University of Colorado
Anschutz Medical Campus
School of Medicine

Internal Medicine

Intern Guide
2014-2015
University of Colorado Guide to Internal Medicine

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Based on a 2007 Gray/Connors Publication
Disclaimer

This handbook is meant to serve as a guide to the practice of internal medicine. All information contained within is believed to be reliable and accurate but is by no means exhaustive on any one topic. The guidelines found within are only recommendations as to the practice of medicine. The editors of this book do not provide any guarantee of their accuracy or completeness.
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A Message From The Chiefs

We think that Colorado is an amazing place to train in medicine, full of a varied array of both medical and non-medical gems. To find them you have to be fully invested in life, both in and out of the hospital. We hope your time here is filled with as much personal and professional satisfaction as ours has been. As your Chief Medical Residents, it is our job to assure this promise is fulfilled. We are committed to helping you in any way we can.

This guidebook has been published annually since 2007 and given to each incoming class of interns. When the guide was described to a non-medical friend of ours he said, “Oh, you mean like CliffsNotes® for doctors?” Exactly. It’s meant to serve as a quick reference during the night and to help give you a push in the right direction toward the correct management of many of the common diagnoses and issues with which you will be confronted this year. That said, it should function as a jumping off point: something to get you started while you dig deeper into the problem.

Keep this reference guide in your pocket and know that you are never more than a page flip away from all those who came before you. Know that, along with your resident, you are following the path of countless doctors who were in your same situation not long before. Feel secure in the understanding that every unusual night float call or patient you see has been dealt with before – that there are answers out there and that you will find them.

You are starting out on something that will change the way you think. It is an experience that cannot be replicated or repeated so take advantage of this opportunity while it is available to you. Call upon those above you. Be judicious, confident and decisive, but never cavalier.

The mastery of medicine takes two things: knowledge and experience. The first will come from the time you are willing to put in to mastering your craft. We want this book to be one of many sources to help you toward your first goal. The second is just that: experience. It will come over time and cannot be rushed, so don’t worry about trying to speed up the process. It’s a life-long learning process, and we promise, it’s happening for you already. You will look back on these years as some of the most difficult and rewarding of your career. We hope that you come away from the process of internship a stronger, more confident person, but realize that you may not necessarily start out that way. Any help that we as the chiefs can provide is yours. Anytime, day or night…just ask.

The Chief Medical Residents
The Eightfold Path Through Internship

1. **Play Nice.** Keep your head. There will be many situations where you may feel the urge to blow up or be less than cordial to your colleagues. Don’t. You’re the doctor now, the leader of the team. People will look to you for calm and confidence during the stressful times…lead by example.

2. **Rely on those above you.** You’re never alone in the hospital, your clinic, or on any other rotation. Call your resident with any and all questions you are unsure of the answer to and the chiefs with any problems that come up…that’s what they’re there for. Remember the old adage: it’s better to wake your resident at 2 AM with a question than at 4 AM to intubate someone.

3. **Pay yourself first.** It’s an old maxim from the economic world and very relevant to the busy lifestyle you have chosen. Get up 15 minutes early and have breakfast; it’s worth hours of fatigue later in the day. If you exercise (and you should), do so the second you get home before the urge to nap sets in. Do something enjoyable on your days off (after sleeping in a little, of course).

4. **Have fun.** Internship is difficult, but fun, especially here in Colorado. When you’re on, you’re on, but on your days off, be off. If you don’t know what to do, call a colleague or friend…you’re all in the same boat and will find plenty of fun ways to spend your free time in this state of ours.

5. **Make time to go to conferences and morning reports.** Time won’t just fall into your lap during your intern year; you will have to carve it out. Most of your payment over these three years comes in the form of educational conferences and lectures from senior faculty. Tap this resource till it’s dry. With the exception of an emergency situation demanding immediate patient care, conference should take priority over all other activities, including rounding.

6. **Communicate.** Effective communication solves just about every misunderstanding in the hospital; conversely, ineffective talk between colleagues and staff will cause plenty of turmoil. When you are having trouble seeing eye to eye with someone, start over by asking what that person is saying, what it is specifically that they want from you and how you can help them solve their (and your) problem. Along these same lines, be honest in all your dealings. If you haven’t done something, own up to it. Lying undermines all communications and puts future dealings between you and others in question. Every resident and attending above you has made mistakes; admitting them in a timely manner will serve you and your patients better than an attempt at concealment.

7. **Learn from your patients.** In the morning, on rounds and every second you can make to have contact with them. This also includes reading after work on the conditions you have seen that day, especially those you may have initially misdiagnosed or were unsure about. Patients have more to teach you (if you listen) than any lecture series or book.

8. **Keep a sense of self.** Internship can be a tough time and it is easy to let the work define you. While we are all here for the primary purpose of becoming great doctors, you were all selected because you were already great people. Don’t let the diversity you demonstrated to get here fall by the wayside. Keep something from your prior life (working out, reading, playing basketball on Saturday, your pre-residency friendships) and always find time for it, even on the busy months. You got here because of who you are and, while internship is certainly transforming in a lot of ways, no job should change your fundamental beliefs or inner self. If you find this happening, come talk to one of the chiefs.
**Resident Mentoring Program**

Residents in the Department of Medicine participate in a mentoring program with four distinct components. The components and the key mentors for each are described below.

1. **Bi-annual career advising and performance review:** residents are required to meet with an assigned core residency faculty member a minimum of twice each year (and interns will be scheduled for an additional meeting during the first 2 months of training). The purpose of these meetings is threefold:
   - To review and provide specific feedback on the resident’s performance and overall well-being to date with a focus on educational and professional goal setting for the short and long-term.
   - To monitor progress towards meeting ABIM requirements to be board-eligible
   - To inquire about their personal well-being
   - To discuss future career aspirations and discuss opportunities within and outside the program to assist the resident in meeting or further investigating career interests
   - To obtain feedback and suggestions from residents on rotations and other aspects of the overall program in order to best meet resident needs over time.

All residents are initially assigned to either the program director or an associate program director. However, they may be referred to others either within or outside of this group for specific career and/or personal mentoring and advising. All of these faculty members have open door policies and encourage meeting regularly with housestaff regarding any issues of concern. The core faculty advisors are:

<table>
<thead>
<tr>
<th>Name</th>
<th>Title</th>
<th>Contact Information</th>
</tr>
</thead>
<tbody>
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</tr>
</tbody>
</table>
2. **Confidential Advising:** This voluntary advising system provides residents with an opportunity to speak to a faculty member regarding any number of issues important to career development, work stresses, health, family and/or personal issues they prefer not to discuss with the program administration. They have no significant role in resident promotion, fellowship selection or hiring and all conversations with them are completely CONFIDENTIAL and SAFE.

<table>
<thead>
<tr>
<th>Name</th>
<th>Mentoring Areas of Interest/ Focus</th>
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</tr>
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<tbody>
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</tr>
</tbody>
</table>

3. **Project or Research-Focused Mentoring:** All faculty within the Department of Medicine have a demonstrated interest in and commitment to mentoring the housestaff. One clear purpose of the bi-annual advising meetings is to help residents identify specific individuals with whom they may share research or other project interests and to facilitate contact with those individuals. For research specific interests, there are also specific “meet and greet events” incorporated into the research curriculum. These provide excellent opportunities to meet with faculty who are interested in having housestaff work with them, and to learn about projects that are currently occurring within the department. Additionally, there is now a database including the faculty members in internal medicine and internal medicine subspecialties and their respective research projects; this is available upon request.

4. **Chief Resident Mentorship Groups:** All the interns will be assigned to one of five chief residents with whom they’ll meet casually throughout the year to discuss personal and career development. These groups will meet on an ad hoc basis, 4-6 times per year, as their members are available.
MENTAL WELLNESS

“The teaching of good doctoring – of patients, of colleagues, of oneself – should begin in the house staff years.”-R. Levin

Your medical training years will be filled with high demand stressors. The importance of simple stress management strategies like making time for some physical exercise, taking advantage of times to catch up on sleep, fueling your body with the most nutritious food you can and scheduling frequent visits with family and friends are vital to your wellness. The following are some of the available resources for you divided up into four categories of wellness: mental, spiritual, physical and nutritional.

**Center for Integrative Medicine - 720-848-1090**

This clinic emphasizes the wellness and healing of the entire person, drawing on both conventional medicine and complementary and alternative medicine (CAM). The services they offer are listed below.

