UCSD Medical Toxicology

Please place your name here: _____
Date of Clerkship:_____

Please Circle Who You Are:
UCSD EM, Navy EM, UCI EM, Rady Peds EM, UCSD IM, UCSD Peds, Navy Critical Care Feloow, UCSD Med Student, Outside Resident, Outside Med Student

Medical Toxicology Rotation

Arranging the Rotation

For **medical students** please contact Beckett Kirk bhkirk@health.ucsd.edu (619-543-3818) who is the medical student rotation coordinator.

For **residents and pediatric fellows** please contact **Maeve-Anne (Mae) Malong** mmalong@health.ucsd.edu (619-543-4627) who is the resident and fellow rotation coordinator.

Medical Toxicology Rotation

Welcome to the Medical Toxicology Rotation. We are happy to have you rotate through and are committed to teaching you as much as we can while you are here. **Included in this packet is a guide for the rotation and also a worksheet with checklists and questions that you will need to return (by email to Dr. Schneir) at the end of the rotation.**

___ABOUT A WEEK BEFORE YOU BEGIN THE ROTATION PLEASE EMAIL Dr. Schneir at aschneir@health.ucsd.edu TO LET HIM KNOW WHEN YOU START SO HE CAN ARRANGE YOU TO BE ASSIGNED A JOURNAL CLUB ARTICLE AND LET YOU KNOW WHERE TO MEET ON YOUR FIRST DAY

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UCSD Medical Toxicology

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UCSD Medical Toxicology

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Weekly Schedule

Monday: 9 AM-10 AM Fellow Chapter Reviews

10 AM to 1200 Toxicology Journal Club

1200 to 1300 California Poison Control Center Conference (alternating Mondays)

Location: MPF building 4th floor conference room at Hillcrest

Note: Mondays are the most important days for rotators to be present. Please do your best not to schedule any other activity this day.

Tuesday: 1st Tuesday: EM conference 0700-1030 AM; La Jolla ACTRI

UCSD Medical Toxicology

auditorium, 1W-210; map at https://maps.ucsd.edu/map/default.htm

2nd Tuesday: EM conference 0700-930 AM; Hillcrest 8th floor conference room 833 (main hospital is on southwest corner)

3rd Tuesday: EM conference 0700-varies; La Jolla LC 145 Med Ed

4th Tuesday: EM conference 1100-varies; Hillcrest first floor main hospital auditorium

5th Tuesday: EM conference 0700-930 AM; Hillcrest 8th floor conference room 833 (main hospital is on southwest corner)

Wednesday:	Rounds (ask day prior when to arrive)
Thursday:	09:30 Poison Center Case Review (MPF 4th floor conference room)
Friday:	09:30 Poison Center Case Review (MPF 4th floor conference room)

Note: Always ask day prior what plan is for next day. Also, timing of bedside consultations and rounds are done based on attendings/fellows schedules.

Dress Code: Most days you will be in the hospital at some point seeing patients so please dress appropriately—wearing a white coat is preferred. Scrubs are fine. Ties are not required (this is San Diego :).

Components of Rotatation

1. **Medical Toxicology Journal Clubs**. Journal Clubs are on Monday mornings between 10:00 and 12:00. There is an alternation each week between review of recent articles and a specific topic. Rotators will be assigned an article to present every Monday. The fellows will try to assign you an article that is relevant to your practice.

   Presenting an article: Please succinctly review. If someone can read the entire article while you are presenting and you are still presenting—you are taking too long ☺

2. **Fellow Chapter Reviews**. Faculty reviews chapters in one of our primary textbooks (Goldfrank's) with fellows. Emphasis is for fellows and no need for rotators to read chapter or prepare but will be good learning for all.

3. **California Poison Control Center Case Conferences**. Every other Monday from 1200 to 1300. Most of medical toxicologists in California discuss cases. Each site (San Diego, San Francisco, Fresno, and Sacramento) alternates presenting cases. All rotators are expected to attend.

4. **Presentations**. Once during the rotation, each rotator is required to do a presentation.

UCSD Medical Toxicology

When: **any time during your time on rotation. You can coordinate with fellows when to present.**

Length: 10 minute maximum (please assure that you do not go longer)

Topic: Toxicology topic of your choice (**except: NOT on KETAMINE, DEXTRAMETHORPHAN, nor on KRATOM).** For any questions regarding relevant topic please discuss with fellows or Dr. Schneir.

Content: Please focus on toxicological aspects. For example, if reviewing a drug, reviewing therapeutic adverse effects and pharmacokinetics is important but we want to know about poisoning/overdoses.

References: Please **obtain, read, and cite primary literature** in your preparation of this (Wikipedia, erowid, UpToDate can sometimes be helpful but are NOT primary literature).

Handout: please do 1-2 page (maximum) handout (no powerpoint).

***_____ **Please email a copy of the presentation to** aschneir@health.ucsd.edu

The rotation is not considered completed until this is received.

5. **Bedside consultations**. The fellows take primary call. **It is expected that during the day until 1400 that rotators will go and see new consultations with the fellow on call.** If there are multiple consults, the rotator may be asked to see the patient first.

