AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
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Shock Tox: Approach to Cardiovascular Death in Overdoses

Emergency physicians are frequently confronted with hypotension from toxins before knowledge of the offending agent. Strategies that apply to cardiovascular compromise from most overdoses will be reviewed. In addition, the few toxins that can benefit from a particular antidote, and their presentation, will be discussed.

- List the most common drug overdoses responsible for refractory shock.
- Develop a treatment plan using both new and established antidotes for cardiovascular compromise from toxins.
- Describe the physiology of heart failure after overdose and the reasons it may not respond to routine treatments.
- Discuss the mechanism and appropriateness of various pressors agents.

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FACULTY

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TOX SHOCK: Cardiovascular Drug Toxicity

ACEP Scientific Assembly 1999
Las Vegas, NV

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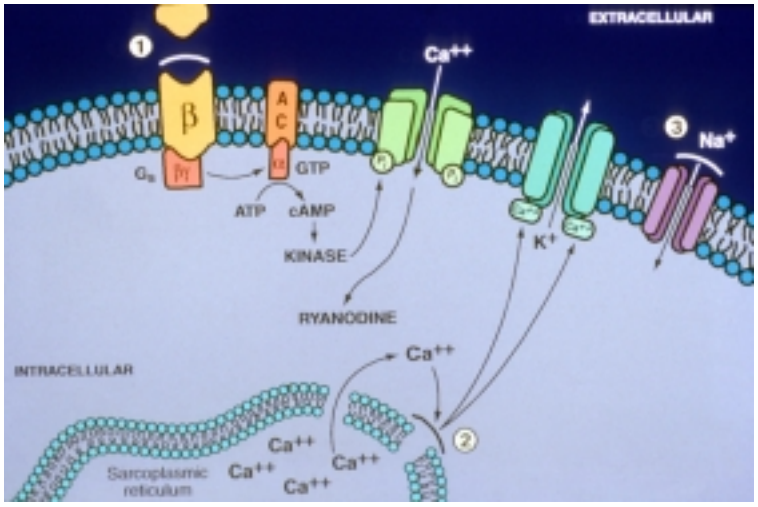
β-Blockers

Mechanism



1. Exaggerated blunting of β_1 & β_2 receptor stimulation
2. Altered calcium and ion dynamics
3. Antagonize fast sodium channels similar to class Ia medications (quinidine)
4. Altered energy substrate (glucose, free fatty acid) utilization (not shown)

Pharmacokinetics

A. Availability: over 30 different β-blockers in regular release & sustained release preparations

B. Onset of action in 1-4 hours

   1. Clinical correlate: **symptoms develop rapidly** following acute ingestion of regular release compounds

C. High Vd and moderate to high protein binding

   1. Clinical correlate: **extracorporeal drug removal not useful** except for atenolol & nadolol (low protein binding & Vd similar to water)

D. Nonspecific hepatic metabolism for fat soluble drugs (propranolol, labetolol, metoprolol)

E. Water soluble drugs undergo renal elimination (atenolol, nadolol, sotalol, timolol)

F. Each agent has slightly different pharmacological profile

Agent	β_1 Specific	Partial agonist	Na^+ channel antagonist
acebutolol	yes	yes	yes
atenolol	yes	no	no
esmolol	yes	no	no
labetolol	no	yes	no
metoprolol	yes	no	no
nadolol	no	no	no
oxprenolol	no	yes	yes
pindolol	no	yes	yes
propranolol	no	no	yes
sotalol	no	no	no

Manifestations

A. **Myocardial depression**

1. **Bradycardia**: typically sinus, but AV block can occur
2. Ventricular dysrhythmia
    a. **Wide complex QRS** with those drugs which also antagonize Na
    b. Ventricular tachycardia, **torsades de pointes** occurs with sotalol which has class III antidysrhythmic properties (prolongied QT)
3. **Hypotension due to decreased contractility**

B. CNS: **coma** & **seizures** due to poor perfusion or direct drug effect. More common following overdose of lipophilic agents (**propranolol**)

C. Pulmonary: despite β_2 antagonism, bronchospasm is rare

D. Metabolic: hypoglycemia is rare

Management

A. What is the optimum therapy for myocardial depression? Answer: No single therapy will consistently reverse cardiotoxicity. Often combined therapies are required. Consider the following:

1. Atropine (cheap, available, & safe, but probably won't work)
2. **Glucagon (3.5-5 mg IV followed by infusion of 1-5 mg/hr) is the current treatment of choice** based on clinical experience & limited animal studies.
    a. Rationale: stimulates cyclic AMP independently of the β-receptor
    b. ADRs: nausea, vomiting, & transient hyperglycemia
3. Norepinephrine (0.5 μg/min & titrate to restore heart rate & blood pressure)
    a. Rationale: catecholamines are a logical therapy. However, their use has met with variable results despite high doses.
4. Amrinone (5 μg/kg/min & titrate)
    a. Rationale: phosphodiesterase inhibitor which inhibits breakdown of cAMP, thereby facilitating maintenance of intracellular calcium.
5. Pacing (bradycardia is often refractory to pacing)