- Acupuncture
- Biofeedback
- Chinese Herbals
- Chiropractic Care
- Educational Workshops and Classes
- Health and Wellness Consultations
- Herbal Supplement/Pharmaceutical Consultations
- Lifestyle/Relaxation Counseling
- Massage Therapy
- Nutrition Counseling
- Psychological Counseling
- Spiritual Counseling
- Tai Chi - Yang Style

**CPHP (Colorado Physician Health Program) - 303-860-0122**

http://www.cphp.org/

The mission of Colorado Physician Health Program is to assist physicians, residents, medical students, physician assistants and physician assistant students who may have health problems which if left untreated, could adversely affect their ability to practice medicine safely.

***Everything you do through CHCP is completely confidential***

CPHP is a non-profit independent organization that is separate from other entities such as the Board of Medical Examiners or the Colorado Medical Society.

Who needs CPHP? Residents with health problems, including medical conditions, psychiatric illnesses, substance abuse, and personal problems.

**Counseling services**

For more information or for an appointment, call the office of Margaret Roath, (303) 315-8159. Biofeedback for stress management is also available. All services are completely confidential.
Students not enrolled in the HSC Student Health Insurance may contact Margaret Roath for assistance in finding a provider.

Student Assistance Office (for referral only) (303) 724-2866
University Mental Health Services (303) 724-1000
Colorado Physician Health Program (303) 860-0122
Housestaff Family Support Group (303) 724-3039
Student Insurance Office (303) 724-7674

Spiritual Wellness

Chaplains are available 24 hours a day, 7 days a week (720) 848-4063. They
...are here to be spiritually and emotionally helpful to you.
...will assist you with prayer, sacraments and other religious rituals.
...will help in the search for meaning in the experience of illness or injury.
...are a resource to reach particular faith group representatives.
...listen, encourage, comfort, care, and support.
...advocate.
The Chapel at Anschutz Inpatient Pavilion, located at the Anschutz Medical Campus is
designed for comfort, peace and healing and is open at all times for meditation, prayer or just
solitude.
http://www.uch.edu/your-visit/patient-services/spiritual-care/index.aspx

Physical Wellness

Anschutz Health and Wellness Center- 303-724-9030
Campus gym and recreational facility with resident rates of approximately $30/month.

CU Sports Medicine - 303-871-2250
http://www.uch.edu/conditions/bones-joints-muscle/sportsmedicine/index.aspx

Campus Recreation at Auraria
Health Sciences Students are welcome to join the Recreation Center at the Auraria Campus
(UCD, MSCD, CCD) for $40/semester.
Spouses/Significant Others/Roommates/etc. are also allowed to join with an HSC Student
sponsor, for $100/semester.
http://www.mscd.edu/~cra/#

Fitzsimons Golf Course
www.golfaurora.com/fitz.htm
(303) 397-1818 (4 days advance notice is required).

Running/Walking
Jogging Trail – a partially paved walking/running pathway follows the inside perimeter of the
entire Fitzsimons site

Intramural Sports - www.uchsc.edu/studentassistance
HSC offers the following for all students on both campuses:
Flag Football...Fall
Volleyball......Fall and Winter
Basketball……Fall and Winter    Softball……Spring

**Nutritional Wellness**

Nutritionist at the Center for Integrative Medicine:  
[http://www.uch.edu/conditions/integrative-medicine/index.aspx](http://www.uch.edu/conditions/integrative-medicine/index.aspx)

[www.eatright.org](http://www.eatright.org) - the ADA web site

[www.quickandhealthy.net](http://www.quickandhealthy.net) - healthy nutrition for busy people

[www.rd411.com](http://www.rd411.com) - wonderful links and tips

**Discount programs**

The **Advantage Program** is sponsored by the HSC Human Resources. The various discounts are listed on this website:  
[http://www.ucdenver.edu/about/departments/HR/EmployeeDiscount/Pages/index.aspx](http://www.ucdenver.edu/about/departments/HR/EmployeeDiscount/Pages/index.aspx)

Discounts are available to Students/Faculty/Staff at HSC.

**Websites**

Finding balance in medical life:  

Healing the Healer:  
[https://www.amsa.org/healingthehealer/index.cfm?secure=yes](https://www.amsa.org/healingthehealer/index.cfm?secure=yes)

Mothers in Medicine:  
Hospital Breakdown

University Hospital/Anschutz Inpatient Pavilion
AIP1 / AIP2

Call Rooms:
- Ward teams: There are 2 call rooms located on the second floor of AIP1 next to the East service elevators and the staff lounge. The door code is 0623.
- MICU call rooms are located in the MICU on the 10th floor of AIP2. The door code is 4444 and badge activated.
- CICU call rooms are located in the CICU on the 3rd floor of AIP2. The door code is 3000 and badge activated.
- Each unit has a staff lounge with lockers to store personal items
- There is a faculty lounge on the 2nd floor of AIP2 across from the East service elevator. The door code is 0623

Computer: The University’s EMR is Epic-based. Login codes are identical and linked to your UCD Webmail login ID and password.

Locations:
- General Medicine Floors:
  - AIP2 9th floor
  - AIP1 6,9,10,12, THRU
- MICU – AIP2 10th floor
- CICU – AIP2 3rd floor
- SICU/NICU – AIP2 2nd floor
- ACE- AIP1 12th floor
- Step-Down – AIP1 10th floor
- BMT/Oncology – AIP1/AIP2 11th floor
- ED: Main Floor of AIP2.
- Dialysis Unit (inpatient): Main floor of AIP1
- Radiology: Basement of AIP1
- Clinical Laboratory services: Leprino 2nd floor

Food:
- Garden View Café (main cafeteria): located in AIP 1, 1st floor, all days 6:30A-1:00A.
- Courtyard Café: located in AOP, 1st floor, M-F 7:00A-2:00P.
- 17th Avenue Restaurants: Jimmy John’s Dazbog, etc. Predominantly open M-F.

Important Numbers and Codes:
- Main Hospital Line: 720-848-0000
- AIP2 10th floor MICU: 85492
- AIP2 3rd floor CICU: 84500
- AIP2 9th floor: 84601
- AIP1 12th floor (ACE): 84751
- AIP1 9th floor (Pulm/Med): 87579
- AIP1 6th floor W (transplant): 84551
- AIP1 6th floor E (Surg): 87680
- AIP1 2nd floor THRU: 84301
- CMR office: x84246
- Lab Main line: 84401
  - Micro: 87084
  - Path: 84421
- Radiology 86343
- Radiology OD: 303-266-6347
- Interpretive Services: 80397
- Pharmacy:81389
- Social Work: 86640
  - Swing (14:30-23:00) 303-266-1385
  - Weekend 303-266-2038
- Pharmacy:81389
- Social Work: 86640
  - Swing (14:30-23:00) 303-266-1385
  - Weekend 303-266-2038
Denver Health Medical Center

**Call Rooms:**
- Ward team call rooms are located on the ground floor of the main hospital across from the ED. The **door code is 5210**. This bank of call rooms has a lounge with a TV, refrigerator, microwave, and couches. It is a good place to relax during some down time.
- MICU call rooms are located in the second floor of the B Tower. Your ID badge is needed to unlock the electronic lock on the door. There is also a very nice lounge with TV and a small kitchen.

**Computer:** Codes and training will be provided. **Lifelink** Clinicals is the main program where CPOE, labs, vitals, medications, I/O’s and ancillary staff reports are located. Within Lifelink you can access **EDM**, a program into which all of the notes (discharge summaries, H&P’s, progress notes, clinic notes, etc.) are scanned. Daily progress notes and H&Ps are handwritten.

**Radiology:** PACS, accessible from your Desktop Portal. Codes will be assigned.

**Locations:**
- General Medicine Floors: 9th, 8th, 6th in the main hospital.
- MICU/CCU: 2nd Floor in the B Tower.
- Step-Down: Several beds on the 3rd floor of the B Tower above the MICU.
- Cafeteria: Basement of main hospital.
- ED: Main Floor of the hospital behind the elevator bank.
- Adult Urgent Care Clinic (AUCC): Main floor of the hospital, next to ED.
- Correctional Care (CCMF): Basement of B Tower.
- SICU: 2nd Floor of the main hospital, above the ED.
- Radiology: Main floor of the hospital by the ED.

**Food:**
- Cafeteria: M-F 6:30A-6:30 P; Holidays and weekends: 11:00A -2:00P.
- SUBWAY: daily 6A-2A.
- Hospital Gift Shop (expensive, but nice snacks and drinks): Main floor of Pavilion C.
- Vending Machines on the 2nd Floor of the main hospital.
- Call rooms on the main floor of the hospital generally have bread, juice, milk, fruit, cereal, and peanut butter/jelly.
- Lunch at Conference every day- 9th floor!