   UCSD EM residents (2 week rotation): it is expected that you take call with the fellow **one entire weekend** during your rotation. Additionally you are expected to take a total of **2 weekday call days** with the fellow during the 2 week block.

   Please list the dates you took call:_____
   Please list the diagnosis/presentation of the patients you evaluated when on call:
   _____

   Navy EM residents and PA fellows, Pediatric EM fellows, and medical students (4 week rotation): It is expected that you take call with the fellow **one entire weekend** during your rotation. Additionally you are expected to take **6 weekday calls** with the fellow during the 4 week block. For pediatric residents and fellows, it would be ideal that you see as many of the pediatric consults as possible even if not on call that day. Let fellow know who will help coordinate. It is required that you provide information below:

UCSD Medical Toxicology

Please list the dates you took call:_____
Please list the diagnosis/presentation of the patients you evaluated when on call:
_____

UC Irvine. UCLA EM, UCSD Internal Medicine, Pediatric/IM and other residents (2 week rotations):

It is expected that during the day until 1400 that rotators will go and see new consultations with the fellow on call. If there are multiple consults, the resident may be asked to see the patient first.

Please list the diagnosis/presentation of the patients you evaluated when doing so:
_____

Coordinating call:

1) The fellows will provide a calendar for you to list when you will be taking call with them. Additionally, on the days you are on call TEXT PAGE the fellow on call and tell him/her that you are on and give them the best number(s) to reach you. If you are on call it expected that you come see the patients even at night.

6. **Phone consultations**. Every **Thursday and Friday at 09:30** the fellows will pull poison center cases that they desire to review. The fellow and faculty will discuss the cases and potentially direct the rotators in contacting the providers, obtaining more information, and providing recommendations.

7. **Daily Rounds**: Timing of rounds is variable.

8. **Didactic Teaching:** on various topics throughout the rotation by faculty and fellows.

Online Teaching Modules. Please complete lectures below and check you have done. If lecture is given live you do not need to do online. Access lectures at website:
https://emergencymed.ucsd.edu/divisions/medical-toxicology/modules.html
If lecture is given live

Emergency Medicine, Internal Medicine, Critical Care Residents and Medical Students:

___Antidepressant Overdoses
___Antidote Update
___Carbon Monoxide
___Hot and Altered: Toxic Causes to Remember
___Snake and Antivenoms

___Wide Anion Gap Acidosis in Toxicology

Pediatric Residents/Fellows:

___ Antidote Update
___ One Pill Can Kill
___ Pediatric Toxins: One Pill Can Kill
___ Tiny People: Tiny Doses
___ Urine Screens for Drugs of Abuse: Review and Rationale Use
___ Wide Anion Gap Acidosis in Toxicology

9. **Reading.** There are 4 articles/reviews that have been placed on our website to read. **Pediatric residents/fellows: read "Toxicology Testing in Kids" and "Methemoglobin". Everyone else: Read all 4 articles.** Please check below box that you have done.

   ___ Toxicology Testing
   ___ Methemoglobinemia
   ___ Serotonin Toxicity
   ___ Charcoal

   Access at http://toxicology.ucsd.edu/Tox%20Rotation.htm

10. **Text.** The latest edition of *Poisoning & Drug Overdose* Editor Kent Olson is a great quick reference, particularly when performing bedside consultations. Many of us have written chapters in the book. One copy will be left in the conference room for all to use as desired (please leave it there). Medical students will be provided a copy to borrow during the rotation-has to be returned to get a grade. Navy residents should have a copy provided/rotated by Navy. UCSD Emergency Residents will have a copy to borrow during the rotation.

11. **Questions**: See syllabus worksheet questions below. Please work on them during the rotation—the didactic teaching, online lectures, articles, and handbook etc. will allow you to answer these. All fellows and faculty are happy to help you with them. Please email the completed packet to aschneir@health.ucsd.edu The rotation is not considered completed until this is received.

Below is Applicable for Medical Students Only

Include a more conceptual overview to the course syllabus
Understand key aspects of the history, physical examination, management, laboratory testing and interpretation of poisoned patients.
Specifically:

UCSD Medical Toxicology

- To identify and distinguish classic toxidromes.
- To correctly interpret common blood gases in poisoned patients.
- Understand why acetaminophen is checked for on intentional overdoses.
- Recognize why properties of carbon monoxide make it so dangerous and the common symptoms of presentation.
- To identify the majority of causes of anion gap acidosis and what is responsible for causing the anion gap.
- To know the common antidotes for various poisonings.

12. Observed History and Physical Examination: At least once during the clerkship it is expected that you perform an observed history and physical examination. This can be observed by either the fellows or faculty.
    ___ Check here that you have done.
    Please detail what the patient was seen for _____.

13. Medical Student Mid-Rotation Feedback: please email Dr. Schneir at aschneir@health.ucsd.edu just after completing the second week of the rotation regarding feedback and this will be provided either by email, phone or directly.
    ___ Check here that you have done.

14. Grading: UCSD is pass/fail/honors. To pass the clerkship attendance and participation in all of the activities is expected and all assignments as detailed in this syllabus must be completed in a satisfactory manner. To achieve honors, performance on all assignments is expected to be excellent. ***To receive their grade students must complete the course and faculty evaluations provided by the School of Medicine. The identity of individual students will not be shared with the course instructors.***

    For now the below task is on hold due to COVID.

