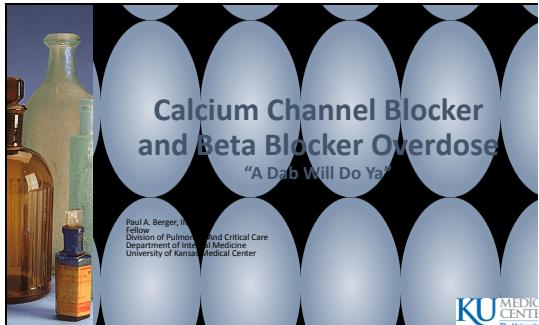
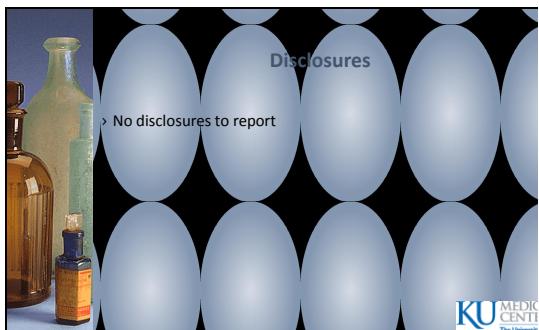


Slide 1



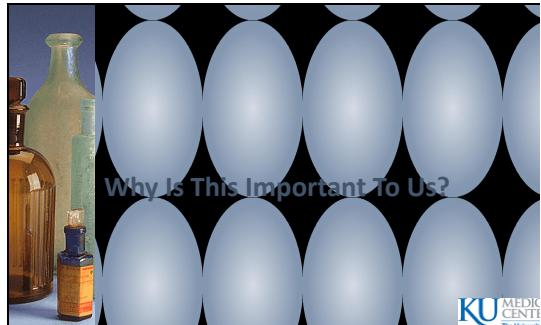
Slide 2



Slide 3



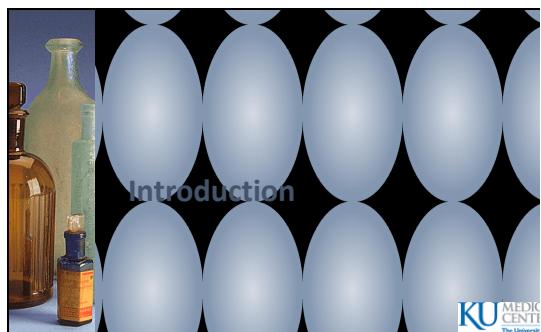
Slide 4



Slide 5



Slide 6



Slide 7



Slide 8

Introduction

- > Increasing prevalence of cardiovascular disease
- > Increasing use of cardiovascular medications
- > Annual Report of the American Association of Poison Control Centers
 102,766 Medication Errors
 CCBs most implicated
- > Identifying and treating patients with toxic effects is complex
- > ABC's and Resuscitative Protocols
- > "Interesting" Therapies

Clinical Toxicology 2014; 52: 935-944
EB Medicine 2014; 16(2)

KU MEDIC CENTER
The University

Slide 9

Pathophysiology and Pharmacokinetics

KU MEDIC CENTER
The University

Slide 10



Slide 11

calcium

- > Atomic number 20
- > Alkaline earth metal
- > Most abundant metal by mass in most animals

Critical role in intracellular messaging and myocyte contraction

KU MEDIC CENTI The University

Slide 12

Calcium Channel Blockers

- > Developed in the 1960's
- > Hypertension, cardiac dysrhythmias, and angina
- > #1 cause of fatal CV medication exposures

Three main classes

- Dihydropyridines (Nifedipine)
- Phenylalkylamines (Verapamil)
- Benzothiadiazines (Diltiazem)

Am J Toxicol 2014; 52: 926-944
J Am Geriatr Soc 2014; 16(2)

KU MEDIC CENTI The University

Slide 13

Calcium Channel Blockers

- > Affinity to L-type Calcium Channels
 - Dihydropyridines: Vascular smooth muscle
 - Phenylalkylamines: Vascular and Cardiac
 - In an OD situation receptor selectivity is lost
- > Blocking of L-type Calcium Channels
 - Interferes with base excitation-contraction coupling
 - Interferes with conformation of troponin-myosin complex
 - Decreases contractility and chronotropy
 - Smooth muscle relaxation

Emerg Med Clin N Am 2014; 32: 79-102
Clin Tox 2014; 52: 939-944
EB Medicine 2014; 16(2)

KU MEDIC CENTI The University

Slide 14

Calcium Channel Blockers

- > Well absorbed in gastrointestinal tract
- > Metabolized via cytochrome system
 - Risk of drug/drug interaction
- Highly protein bound
- Large volume of distribution
- LD is not altered in OD situations

Emerg Med Clin N Am 2014; 32: 79-102
Clin Tox 2014; 52: 939-944
EB Medicine 2014; 16(2)

KU MEDIC CENTI The University

Slide 15

Calcium Channel Blockers

Calcium channel blockers
Benzothiazepine
Diazepam
Dihydropyridines
First generation
Nifedipine
Nifedipine
Second generation
Felodipine
Amlodipine
Nimodipine
Nisoldipine
Third generation
Amlodipine
Clevidipine
Phenylalkylamine
Venlafaxine ^a
= Sodium channel inhibition.

Emerg Med Clin N Am 2014; 32: 79-102

KU MEDIC CENTI The University

Slide 16

Beta Blockers

- > Developed in the 1960's
- > Hypertension, CHF, thyrotoxicosis, angina, ACS, and essential tremor

Two Beta Receptors of Importance

- Beta-1: Cardiac myocytes
- Beta-2: Smooth muscle, vasculature, skeletal muscle

Emerg Med Clin N Am 2014; 32: 79-102
Clin Tox 2014; 52: 926-944
B Medicine 2014; 14(2)

KU MEDIC CENTI
The University

Slide 17

Beta Blockers

- > Beta-1
 - G coupled, cyclic-AMP receptors
 - Calcium release from sarcoplasmic reticulum
 - Increases in heart rate and chronotropic
- > Beta-2
 - Less well understood
 - Smooth muscle relaxation

Selective or non-selective

- Non-selective: Propranolol
- Selective: Metoprolol

Emerg Med Clin N Am 2014; 32: 79-102
Clin Tox 2014; 52: 926-944
B Medicine 2014; 14(2)

KU MEDIC CENTI
The University

Slide 18

Beta Blockers

- > Lipophilicity (Propranolol)
 - Increased CNS penetration
 - Increased risk of seizures (Therapeutic dosing)
 - Decreased level of consciousness (Overdose)
- > Membrane stabilizing activity (Propranolol vs Acebutolol)
 - Sodium channel blockade
 - QRS widening
 - Increased risk of dysrhythmia
 - Seizure activity and diminished consciousness