**Important Numbers and Codes:**

<table>
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<tr>
<th>Description</th>
<th>Code</th>
</tr>
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<tbody>
<tr>
<td>Main Number</td>
<td>303-436-6000</td>
</tr>
<tr>
<td>Main Call Room Door Code</td>
<td>5210*</td>
</tr>
<tr>
<td>MICU Door Code</td>
<td>159</td>
</tr>
<tr>
<td>CMR: 303-602-8626</td>
<td></td>
</tr>
<tr>
<td>Lab: 303-602-5200</td>
<td></td>
</tr>
<tr>
<td>Micro: 303-602-5205</td>
<td></td>
</tr>
<tr>
<td>MICU/CCU: 303-602-1460</td>
<td></td>
</tr>
<tr>
<td>Step-Down Unit</td>
<td>303-602-1425</td>
</tr>
<tr>
<td>9th Floor</td>
<td>303-602-8700</td>
</tr>
<tr>
<td>8th Floor</td>
<td>303-602-8800</td>
</tr>
<tr>
<td>6th Floor</td>
<td>303-602-8900</td>
</tr>
<tr>
<td>Inpatient Pharmacy</td>
<td>303-602-9200</td>
</tr>
<tr>
<td>ED</td>
<td>303-436-8100</td>
</tr>
<tr>
<td>Radiology</td>
<td>2-4144, US: 2-4135, CT: 2-4149, MRI: 2-4128</td>
</tr>
</tbody>
</table>
Denver VA Medical Center

Call Rooms: Located on the 5th Floor VA, rooms 5A162, 5B106, 5B110, 5B111.
   Door codes: 5A162 = 143
   5B106, 5B110, 5B111 = 1430

Resident Lounge: 5A161, code 1430

OBMT: This is primarily an outpatient rotation with some overnight call to provide night float to the VA floor teams. There is a separate morning report at 7:30 AM in 1B135 (Code 1-3-5-2) on the first floor every weekday. Interns and residents will do various outpatient clinics during the day as assigned at the beginning of the month.

Computer: Codes are assigned during an orientation on the first day of every month for new interns and new residents. Computer problems: CPRS (2777) or Help Desk/IRMS (4681).

Data: Vitals: Essentris application, I&O’s as well. Labs/Meds can be checked in CPRS. Check CPRS administration record to determine med dosing.

Social Workers:
   Teams A+C  303-687-0314
   Teams B+D  303-613-0789
   General Phone number for SW: 2558

Locations:  
   Basement: Cafeteria/Canteen, prosthetics dept., path to the nursing home
   1st floor: Subspecialty 2 clinic, OBMT conference room, OBMT A,
      Starbucks behind the police station
   2nd floor: Radiology dept.
   3rd floor: Dental dept., lab services
   4th floor: SICU, general med/surg on 4 south
   5th floor: Primary medicine/telemetry floors on 5 north and south, MICU
   6th floor: OBMT B clinic, subspecialty 3 & 4 clinics
   7th floor: Inpatient psych services
   8th floor: OBMT C clinic

Phone Numbers:
Main 303-399-8020                             Lab 2213
Inpatient CMR 2390                             Micro 2638
OBMT CMR 2058                                 Path 2628
MICU 3344                                      National Teleradiology Program 877-780-5559
5N 2827                                        Radiology 2430
5S 2338                                        US 2204
4S 3881                                        CT 2443
SICU 2879                                      MRI 4166
Firm A OBMT 3862/3874/3878                     Nuclear 2454
Firm B OBMT 3082/4062/4112                     IR 2445
Firm C OBMT 3570/5287                          Cath Lab 5865
ED 2353                                        Echo 2378
Inpt pharmacy 303-613-0404   Endoscopy lab 4606

People to know:
PICC pager: 303-413-2286
Silver Bullet pager: 303-402-3717
Transfer Coordinator: 5284
CPRS help pager: 2777 call day or night with any problem with computer system

Cafeteria Hours:  M-F 7:00AM-4:00PM in the basement.

Presbyterian/St. Luke’s

Here’s all the info you need to know at PSL.

Parking: Free parking in the underground lot. Use any of the spots except the handicap and short term parking. Take the elevators on the right up to the main floor of the hospital.

Call Rooms:  4th floor. Take main elevator to 5th floor and then walk down to 4th floor. Code is 2565. Computer, phone in each sleep room (code 2565). Refrigerator, lockers available.
Intern Lounge: Located on the 1st floor. Code is 2565.

Noon Report: 12:00 in Colorado South or alternate published room.

Daily Schedule:
6:00- 7:30AM: Cross cover signout and pre-rounding.
7:30-8:30AM-Morning Handoffs: Occurs in the doctors’ lounge.
8:30-9:30AM: Team Rounding
9:30-10:30AM: SIBR (interdisciplinary rounds)
Pagers should remain on until 6:00pm unless you are post-call.

Computers: The PACS helpline is X7721 (839-7721). PACS is available on most computers. There is a larger screen in the C pod of the ICU. Physicians Help Desk #720-612-6022 (24/7)

Meditech helpline is X7898 for most Meditech issues. For Meditech passwords, Linda Akbarzadeh is the contact at X 6329.

Data: HCarePortal for data collection, Dictations, prior admits.
Orders go on paper as do daily notes.

Locations:
ICU-2nd floor
Teaching Team Medicine 5th and 9th Floors
* B elevators are at the top of the escalators through the main entrance

Cafeteria Hours: 7am-7pm; 7am-2pm Weekends.

Food: $103 provided for the month. Max of $15/day. Just give your food code at the register when you are checking out. PSL employees also get a 25% discount so make sure you get your
PSL badge and wear it. Lunch is provided >90% of the time M-F. Can use Dining money at Jazzman’s Coffee, which has great hours!

**Dictation:** INTERNS DICTATE ALL H&Ps. (Not as bad as it sounds. You will learn very fast.) Make sure you use the complete format including family history and complete review of systems for billing purposes. Upper level residents dictate the discharge summaries.

**Protocols:** PSL has protocols with associated order sets for ventilated patients, heparin drips, sepsis, pneumonia, MI, electrolyte replacement, insulin drips and DKA, neutropenia, stroke, bone marrow transplant, etc. Please use them.

**Telephone numbers:**

- ER 4111
- CMR 6376
- ER door code ___ # (month/year)
- ICU pod D 6471, pod A 6472, pod B 6470
- 5th floor 7590
- 3b onc 4936
- radiology 6522
- rad report line 303-869-1800
- echo report line 800-810-0342, then your ID#, then * for work type, then 2 to listen, then enter account number followed by #
- Long distance: 1+##6277

**Rose Medical Center**

**Format:** Residents, generally R3, will be in the emergency department for 10 hour shifts through the month

**Computer:** All codes for the Meditech system (also used at PSL) are given out on the first day of your rotation. In the emergency department, there is a special computer system that you will be trained to use for chart documentation on the first day of the rotation

**Data:** Vitals/Labs/Meds: computer. Codes for ED computer system are given at the beginning of the rotation.

**Radiology:** Radiology dictation line: 303-320-7421 or ext 7421
- Enter ID#
- Enter Password
- Enter pts birthday (e.g., May 4, 1953. Enter 05041953)
- Obtain codes via ext 2023 if not given to you at orientation.
- Viewing: PACS in MICU. Codes given at beginning of rotation.

**Locations:** Basement – cafeteria, grand rounds room
- 1st floor – ED, radiology dept., “Little Miss Latte” café
2nd floor – step down unit, OR suites, bridge to Pavilion area (long term stay pts and some post op recovery)
3rd floor – surg beds
4th floor – main medical floor, medicine conference room, ICU
5th floor – secondary medical floor
6th floor – special “VIP” patient rooms

Phone Numbers:
Main: 303-320-2121   CMR: 2977
Information systems: 2776   Endoscopy lab: 2066
Lab: 2364   Micro: 2360
Path: 2250   Inpt pharmacy: 2167
Cath Lab: 2238   Intermediate Care Unit: 2273
Radiology: 2290   Rads back desk: 2574
US: 2296   CT: 2815
MRI: 2582   Nuclear med: 2295
IR: 7540   2 PAV: 7200
MICU 2226   4 central: 2475
4 north: 2470   5 central: 2575

ER door code: 6789 (subject to change) ED back line: 5400

Cafeteria Hours:
- Breakfast: 6:30AM-10:30AM, 7 days a week
- Lunch: 11:00AM-4:00PM M-F, 11:00AM-2:00PM Weekends.
- Dinner: 4:30PM-7:00PM M-F

- There are no food credits for residents during the Rose month
- As mentioned above, there is a small coffee/sandwich shop on the first floor called Little Miss Latte is open from 7:00am often until 02:00 am for sandwiches, breakfast foods, coffee, and snacks.
Conducting Yourself On The Wards

The first and most important thing to remember is to be courteous. Everyone has a common goal: taking care of the patient. This can be difficult to remember when tired and under stress, but it’s true. When nurses call you, be polite and respond quickly. Answer all pages in a timely manner. The person who needs something from you now may be the one you need something from later.