15. **Poison Center:** For now due to COVID the below assignment with the poison center is on hold.. Please email Lee Cantrell, the managing director to arrange a time during the month to meet at the actual poison center. **Do not show up unannounced!!** His email is: lcantrell@calpoison.org It is located in the main hospital (first floor west of the reception desk in the lobby room 1-145 in southwing code to get in is 543). **Medical students are required to visit at least once during the rotation and listen to at least 5 calls. Please list nature of the 5 calls you listened to below:**
    1. _____
    2. _____
    3. _____
    4. _____
    5. _____

UCSD Medical Toxicology

Questions for Rotators on Medical Toxicology Rotation UCSD

(Internal Medicine Residents Please Skip Any Peds Questions)

1. Routine blood tests and their interpretation are generally far more important than specific toxicological testing. Blood gases although not routinely needed, can give critical information quickly in poisoned patients.

   Simple, clinically helpful blood gas reading rules:

   -for every acute rise in pCO2 of 10, the pH will decrease about 0.1
   -for every acute decrease in pCO2 of 10, the pH will increase about 0.1
   -*in an acute metabolic acidosis with normal respiratory compensation, the second 2 numbers of the pH will roughly equate with the pCO2*; example 7.30/30*
   -the pH should reflect the primary blood gas abnormality and overcorrection is not expected. Example: if there is a primary respiratory acidosis with subsequent renal compensation, the pH will be below 7.40, not above.

   Interpret the following blood gases (acid/base disturbance and whether compensation is present).

Gas	Interpretation	Medical Condition?
pH 7.39 PCO2 60		

The following can be associated with specific drug toxicity.

	Interpretation
pH 7.20 pCO2 60	

In the next three situations, outwardly all are breathing fast and deep. A lot of clinical clues can be used to help distinguish the causes of all three but just looking at the respiratory pattern one would not be able to know what the actual blood gas abnormality is—hence the importance of the blood gas as the etiologies of each can be quite different. Interpret each blood gas and name agents than can cause.

	Interpretation	Agents That Can Cause
pH 7.20 pCO2 20		
pH 7.60 pCO2 20		
pH 7.46 pCO2 20		

Salicylates will NOT cause which *one* of the above blood gases?_____

Toxic alcohols cannot cause which *two* of the above blood gases?_____

Further comment. The most common blood gas caused by salicylates is a primary respiratory alkalosis and metabolic acidosis. This characteristic blood gas abnormality is very rare with anything else. For this reason, it is emphasized. The key is to recognize the patient with it and interpret the blood gas correctly. A huge clue to correctly interpreting the classic blood gas abnormality with salicylates is to know that in a primary respiratory alkalosis the pH will roughly increase 0.1 for every drop in pCO2 of 10. If the expected pH is much lower….there must be a metabolic acidosis too!

2. Winters equation estimates what the expected pCO2 will be in the setting of an acute metabolic acidosis with normal respiratory compensation. It utilizes the measured HC03 on a chemistry (blood gases calculate HCO3).

   ? pCO2 = 1.5 X (HCO3) + 8 (+ 3)

   In an acute metabolic acidosis with normal respiratory compensation, and a serum HCO3 of 10:
   What would be the predicted pCO2?_____
   Therefore given the blood gas rule described above*, what is the predicted pH?_____

   Toxidromes

   Toxidromes are groups of signs and symptoms (some with characteristic lab values) caused by particular substances, groups of substances. Match the following ones (each has ***one*** best answer). Certain conditions that are not quite toxidromes but present with characteristic combination of history, physical examination and lab testing are added. Certain physical examination findings are quite unique and may help distinguish similar toxidromes. Unique ones include clonus, fasciculations, piloerection, rigidity, and tremors.

   alcoholic ketoacidosis
   antimuscarinic
   cholinergic
   cyanide
   opioid
   opioid withdrawal
   neuroleptic malignant syndrome
   salicylates
   sedative/hypnotic
   sedative/hypnotic withdrawal
   serotonin toxicity
   sympathomimetic
   toxic alcohols

Depressed level of consciousness, hypoventilation, miosis: _____

Tachycardia, hyperthermia, mydriasis, dry skin, picking behavior, delirium, urinary retention: _____

Tachycardia, hypertension, hyperthermia, mydriasis, diaphoresis, agitation without delirium (latter can occur however), no muscular rigidity, no hyperreflexia:_____

Tachycardia, hypertension, hyperthermia, mydriasis, diaphoresis, agitation, delirium, no rigidity, prominent tremors:_____

Coma, tachypnea, normal blood pressure, normal blood glucose, vbg pH 7.14 pCO2 14_____

Nausea, vomiting, tachypnea, tachycardia, normal blood pressure, normal mental status, pH 6.95 pCO2 16:_____

Mydriasis, sneezing, yawning, piloerection: _____

Tachycardia, potential hyperthermia, tachypnea, diaphoresis, confusion vbg 7.46 pCO2 20_____

Tachycardia (or bradycardia), miosis (rarely mydriasis), salivation, lacrimation, urinary incontinence, diarrhea, emesis, fasciculations:
_____

Sudden onset (as in minutes after ingestion or inhalation), depressed level of consciousness, apnea, hypotension, normal sized pupils
_____

Tachycardia, mydriasis, hyperthermia, confusion, tremors, diffuse hyperreflexia and clonus: _____.