Emerg Med Clin N Am 2014; 32: 79-102
Clin Tox 2014; 52: 926-944
B Medicine 2014; 14(2)

KU MEDIC CENTI
The University

Slide 19

Beta Blockers

Selected β -adrenergic antagonists

Selective β_1 -Antagonists	Nonselective β_1 - and β_2 -Antagonists	β_1 - and β_2 -Antagonists with α_1 -Antagonism
Acebutolol ^{a,b}	Carteolol ^{c,d}	Carvedilol ^e
Atenolol	Levobunolol ^c	Labetalol ^{b,f}
Betaxolol ^{b,c}	Metipramol ^f	
Bisoprolol	Nadolol ^f	
Esmolol	Oxprenolol ^{b,g}	
Metoprolol ^a	Penbutolol ^b	
	Pindolol ^{b,h}	
	Propanolol ^b	
	Sotalol ^{b,i}	
	Timolol ^b	

^aVigorod 2014; 32: 79-82
^bWong Med Clin N Am 2014; 32: 79-82
^cEB Medicine 2014; 16(2)
^dKU MEDIC CENTR The University
^eJ. Med. Toxicol. (2010) 6:100–105
DOI 10.1007/s13181-010-0072-z
^fORIGINAL STUDY
^gVerapamil Inhibits the Glucose Transport Activity of GLUT1
Larry L. Louters • Nathan Stehouwer •
Janelle Rekman • Andrew Tibball • Alexandra Cok •
Christopher P. Holstege
^hVerapamil Toxicity Dysregulates the
Phosphatidylinositol 3-Kinase Pathway
Laura K. Bechtel, PhD, Doris M. Haverstick, PhD, Christopher P. Holstege, MD

Slide 20

The Role Of Calcium In Myocardial Contraction

EB Medicine 2014; 16(2)
KU MEDIC CENTR The University

The diagram illustrates the intracellular calcium signaling pathway in myocardial contraction. It shows the entry of extracellular calcium (Ca^{2+}) through L-type calcium channels. This calcium binds to calmodulin (CaM), which then activates myosin light chain kinase (MLCK). MLCK converts ATP to ADP and inorganic phosphate (Pi), activating myosin light chain (MLC2). Simultaneously, calcium also activates phosphatase, which dephosphorylates MLC2, leading to its inactivation. The G-protein (G_i) is shown coupled to the receptor (R).

Slide 21

Verapamil Inhibits the Glucose Transport Activity of GLUT1

J. Med. Toxicol. (2010) 6:100–105
DOI 10.1007/s13181-010-0072-z
ORIGINAL STUDY
Verapamil Toxicity Dysregulates the Phosphatidylinositol 3-Kinase Pathway
Laura K. Bechtel, PhD, Doris M. Haverstick, PhD, Christopher P. Holstege, MD

Slide 22

The Toxic Effects

- Calcium Channel Blockers
 - Inhibition of Calcium influx through L-type channels
 - Inhibition of myocardial fast sodium channels
 - Inhibition of insulin release from pancreatic islet cells
 - > Hypoglycemia and Hyperglycemia
 - Inhibition of glucose uptake by peripheral tissues
- Beta Blockers
 - Inhibition of beta receptors → reduction in Catecholamines
 - Inhibition of Calcium influx through L-type channels
 - Inhibition of myocardial fast sodium channels

Clinical Toxicol 2014; 46: 277-283
Georg Mat Clin N Am 2014; 32: 775-782
Clin Tox 2014; 52: 926-944
J Medicine 2014; 16(2)

KU MEDIC CENTI
The University

Slide 23

The Toxic Effects

- Calcium channel blockade triggers the heart to change metabolism
 - Carbohydrate metabolism (Stressed State)
 - Free fatty acid oxidation (Non-Stressed State)

Georg Mat Clin N Am 2014; 32: 775-782
Clin Tox 2014; 52: 926-944

KU MEDIC CENTI
The University

Slide 24

The Toxic Effects

- Profound vasodilation!!!
- Decreased systemic vascular resistance!!!
- Bradycardia!!!
- Conduction delays!!!
- Tachyarrhythmias!!!
- Metabolic acidosis!!!
- Altered mental status!!!
- Seizures!!!

SHOCK!!!

:(

KU MEDIC CENTI
The University

Slide 25



Slide 26

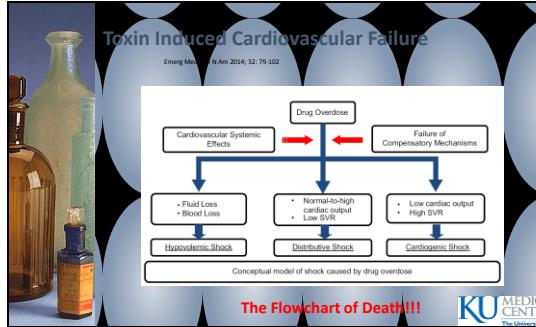


Slide 27

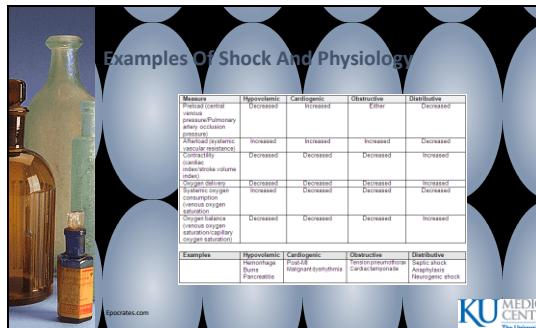
ACVE	Mechanism	Pathophysiology
Mycardial injury	Decreased myocardial O ₂ supply Increased myocardial O ₂ demand	Coronary artery vasospasm Decreased O ₂ carrying capacity Hyperthermia, agitation Tachycardia, hypertension Inhibition of oxidative phosphorylation
Shock	Decreased intravascular volume Decreased SVR Diminished myocardial contractility	Fluid losses Gastrointestinal hemorrhage Vasodilation β-Adrenergic antagonism Ca ²⁺ /Na ⁺ channel blockade
Ventricular dysrhythmia	Mycardial sensitization Triggered beats	O ₂ delivery compromise K ⁺ channel blockade Premature contractions Intracellular Ca ²⁺ release