B. General

1. Decontamination
    a. Activated charcoal
    b. Consider whole bowel irrigation following charcoal for sustained release preparations (unstudied)
    c. Lavage if comatose & intubated
2. CNS depression & seizures
    a. Airway support
    b. Correct hypoxia, assess glucose, & maintain organ perfusion
    c. Benzodiazepines for seizures
3. Hemodialysis: consider for atenolol or nadolol overdose
4. Wide complex dysrhythmia: serum alkalinization with sodium bicarbonate
5. Torsades de pointes: magnesium or pacing
6. For sustained release products, monitor > 12 hrs, as onset of symptoms may be delayed

What's New with β-blocker overdose therapy?

In a canine model of severe propranolol toxicity, high dose insulin with euglycemia conferred greater survival than glucagon, epinephrine, or sham treatment. In this model, 6/6 insulin animals survived compared to 4/6 glucagon, 1/6 epinephrine, and 0/6 sham treated animals. Human clinical trials are pending.

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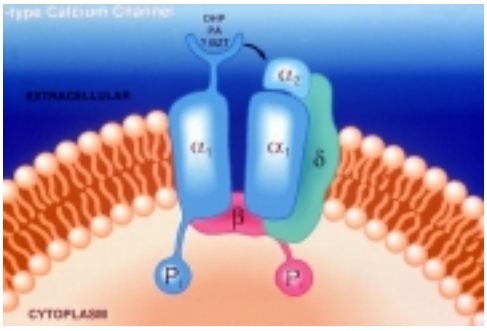
Calcium Channel Antagonists (CCB's)

Mechanism



A. Inhibit calcium entry into smooth muscle & cardiac myocytes by binding to the L-type calcium channel. Decreased calcium impairs excitation-contraction coupling
B. Alters primary myocardial energy source from free fatty acids to glucose (not pictured)

C. Inhibits insulin release by the pancreas, therefor glucose is not usable (not shown)

Pharmacokinetics/Pharmacology

A. Three classes of CCB, available as regular release & sustained release preparations, each class having different pharmacological effect in therapeutic doses

	Dihydropyridine (nifedipine)	Benzothiazepine (diltiazem)	Phenylalkylamine (verapamil)
periph resistance	++	-	+
A-V node conduction	-	+	+
contractility	-	+	++

B. Onset of action is rapid, within 1/2-2 hrs after regular release
    1. Clinical correlate: symptom onset within several hours for regular release and symptom onset delay 6-12 hours for sustained release preparations

C. High protein binding & large Vd
    1. Clinical correlate: extracorporeal drug removal of little benefit

D. Verapamil & diltiazem have active metabolites
    1. Clinical correlate: potential for prolonged symptoms

Manifestations

A. **Myocardial depression**
    1. **Bradycardia with AV block, asystole**
    2. **Hypotension** due to decreased contractility & peripheral vasodilation

B. CNS: **coma & seizures** due to poor perfusion

C. Metabolic: **hyperglycemia** & acidosis

Management

A. What is the optimum therapy for myocardial depression? Answer: there is no single agent that consistently reverses cardiotoxicity.
    1. Atropine (safe, cheap, but unlikely to help)
    2. Crystalloid bolus with normal saline

a. Useful for hypotension from peripheral vasodilatation in mild cases.

3. Calcium (10 ml of 10% solution IV bolus, then 20-50 mg/kg/hr infusion)
    a. Rationale: exogenous calcium may overcome inhibition of calcium channel. However, results often disappointing.
    b. The effect of a calcium bolus alone is transient.
4. Norepinephrine (0.5 μg/min & titrate to restore heart rate & blood pressure)
    a. Variable results
5. Glucagon (3.5-5 mg IV, followed by 1-5 mg/hr infusion)
    a. Rationale: increase cAMP
    b. Variable results
6. Electrical pacing

B. General supportive care
1. Monitor patients for at least 12 hr following ingestion of sustained release agents, as symptoms can be delayed
2. Give activated charcoal for GI decontamination & consider whole bowel irrigation following charcoal for ingestion of sustained release compounds
3. Pharmacokinetics do not lend to beneficial extracorporeal drug removal
4. Seizures: improve cerebral perfusion, correct hypoxia

What's new for calcium channel blocker therapy?