Secondly, be professional. Dress professionally and act (even though you may not feel like it the first few days) like a doctor should. Professionalism includes not only your attitude and your dress but also taking care of all your tasks before you leave the hospital every day. This includes getting all notes in the chart early, updating your sign-out, dictating any discharge summaries that are assigned to you and making sure everything your patient needs has been followed up on personally. You are ultimately the one the patient will look to for help, guidance, compassion and answers – make them feel comfortable and confident doing this. Don’t argue and complain, even if you think you are right about a trivial matter – it doesn’t do you or the patients any good to have an angry, resentful staff surrounding you.

As for the people you work with in the hospitals, it is a great idea to introduce yourself to as many of them as possible when starting a new rotation…nurses, ward clerks, techs. Many have been there for years and a good working relationship can be vital to getting things accomplished in a friendly work environment.

These things can be tough to remember in the heat of battle, especially when you don’t see everyone else around you playing by the same rules. But you’re the doctor now, the captain of the team, and it’s your job to rise above all things petty and lead your team to victory.

General Order Writing

- All medications need a dose, route (PO, PR, IV) and a schedule (q6 hours)
- All blood pressure medications should have parameters for when they should be held (i.e. hold labetalol for SBP < 120 or HR < 60)
- Be careful with writing too many PRN medications on your admission orders; while this can be a time saver later, there are many conditions you would like to be called with (agitation, anxiety, hypertension) when they occur
- Verbal orders are often required in emergency situations but should be avoided otherwise as they are fraught with obvious problems; do not expect every nurse to take a verbal order from you in the night if the situation does not absolutely require it. These orders need to be signed within 24 hours.

Admit Orders

(Editor’s Note: While our major hospitals now have electronic order entry and admission ordering templates, this section is still included for (a) historical reference and (b) because
computers can and do sometimes go down. The work of the intern will not go away just because the computer did; you may need to refer to this section!

While a specific patient’s needs may call for special orders to be written on admission, here is a general guideline for what needs to be in every set of admit orders – other things may need to be added on but the following order categories have to be covered for every patient. The mnemonic for this is usually referred to as ACDA VAN DISML.

Note: Many common diagnoses (community acquired pneumonia, myocardial infarction) have order sets preprinted for you. These are not designed to take the thinking and decision making out of your hands but rather to ensure that all important aspects of their care are initiated from the time of admission. It is required of you that you use them if available, but be sure to review them carefully for each individual patient as no order set can address every individual patient’s needs.

A – Admit to: the name of your service (Medicine) followed by attending name, resident name, intern name and pager number
D – Diagnosis
C – Condition: critical, guarded, fair, stable, good are your options
A – Activity: bed rest, ambulate with assistance, ad lib, etc.
V – Vitals: how often you want vitals done (usually write “per routine” here)
A – Allergies
N – Nursing: a good place to list telemetry, Foley placement, NG usage, etc.
D – Diet
I – IV fluids: list the rate, type of fluid and how much. Always write an endpoint for fluids.
S – Studies: list all radiology and non-invasive cardiology stuff here (don’t forget to include a radiology order form for each study ordered)
M – Medication Orders
L – Labs: don’t count on the ER to have done things for you, order everything you think the patient needs, lab will call you if it’s a duplicate

End every set of admit orders with a Call House Officer “Call H.O.” section which gives the nurses parameters that, when met, they are to call you about. 

E.g. Call H.O. if temp > 101.5, SBP > 160 or < 100, DBP > 90 or < 60, HR > 110 or < 60, new chest pain, altered mental status.

Sign, date and time your orders and stamp them.
Sign-out

Sign-out occurs differently across the various hospitals. In some situations a designated time and place are arranged, in others you can sign-out whenever all your work for the day is finished, so check with your chiefs on the first day to find out how it’s done in the hospital you are rotating through.

Sign-out has the potential to be a place where “the ball gets dropped”; don’t let this happen to you. Use the form below which includes a general template as well as an example patient to be sure everything that needs to be included is in there. This will be your last chance to tell the on-call intern everything he or she needs to know about what to do for your patients overnight so make it count.

Several sights have software in place for generating sign-out. Where this is not the case, there are usually standardized templates similar to the one below.

<table>
<thead>
<tr>
<th>Patient Name</th>
<th>MR number</th>
<th>HPI: list the age, sex, admitting diagnosis and major current issues</th>
<th>Medications: list all meds, in order of importance</th>
<th>Studies: list all studies to date and their results</th>
<th>Active Issues: list all problems you are currently addressing as well as active chronic problems</th>
<th>To Do: list anything that needs to be followed up by cross cover or things to watch out for Primary Team: list issues for the primary team that need to be addressed the next day</th>
</tr>
</thead>
</table>

The most important aspect of the sign-out is **assuring that the overnight person knows what to do** in case an issue with your patient occurs overnight. Dr. Eugene Chu, formerly at Denver Health has developed a good sign-out method that should be observed every time you check out for the evening. The mnemonic is **SAIFIR**:

- **S** – Summary (to be given in 1 or 2 sentences)
- **A** – Active issues (things the team is addressing that may come up overnight)
- **I** – If/then (a series of scenarios that may occur and what to do about it)
- **F** – Follow up (all labs, test, etc. specifically that need to be followed up)
- **I** – Interactive questions (from the receiving intern to the handing-off intern)
- **R** – Read back (confirmation by the receiving intern of all he/she has heard)

You will receive further signout training during your first week of internship.
Pagers

How To Page

Seems obvious but just in case:
1) Call the number you want and wait for the beep.
2) Put in the number at which you would like to be called back.
3) Press the * button and put the last 4 digits of your pager in. This way if a call cannot be returned immediately, the person can page you back later when you may not be by the same phone.

Changing Your Message

Find the 4 digit code on the back of your pager. It is the last 4 digits in the 9 digit number.

Once you have the code....

1. Call your pager.
2. Press "0"
3. Enter your 4 digit password. At this point, you can hit "3" to listen to voicemail, "2" to delete voicemail, or "30" to record your message. If you hit 30....
4. Record message after the beep
5. Hit "0" again to save message.

This can be confusing the first couple times but the learning curve is steep. After that, it’s very useful. Not only saves you from getting called at night, but helps direct the nurse/radiologist on whom to call when you are off.

Forward Your Pager

As a rule of thumb, on inpatient months you should be either answering your pager or have it forwarded to someone else. This streamlines nurse – physician communications in off-hours.

To forward you pager:
1. Call your pager
2. Press “0” followed by your 4 digit password
3. Then press “16”
4. Follow the prompts: Press “6” followed by the pager # you are forwarding to. Finish with the # sign. It will then prompt you to confirm the number
5. To cancel pager forwarding, follow steps 1-3 then press “3” to cancel

An Ideal Admission Note (H+P)
This is the cornerstone of the patient’s admission as far as medical decision making goes. It is the single most referred to note in the chart, the one every consultant, nurse, and co-resident will turn to for information on your patient. As an internist, it is also your stage, the place where your thoughts and conclusions shine, so take time with this document.

**HPI:** The first sentence should always be of the “67 y/o woman with known CAD and DM II who presents with crushing, substernal chest pain” variety. Keep it very short and to the point, including only those facts which directly relate to your ultimate diagnosis.

The next paragraph should include a thorough analysis of the patient’s complaint using the **FAR COLDER** system:

- **Frequency**
- **Character**
- **Associated symptoms**
- **Onset**
- **Location**
- **Duration**
- **Exacerbating factors**
- **Relieving factors**

The next paragraph should follow from the conclusions you’ve derived from the first. In it, ask and answer all the pertinent review of systems (+fever, +SOB, no N/V) that relate to the condition you believe the patient has. The HPI, when done well, should generate the right answer to a diagnostic dilemma before any test is ordered more than 90% of the time.

**PMH:** include medical conditions, when they were diagnosed, as well as surgeries and when they occurred

**Allergies:** include the name of the offending agent as well as the specific reaction it causes

**Meds:** many patients do not know the meds they are taking; the best way to get this list correct is to have a family member bring in all their pill bottles. Short of that you may have to use the most recent clinic note available in the chart

**Social History:** include drug, alcohol, tobacco use status as well as family support system and name of PCP.

**Review of Systems:** this section is designed to be the answer to the question, “Is there anything else bothering you today?” It should not include any repetition of things already mentioned in the PMH or the HPI.