J Emerg Med Clin N Am 2014; 32: 7-102

Slide 28



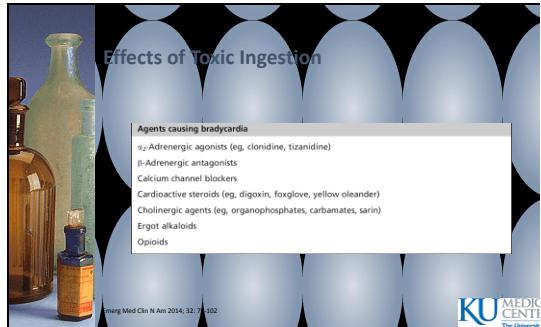
Slide 29



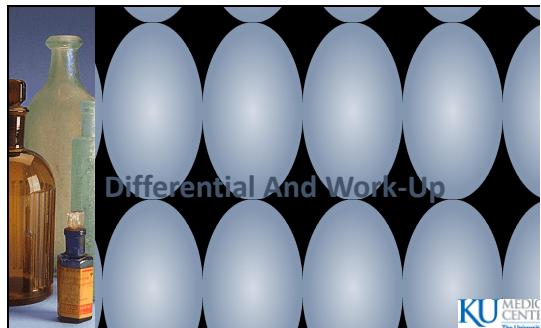
Slide 30



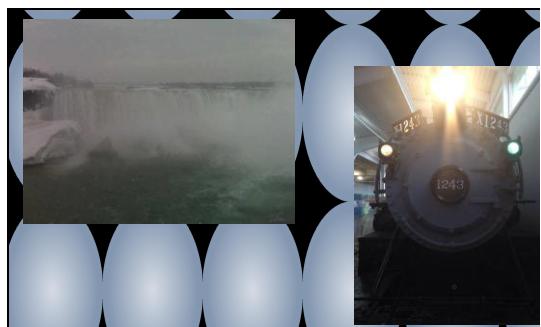
Slide 31



Slide 32



Slide 33



Slide 34

Differential Diagnosis

- > Short list with Bradycardia and Hypotension
- > Acute Coronary Syndrome (Inferior MI with various blocks)
- > Hyperkalemia
- Endocrine Disorders (Hyperthyroid)
- Other

Poisoning

- Clonidine
- Cholinergic toxicity

Clinical Toxicol 2011; 49: 277-283
Am J Med Clin N Am 2014; 32: 79-102
Clin Toxic 2014; 52: 926-943
J Emerg Med 2014; 56(2)

KU MEDIC CENTI The University

Slide 35

Clinical Work-Up

- > ABC's
- > IV Access
- > EKG
- Review medications!!! (Regular vs Sustained Release)
- Consider the patient's history (Blood Glucose)
- CT Head with contrasted meninges status
- Consider the Blood Sugar Level
- > Identify the "type" of shock if unstable

KU MEDIC CENTI The University

Slide 36

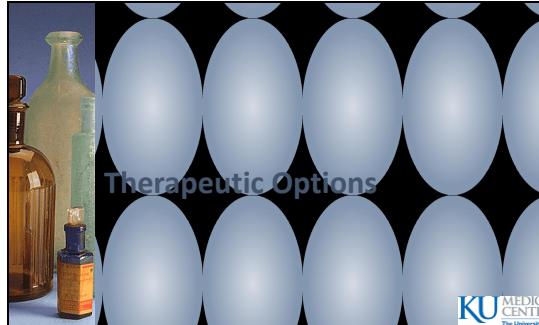
Clinical Work-Up

- > Variability of presentation depending on ingestion
- > Verapamil and Diltiazem -> Bradycardia and Heart Blocks
- > Nifedipine -> Hypotension +/- Reflex tachycardia
- Beta Blockers -> 1st degree heart block w/ prolonged QRS interval
- High risk of atrioventricular dysrhythmias

Clinical Toxicol 2011; 49: 277-283
Am J Med Clin N Am 2014; 32: 79-102
Clin Toxic 2014; 52: 926-943
J Emerg Med 2014; 56(2)

KU MEDIC CENTI The University

Slide 37



Slide 38



Slide 39



Slide 40

GI Decontamination

- > Prevention of absorption (Seems like a good idea)
- > Caution
- Do not induce emesis
- Activated charcoal
- Non-specific product
 - 1-2 hours after ingestion (most effect ~ 6 hours)
 - 50% reduction at 2 hours from ingestion
 - 25 – 100 grams + Sorbitol

Clinical Tox 2011; 49: 277-283
Emerg Med Clin N Am 2014; 32: 79-102
Clin Tox 2014; 52: 926-944
J Medicine 2014; 16(2)

KU MEDIC
CENTI
The University

Slide 41

GI Decontamination

- > Whole bowel irrigation
 - Sustained release medication
 - Hemodynamically stable
 - Polyethylene glycol 1500 – 2000 mL/h (until clear)
 - Beware:
 - > Pronounced hypotension
 - > Respiratory depression
 - > Normal respiration

Clinical Tox 2011; 49: 277-283
Emerg Med Clin N Am 2014; 32: 79-102
Clin Tox 2014; 52: 926-944
J Medicine 2014; 16(2)

KU MEDIC
CENTI
The University

Slide 42

High dose insulin therapy, an evidence based approach to beta blocker/calculum channel blocker toxicity

Christina Woodward, Ali Pourmand¹ and M...
Insulin-Glucose as Adjunctive Therapy for Severe Calcium Channel Antagonist Poisoning

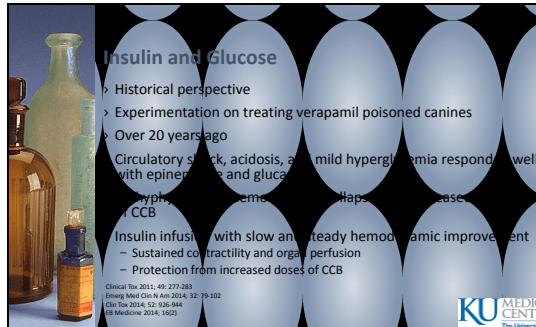
Tony H. Yuan; William P. Kerns II; Christian A. Tomasze
Marsha D. Ford; Jeffrey A. Kline

High-dose insulin therapy in beta-blocker and calcium channel-blocker poisoning

KRISTIN M. ENGBRETSSEN¹, KATHLEEN M. KACZMAREK², JENIFER MORGAN², and JOEL S. HOLGER³

¹Emergency Medicine Department/Clinical Toxicology Service, Regions Hospital, St. Paul, MN, USA
²College of Pharmacy, University of Minnesota, Minneapolis, MN, USA
³Emergency Medicine Department, Regions Hospital, St. Paul, MN, USA