High dose insulin with euglycemia is a superior antidote in canine models of acute and chronic verapamil toxicity compared to glucagon & epinephrine. Insulin was utilized as an adjunct to standard therapy in several severe overdoses (4 CCB and 1 combined CCB & β-blocker) with good results. However, these are uncontrolled cases. A clinical trial is pending.

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Cyclic Antidepressants

Despite awareness of the potential severity of cyclic antidepressant (CA) toxicity and development of pharmacologically different antidepressants, CA overdose continues to result in significant mortality.

Mechanism

A. Seizures: complex interaction of effects on serotonin, NE and GABA
B. Sinus tachycardia: anticholinergic effects
C. Dysrhythmias: fast - inward sodium channel blockade which decreases the slope of phase 0 depolarization, prolonging the QRS interval and leading to reentrant ventricular dysrhythmia, asystole
D. Hypotension: peripheral α-blockade & myocardial depression

Pharmacokinetics

A. Rapid absorption and peak drug levels
    1. Clinical correlate: precipitous onset of symptoms, usually within 6 hr
B. Large volume of distribution

1. Clinical correlate: limited utility of extracorporeal drug removal

C. High percent of protein binding

1. Clinical correlate: limits extracorporeal drug removal
2. Ratio of free to bound drug is pH dependent (acidemia ↑ free drug & **alkalemia ↓ free drug**)

Clinical manifestations: primarily affects 3 systems (ANS, CNS, Cardiovascular)

A. ANS: anticholinergic syndrome

B. CNS: altered mental status with coma (30 %) & seizures (8 %)

C. Cardio-pulmonary

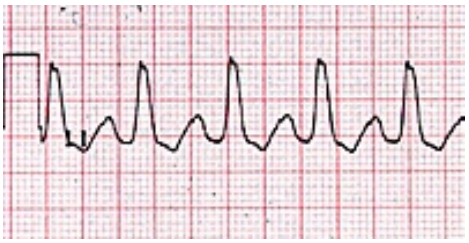
1. Tachycardia (51%)
2. Conduction delay (21 %)
3. Dysrhythmia (6 %): ventricular fibrillation & tachycardia, atrial dysrhythmias
4. Hypotension (15 %)
5. Noncardiogenic pulmonary edema

ECG changes: (validated for limb leads only)

A. Wide QRS

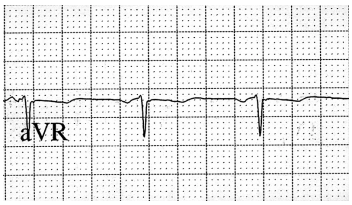
1. > 160 ms: risk of ventricular dysrhythmias
2. > 120 ms: risk of seizures

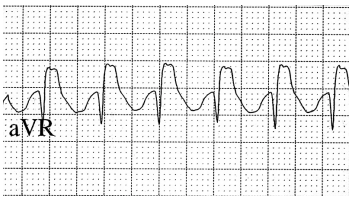
Lead II



B. **R or R:S ratio in aVR** for predicting seizures & dysrhythmias

1. R > 3 mm: sens = 81% & neg pred value = 94%
2. R:S > 0.7: sens = 75% & neg pred value = 92%





normal AVR terminal R forces in AVR

Laboratory

A. Serum levels: not useful
    1. Not available quickly
    2. **No correlation between major symptoms and serum levels**

Management

A. Initial evaluation in area with continuous monitoring of vital signs & ECG
B. General supportive care
    1. airway patency
    2. hypotension: fluids and vasopressors with α-agonist activity
    3. seizures: benzodiazepines, phenobarbital and, lastly, dilantin
    4. malignant dysrhythmias (v-tach or fib): alkalinization, lidocaine and, lastly, phenytoin

C. GI decontamination
    1. Ipecac is contraindicated due to risk of precipitous mental status change/seizure and subsequent aspiration
    2. Lavage if intubated or ingested a "massive" amount
    3. **All patients get activated charcoal** to prevent drug absorption

D. Specific therapy: Serum alkalinization
    1. Mechanism
        a. Restoration of sodium channels (probably the most important)
        b. Increased protein binding and reduced free drug
    2. Which is more efficacious: giving bicarb or hyperventilating?
        a. Both are effective alone in animal models, but **more efficacious when combined to achieve a serum pH 7.45 - 7.50**
    3. Indications
        a. **QRS > 120 ms**
        b. Malignant dysrhythmia **(v-tach, v-fib, rhythm causing hypotension**)
        c. **Acidosis** (treat primary source also $\rightarrow$ seizure, hypotension)

4. Method of administration
    a. If awake: bicarb bolus (1-2 mEq/kg) and saline infusion
    b. If comatose: bicarb bolus, normal saline infusion and hyperventilate
    c. If pulmonary edema present or patient won't tolerate sodium load → hyperventilate