**Physical Exam, Vitals and Laboratory Studies**

**Assessment and Plan:** this is where you put your money down on a diagnosis, explain why you think it is correct, other things you considered and excluded and what you plan to do about it.

- write this out in a problem-based format touching on the chief complaint as well as anything else you will be treating including chronic active conditions
- explain your reasoning thoroughly, **DO NOT JUST USE BULLET POINTS**
- address COR status

Sign, date and time your note and stamp it (if paper).

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**An Ideal Progress Note**
While it may seem like a chore to write a note on each patient every day it is both required and important. The progress note should be a concise record of the events of the last 24 hours in that patient’s hospitalization. It should be something that you and your colleagues can refer to in order to know exactly what has happened to the patient, any recent changes or events and what you have done or plan to do. Imagine that you are writing it so that a consultant can be kept up to date or a cross cover intern in the middle of the night can know exactly what is happening with a patient they may not be very familiar with.

Remember that cutting and pasting in our electronic records, while seemingly convenient, encourages you to make errors of omission, increases mistakes, and perpetuates old, possibly inaccurate information. Use the cut and paste very sparingly, if at all.

Date: xx/xx/xxxx
Time: 00:00
Service: primary medicine team

Subjective: a quick outline of important events in the last 24 hours, issues the nursing staff has mentioned that the patient may not have, any updates on their condition that the patient mentions to you (worsening nausea, improved pain)

Vitals: they are vital and must include numbers in a range of lows and highs over the last 24 hours; afebrile is not a vital sign.

I/O’s: include totals in and out as well as specifics (what amount out was urine, how much was stool, how much was vomitus)

Meds: copied into the note and updated daily from the most recent MAR (not from yesterday’s note even though it may be easier to find in the morning)

Physical Exam: you must document at least a 6 system exam every day at a minimum with other areas examined as required based on the patient’s symptoms

Labs: include daily labs, pending or completed culture results including sensitivities, any new radiographic studies, results of any procedures performed since the last note you wrote

Assessment: a quick, one-liner including the patient’s age, sex and primary problem

Plan: List this out by the issues or in the case of an ICU patient, in a system-based format.
  • each problem should be listed separately along with what you plan to do about it that day and what was done about it the day before
  • any problem which has resolved in the last 24 hours should be listed one last time to acknowledge its resolution and then can be dropped from the list
  • if you don’t know the specific plan on an issue, you may write, “to be further discussed with team” – an addendum note can always be completed later
  • be sure to mention prophylaxis daily (nexium, SQ heparin, SCD’s) in every note
  • always end your note with COR status

Sign, date and time your note and stamp it (if paper).
An Ideal Procedure Note

As an example a note for a thoracentesis follows although this note is acceptable for any procedure with small changes.

Operator: Your name, name of supervising resident
Procedure: Left sided, diagnostic and therapeutic thoracentesis
Consent: “Consent was obtained and on the chart at time of procedure.”

“Area was prepped and draped in the usual sterile fashion. Area was numbed with 1% lidocaine using a 25 gauge needle and the procedure was begun. A catheter over a needle was inserted over the rib until a clear, straw color fluid was obtained. The catheter was then advanced as the guide needle was removed until the catheter was advanced fully into the thorax. 1,500 cc of fluid was removed and the catheter was taken out. Estimated blood loss (EBL) = 2 cc. The patient tolerated the procedure without issue. A CXR was ordered to assure no complications.”

Tests sent: cell count, gram stain, pH, AFB

Sign, date and time your note and stamp it (if paper).

An Ideal Death Note

“Called to see patient at 00:00 by the nurse for complaint of (labored breathing, decreased wakefulness, agitation). Found patient to be (unresponsive, Cheyne-Stokes breathing, without signs of life). I examined the patient and noted no respirations, no heart sounds, and no withdrawal to pain. Pupils were fixed and dilated. Telemetry confirmed no signs of cardiac electrical activity; a strip was printed and placed in the chart.

Time of death: 00:00.
Next of kin was notified.”

Sign, date and time your note and stamp it (if paper).
The Bane of Residency – Discharge Summaries

OK, one of the least fun things you will have to do during your residency is a discharge summary...in fact, lots of them. On the other hand, when a patient is admitted to your service, the first thing you will scour the records for and read is the last discharge summary on that person. So it’s important. If you don’t feel like doing one right as the person is being discharged, think about the next doc who will take over their care, how much having that document will impact that patient’s care and reconsider.

When you are dictating a summary (done at some of our hospitals instead of typing), speak reasonably slowly and clearly into the phone. Do not do them from home on a cell phone...the reception is not adequate for dictation and you’ll end up having to do it again. When you are done, write down the dictation number along with the patient’s name and medical record number – keep these for about a month after you’re off service in case a file gets lost (as it occasionally does). Having this record will keep you from having to do the whole thing again...from memory...not fun.

To make things a little easier for you and to help keep anyone from getting behind, here’s a template you can read from into the phone, adding in the details about your specific patient as you go.

(Please see the sections on the specifics of each hospital for codes, instructions, etc.)

Your Name:
Attending Name:
Patient's Name:
Medical Record Number:
Date of Admission:
Date of Discharge:

Admission Diagnosis: list the primary diagnosis first followed by all other conditions which were acute and treated on that admission, leave out chronic conditions
Consultations Called: list all, if none, state “None”
Procedures Performed: same as above

Hospital Course: as descriptive as possible including how the patient presented, what you did for them, how they responded, what you did next
Diagnoses at Discharge: list all conditions treated; in this section you will start with the thing that brought the patient into the hospital, move on to other acute issues and then include all chronic conditions for which a medicine or therapy will be provided on discharge, even if it is the same one they came in on
Medications at Discharge: list all meds, old and new
Diet on Discharge
Activity on Discharge
Disposition: “discharged to home”, “discharged to Autumn Sunset Nursing Home”, etc.
Follow Up: list all follow up appointments with dates and times
The Experienced Approach to: Night Float/Cross Cover Issues

Your most important job at night, other than admitting and caring for your own patients, is helping to care for other’s patients while they are out of the hospital. Whenever you are in doubt about any issue over night, call your resident.

Tachycardia –
1. Get vitals first. If unstable, manage patient via ACLS guidelines. Call a Rapid Response or potentially a code if you need help.
2. Go see the patient. Is this new? Are they symptomatic? Order stat EKG.
3. Compare EKG to old. See cardiology section for algorithm on how to diagnose rhythm and when to use adenosine and vagal maneuvers.

Bradycardia –
1. Get vitals first. Go see the patient.
2. If they are symptomatic (dizzy, chest pain, syncope) or hemodynamically unstable, follow ACLS guidelines. Put patient in Trendenlenberg. Call a Rapid Response or potentially a Code if you need help.
3. If stable, order atropine to the bedside and consider placing the patient on telemetry.
4. Place pacer pads on the patient (can always take them off).
5. If ECG shows either Type II second degree or 3rd degree AV block, consider transcutaneous pacing and possibly a transvenous pacer. Call Cardiology ASAP, and transfer to ICU.
6. If patient is stable and not symptomatic, take a quick look at the chart to try and determine why this might be happening.
7. DDX:
   - Meds: β-blockers, Calcium-channel blockers, digoxin, amiodarone, clonidine.
   - Cardiac: sick sinus syndrome, inferior MI, vasovagal (usually transient), 2nd or 3rd degree AV block, junctional rhythm.
   - Autonomic N.S: neurocardiogenic syncope, carotid-sinus hypersensitivity, cough/micturition/emesis/defecation induced.
   - Other: idiopathic degeneration (aging), infiltrative disease in the conducting system (sarcoid, amyloid), collagen vascular disease, surgical trauma, endocarditis, hypothyroidism, hypothermia, increased intracranial pressure (Cushing’s reflex), hyperkalemia, hypokalemia, OSA, normal variant (marathon runner).
8. Management (if stable):
   - Take a focused H&P and look at the medication administration record.
   - If you think this is medication induced, consider holding a dose of the med if stable. Consider calcium or glucagon administration if you believe it to be secondary to the calcium channel or beta blocker the patient is taking.
   - Put patient on telemetry to track trends.
   - Consider sending electrolyte panel.
**Hypotension**

1. Start with your ABC’s. Is this person symptomatic (dizzy, chest pain, unconscious…)?
2. See the patient immediately. If unstable, call a Rapid Response or potentially a Code.
3. Get the rest of the vitals. Is there evidence of shock (distributive, cardiogenic, hypovolemic)?
4. Make sure the blood pressure is real (if the patient is stable). Measure it yourself with a manual cuff. Make sure all vitals are current.
5. Is this patient’s blood pressure always 80/40 and they feel fine (you will probably see this on the cardiology service)?
6. Calculate the MAP: MAP <60 is associated with decreased perfusion to vital organs.
7. Determine what your IV access is: Make sure there are two large bore IV’s and start Normal Saline wide open while you are thinking, unless the patient is in cardiogenic shock.
8. Consider need for a central line early. If you are going to give pressors, they need a central line stat. Pressors are dangerous to use via peripheral access, and some hospital policies flatly prohibit it.
10. If BP is undetectable, remember that if you can feel a femoral pulse the SBP is >80 and if you can feel a carotid pulse the SBP is greater than 60.
11. Place patient in Trendelenburg.
12. Labs and Studies: CBC, chem.-7, LFT’s, 2 sets of blood cultures, UA with culture, lactate,? BNP, troponin (if you think is cardiac), CXR, EKG, ABG.
13. Broad-spectrum antibiotics if you think this is septic/distributive shock.