Slide 43



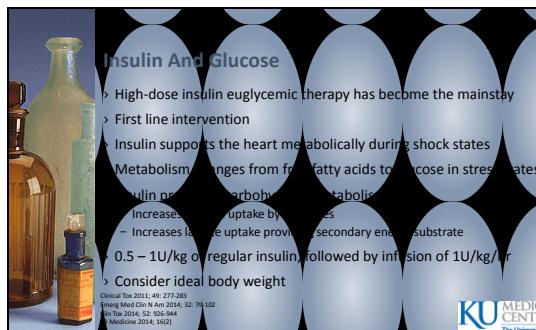
Insulin and Glucose

- > Historical perspective
 - > Experimentation on treating verapamil poisoned canines
 - > Over 20 years ago
 - Circulatory shock, acidosis, and mild hyperglycemia respond well with epinephrine and glucose
 - Hypotension, tachycardia, tachypnea, hypoglycemia, and metabolic acidosis
 - CCB
- > Insulin infusion with slow and steady hemodynamic improvement
 - Sustained contractility and organ perfusion
 - Protection from increased doses of CCB

Clinical Toxicology 2011; 49: 277-283
Emerg Med Clin N Am 2014; 32: 79-102
Clin Toxic 2014; 52: 926-944
J Clin Medicine 2014; 3(6)

KU MEDIC CENTI The University

Slide 44



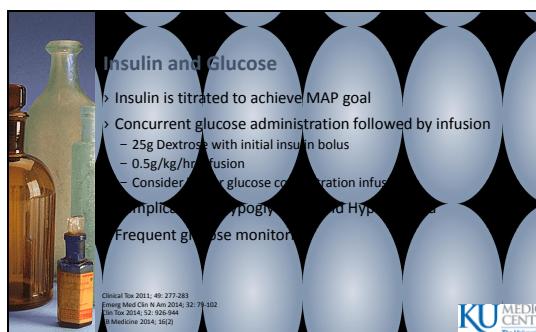
Insulin And Glucose

- > High-dose insulin euglycemic therapy has become the mainstay
- > First line intervention
- > Insulin supports the heart metabolically during shock states
- Metabolism changes from free fatty acids to glucose in stress states
- > Insulin promotes carbohydrate metabolism
 - Increases glucose uptake by tissues
 - Increases lactate uptake providing secondary energy substrate
- 0.5 - 1U/kg of regular insulin, followed by infusion of 1U/kg/hr
- > Consider ideal body weight

Clinical Toxicology 2011; 49: 277-283
Emerg Med Clin N Am 2014; 32: 79-102
Clin Toxic 2014; 52: 926-944
J Clin Medicine 2014; 3(6)

KU MEDIC CENTI The University

Slide 45



Insulin and Glucose

- > Insulin is titrated to achieve MAP goal
- > Concurrent glucose administration followed by infusion
 - 25g Dextrose with initial insulin bolus
 - 0.5g/kg/hr infusion
 - Consider low glucose concentration infusion
- Implications of Hypoglycemia and Hyperglycemia
- Frequent glucose monitoring

Clinical Toxicology 2011; 49: 277-283
Emerg Med Clin N Am 2014; 32: 79-102
Clin Toxic 2014; 52: 926-944
J Clin Medicine 2014; 3(6)

KU MEDIC CENTI The University

Slide 46



Calcium

- > Seems like a natural reversal agent
- > Evidence is weak
- > Dosage not well defined / no dose-effect relationship
- 10 – 20mL of 10% CaCl₂ or 30 – 60mL 10% Calcium gluconate
 - Which provides more calcium?
 - CaCl₂ via IV or nasogastric. Calcium gluconate peripheral IV.
 - CaCl₂ syrup available in Australia. Calcium gluconate usually delivered in 10% solution
- Single dose?
 - Efficacy not well defined
 - Too much Ca⁺⁺ can be harmful

Clinical Toxicology 2011; 49: 277-281
Emerg Med Clin N Am 2014; 32: 79-102
Clin Toxicol 2014; 52: 926-944
Crit Care Med 2014; 16(2)

KU MEDICAL CENTER The University

Slide 47



Vasopressors

- > Commonly used, variable success
- > Not necessarily failure of treatment, too profound of toxicity
- > Standard dosing may not be adequate
 - Push the limits
 - No single drug has been shown to be superior

Clinical Toxicology 2011; 49: 277-281
Emerg Med Clin N Am 2014; 32: 79-102
Clin Toxicol 2014; 52: 926-944
Crit Care Med 2014; 16(2)

KU MEDICAL CENTER The University

Slide 48



Vasopressors and Inotropes

TABLE 3

Agent	Common Name	Mechanism	Effects	Uses in Shock
Dobutamine		β ₁ agonist	Increased CO	Cardiogenic, Septic, Hemorrhagic
Dopamine		Low doses: DA-agonist; High doses: α ₁ , β ₁ agonist	Increased CO and mild increase in blood pressure	Cardiogenic, Septic, Hemorrhagic
Epinephrine		α ₁ , α ₂ , β ₁ agonist	BPH, SVR, and CO	Cardiogenic, Septic, Hemorrhagic
Norepinephrine	Levoephedine	α ₁ , β ₁ agonist	Vasoconstriction	Cardiogenic, Septic
Therapy/drive		α ₁ agonist	Isotonic, BP	Septic
Vasopressin		VI, V2 agonist	Vasoconstriction and anti-diuretic	Septic

www.aazemrnsa.org

KU MEDICAL CENTER The University

Slide 49

Glucagon

- > Studied since the 1960's
 - Increased chronotropic and inotropic effects
- > Produced by the pancreas, glucose homeostasis
- Increases Ca²⁺
 - Decreased CCB / BB OD
 - > Negative inotropic and chronotropic effects
- Passes through catecholamine driven phosphorylation of cAMP
- Early use may provide benefit
- 3 – 5mg over ~2 min, repeat – 10mg, main 2 – 5mg/h

> Nausea / Vomiting

Clinical Toxicology 2011; 49: 277-283
Emerg Med Clin N Am 2014; 32: 79-102
Clin Toxic 2014; 52: 926-944
EB Medicine 2014; 16(2)

KU MEDIC CENTI The University

Slide 50

Atropine

- > Used for the bradycardic and hypotensive patient
- > Rarely effective in CCB or BB OD
- > Studies provide limited evidence
- 0.5 – 1mg qv every 2 minutes up to 3mg may be trialed

Clinical Toxicology 2011; 49: 277-283
Emerg Med Clin N Am 2014; 32: 79-102
Clin Toxic 2014; 52: 926-944
EB Medicine 2014; 16(2)

KU MEDIC CENTI The University

Slide 51

Sodium Bicarbonate

- > Widened QRS
 - Indicative of sodium channel blockade
- > If the QRS shortens, consider infusion
- Not routinely used for treatment of CCB or BB OD