Depressed level of consciousness, hypothermia, minimal respiratory depression, (although respiratory depression can definitely occur), loss of airway tone_____.

Hyperthermia, confusion, diffuse muscular rigidity _____.

3. Tricyclic antidepressants have many properties that manifest clinically in overdose.

   The first three properties are the most important and are also shared by diphenhydramine:

UCSD Medical Toxicology

1. **Antimuscarinic (antagonize muscarinic acetylcholine receptors)**

   Clinical manifestations: confusion/coma, mydriasis, dry skin, tachycardia, urinary retention.
   Treatment: supportive

2. **Na+ channel blockade**:
   Clinical manifestations: QRS prolongation, possible dysrhythmias, convulsions.

   Principle treatment:
   QRS prolongation?_____ convulsions?_____

3. **Alpha-1 blockade** (peripheral vasodilation)

   Clinical manifestation: hypotension
   Treatment (after assuring not hypovolemic)?_____.
   ---

4. Reuptake inhibition of dopamine, norepinephrine.

   Clinical manifestations: initial hypertension; tachycardia

   comment: since dopamine is converted to norepinephrine, intravenous dopamine may be less effective; first pressor of choice is norepinephrine (clue to above question)

5. GABA antagonism:

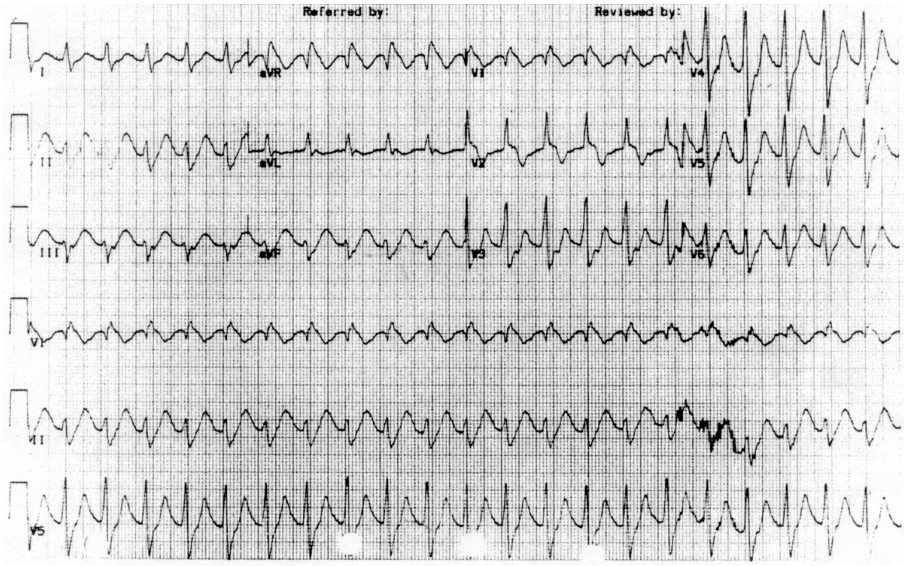
   Clinical manifestation: higher risk convulsions

6. K+ channel blockade:

   Clinical Manifestation: QT prolongation

   comment: avoid administering QT prolonging agents ex. haloperidol, droperidol; tachycardia (see why from other properties) helps decrease risk of torsade which is very rare with acute overdose.

The ECG below demonstrates many of the classic findings that tricyclic antidepressants may manifest. In fact, many sodium-channel blocking drugs (example: diphenhydramine, flecainide, lamotrigine, venlafaxine) may cause similar findings.



These include:
1._____
2._____
3. Terminal rightward axis manifested **by large S wave in I and large R wave in aVR**. Why does this finding occur? Right bundle is more susceptible to sodium channel blockade. Clinical Pearl: unusual to have left bundle pattern from acute sodium channel blockade.

4. Regarding why we check for acetaminophen concentrations in all patients with an intentional overdose (even if we are not suspecting acetaminophen).

   1. Super common ingestion, in many different OTC preparations? T or F
   2. Nonspecific symptoms/clinically silent (major exception is massive OD's) ? T or F
   3. Can cause liver failure and death. T or F
   4. Incredibly effective antidote N-acetylcysteine. T or F
   5. Easy lab test to check. T or F

5. Carbon monoxide binds to iron (in hemoglobin, myoglobin, and cytochromes) and inhibits both the transport of oxygen to cells and utilization of oxygen within cells. True of False regarding properties of CO gas itself that contribute to why it is so dangerous.
   T or F colorless
   T or F odorless
   T or F tasteless
   T or F non-irritating

UCSD Medical Toxicology

Clinical Pearls:
In addition to the properties of carbon monoxide itself, the variety of nonspecific symptoms that it causes make it missed too frequently by health care providers with potentially devastating consequences (patient or family goes back into environment and potentially die). It should be kept on the differential for nonspecific headache and other nonspecific symptoms including nausea, vomiting, malaise etc. In the found down, comatose, confused etc. patient, checking for carboxyhemoglobin upon presentation can potentially cinch a diagnosis, completely change management, including preventing extensive unnecessary workup. The best chance of making the diagnosis is at arrival as the carboxyhemoglobin % will progressively decrease over time.