Pitfalls in CA poisoning: the following may cause **complications**

A. Ipecac may result in vomiting with subsequent **aspiration pneumonitis** in the face of precipitous decline in mental status
B. Atropine worsens anticholinergic effects, but OK for bradycardia with hypotension
C. Physostigmine may worsen conduction block and cause **asystole**
D. Flumazenil: antagonizes protective effect of GABA possibly causing **seizures**

Disposition

A. Admit any patient with major manifestation (altered MS, seizure, QRS > 100 ms, hypotension, or persistent tachycardia)
B. If, after a 6 hour ED observation period, a patient is asymptomatic or has minor symptoms (sleepy or only a narrow complex tachycardia) that resolve, the patient gets charcoal, and has a normal repeat ECG, they are safe for discharge to psychiatric facility.

What's new for CA poisoning?
Initial human clinical trials are underway utilizing tricyclic antidepressant specific fab antibody therapy. This therapy is similar to treatment of digoxin toxicity with Digibind®.

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Digitalis

Mechanism of toxicity: exaggerated therapeutic effect

A. Inhibition of Na-K ATPase which ultimately increases cytosolic calcium
    1. Since Na-K ATPase acts to maintain intracellular K, inhibition results in loss of K extracellularly. Thus, ↑ **extracellular K becomes an important marker of acute toxicity**
B. Electrophysiological effects
    1. Atria & ventricles: ↓ conduction velocity & ↑ automaticity
    2. AV node: ↓ conduction velocity & ↑ refractoriness
C. Vagotonic thereby contributing to bradycardia

Pharmacokinetics

A. Volume of distribution: varies with age but is approximately 6 l/kg for adults with normal renal function
B. Renal elimination (60-80% unchanged)
C. Metabolism: small amount undergoes enterohepatic circulation. Multiple doses of charcoal may enhance elimination by interfering with this path
D. Drug interactions: **several drugs ↑ serum [digoxin] including verapamil, quinidine, spironolactone, diltiazem, amiodarone, erythromycin, tetracycline**

Manifestations of toxicity: literature reflects experience with chronic toxicity

	acute toxicity	**chronic toxicity**
history	intentional or accidental **usually healthy**	elderly with **underlying medical problems**, diuretic use, dehydration, renal dysfunction, concurrent medication use
CNS	H/A, confusion, coma	fatigue, confusion, hallucinations, **color halos**, coma
GI	nausea, vomiting	**anorexia, nausea, vomiting**, abdominal pain
cardiac	bradydysrhythmias such as atrial tachycardia with AV block	**ventricular dysrhythmias** bradydysrhythmias atrial tachycardia with AV block
K^+	**↑ K**	normal or ↓K
[digoxin]	↑	normal or mildly ↑

Laboratory aids

A. ECG: see above tables

B. **[K^+]>5.0 mEq/l**: Of 91 moderate to severe digitoxin patients (81 acute), no patients with initial arterial [K] > 5.5 survived & no patient with [K] < 5.0 died

1. **[K] may be normal or low in chronics** due to other diseases or concurrent diuretic use

C. Digoxin levels

1. Therapeutic: 0.5-2.0 ng/ml
2. There is no panic serum level that demands treatment

Factors to consider:

a. Time of serum level with respect to ingestion. A digoxin level of 10 ng/ml obtained 1 hr after ingestion (during distribution of the drug), is not as worrisome as a level of 10 ng/ml drawn 10 hrs post-ingestion (steady state)

b. 10 % of symptomatic chronics have therapeutic values

c. availability of testing

3. False + assay due to endogenous digoxin-like immunoreactive subs (DLIS)
   a. Pregnancy
   b. Chronic renal failure

c. Liver disease with hyperbilirubinemia
d. Spironolactone use
e. Treatment with Fab: **following treatment, [dig] will be elevated 10-20 fold** (in one case the post-Fab [dig] = 200 ng/ml). **Most assays don't differentiate free versus bound digoxin**. There is no reason to get levels after giving Fab!

Management

A. Decontamination
1. One dose of activated charcoal

B. **Fab (digoxin specific antibodies)**: **primary treatment for significant digitalis poisoning**
1. Synthesized: IgG from sheep exposed to digoxin is harvested & cleaved into Fab & Fc portions (Fc, the antigenic portion of the IgG is discarded)
2. Mechanism: Fab binds digoxin in the interstitium & intravascular space
3. **Indications**
a. **Malignant ventricular dysrhythmia**
b. **Bradycardia with hemodynamic compromise**
c. **[K] > 5.0 mEq/l**
4. Dosing: three methods to determine the dose
a. Known amount ingested:

(amount in mg)(0.8 bioavailable) / (0.6 mg/vial) = # vials Fab

b. Known serum [dig]:

([dig] in ng/ml)(Vd 5.6 l/kg)(weight in kg) / (1000)(0.6 mg/vial) = # vials Fab

c. Empiric (IE: sick, unknown amount, & [dig] not readily available): **10-15 vials**
5. Administration
a. Give IV over 30 min via 0.22 micron filter
b. IV push if arrest imminent
6. Efficacy: of 150 patients treated with Fab
a. **80% resolved, 10% improved**, & 10% had no response
b. Of the 10% (15) nonresponders: 5 were moribund, 5 were later determined not related to digoxin, 4 were deemed unlikely related to

digoxin, **leaving 1 true nonresponder**

c. Time to initial response = 19 (0-60) minutes

d. Time to complete response = 88 (30-360) minutes

7. Safety

a. Allergy: **4/717 patients developed pruritic rash or urticaria after Fab**. All 4 responded to antihistamine treatment. **All 4 gave a history of medication allergy or asthma**. Thus Fab appears **safe with only slight risk of allergic reaction in those patients with previous allergies**.

b. Other reported ADR's in 150 patients:

i. hypokalemia in 6/150

ii. exacerbation of CHF in 4/150

C. Supportive measures (if digibind not available)

1. Hyperkalemia

a. Insulin & glucose

b. HCO_3

c. Albuterol

d. Kayexalate

e. **Exogenous calcium is contraindicated in digoxin poisoning**: there is already excess cytosolic calcium via Na-K ATPase inhibition & compensation by Na-Ca exchange (the Na-Ca exchanger moves excess Na out in exchange for Ca)

2. Bradycardia

a. Atropine: vagolytic, cheap, readily available

b. Pacing: In 92 patients with digitalis toxicity, pacing had a higher failure rate (23%) compared to Fab (8%). Pacing also had more complications versus Fab (13% deaths vs no deaths in Fab group)

3. Tachydysrhythmia

a. Phenytoin or lidocaine ↓ automaticity without affecting AV conduction

b. Propranolol (may exacerbate CHF)

4. Magnesium (↓Mg will ↑ myocardial digoxin uptake)

a. 2 gm over 20 minutes (no studies defining exact dose)

b. Contraindicated in bradycardia or conduction block

Special situations

A. Do renal patients treated with digibind get rebound toxicity? In 28 functionally anephric patients treated with Fab, only 1 patient developed 2° & 3° AV block 10 days later. No symptoms developed.

B. What about digoxin toxicity & Fab use in pediatrics?

1. Kids tolerated intoxication better than adults
2. Potentially toxic dose: 0.1 mg/kg or steady state [dig] > 5.0 ng/ml
3. Most common ECG finding is 2° AV block
4. Severe poisoning tended to be chronic & in children with underlying cardiac disease
5. **Suggested indications for Fab in children**:
    a. Known intoxication (0.1 mg/kg or steady state [dig] > 5/0 ng/ml
    b. Progressive symptoms
    c. life-threatening dysrhythmia
    d. [K] > 6.0 mEq/l

C. Pregnancy: no published cases of Fab use in pregnancy

D. Are there any naturally occurring sources of digitalis or cardiac glycoside poisoning which have similar manifestations to digoxin toxicity & may respond to Fab?

1. Foxglove (*Digitalis purpurea*)
2. Oleander (*Nerium oleander*)
3. Red Squill (*Urginea maritima*)
4. Lilly of the Valley (*Convallaria majalis*)
5. Bufotoxin: secreted by frog skin
    a. From toad licking (*Bufo marinus & alvarius*)
    b. Chinese herbal medicines may contain bufotoxin

Pitfalls in digitalis toxicity

A. Failure to recognize toxicity: 10% of chronic intoxications have normal [dig]

B. Don't give calcium in a patient suspected of digitalis toxicity

C. Don't give a class Ia antidysrhythmic agent (quinidine) as they may worsen conduction problems

D. Forced diuresis & dialysis are not beneficial

E. Don't be alarmed by high [dig] following Fab treatment

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Clonidine

Pharmacology

A. Imidazole antihypertensive which **stimulates α_2, presynaptic receptors in the vasomotor center** of the brainstem (locus ceruleus, nucleus ambiguus, & nucleus of the solitary tract). α_2 receptor stimulation results in negative feedback, decreasing release of vesicles containing norepinephrine & sympathetic outflow.

B. B. Alleviates opioid withdrawal symptoms by decreasing noradrenergic outflow from locus ceruleus (where opioid receptors are also concentrated).