Clinical Toxicology 2011; 49: 277-283
Emerg Med Clin N Am 2014; 32: 79-102
Clin Toxic 2014; 52: 926-944
EB Medicine 2014; 16(2)

KU MEDIC CENTI The University

Slide 52



Phosphodiesterase Inhibitors

- > Amrinone, Enoximone, Milrinone
- > Prevent degradation of cAMP
- > Possible efficacy with concomitant use with Glucagon

Side effects: Hypotension
May paradoxically increase the arrhythmia in already hypotensive patients
Long half-life

Clinical Toxicology 2011; 49: 277-283
Emergency Medicine Clinics of North America 2014; 32: 79-102
Clinical Toxicology 2014; 52: 926-944
Journal of Medical Toxicology 2014; 10(2)

KU MEDICAL CENTER The University

Slide 53



Pacing

- > Transvenous or Transthoracic
- > Consideration for patient refractory to other therapies
- > Goal HR ~ 50 – 60 bpm

Efficacy is uncertain
While Hemodynamic support may be necessary

Clinical Toxicology 2011; 49: 277-283
Emergency Medicine Clinics of North America 2014; 32: 79-102
Clinical Toxicology 2014; 52: 926-944
Journal of Medical Toxicology 2014; 10(2)

KU MEDICAL CENTER The University

Slide 54



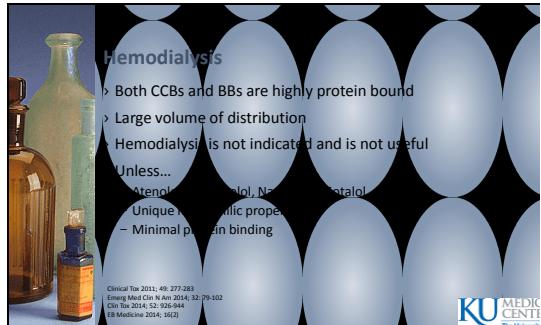
ECMO / IABP

- > Extracorporeal Membrane Oxygenation
- > Itra-Aortic Balloon Pump
- > No clear guidelines
 - ARDS Studies
 - Few reports of good outcome in patients refractory to all other therapies

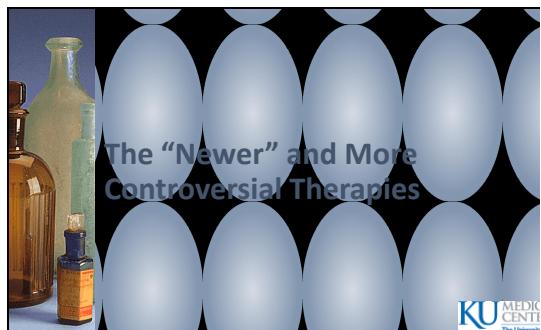
Clinical Toxicology 2011; 49: 277-283
Emergency Medicine Clinics of North America 2014; 32: 79-102
Clinical Toxicology 2014; 52: 926-944
Journal of Medical Toxicology 2014; 10(2)