**Hypertension**

1. Take a deep breath. Urgent action is only required if you think the patient has hypertensive emergency.
2. Re-check the reading manually. Check the other vital signs. Focused H&P. Quick chart biopsy. What do they take at home?
3. Review the vital sign trends. Is this new? Occasionally you will be called with what seems like a concerning blood pressure, when the patient lives at the BP.
4. Uncontrolled Hypertension (formerly Hypertensive Urgency): SBP > 220 or DBP >125. No end organ damage.
5. Hypertensive Emergency: Hypertension + end organ damage. The BP does not have to meet certain cut-offs. Look for end organ damage:
   - Brain: AMS, lethargy, stroke, seizure
   - Eye: change in vision, papilledema, flame hemorrhages
   - Heart: CP, heart failure, ECG with strain or ischemic changes, SOB
   - Renal: low urine output, edema, elevated Cr, hematuria.
6. DDX:
   - Withdrawal: ETOH, β-blockers, ACE-I, clonidine. Did someone stop their meds or lower their doses when they were admitted?
   - Drugs: cocaine, amphetamines
- Cushing’s reflex?
- RAS, ESRD, renal failure
- Drug Interactions
- Pregnancy: eclampsia, pre-eclampsia
- Aortic dissection, coarctation of the aorta
- Endocrine: pheochromocytoma, Cushing’s syndrome, thyrotoxicosis.

7. Treatment:
- Hypertensive Emergency: ICU admission. Lower BP by no more than 25% in the first 6 hours and then to a goal of 105 DBP over 6-12 hours. Patient will likely need nitroprusside (best choice for most) or labetalol gtt and an arterial line. Consider nitroglycerine gtt if patient has cardiac ischemia. Also consider giving an ASA if no contraindications.
- Uncontrolled Hypertension (formerly Hypertensive Urgency): this must be evaluated in the context of the patient with attention paid to the time course of the hypertension. If felt to be a chronic problem, consider either not treating or treatment with medications which would be appropriate for long-term use as an outpatient (thiazide diuretics, ACEI, calcium channel blockers). If the time course is felt to be more acute, consider use of hydralazine: 10 mg IV q 6 hours. PO hydralazine can be used as well, but you will need to use higher doses such as 25 mg PO. In the

Fever

1. Temp > 38.3°C and greater than 38°C in neutropenic/transplant/dialysis patients.

2. Broad DDX:
   - Infection: PNA, UTI, line infection, cellulitis, VAP, osteomyelitis, endocarditis, myocarditis, pericarditis, encephalitis, meningitis, abscesses (esp. if post-op), sinusitis, prostatitis, C. difficile diarrhea, fungal (esp. if immunosuppressed with lines), decubitus ulcers.
   - Inflammation: collagen vascular diseases, neoplastic, mucositis.
   - Drug Fever: beta lactams, amphotericin, chemotherapy, neuroleptic malignant syndrome, malignant hyperthermia of anesthesia… to name a few.
   - Clot: PE, DVT, thrombophlebitis.
   - Neurologic: spinal cord injury, hypothalamic injury, intracranial hemorrhage, seizures, subdural hematoma.
   - Endocrine: adrenal insufficiency, thyrotoxicosis.
   - Miscellaneous: Alcohol/drug withdrawal, aspiration, transfusion reaction, hemotoma, pancreatitis, MI, acalculous cholecystitis, ischemic bowel, fat emboli, transplant rejection, gout/pseudogout, GI bleed.
   - Atelectasis does not cause a high fever; this is a myth, so keep looking.

3. If the patient develops fever after 72 hours in the hospital, then likely nosocomial infection or drug-induced fever:
   - Common Infections: UTI (esp. with foley catheters), central venous or peripheral catheter infections, pneumonia, wound infection, C. difficile colitis.
   - Less Common Infections: decubitus ulcers, sinusitis, acalculous cholecystitis.
4. **Work-Up:**
- Determine whether patient is stable or unstable → Ask nurse for V.S. over the phone. Assess patient immediately at the bedside if hypotensive, tachycardic, tachypneic. Document your findings in the chart with a short note. If unstable, call your resident, as patient will likely need to be transferred to the ICU. If hypotensive, start normal saline IV bolus and put patient in Trendelenburg position.
- Perform a focused history and physical.
- **Order two sets of peripheral blood cultures.** If patient has a line, and peripheral blood cultures are difficult to obtain, it is O.K. to get one of the two sets through the line. If blood cultures have been done within 24 hours and patient is stable, then only re-culture if unstable.
- Determine what other tests should be ordered according to clinical findings. If an infectious etiology seems likely, urine and lung sources can be evaluated with a UA with culture and CXR.
- Keep your differential broad. Consider non-infectious etiologies including drug fever, PE, transfusion reactions… (See above).

5. **Treatment:**
- If patient is unstable, start broad-spectrum antibiotics or start directed antibiotic therapies if you have identified the source of the fever (i.e. PNA, UTI).
- See Oncology section for neutropenic fever.
- Follow algorithms for bone marrow/stem cell transplant patients.
- If patient is stable and you do not have a source, you can withhold antibiotics until the source is determined.

6. **Specific Therapies:**
- If known neutropenic fever, – see oncology section for recommended regimens.
- If suspected pulmonary source – is this community acquired, hospital acquired, health care associated, or ventilator associated? Next, consider patient’s risk factors for multi-drug resistant organisms. These pieces of information will help you determine an appropriate regimen.
- If suspected intra-abdominal source, need to cover for polymicrobial infection (gram negatives, enterococcus and anaerobes). Consider carbapenems or beta lactam/beta lactamase inhibitor like piperacillin-tazobactam.
- If urinary source, will need coverage for gram negatives.
- If suspected soft tissue infection, add vancomycin for coverage of MRSA (good first line therapy in cellulitis).
- **Antipyretics:** acetaminophen 500 mg to one gram PO (write not to exceed 3 grams per day and if patient has liver disease not to exceed 2 grams per day). Be careful with ibuprofen especially if patient has ARF, ESRD, surgery is planned, GI bleed, anticoagulated.

Low Urine Output (UOP) – defined as less than 0.5 cc/kg/hr

1. **Fluids, not lasix.** With rare exceptions (extreme volume overload such as in end-stage CHF patients come to mind), the answer to low urine output is almost never to give lasix.

2. Consider whether or not they have a Foley in place. In patients without a Foley, not urinating for 4-6 hours may be normal (you don’t give hourly urine output normally, do you?). In this case, if you or the nurse are still concerned about their urine output, order a quick bladder scan or PVR to see how much is in there. If nothing, see #3. If a lot, see #4. If 100-200 cc’s you can safely continue to monitor closely for another couple of hours.

3. **Determine the cause.** Causes to consider:
   - Poor flow to the kidneys because of a bad or worsening pump (the heart)
   - Poor flow because of hypovolemia
   - Poor flow because of distribution (sepsis/shock)
   - Renal failure (a variety of causes, either new or worsening)

4. **Obstruction not allowing any urine to get out.** Place Foley or straight cath and continue to assess.

Hypoxia

1. See the patient immediately! Start with ABC’s. Does this patient need to be intubated right now? What are the vital signs? How much oxygen are they on?

2. Look at the trend. Is this immediate or has their oxygen requirement steadily been increasing?

3. Call a Code if patient has stopped breathing, is unstable, is blue…after making sure of their cor status.

4. Make sure it is real. Switch extremities, switch probes.

5. Diagnostic tests: CXR, ABG stat (goal PaO2 is greater than 60). Then order: EKG, CBC, chem.-7, troponins. Perform a focused history and physical- is this patient in heart failure, no breath sounds on one side indicating a PTX …

6. **DDX:**
   - Pulmonary: pneumonia, PTX, PE, aspiration, bronchospasm (asthma, COPD, drug reaction), upper airway obstruction, ARDS, flash pulmonary edema.
   - Cardiac: MI/ischemia, CHF, arrhythmia, tamponade.
   - Metabolic: sepsis, acidosis
   - Hematologic: profound anemia, methemoglobinemia (low sat, normal PaO2)
   - CNS: stroke, medications (benzos, opiates), AMS.