C. Peripheral α agonist

Pharmacokinetics

A. Availability: regular release tabs & timed release patch (toxicity reported from both)
B. Onset of action: 30-60 min
C. Elimination T1/2: 8 hours
D. Primary renal elimination

Manifestations of toxicity

A. CNS: **coma, apnea, miosis**, hypothermia
B. Cardiovascular: **sinus bradycardia**, initial, brief hypertension (due to peripheral α agonism), & then **hypotension**

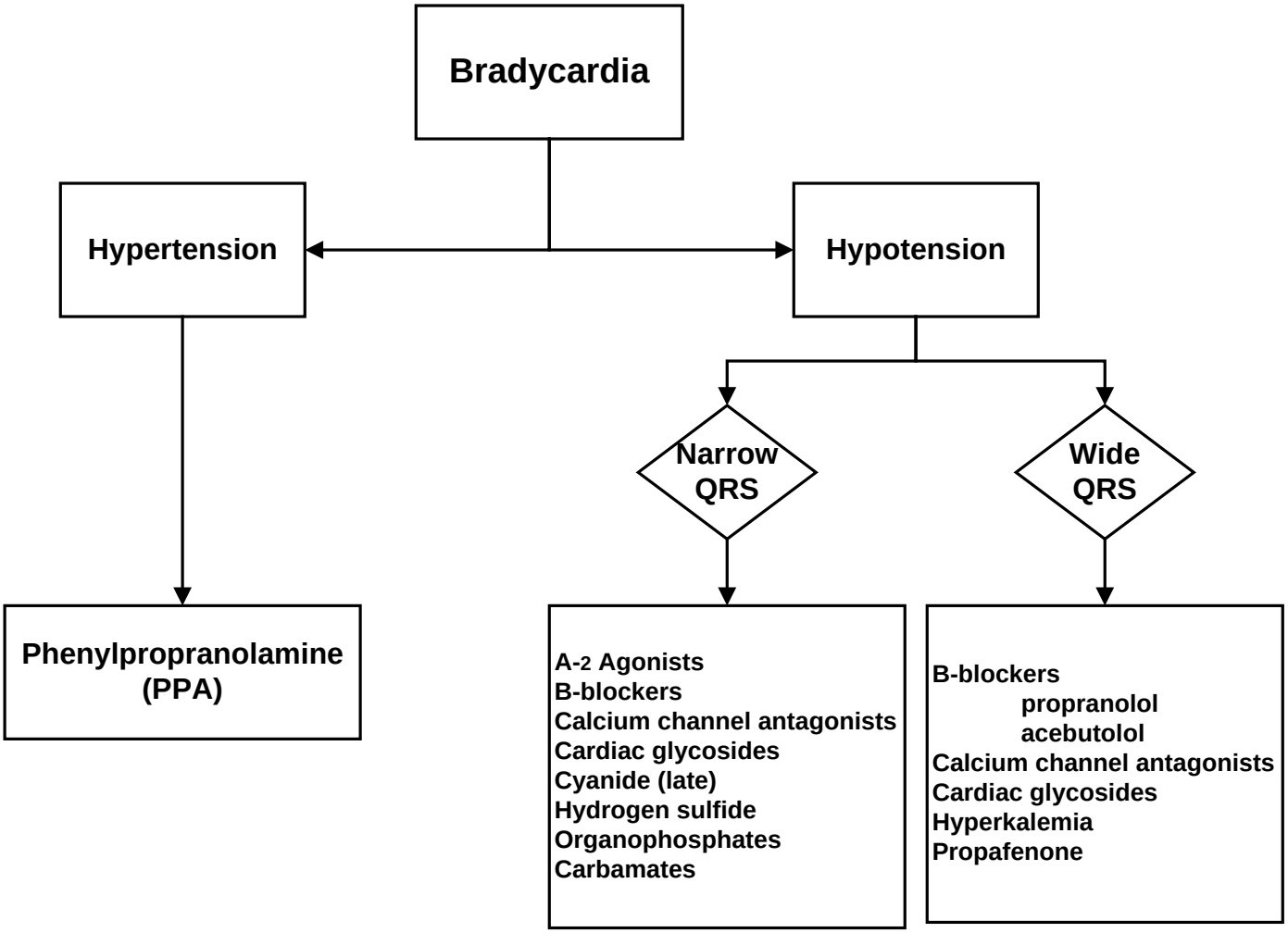
Management

A. Decontamination
    1. Activated charcoal
    2. Consider whole bowel irrigation after charcoal for ingestion of timed patch
B. CNS depression
    1. Control airway
    2. Naloxone: used successfully in anecdotal cases
C. Bradycardia
    1. Atropine is usually sufficient
D. Hypotension
    1. Crystalloid infusion
    2. Dopamine infusion
    3. Naloxone
        a. reverses the antihypertensive effect of clonidine in rats & humans