KU MEDICAL CENTER The University

Slide 55



Slide 56



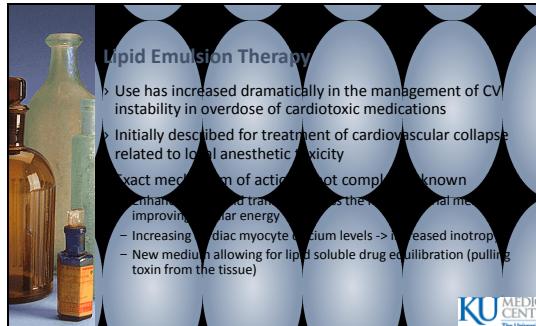
Slide 57



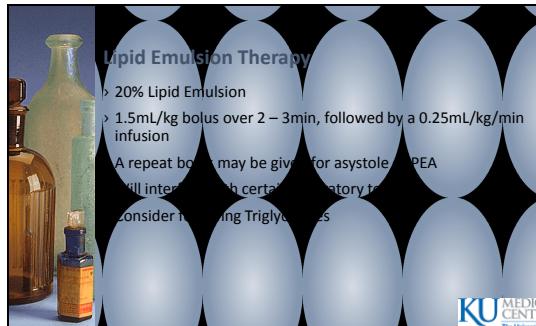
Slide 58



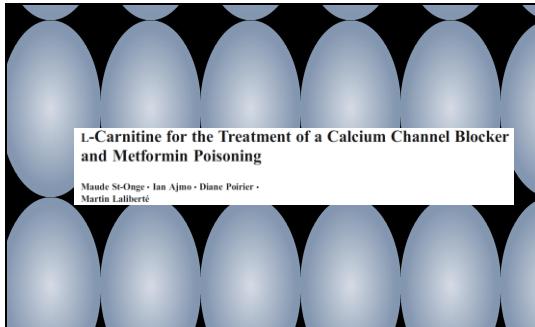
Slide 59



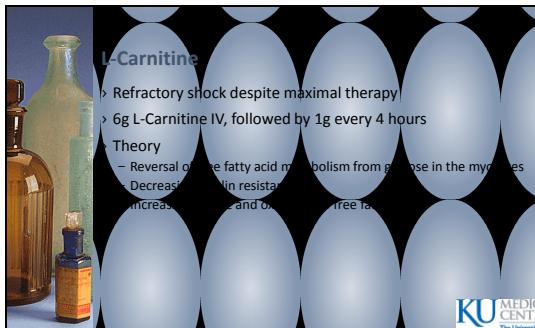
Slide 60



Slide 61



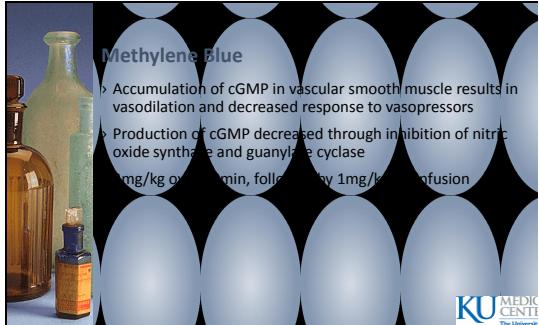
Slide 62



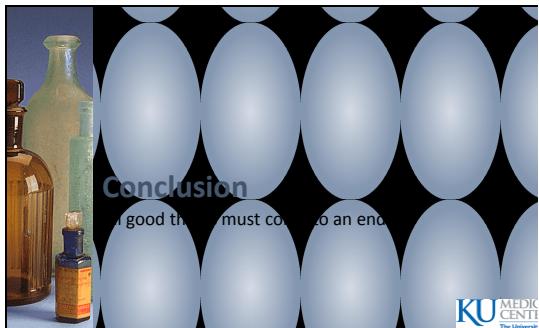
Slide 63



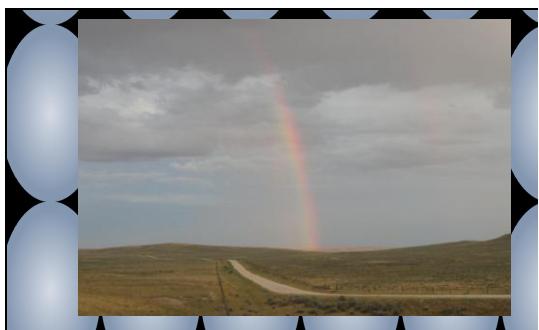
Slide 64



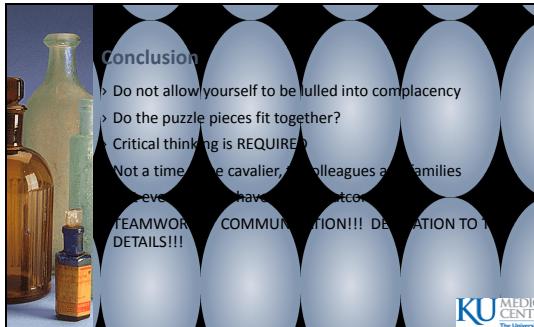
Slide 65



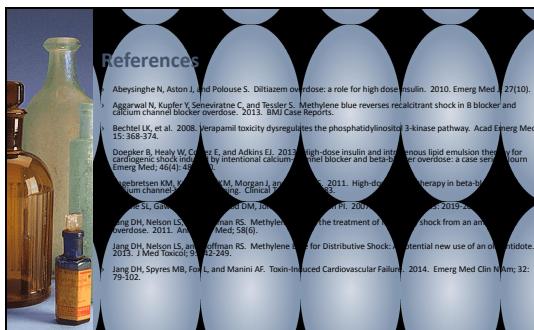
Slide 66



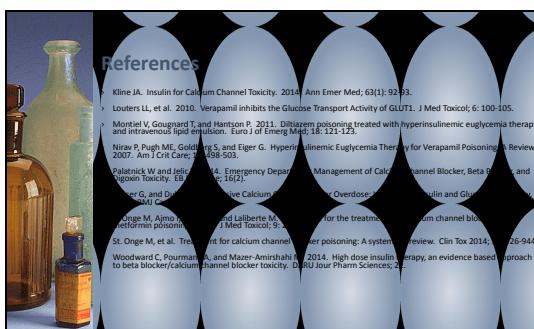
Slide 67



Slide 68



Slide 69



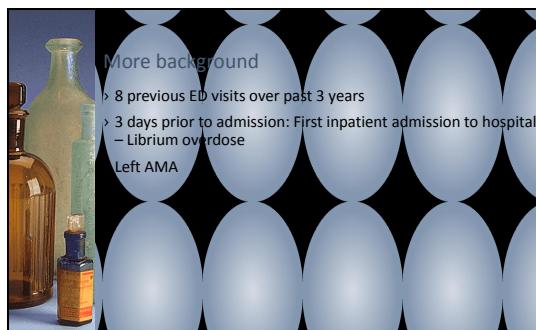
Slide 70



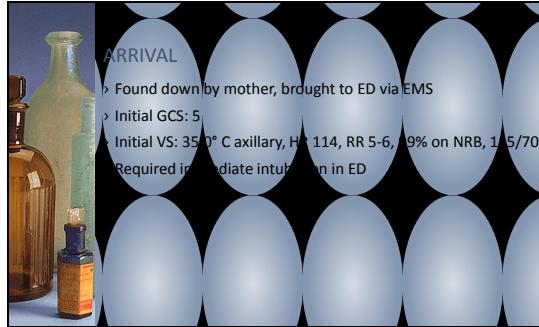
Slide 71



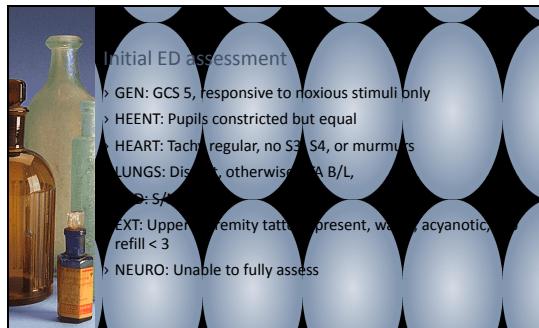
Slide 72



Slide 73



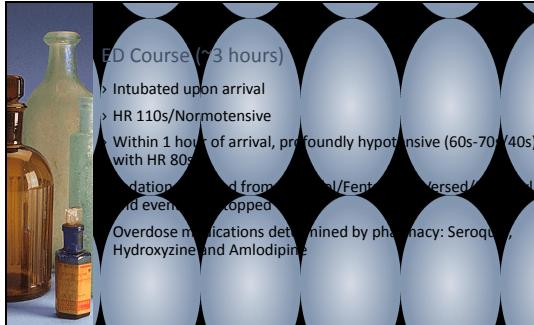
Slide 74



Slide 75



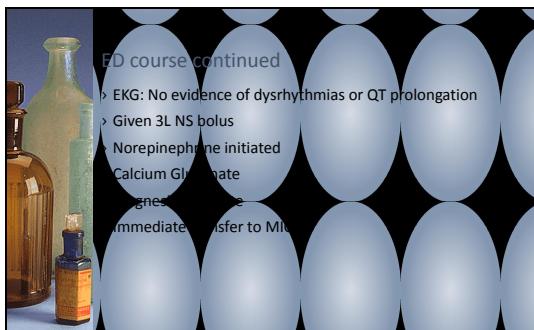
Slide 76



ED Course (~3 hours)

- > Intubated upon arrival
- > HR 110s/Normotensive
- > Within 1 hour of arrival, profoundly hypotensive (60s-70s/40s) with HR 80s
- > Bradycardia and from Atropine/Fentanyl/Versed/Atropine and even stopped
- > Overdose medications determined by pharmacy: Seroquel, Hydroxyzine and Amlodipine

