallow)

MSE: obtunded, minimal response

- **Primary ABCD Survey?**
- **Secondary Survey, recheck ABCD?**
- **Airway:** intubate?
- **Breathing?**
- **Circulation?**
  - ECG: profound bradycardia, idioventricular rhythm
    - Start 2 large-bore IVs, begin fluid challenge (“10 to” 20 cc/kg) = 1 liter
    - Give “Coma Cocktail” as part of “C”:
      - DONT regimen: Dextrose, Oxygen, Narcan, Thiamine
      - D5W, IV
      - Narcan 2 mg IV (less if IV drug user: 0.2 to 0.4 mg IV)
      - Thiamine 100 mg IV (fugedabudit)
- **Develop a differential diagnosis; decontaminate, define toxidrome, drug-specific therapy**

- **Differential Diagnosis**
  - Friend presents 2 empty bottles:
- Verapamil (calcium channel blocker)
- Propranolol (β-blocker)
- Correct diagnosis?
- What else can cause profound bradycardia, low BP?
- Are you certain that this patient has been poisoned?
- Did the patient respond to the coma cocktail?
  - Acute poisoning?
  - Drug withdrawal?
  - Central nervous system pathology?
    - Meningitis, abscess, encephalitis
    - Seizure (ie, generalized tonic)
    - CVA, hematoma, contusion
    - Sepsis?
- **toxidrome**
  - Bradycardia (± AV block)
  - Hypotension
  - Altered level of consciousness
  - Hyperglycemia/hypoglycemia
- **Treatment**
  - Ventilate
  - Coma cocktail → check response → intubate if needed
  - transcutaneous pacemaker
  - Fluids:
- Start 2 large-bore IVs
- Start wide open; check BP and HR q 5 to 10 min
- Monitor volume; do not overload!
- Start pressors if needed. Which one? How much?
  - **Standard ACLS**
    - Atropine — often not effective
    - Isoproterenol alone — may drop pressure more
    - Pacemaker (external or internal)
    - Dopamine
    - Epinephrine
  - **Toxicologic Approach**
    - High-dose pressors (ultra)
    - Class IIb (possibly useful): calcium, glucagon, insulin pump, amrinone, circulatory-assist devices
      - Glucagon: 5 to 10 mg IV bolus: 3 to 5 mg/h drip
      - Calcium: 1 to 3 g, slow IV bolus
      - Insulin/glucose drip: 20 U reg bolus, 0.5 to 1.0 U/kg per hour drip; glucose 25 g bolus, 20 g/h
      - Amrinone: 0.75 mg/kg 2 to 3 min; 5 to 10 mg/kg per minute
    - Consult a medical toxicologist
    - If at high risk for death: consider heroic therapies
Hemodialysis/hemoperfusion not useful!
Intra-aortic balloon pump
Extracorporeal life support “bypass”

Case 2
- 30-year-old woman; brought into ED by EMS; Boyfriend reports recent anxiety, stress; empty bottles of Ativan and whiskey found

Primary ABCD Survey
- Airway: open; saliva accumulating; gurgling; smell of alcoholic beverage on breath
- Breathing: respiration = 8 sonorous; “decreased gag”
- Circulation: BP = 80/50, Sinus tach @ 130 bpm
- Defibrillation: VF not present

Secondary ABCD Survey
- Airway: clearly needs intubation performed
- Breathing: ETT placement confirmed 2 ways
- Circulation:
  - 2 large-bore IVs, begin fluid challenge
- Develop differential diagnosis; note major findings:
  - ↓ LOC, RR, and BP
- ↑ HR
- Not consistent with benzodiazepine OD!

**Drug-Specific Therapy**
- DONT: coma cocktail is non-specific: D50W, Oxygen, Narcan, Thiamine

**Benzodiazepine antagonist**
- Flumazenil (Romazicon) blocks BZD binding to GABA receptor; 0.2, 0.3, 0.5, 1.0 slowly, up to 3 mg
- **Adverse effects**
  - BZD withdrawal (mild ➔ severe)
  - Removes masking of seizures by BZDs from coingestants (eg, from TCAs)
  - Unmasks ventricular arrhythmias
- **Contraindications for Flumazenil**
  - History of seizures
  - Recent myoclonus or seizure episode
  - Known addiction to short-acting BZDs
  - Heavy/long-term BZD abuse
  - Coingestion of epileptogenic drugs (TCAs most common)