6. T or F Headache is the most common symptom of carbon monoxide poisoning.

7. Name two screening questions that can help determine if the symptoms a patient has are related to carbon monoxide poisoning, separate than if they have a CO detector at the location of potential exposure which is probably the most reliable one.

    1. _____
    2. _____

8. Give two reasons why urine drugs of abuse screens are nearly worthless in managing the poisoned (or potentially poisoned) patient or the patient with significant altered level of consciousness? Despite being sent often by physicians there is not a good rationale for sending drug screens on an overdose patient or one with altered level of consciousness. This is also well supported by literature. As you will see on the rotation we manage patients optimally without urine drug screens. If you think otherwise please discuss with me ☺!!

    a._____
    b._____

    Comment: opioids agonize the opioid receptor; opiates are derived from opium and have a structure of morphine or similar. Many opioids (fentanyl and analogs, tramadol, methadone) agonize the opioid receptor but structurally are distinct from opiates. Is why they will not trigger a positive on a urine drugs of abuse screen for opiates. The diagnosis of opioid poisoning is based on clinical grounds and response to naloxone, not on a drug screen.

    Comment: quantitative ethanol testing can be helpful to correlate with a patients clinical presentation, of course being aware of the potential for tolerance in heavy drinkers. Drug screens are qualitative and intentionally made to detect a very low level of drug. In the absence of a false positive they can determine recent use but NOT intoxication. Imagine if ethanol was tested for qualitatively (yes/no). The same yes could be 10 mg/dL or 440 mg/dL. This would provide inconsequential information regarding a patient in front of you. Think of urine drugs screens as analogous to that.

9. How do you calculate (the formula) the serum osmolarity?
   Note: Serum osmolality is measured in the lab by freezing point depression. Certain agents such as the toxic alcohols, isopropanol and ethanol can cause an osmol gap—that is a large (typically >10) difference between the measured and calculated osmoles. The common formula you likely used above is an estimate and is appropriate to use but is not the most accurate one. The number 18 is the molecular weight of glucose divided by ten and the number 2.8 is an estimate of the molecular weight of urea divided by 10. When serum osmolality is ordered it is important to check an ethanol at the same time as it is such a common cause of an elevated osmol gap and can be incorporated into the calculation by dividing the ethanol concentration by its molecular weight (46) by ten so 4.6—actually better to use a slightly lower number it turns out—highlighting that all of this calculation is simply an estimation.

10. T or F. The absence of an osmol gap can exclude the presence of a toxic alcohol ingestion.
    Clue: Only the parent compound and not the toxic metabolites will create an osmol gap. As the poisoning progresses and the parent compound is metabolized, the osmol gap will decrease and the anion gap with increase. We do not know what one's baseline osmol gap is prior to a potential ingestion and it turns out that some people have negative osmol gaps (based on difference between estimate by calculation and lab measurement). Lastly, as an example: if a concentration of ethylene glycol is 36 mg/dL an estimate of how many osmoles it will create would be to divide 36 by the molecular weight of EG by 10 = 6.2. Not many osmoles is it!?

11. T or F Acetaminophen can effectively treat hyperthermia.

12. Name 5 toxidromes/clinical syndromes induced by drugs that can cause hyperthermia. (hyperthermia reflects thermoregulatory failure and is NOT a fever that is generally prostaglandin and or cytokine mediated via the hypothalamus). Much of drug-induced hyperthermia relates to excess muscular activity. A huge exception are agents that cause hyperthermia via uncoupling of oxidative phosphorylation.

    1. _____
    2. _____
    3. _____
    4. _____
    5. _____

10. Name the serotonin, norepinephrine reuptake inhibitor (NOT a tricyclic antidepressant) that has sodium channel blockade and therefore can cause QRS prolongation. _____.

UCSD Medical Toxicology

13. What is the treatment for tricyclic antidepressant-induced convulsions? (answer is NOT sodium bicarbonate)_____

14. What is the treatment for diphenhydramine-induced QRS prolongation?_____

15. T or F Carboxyhemoglobin and methemoglobin can be measured accurately on venous blood.

16. T of F Alcoholic ketoacidosis is typically characterized by a relatively normal mental status.

17. Hyperammonemia in the absence of hepatotoxicity is characteristic of which drug?_____ Name the antidote for it._____.

18. Regarding rattlesnake bites:

    1. T or F Antibiotics are indicated prophylactically.
    2. There are two potentially abnormal laboratory findings that are the main focus of initial and serial monitoring in patients with rattlesnake bites. One is seen on a hematologic test and is NOT hemolysis nor DIC which have never been described with rattlesnake bites but is _____. The other is _____(and is not the INR nor PT nor PTT neither of which appear to add information beyond this test).