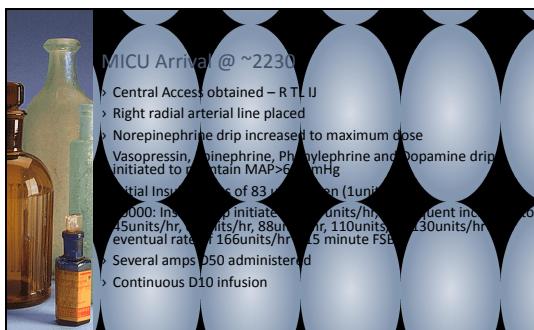
Slide 77



ED course continued

- > EKG: No evidence of dysrhythmias or QT prolongation
- > Given 3L NS bolus
- > Norepinephrine initiated
- > Calcium Gluconate
- > Immediate transfer to MICU

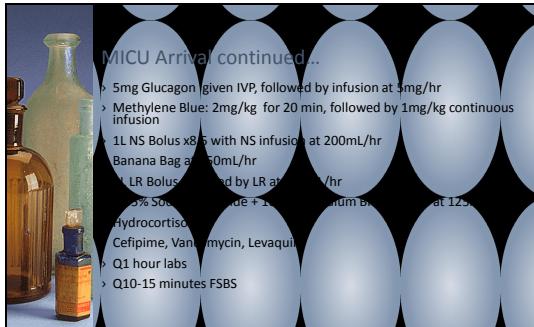
Slide 78



MICU Arrival @ ~2230

- > Central Access obtained – RT-LU
- > Right radial arterial line placed
- > Norepinephrine drip increased to maximum dose
- > Vasopressin, Epinephrine, Phenylephrine and Dopamine drip initiated to maintain MAP>65mmHg
- > Initial Insulin bolus of 83 units regular (1unit/kg)
- > Dextrose 2000: Insulin drip initiated at 4units/hr, subsequent increase to 4units/hr, 8units/hr, 88units/hr, 110units/hr, 1230units/hr, eventual rate of 1166units/hr, 15 minute FSB
- > Several amps D50 administered
- > Continuous D10 infusion

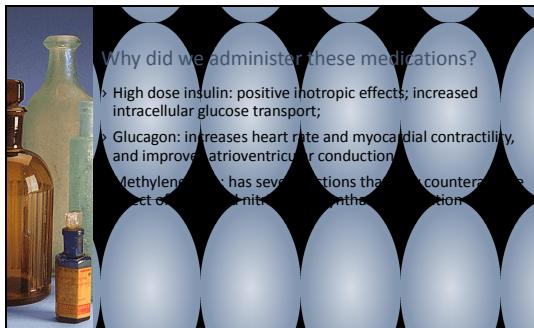
Slide 79



MICU Arrival continued...

- > Smg Glucagon given IVP, followed by infusion at 5mg/hr
- > Methylene Blue: 2mg/kg for 20 min, followed by 1mg/kg continuous infusion
- > 1L NS Bolus x8 w/ NS infusion at 200mL/hr
- Banana Bag at 50mL/hr
- LR Bolus followed by LR at 100mL/hr
- 5% Sodium Bicarbonate + 10% Dextrose + Calcium Bicarb at 125mL/hr
- Hydrocortisone 100mg IV
- Cefipime, Vancomycin, Levaquin
- > Q1 hour labs
- > Q10-15 minutes FSBs

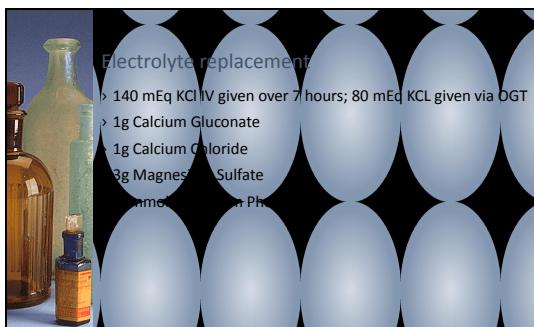
Slide 80



Why did we administer these medications?

- > High dose insulin: positive inotropic effects; increased intracellular glucose transport;
- > Glucagon: increases heart rate and myocardial contractility, and improves atrioventricular conduction
- > Methylene Blue: has several actions that can counteract the effect of nitro- and amine drugs on the myocardium.

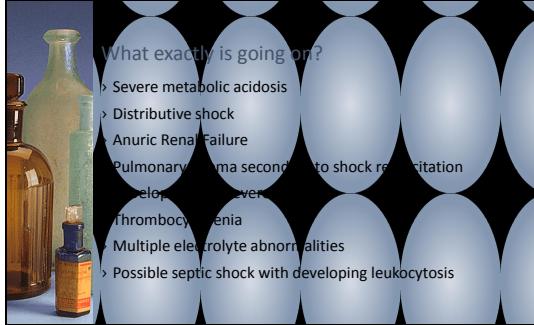
Slide 81



Electrolyte replacement:

- > 140 mEq KCl IV given over 7 hours; 80 mEq KCl given via DGT
- > 1g Calcium Gluconate
- > 1g Calcium Chloride
- > 3g Magnesium Sulfate
- > 100mL 5% Dextrose + Ph

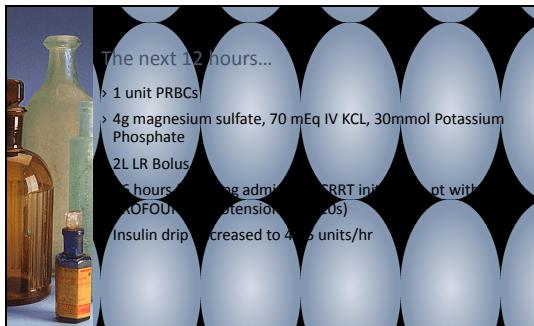
Slide 82



What exactly is going on?

- > Severe metabolic acidosis
 - > Distributive shock
 - > Anuric Renal Failure
 - Pulmonary edema secondary to shock resuscitation
 - > Development of hypotension
 - > Thrombocytopenia
 - > Multiple electrolyte abnormalities
 - > Possible septic shock with developing leukocytosis

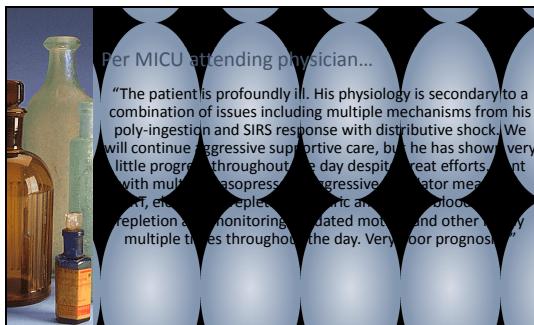
Slide 83



The next 12 hours...

- 1 unit PRBCs
 - > 4g magnesium sulfate, 70 mEq IV KCL, 30mmol Potassium Phosphate
 - 2L LR Bolus
 - Hours after admission to RRRT initiation with AFOURNED extension (20s)
 - Insulin drip increased to 4-6 units/hr

Slide 84



Per MICU attending physician...