**Critical Points**
- Hemodynamic compromise unexpected in BZD OD; when observed search for another cause
- Specific central BZD receptor antagonist is available: flumazenil
- May unmask seizures or arrhythmias
- Not recommended if
  - Any recent or remote seizure-like activity
  - Short-acting benzos
  - History of heavy benzo abuse
  - Coincidence of epileptogenic drug (TCAs, cocaine)
  - Not indicated in coma of unknown etiology

- **Case 3**
  - 23-year-old zoology graduate student; application rejected by medical school; “Dumped” by girlfriend 2 days later
  - Took bottle of “some sort of mood lifter” 3 hours ago
  - Friend discovered him somnolent, unarousable, “barely breathing”
  - 911 called; transported by EMS unit

- **Primary ABCD Survey**
  - Not in full cardiac arrest
  - Multiple episodes of nonsustained VT occur
  - One shock converts episode of sustained VT
Secondary ABCD Survey
- Needs intubation and hyperventilation
- Oxygen, IV, monitor, fluid challenge
- Continuous grand mal seizures begin

Toxidrome for TCA OD
- Anticholinergic effects
  - Hyperthermia, blurred vision, flushed skin, hallucinations, tachycardia, status seizures
  - Quinidine-like effects
- Negative inotrope, prolonged QT, ventricular arrhythmias (e.g., torsades de pointes)
- α-Adrenergic blockade effects
  - Hypotension
- CNS effects
  - Seizures, coma
- Mild-to-moderate toxicity
  - Drowsiness → lethargy
  - Slurred speech
  - ↑BP, ↑HR
  - Hypoventilation
- Severe toxicity (rapid progression)
  - Coma
  - Seizures
  - Tachy → Arrhythmias
  - Hypotension

TCA OD Rx
- Systemic alkalinization (achieve and sustain)
- Target pH: 7.45 to 7.55 (nl. 7.35)
- Bicarbonate: superior to hyperventilation

**Complications: severe TCA toxicity**
- Marked conduction disorders (QRS >120 ms)
- Marked tachycardia/bradycardia
- Ventricular arrhythmias
- Significant hypotension
- Seizures or coma

**Nurse hands you 12-lead: ? EKG #1**
- Drugs that induce torsades
  - YES: terfenadine (Seldane), astemizole (Hismanal)
  - NO: loratadine (Claritin), cetirizine (Zyrtec), or fexofenadine (Allegra)
  - Erythromycin, Class IA and III antiarrhythmics, TCA, phenothiazines, pentamidine, cisapride

- Symptoms of torsades
  - Dizziness, syncope, palpitations, sudden death

- Risk factors
  - Excess drug (iatrogenic or intentional)
  - Drug-induced impaired elimination
  - CYP3A4 inhibition (imidazoles, macrolides, SSRIs, protease inhibitors, cimetidine, grapefruit,
quinine, zafirlukast, zileuton), hepatic failure
- ↓K, ↓Mg, heart disease, congenital long-QT syndrome

- **Treatment of Torsades**
  - Correct ↓K, ↓Mg
  - Overdrive pacing
  - Pacemaker (external or internal)
  - Isoproterenol — best avoided
  - Antiarrhythmics
    - Magnesium or lidocaine?

- **TCA OD EKG Effects**
  - Typical ECG changes
  - Prolonged QRS and QT intervals
  - Rightward terminal QRS-axis deviation
  - Bundle branch block
  - AV blocks
  - Ventricular arrhythmias
  - Pulseless electrical activity

- **TCA Poisoning Summary**
  - Alkalinize severe cases — bicarbonate is best antidote!
  - Use high-dose pressors when necessary
  - Avoid procainamide and physostigmine
  - Stop TCA seizures with bicarb, benzos, Dilantin
➢ Use circulatory-assist devices in cases refractory to maximal medical therapy
➢ If cardiac arrest occurs, perform prolonged CPR:
  ▪ Buys time for drug to dissipate
  ▪ Good neurologic outcome possible
  ▪ Good heart; good conduction system

❖ Case 4
➢ 30-year-old man; police say he “swallowed a handful of rocks”
➢ Complains of “crushing” chest pain; shortness of breath for 1 hour
➢ Pale, diaphoretic, clutching chest, agitated, delirious
➢ HR = 140, BP = 160/120, RR = 30, T = 39°C

❖ Primary ABCD Survey
➢ ABCD: clear

❖ Secondary ABCD Survey
➢ A: No intubation required
➢ B: Oxygen given
➢ C: IV fluids, monitor, cool him down
➢ D: Develop a differential diagnosis; decontaminate, define toxidrome, drug-specific therapy