19. Routine lab tests are critical in the evaluation of certain poisonings and usually more important than any specific lab testing. Name the below lab abnormality.

    a. Precedes cardiac manifestations of *acute* cardiac glycoside poisoning _____.

    b. Precedes cardiovascular manifestations of calcium channel blocker poisoning_____. Answer is NOT hypocalcemia.

    c. First laboratory evidence of systemic fluoride poisoning is _____. Hypomagnesemia and delayed onset hyperkalemia may also occur.

    d. Expected electrolyte (not glucose) with significant methylxanthine (caffeine or theophylline poisoning). _____.

    e. Typically present in acute poisoning with chloroquine or hydroxychloroquine and is thought to be due to a transcellular shift. _____(clue: same answer as d above).

UCSD Medical Toxicology

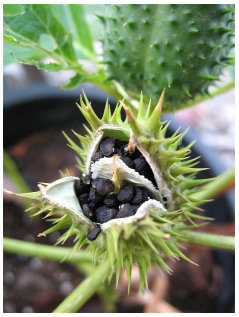
20. Multiple plants have cardiac glycosides (either digoxin or very similarly acting drugs). Two of them are below.

1. Name the plant? Clue: It is in the median of I-5 throughout California. Accidental ingestions by children are universally benign as vomiting occurs and very little is ingested.
_____.

2. _____. Clue: scientific name derives from the fact that flower can hold your digit.

UCSD Medical Toxicology

21. What type of toxidrome does the following plant induce when ingested (typically seeds are made into a tea?_____

22. The following plant will cause the same toxicity. What is its name? _____.

23. What toxin is derived from this bean/seed?

(Clue: it was successfully utilized in a weaponized umbrella by an assassin in London. A model of the umbrella is in the spy museum in Washington D.C.) Ingestions of the bean are generally benign as the hard shell is thought to prevent absorption of the toxin.)

UCSD Medical Toxicology

24. An injection drug user presents with ptosis, mydriasis, and has dysphagia. This is a really close mimick of myasthenia gravis as both cause a descending paralysis. In anyone with weakness a good neurological exam including looking for ptosis should be done. What toxin are you concerned about?
_____.

25. Toxicity from this agent can closely mimic tetanus in that it causes spasms in response to minimal stimuli. _____.

Clue: in the U.S. can be purchased to kill gophers.

Interesting: *Clostridium botulinum* and *Clostridium tetani* both have toxins that prevent the release of neurotransmitters. Botulinum toxin prevents from release of acetylcholine from muscarinic receptors (autonomic effects) and at the neuromuscular junction (weakness). Tetanospasmin prevents the release of glycine. The toxin for the answer in 23 antagonizes the glycine receptor.

26. T or F Opioid withdrawal is typically associated with an altered level of consciousness.

27.

a. How many grams of dextrose in a U.S. ampule of D50? _____
How many kilocalories is this? _____. Not many calories is it!! Is one of reasons so important to feed someone after correcting glucose.

b. How many milligrams/grams of calcium chloride (not the amount of just calcium but the calcium chloride) are in a U.S. 10% ampule of it? _____.

c. How many milligrams per mL is 0.5% bupivacaine? _____.

If having trouble with above please read this: At least in the U.S.: % in medicine refers to weight in grams per volume 100 mL. 1% lidocaine for example is 1 gram/100 mL = 1000 mg/100 mL = 10 mg/mL. An ampule just refers to the container the drug/glucose is in. At least in the U.S. an ampule of D50 is 50 mL, an ampule of 10% calcium chloride or calcium gluconate is 10 mL. Remember that 1 gram of carbohydrate is 4 kilocalories.

28. Name 2 drugs that when administered therapeutically to a patient who is on lithium can produce lithium toxicity. Lithium is not metabolized, is almost 100% eliminated renally and has a narrow therapeutic index.

1. _____
2. _____

UCSD Medical Toxicology

29. Name 2 other drugs (in addition to lithium) that are particularly susceptible to drug-drug interactions. Clue: commonalities of these drugs include a narrow therapeutic index and that they are typically monitored by a drug level and/or other lab test.

    1. _____
    2. _____

30. Roughly what is the average amount of ethanol metabolized per hour (mg/dL)? Beyond concentrations of 20 mg/dL, as enzymes are saturated ethanol is eliminated by 0 order kinetics (fixed amount of drug per time), rather than 1st order kinetics (half-life based) When studied in an ED population of children, adolescents and adults the range is surprisingly narrow. _____

31. Name 3 characteristics of drugs make them amenable to removal by hemodialysis?

    1. _____
    2. _____
    3. _____

32. A patient presents with coma after being found down. Lab testing reveals an AST of 450, an ALT of 200, normal total bilirubin and alkaline phosphatase and an elevated creatinine. (Ethanol has nothing do with answer, nor does acetaminophen nor asterixis, nor toxic alcohols) A clue is that skeletal muscle has AST and ALT in it. The answer to number 2 below is not rhabdomyolysis but an emergent condition that may be present if rhabdomyolysis is present and the comatose patient cannot complain about.