Slide 85



24 hours later...

- > Opened eyes to voice, began following simple commands
- > Net + 28L fluid!!!
- > Severe ARDS, paralyzed with Cisatracurium due to dysynchrony with vent
- > Only 2 pressors (Norepinephrine and Vasopressin)
- > Elevated blood pressure (highest 25)
- > Shock Liver
- > Insulin drip off, D50 q4hrs for hypoglycemia

Slide 86



Nursing care the first 24 hours...

- > 4-5 RNs first several hours
- > 12-16 IV pumps running with continuous medications
- > MULTIPLE titrations/rate changes/IV bag changes
- > Q1 hr labs
- > Q15 minute - 1 hour FSBS
- > Ventilator management
- > Hemodynamic assessment/titrations
- > CRRT initiation/management
- > General nursing care/assessment
- > Other medications

Slide 87



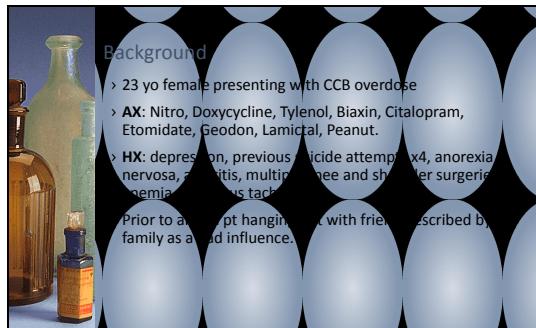
Overall course

- > 4 days later: Off all vasopressors
- > 5th day: STAT urology consult due to significant penile/scrotal edema
- > 8 days later: CRRT stopped
- > 9th day: Extubated to nasal cannula (significant delirium/Confusional Nephropathy)
- > 11th day: UWAS monitor off
- > 12 days later: Transferred to floor
- > 16th day: transferred to inpatient psychiatry
- > 22 days later: Discharged home.

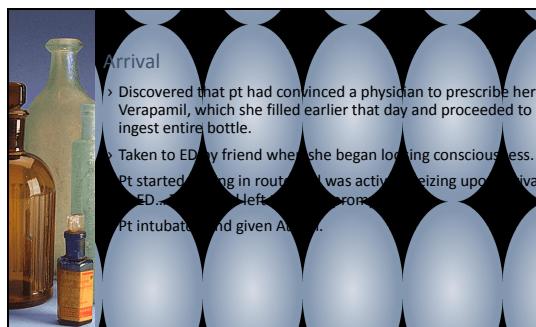
Slide 88



Slide 89



Slide 90



Slide 91

Labs on Arrival

- > Lactate 14.4
- > ABG- pH 6.74, CO₂ 48, PaO₂ 94, HCO₃- 6.5
- > Glucose 483
- Cr 1.92
- 1.7
- Anion gap
- Ca initially high and quickly dropped

Slide 92

ED Adventure

- > Pt in 3rd degree block with HR ~20 and SBP ~60.
- > Given calcium, activated charcoal, Epi, bolus of LR, Atropine, 20% lipid emulsion therapy, and high dose insulin therapy.
- Pt bolused with 70 units of insulin and started on gtt at 7 u/hr.
- Intravenous defibrillator placed after extubation, pacing failed.

Slide 93

Some Rationale

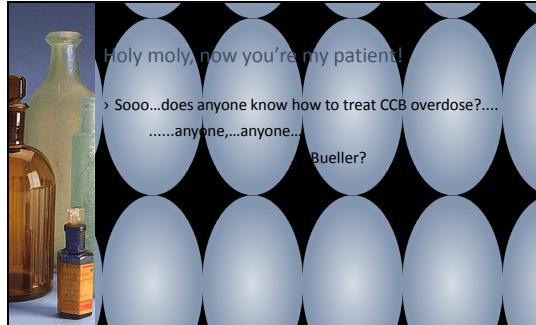
Effects of CCB overdose:

- > Hypotension from negative effects on inotropy, chronotropy, and peripheral vascular tone.
- > Hyperglycemia from blockade release of insulin.

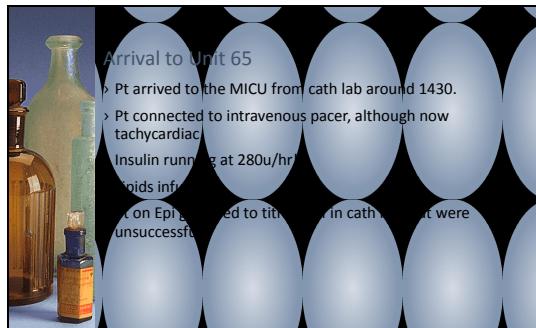
Effects of treatment:

- > High dose insulin has inotropic effect.
- > Fat emulsion creates lipid sink.

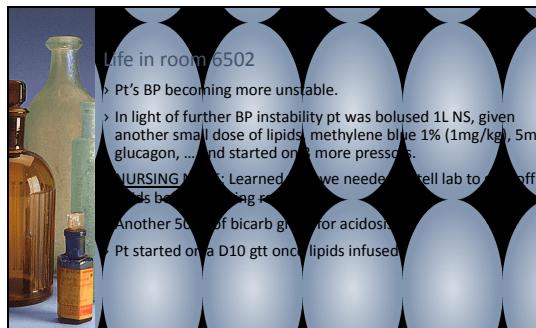
Slide 94



Slide 95



Slide 96



Slide 97



Overnight...

- > Pt also now requiring large amounts of KCl replacement due to intracellular shifts....200mEq over the next 24hrs
- > Overnight pt was weaned off one pressor, but then had to be restarted once she began waking up and needed sedation.
- > Pt went through 47 bags of insulin in 24hrs and another 30mls of KCl.

Slide 98



The next day...

- > Pt off all vasoressors.
- > MANY electrolyte replacements
- > D20 gtt started to maintain blood glucose > 100 with insulin therapy.
- > Blood sugar checks q30min.
- > Insulin titration rate → 10% as pt tolerated.
- > Pt still had severe metabolic acidosis.
- > Pt had a lactate >2 for 4 days.

Slide 99



Her Admission

- > Insulin gtt titrated off after 72hrs.
- > Pt's K initially high after insulin stopped, but resolved on its own as AKI resolved.
- > Pt extubated after 48hrs, FIM status in 5 hours, and d/c'd after 6 day admission.
- > Pt developed delirium in recovery where co-oximetry pacer d/c'd on washout.
- > Pt thought by psych to have a conversion disorder.