❖ Hyperdynamic/Hyperadrenergic Agents
➢ Cocaine (crack)
➢ Ketamine/phencyclidine (PCP)
- Amphetamine/methamphetamine (ice, crystal meth)
- Ephedrine (ephedra) and derivatives, β2-agonists
- Caffeine, nicotine, theophylline
- Dextromethorphan

**Adrenergic Agent OD Complications**
- Angina/infarction
- Dissecting aorta
- Seizures
- Intracranial bleed
- Rhabdomyolysis

**Major Problems to Consider**
- Monitor shows ventricular tachycardia: symptomatic?
- High blood pressure: urgency vs emergency?
- Chest pain: r/o angina; cocaine-induced AMI? vs dissection
- Agitation and delirium
- Hyperthermia: increases delirium
- Cocaine: associated with lowered seizure threshold

**Critical Actions**
- Reduce the hyperdynamic state
- Monitor/reduce temperature
- Physical cooling: spray and fan
- Dantrolene 1 to 10 mg/kg
- Prevent or treat seizures
- Restrain to prevent harm
Chemical restraints preferred over physical restraints
- Agents: BZDs, haloperidol, or droperidol

- Drug-Induced Acute Coronary Syndrome Treatment
  - Benzodiazepines: first-line agents
  - Nitrates (NTG)
  - Phentolamine (Regitine): α-blocker
  - Avoid propranolol (or other pure beta blockers): leaves unopposed α-adrenergic stimulation; may ↑BP
  - IV thrombolytics: Caution! PTCA preferable to systemic thrombolytics

- Case 4 EKG: (see EKG #2)
- Overdose With Adrenergic Agents
- Summary
  - Hyperthermia: cooling and dantrolene
  - Seizures: BZDs, phenobarbital
  - Delirium: BZDs, droperidol
  - Drug-Induced acute coronary syndrome: BZDs, nitrates, phentolamine
  - ST elevation with enzyme release:
    - PTCA preferred over IV thrombolytics
  - ↑BP: BZD, nipride, phentolamine, labetalol
  - Avoid propranolol

- Case 5
Scene: 40-year-old woman drank “eucalyptus tea” 2 hours ago; Confused, pale, HR 30 bpm, BP nonpalp, RR 36/min

Prehospital: O₂, IV, naloxone, D₅₀W, shock trousers

ED: 300 mL saline, atropine 0.5 × 4 → no response Isoproterenol 2 μg/min → VT (140/min) → VF

Primary ABCD Survey

Secondary ABCD Survey

Differential Dx: Drugs → Bradycardias

Oxygen–IV–monitor–fluid challenge
Tank (content)–tank (size)–rate–pump

β-Adrenergic blockers
Calcium channel blockers
Digoxin or other cardiac glycosides
Quinidine and other antiarrhythmics
Opiates
Gamma-hydroxybutyrate (GHB)
Organophosphates, carbamates
Clonidine

Cardiac Glycoside Plants

Foxglove (Digitalis purpurea)
Lily of the valley (Convallaria majalis)
Oleander (Nerium oleander)
Red squill (Urginea maritima)
Yellow oleander (*Thevetia peruviana*)

**Digoxin Toxidrome**
- CV: Bradycardia, heart block, junctional tachycardia, PVCs, VF, VT, ↓BP, asystole
- GI: Anorexia, nausea/vomiting, cramps
- CNS: Lethargy, delirium, weakness, hallucinations, paranoia, agitation
- Ocular: Yellow halos (chronic cases)

**Dig Tox Management**
- activated charcoal
- ↑GI absorption
- ↓Enterohepatic recirculation
- Fix ↓K, ↓Mg, ischemia
- Treat ventricular arrhythmias: Lidocaine, Mg+, phenytoin
- Bradycardia: atropine, pacemaker
- Digoxin-specific antibody

**Dig Fab Fragments (Digibind)?**
- Life-threatening CV toxicity
- K+ >5.5 mEq/L (except CRF)
- Steady-state level >10 ng/mL?
- Ingested dose >10 mg (adult)?
- A caution: If digoxin toxicity is questionable, confirm diagnosis, especially if calcium channel blocker overdose is possible.

**Cardiac Arrest Due to Drug ODs “Codes Due to Poisoning”**
- Consider medical toxicology consultation early
- Consider prolonged CPR
- Sodium bicarbonate
- Use for any wide-complex rhythms
- High-dose pressors may be required
- Inotropes if refractory to high-dose pressors
- Insulin pump, glucagon, amrinone
- Circulatory-assist devices: last resort