    1. What additional lab test should these lab abnormalities trigger you to check?_____
    2. What condition should be checked for on this patient (generally found on their extremities)?_____

33. Name three agents for which hemodialysis is commonly used to treat severe toxicity.

    1. _____
    2. _____
    3. _____

34. The presence of a wide anion gap acidosis is ultimately caused by the excess of either lactic acid, ketoacids (acetoacetate and/or beta-hydroxybutyrate), or an organic acid. Examples of organic acids include urea, formic acid (methanol metabolite), glycolic acid (ethylene glycol metabolite). For the following list give the primary cause of anion gap acidosis (ketoacid and or lactate, or other organic acid). **One answer has both! (if you do not have an answer with both you are missing it).**

Acetaminophen =_____
Clue to above: both acetaminophen and its major toxic metabolite NAPQI inhibit oxidative phosphorylation. This explains why in massive overdoses of acetaminophen, coma and severe acidosis occurs. In most typical ODs with acetaminophen a mild acidosis is present due to presence of this. Also, answer is NOT 5-oxoproline which is well described in certain people but is quite rare.
Alcoholic ketoacidosis = ketoacid
Methanol = _____ (is not lactate or ketoacid)
Metformin= _____
Urea = urea (duh!)
DKA = ketoacid
Phenformin = _____
Propylene glycol=_____
Isoniazid = _____
Regarding above, ketoacidosis described with INH poisonings but is mainly NOT from ketoacidosis. **Find another answer** that much more commonly causes both a lactic and ketoacidosis.
Inhibitors of Oxidative Phosphorylation (cyanide, carbon monoxide)=_____
Iron = _____
Lactate = lactate (duh!)
Ethylene glycol = _____ (is not lactate or ketoacid)
Salicylates = _____
Note: salicylates themselves contribute some to anion gap but is not part of this answer. Clue to above: in poisoning, salicylates uncouple oxidative phosphorylation so some anaerobic metabolism occurs that generates…… Glycogen and fat are also broken down for energy.

35. A patient has a significant anion gap acidosis. The absence of the following clinical findings would exclude acute poisoning from which of the following agents found above.

    Vomiting and diarrhea?_____
    Convulsions?_____

36. What is the main reason patients with acute isoniazid poisoning have a metabolic acidosis (the presence of this clinical finding is responsible for the answer above)? An animal study demonstrated this in that the animals that were poisoned but paralyzed did not develop an acidosis. _____

37. What is the major clinical manifestation of toxicity associated with abuse (people may snort it) and overdose of bupropion (tachycardia and hallucinations typically precede it)? _____.

38. Inhaled abuse of this drug leads to both functional and depleted vitamin B12 deficiency leading to demyelination and megaloblastic anemia. The agent oxidizes

the cobalt in vitamin B12 making it inactive and it is thought that the body tends to clear the non-functional B12 so levels can actually be low. Patients often present with ataxia due to demyelination of the posterior columns causing proprioceptive difficulty. _____.

39. T or F QRS prolongation in the setting of poisoning is evidence of sodium channel blockade.

40. A patient presents after accidentally ingesting a "heart" medication. The ECG reveals occasional PVC's and the serum potassium is 6.0. The most likely agent is?
    _____

41. A patient accidentally ingests an unknown medication. Physical examination reveals sedation, miosis, and respiratory depression. Naloxone administration reverses all of the adverse effects. What medication could induce these symptoms that is NOT an opioid? _____. (methadone and dextromethorphan are opioids and not the correct answer).

    Clue: any drug in this class of alpha-2 agonists can cause the above.

42. Bradycardia and hypotension can be caused by many drugs including beta blockers, calcium channel blockers, alpha-two agonists, and cardiac glycosides.

    Match the physical examination or laboratory finding with each.

    1. Miosis: _____.
    2. Hyperglycemia (universal and occurs even prior to hypotension):_____.
    3. Hypoglycemia (well reported but rare):_____.
    4. Hyperkalemia (two answers):_1)_____2)_____.

43. Regarding beta blockers and calcium channel blockers. In overdose which one would generally be expected to manifest with cold and clammy skin?_____.
    versus warm and dry skin? _____.

    Clue: which causes pure cardiac effects and which also vasodilates? Vasodilation in the setting of decreased chronotropy, dromotropy, and inotropy is especially bad and is why this class of drugs is one of the worst to overdose on.

44. Name 6 agents that have been used in date rape. Make sure not to miss the one that is by far the most common. Clue: it is in many hand sanitizers.
    1. _____
    2. _____

3. _____
4. _____
5. _____
6. _____

45. Name 2 drugs that commonly cause methemoglobinemia. (lidocaine and nitroglycerine only rarely do and are not the right answers).
    1. _____
    2. _____
    3. _____

46. A patient appears "drunk" but has no ethanol present. A chem 7 is normal (no acidosis) but an osmol gap exists and ketones are positive in the urine. What is the most likely agent (It is NOT methanol nor ethylene glycol as both will eventually cause an acidosis and will not produce ketones)? Clue: causes a ketosis but no acidosis_____.

47. T or F In treating someone for symptomatic hypoglycemia, thiamine administration should precede glucose administration.

    Note on above: Some still teach this incorrectly despite extensive reviews debunking the myth. Thiamine enters cells much more slowly than glucose.

48. T or F Fluorescein is added to ethylene glycol (antifreeze) so physicians can identify the presence of it in the urine.