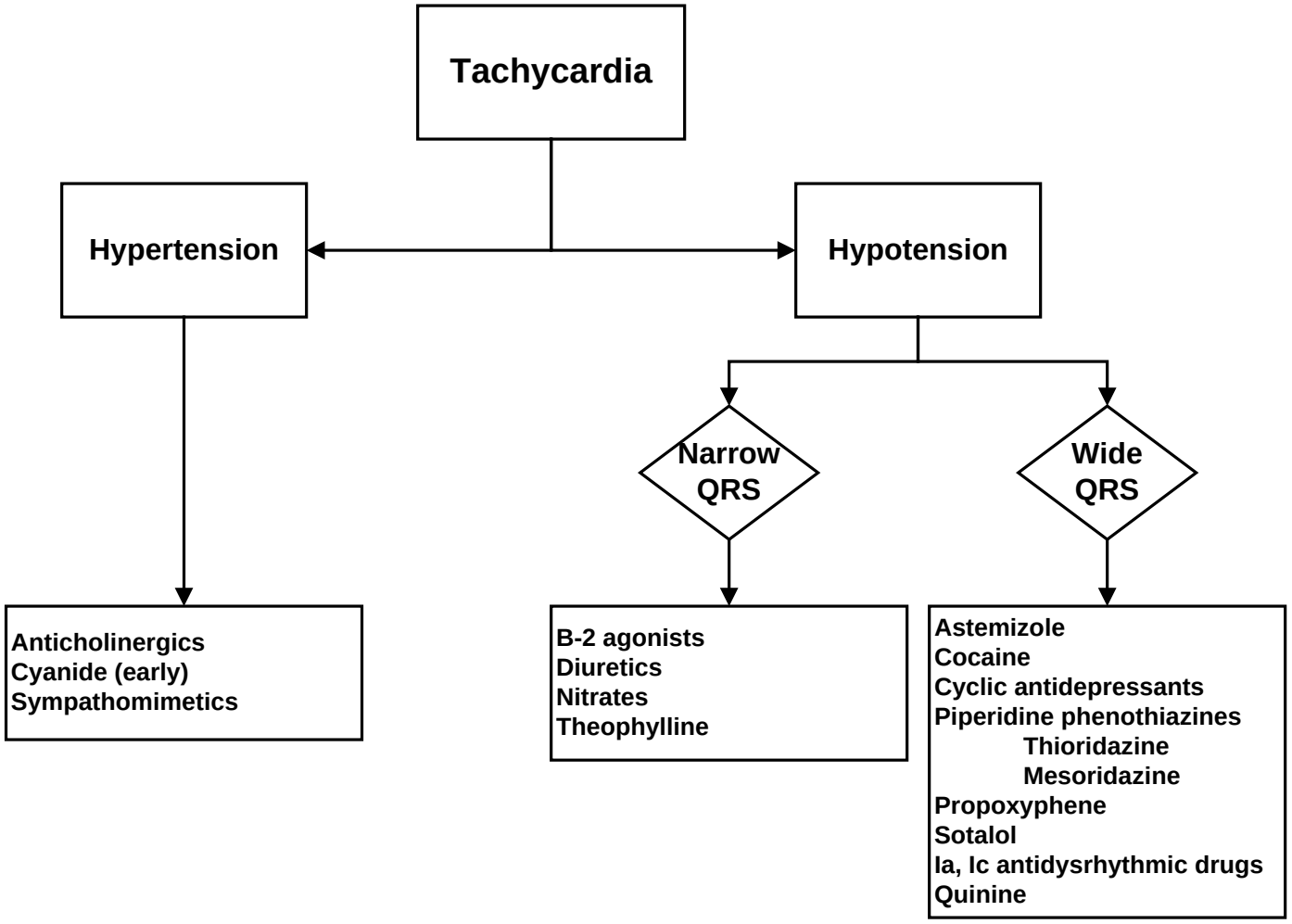
References

1) Anderson RJ, Hart GR, Crumpler CP, et al. Clonidine overdose: A report of six cases and review of the literature. Ann Emerg Med 1981;10:107-12
2) Caravati EM, Bennet DL. Clonidine transdermal patch poisoning. Ann Emerg Med 1988;17:175-6
3) North DS, Wieland MJ, Peterson CD, et al. Naloxone administration in clonidine overdosage. Ann Emerg Med 1981;10:397
4) Aghajanian G. Tolerance of locus coeruleus neurons to morphine and suppression of withdrawal response by clonidine. Nature 1978;276:186
5) Farsang C. Naloxone reverses the antihypertensive effect of clonidine. Br J Phamacol 1979;67:161-4

Bradycardia Diagnostic Algorithm



Tachycardia Diagnostic Algorithm



Toxicology Lab Case Wrap-Up: Answer Key
ACEP Scientific Assembly
Oct 11-14, 1999
Russ Kerns, MD

Station 1:

1) She is right – Mercury poisoning
2) The most common intentional exposure is likely a heavy metals (Arsenic, Mercury, Thallium, Selenium)

Station 2:

1) Capsaician
2) Treat like a local thermal injury
3) Bronchospasm due to pepper spray

Station 3:

Boletus species – edible

Station 4:

Amanita species – hepatotoxic and potentially lethal

Station 5:

Chlorphyllum – may cause severe gastroenteritis, but not lethal

Station 6:

??? species – but I would not eat it!!!