7. **Treatment**
   - Give them as much oxygen as you can.
   - Consider Non-invasive positive pressure ventilation (BiPAP or CPAP) if they do not have altered mental status and they are relatively stable (especially for hypercapnea).
   - Intubate those who cannot protect their airway or who do not turn around with oxygen- remember to check Cor Status.
   - RT can be our best friend in these situations.
   - Treat the underlying cause (i.e. furosemide and CPAP if heart failure, abx for PNA).
   - Give albuterol nebs to those who are wheezing.
Electrolyte Replacement

Rough goals for lytes replacement (especially in cardiac patients or anyone with a history of arrhythmia) are K = 4.0, Mg = 2.0, Phos = 3.0.

*Be very, very careful replacing lytes in a renal patient with poor ability to process them.*

**Potassium:** Can be given PO (very effective, a little hard on the stomach at doses above 40 meq) or IV (should be given with lidocaine as it burns going in).

Expect a result as follows:
- K = 3.5 – 4.0……..to raise the level by 0.1 will take 10 meq
- K = 3.0 – 3.4……..to raise the level by 0.1 will take 20 meq
- K = 2.5 – 3.0……..to raise the level by 0.1 will take 30 meq
- K = 2.0 – 2.4……..to raise the level by 0.1 will take 40 meq

Never give more than 10 meq IV per hour as it can cause arrhythmias/death.

Give via PO route whenever possible, by both routes if the K is less than 2.5 and there is a high risk for the hypokalemia causing an arrhythmia or if multiple PVC’s are noted on telemetry.

If hypomagnesemia is present as well, this must also be corrected. Low magnesium levels can cause patients to be refractory to potassium repletion.

**Magnesium:** Can be given PO (also a little hard on the stomach, may cause diarrhea)

500-1000 mg per dose, up to TID or IV 1-4 g per dose. Expect approximately a 0.1 increase in Mg levels for every gram given IV. The max you can give in a single IV dose is 4g if the Mg is < 1.5.

**Phosphorus:** Can be given via PO or IV routes, equally effective. A great way to raise the phos in a patient who can eat is to add 1-2 containers of skim milk to each meal. PO dosing is 500 mg BID – TID, IV dose is 10 mmol IV x 1, repeat as needed.

Phosphorus IV comes in two flavors: NaPhos and KPhos. Both contain fairly negligible (7 meq) of their respective carriers but avoid KPhos if the K is high and NaPhos if the Na is high.

Never attempt to use KPhos to replete the K.

**Calcium:** Remember that calcium needs to be corrected for the patient’s albumin based on the following formula prior to replacing it:

\[
Ca = serum \ Ca + 0.8 \ mg/dL \ for \ every \ 1 \ g/dL \ of \ albumin < 4.0
\]

If you are still unsure of the true Ca level or want to check more accurately, you can order an ionized calcium prior to replacement.

Replacement can be accomplished via PO or IV routes. Either 1 gram of CaCl (13.6 mEq of elemental Ca) or 1 gram Ca gluconate (approx. 4.5 mEq of elemental Ca) are acceptable IV preparations, noting the difference in strengths.
Ca gluconate 500-1000 mg PO TID is a standard PO dosing (as is the use of TUMS OTC 500 mg PO TID which will provide 30mEq of elemental Ca).

**Heparin Drip Adjustment**

Heparin drips are being used less and less frequently as low molecular weight heparin becomes more acceptable for use in PE, DVT and other common conditions but you will still need to know what to do with one for your post-cardiac cath patients, those with unstable angina and some others.

Adjusting the drip is a little bit of science and a lot of art, with the final answer being to check q6h PTT’s and monitor carefully for bleeding. Most hospitals have protocols that allow the nurse to adjust appropriately.

**Loading dose:** 60 - 80 units/kg IV x 1
- 60 units/kg for unstable angina and MI, 80 units/kg for treating a known or suspected DVT or PE

**Starting dose:** 12-18 units/kg/hr continuous infusion
- again, the lower dose for UA and MI, the higher for known clot

**Adjustment Table**

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<tr>
<th>apt</th>
<th>Rebolus</th>
<th>Stop Time</th>
<th>Drip rate change</th>
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</thead>
<tbody>
<tr>
<td>&lt; 35</td>
<td>80 units/kg</td>
<td>-</td>
<td>↑ 4 units/kg/hr</td>
</tr>
<tr>
<td>35 - 45</td>
<td>40 units/kg</td>
<td>-</td>
<td>↑ 2 units/kg/hr</td>
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<tr>
<td>46 – 59</td>
<td>-</td>
<td>-</td>
<td>↑ 2 units/kg/hr</td>
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<tr>
<td>60 – 80</td>
<td>-</td>
<td>-</td>
<td>-GOAL RANGE</td>
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<tr>
<td>81 – 100</td>
<td>-</td>
<td>30 minutes</td>
<td>↓ 1 unit/kg/hr</td>
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<tr>
<td>101-120</td>
<td>-</td>
<td>60 minutes</td>
<td>↓ 2 units/kg/hr</td>
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<tr>
<td>&gt; 120</td>
<td>-</td>
<td>90 minutes</td>
<td>↓ 3 units/kg/hr</td>
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**Hint:** If you are called with a very high PTT (>200) on a recently started drip or on a patient who has had a stable drip rate for days, the person drawing the level may have pulled the blood out of a heparinized line. It is reasonable and OK to ask them to redraw it via a peripheral stick before making any changes unless the patient is clinically over-heparinized (i.e. bleeding).

**Nausea/Vomiting**

1. First of all figure out what they are vomiting and why. Could they be obstructed- if so, get a 3-Way of the abdomen. Examine them and make sure something more serious isn’t going on. If they are not vomiting blood, consider the following recommended antiemetics. Remember many of these drugs can have significant extra-pyramidal effects so be careful.

- Zofran 4 mg IV q 8 hours
- Phenergan 12.5 mg to 25 mg IV or PO q 6 hours prn. Can also be given as a suppository.
- Compazine 5 mg to 10 mg PO q 6 t 8 hours. 25 mg suppositories are also available. Can be given IV/IM at doses of 5 to 10 mg.
- Reglan (a good motility agent as well). Start 10 mg Q 6 hours. Try to avoid this drug in elderly patients.
- Low dose benzos such as ativan can be used, but again do not give to elderly.

Chest Pain

For more specific info see the cardiology section but this should get you started.

1. Order an EKG, CXR and a new set of vitals while you are on your way to see the patient.
2. Obviously your first concern is the heart, lungs and vessels as they are the things that could lead to acute collapse of the patient if missed or not addressed in a timely fashion. Consider this as you review the nature of the pain and the patient’s history.
3. Then, think about what’s in the chest that could be hurting, layer by layer:
   - Derm – herpes zoster (pain will start approx. 48-72 hours before the rash appears).
   - Musculoskeletal – fractured rib (coughing, trauma), pulled muscle, costochondritis.
   - Nerves – nerve root pain from a compressive spinal lesion (cancer).
   - Heart – angina, MI, arrhythmia causing poor coronary perfusion, pericarditis.
   - Vessels – aortic dissection, pulmonary embolus.
   - Lung – pneumothorax, pneumonia, pleurisy from an effusion.
4. Other potential etiologies of chest pain:
   - GI – esophagitis (pill or other), GERD, esophageal spasm, peptic ulcer.
   - Hepatology – hepatitis with capsular strain, Fitz-Hugh-Curtis syndrome.
   - Other – pancreatitis, splenic rupture or infarct, gallstones.

Then go back and make sure it’s not the heart again.

Rising Creatinine/Acute Renal Failure – see the renal section

Falls

1. See the patient and examine them with a full neurologic exam.
2. If at all in doubt about head trauma order a **non-contrast head CT to be done STAT**.
3. Order any other radiology as directed by your exam (hip films, wrist films) and make sure it gets done that night, not in the morning, unless you are confident in splinting the joint appropriately until further evaluation.
4. Attempt to figure out if there is a correctable or treatable reason as to why they fell. Look at the medicine list.
Insomnia

1. First and foremost, find out why the patient can’t sleep; there’s usually a good answer that might not require medication.
2. Choose a medicine designed for sleep; do not rely on the side effects of medications for other conditions (i.e. Seroquel, Benadryl, amitriptyline, Ativan)
   
   Good Options:
   - Trazodone 25 mg po qhs PRN
   - Ambien 5 mg po qhs PRN (use with caution in elderly)

   *Hint: Always remember to give people over age 65 half the dose you would normally use.
3. Assure good day/night cycle in their room by asking the nurses to turn the TV off after midnight, turn the lights out after 10 PM, turn the lights back on during the day and have them open their blinds in the AM as well. This may help avert the same call the next night.