    There are multiple studies on the clinical utility of using a Woods lamp to detect fluorescein in the setting of possible ethylene glycol detection. It is NOT reliable and should not be used!

49. A patient has a generalized convulsion while out boating with his family. He presents confused with normal vital signs. Name the potential non-ingested toxin that needs to be considered._____. If you get this wrong you did not watch all of the lectures ☺.

50. A patient ingests pills that are used to treat his mothers "positive ppd" and develops convulsions. What is the antidote?_____

51. T or F The presence of vomiting and diarrhea within 5 hours after a mushroom ingestion predicts the ingestion of a *non-hepatotoxic* mushroom. (This is THE most important clinical question regarding hepatotoxic mushrooms)

52. T or F Lead toxicity predominantly manifests as a sensory neuropathy. Clue: most drug induced neuropathies are sensory, is this the exception?

53. What is the predominant reason iron poisoning causes a lactic acid metabolic acidosis? (Clue: is NOT ferric conversion to ferrous and release of hydrogen, nor effect on oxidative phosphorylation) _____

54. T or F There is no benefit of beginning N-acetylcysteine treatment for the vast majority of acute acetaminophen overdoses at 0-4 hours s/p ingestion as compared with 4-8 hours.

    Clinical Pearls: In patients you are not suspicious of having ingested acetaminophen, checking a concentration on arrival is fine. Acetaminophen is rapidly absorbed and excluding the presence in the blood excludes recent ingestion of it. If you know the patient ingested acetaminophen, however, draw the level at 4 hours (nomogram starts at 4 hours—checking a level earlier is generally not helpful (unless in the rare massive overdose). Beginning the antidote N-acetylcysteine prior to 8-9 hours appears to be optimal timing. Empiric administration of IV NAC has caused fatal anaphylactoid reactions in patients who never even need it!! Empiric administration (prior to level returning) is appropriate if good story of ingestion greater than 150-200 mg/kg and by time NAC will be begun is beyond 9 hours.

    Antidote specific:

55. This drug antagonizes the release of preformed insulin and is used after glucose to treat sulfonylurea toxicity (diazoxide can do this but is not nearly as effective nor likely as safe as this agent:_____

56. This drug bypasses the beta receptor and is used to treat beta blocker toxicity:_____. Starting bolus dose is 5 to 10 milligrams.

57. Very high dosing (1-2 unit/kg bolus followed by 0.5-1 unit/kg/hour) of this drug is used to treat calcium channel blocker poisoning:_____.

58. The incredibly effective antidote for acetaminophen poisoning is:_____

59. This drug is a an acetylcholinesterase inhibitor and can be used to reverse antimuscarinic-induced delirium:_____

60. This drug blocks alcohol dehydrogenase and is used to prevent the metabolism of ethylene glycol and methanol:_____.
    _____ is used to remove the toxic alcohol and its toxic metabolites.

61. The current favored antidote for cyanide poisoning is a vitamin B12 precursor:_____. It is bright red and when given turns the skin and plasma red.

62. The treatment for methemoglobinemia is:_____. Give a blue drug to treat a blue patient.

63. The drug used to treat malignant hyperthermia:_____.

64. Administered in organophosphorous poisoning. Correct endpoint is drying of secretions:_____.

Toxicology Unknowns (Classic presentations: name the poison/syndrome)

1. COPD patient presents with convulsion, tremors, tachycardia, wide pulse pressure and is noted to have hypokalemia. (answer not albuterol or other pure beta agonist which could do same thing) _____
2. Patient with bipolar disorder presents tremulous, confused, hyperreflexic. One is a syndrome/toxicity_____ one is toxicity from drug they could be on_____.
3. Psychiatric patient who has had no changes/additions of any medications presents with severe rigidity, confusion, elevated CPK and a rectal temperature of 107 F. _____
4. Patient presents following the *ingestion* of a therapeutically used, non-radioactive medication, with severe vomiting and diarrhea and subsequently develops multi-system organ failure and alopecia. Thallium is not answer as although causes alopecia, GI symptoms are not prominent but neuropathy is. What is drug?_____

Pediatric Specific Questions (Internal Medicine can Skip)

65. T or F Initial dosing of antivenom for rattlesnake envenomation is identical in children and adults.
66. What is the pediatric dosing of glucose for hypoglycemia? For answer use mL of D(?)/kg as you would literally order it.
    1. Neonates? _____
    2. Children? _____
67. How do you dose activated charcoal to children? _____
68. What toxic pharmaceutical additive has been occasionally added to acetaminophen and has caused outbreaks of pediatric deaths characterized by renal failure? _____.

69. A child presents with ataxia and hypoglycemia. The hypoglycemia is corrected but the patient is still ataxic. Name the most likely agent (it is NOT a sulfonylurea, nor is it insulin as why then would child be persistently ataxic?; additionally is NOT ethylene glycol which can cause ataxia but would not expect hypoglycemia!)_____.

70. Child presents with significant vomiting and diarrhea. KUB reveals pills in stomach. Toxin? _____.

71. Name 3 non-beverage sources of ethanol:
    1. _____
    2. _____
    3. _____