Station 7:

1) Anticholinergic poisoning from Jimson Weed
2) Treatment includes intravenous fluids, benzodiazepines, foley catheter, and physostigmine for severe agitation

Station 8:

1) Disulfiram reaction: disulfiram inhibits aldehyde dehydrogenase resulting in acetaldehyde accumulation (aldehydes are noxious compounds – flushing, agitation, headache, nausea and vomiting)
2) Toxin-induced flushing reactions:

Disulfiram-like reactions: DDC, metronidazole, coprinus mushroom, chloral hydrate
Anticholinergic drugs and plants
MAO inhibitor and food interactions
MSG (Chinese restaurant syndrome)
Food poisoning (scrombroid)

Station 9:

1) hydrofluoric acid
2) intraarterial calcium if topical calcium does not rapidly relieve pain. Consider intravenous magnesium also.

Station 10:

1) Hypocalcemia, acidosis, coagulopathy, cardiac dysrhythmia
2) Give exogenous calcium and magnesium

Station 11:

1) Hyperglycemia is a common finding with CCBs
2) Calcium, glucagon, vasoactive agents, pacer, kitchen sink

Station 12:

1) Hyperkalemia is asoociated with acute digoxin toxicity. Serum K+ > 5.0 is an indication for antidotal therapy
2) Digoxin specific antibodies

Station 13:

1) Wide complex tachycardia is due to sodium channel blockade and the most common drug with sodium channel antagonism is cyclic antidepressants
2) Treat with sodium bicarb boluses for QRS > 120 ms or if dysrhythmia occurs
3) Cocaine, propoxyphene, thioridazine, mesoridazine, Class 1a & 1c antidysrhymics, quinine

Station 14:

1) Some beta-blockers (propranolol and acebutolol), calcium channel blockers, and propafenone have multiple toxic mechanisms such as b-blockade and sodium channel blockade. These agents may cause wide complex bradycardia. Digoxin may also cause wide complex bradycardia.

2) This patient took acebutolol and was treated with glucagon

Station 15:

1) Nasotracheal intubation
2) Antivenin

Station 16:

Venomous (Copperhead)

Station 17:

Answer: give antivenin to the child only. Coral snakes have small teeth and must chew to envenomate. Therefore mom doesn't need treatment. Treatment is indicated if neurologic symptoms are present or if there is a history of the snake chewing or hanging on. Elapidae have primarily neurotoxicity, not local toxicity or coagulopathy. Neurotoxicity may occur without any local bite findings.

Stations 18-22:

Sulfur:	hydrogen sulfide ("knock-down gas")
Almonds	cyanide
Garlic	**organophosphates**, arsenic
Peanuts	Vacor (PNU), a rodenticide that acts like streptozocin (pancreatic toxin) leading to diabetic ketoacidosis
Wintergreen	methyl salicylate

Station 23:

1) Chronic salicylism
2) Peptobismol
3) Hemodialysis
4) Salicylate, opioids, phenothiazines, CCBs, crack cocaine, inhalation injury, organophosphates

Station 24:

Ferric chloride when added to urine containing salicylate will turn the urine purple

Station 25:

Poinsettias are not toxic

Station 26:

Iron, potassium, body packers/stuffers (foreign bodies), heavy metals

Station 27:
Bicarbonate-wasting renal tubular acidosis (reversible after removing patient from exposure)

Station 28:

1) inhibit alcohol dehydrogenase (etoh or fomepizole)
2) dialysis

Station 29:

Despite an unimpressive CXR (? RML infiltrate) he is symptomatic and needs admit for supportive pulmonary care

Station 30:

1) Delayed neurological sequelae (secondary Parkinson's)
2) Basal ganglia

Station 31:

1) Pneumomediastinum
2) AMI, PTX, crack lung, aortic dissection

Station 32:

Botulism

Station 33:

Fire Ants

Station 34:

Antibiotics are the correct choice. None of the other listed treatments are of proven efficacy.

Station 35:

Opioids and skeletal muscle relaxants (benzodiazepines and magnesium) are most effective. If refractory pain or hypertension develop, antivenin is available.

Station 36:

Oleander contains a cardiac glycoside very similar to digoxin

Station 37:
Hot water immersion

Station 38:

1) Pull off tentacles with a sandy towel
2) Baking soda
3) Analgesics and antihistamines