Hypoglycemia/Hyperglycemia – see the endocrinology section

Altered Mental Status

Needless to say, the causes are many and each needs to be carefully considered. The mnemonic is not very helpful (probably easier to just flip this page open until these things stick in your head as opposed to trying to memorize them cold). In the hospitalized patient, the cause is usually multifactorial.

Mnemonic: SHE STOPs for TIPS on AEIOU

<table>
<thead>
<tr>
<th>SHE</th>
<th>STOPs</th>
<th>TIPS</th>
<th>AEIOU</th>
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<tbody>
<tr>
<td>Sepsis</td>
<td>Hepatic Enceph.</td>
<td>Stroke</td>
<td>Trauma</td>
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<td>Electrolytes</td>
<td>Temp</td>
<td>Oxygen</td>
<td>Infection</td>
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<td>Psych</td>
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<td>Porphryia</td>
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<td></td>
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<td>Inflammation</td>
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<td></td>
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<td></td>
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<td>Uremia</td>
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</tbody>
</table>

*Hint: Finding one of these reasons in the chart does not rule out the others...make sure you have considered all the possibilities as there are many and some patients will have more than one.
Dyspnea

Remember first of all that dyspnea is a symptom, not a diagnosis or disease. Your job in the patient complaining of dyspnea, or shortness of breath, is to figure out why. A good approach is to think of dyspnea in four broad categories, each of which can be investigated quickly with just four tests.

**Pulmonary Causes:** the largest group, a good place to start
- pneumonia
- pneumothorax
- pulmonary embolus
- COPD
- aspiration
- mechanical obstruction
- ARDS

**Cardiac Causes:** the next largest and most common
- CHF
- myocardial infarction
- tamponade
- arrhythmia

**Hematologic Causes:**
- hemoglobinopathies
- cyanide toxicity
- very severe anemia

**Acid/Base Disturbances:**
- metabolic acidosis
- respiratory alkalosis

**Psychiatric Causes:**
- panic attack

Using this list from the top down will not give you every answer, but it will allow you to quickly reason through 18 of the more common causes.

As for figuring out which on it is, four tests will help rule in or out each of the above listed disorders (with the exception of psych causes, which are always a diagnosis of exclusion).

- CBC
- CXR
- ABG
- EKG

Order these over the phone or as soon as you get to the patient in all calls for new-onset dyspnea; while you are examining them the results will be cooking and you’ll be well on your way to solving the problem.

*Hint: Do not disregard dyspnea in the face of a normal O2 sat; it takes a lot to change the saturation and can fool you into thinking nothing is going on.*

Adapted from J. Wiese, Tulane Internal Medicine Guide, 2003

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**Combative Patient**

1. Go see the patient and figure out what is going on. Is this a psychiatric issue or is it medical?
2. If you feel like you and the staff are in danger of physical injury, call security right away. Do not try and restrain the patient yourself. It is not your job to physically restrain anyone; don’t attempt to do that. If the patient is trying to leave the hospital and you believe them to be a danger to themselves AND you believe this to be primarily psychiatric, place them on a Mental Health Hold. Remember that a MHH is not necessary to prevent someone from leaving the hospital AMA when the cause is
medical; in this case, you must simply document the lack of capacity from a medical cause (delirium, intoxication, etc).

3. If you feel this is not psych, then do the AMS work-up recommended in the preceding section.

4. Try and re-orient the patient before using chemical or physical restraints.

5. Chemical restraints:
   - Haldol should be your first choice. Start at low doses especially in the elderly, such as 0.5-1 mg IV X 1. Can be given IV/IM/PO. Do not give doses larger than 5 mg at one time.
   - If for some reason haldol cannot be used, try zyprexa. There is a fast acting form “Zydis” that can be given in a pinch. However, remember that atypical antipsychotics have been associated with increased mortality rates in the elderly.
   - Risperidone is another option, but this also carries to increased mortality rate discussed above.

6. Physical restraints: if at all possible, try not to restrain a patient, but if needed try to use the least restrictive. Try a Posey vest or a distraction apron first; a distraction apron has a lot of buttons, zippers, and things for someone to play with and works well for the elderly. Also consider using mittens if the concern is that the patient will pull something out. The nurses are really the best at determining what will help a patient. Please sign your restraint orders in a timely manner.

Deceased Patient

You will be called to pronounce a patient who has died at some point. The nurses know that we don’t do this a great deal and have streamlined the process for you. Interns may pronounce death without their residents present but if you have concerns or it is your first time, call your resident to assist.

1. Find the charge nurse, let them know you will be the doctor pronouncing the patient and will need all the necessary paperwork.

2. Examine the patient for signs of life and note the time you finish your exam – this is the time of death.
   - if the family is in the room and upset, speak with them before approaching their recently-deceased relative; let them know what you will need to do and why. This can be difficult, particularly when you are cross covering and have never met the family. The vast majority of the time, they will be fine with you examining their loved one once you explain your role. However, if the family is very upset with your presence, leave the room and get a nurse to come back in with you to help console the family while you make the pronouncement
   - check heart and breath sounds with your stethoscope; while you are doing this, out of sight of the family, firmly squeeze the patient’s nail bed between your fingers to assess withdrawal from pain; then quickly check pupillary light reflexes in both eyes
   - you do not say the time of death out loud like on TV, just note it in your head

3. Fill out the Death Packet provided for you by the charge nurse. It is very self-explanatory but the charge can help if you have questions. However, do not sign the form, only an attending can do that in the morning.

4. Call the coroner using the number provided in the death packet. You’ll want to have the patient’s chart with the face sheet for this as there are a lot of questions to be answered.
5. You are in charge of asking the family for permission to do an autopsy and should make an effort to obtain one in all cases.
6. Write the death note as outlined in the “Ideal Death Note” section of this guide.
7. If appropriate and you feel it is needed, go back and offer your and the hospital staff’s support to the family, i.e. see if they need tissues, coffee, have any questions.
8. Ensure that next of kin is either present at the bedside or has already been notified. If there is a question, call them yourself.

You do not get involved in the procurement of organs, it is a conflict of interest.

The Experienced Approach to: Procedures

Central Lines

Central lines are important ways to get access in a patient requiring pressors for any reason, someone in whom immediate, multiple points of access are needed and in those for whom no other sites of access are available in an emergency. They should always be performed in a very sterile fashion (except in Code situations when time is of the essence) and an ultrasound should be used whenever possible.

They are very useful when done correctly although inserting one carries with it a certain amount of risk. The femoral approach is best reserved for Code situations when interosseous access is unavailable for some reason, and when other access sites cannot be used. It is the least sterile of the options in the long term. The other two sites, the internal jugular and the subclavian, can be used interchangeably based on operator experience and the patient. For example, an IJ is safest in a patient on positive pressure ventilation and in very thin patients. An IJ has very few drawbacks, although a very agitated patient may thrash their head around a lot and a person with a large neck can be technically challenging.

While the sites may vary, the technique is the same. Below is a description of the Seldinger technique for reference and a few helpful hints to be reviewed prior to the procedure.

1) Obtain consent and place it on the chart.
2) Use the ultrasound to locate the vein if using the IJ. Compress the vasculature to identify artery and vein.
3) Completely dress yourself for the procedure; this means sterile gown, gloves, protective eyewear, and cap. This is required for all central lines.
4) Prep and drape the area in question in the usual sterile fashion.
5) Numb the skin over the area as well as your intended path to the vein with the 1% lidocaine included in all the kits. For IJ approaches you can usually find the vein with this needle as well.
6) Flush the catheter in all its ports with sterile saline so no air is left in it.
7) Cover the Ultrasound probe with a sterile sleeve.
8) Now use the insertion needle, the largest one in the kit, to follow the path you just numbed in an effort to find the vein. Hold the Ultrasound in your other hand and keep an eye on both your needle and the vein on the ultrasound monitor. Always draw back as you are advancing so you will know when you are in the vein (the blood will fill the syringe quickly).
9) Once you are in and getting good flow back, remove the syringe from the needle, leaving the needle exactly where it is. Often blood will flow briskly from the back of the needle but not in every case, depending on the pressure and volume status of your patient. If blood is squirting from the needle in a pulsatile fashion you likely have canulated the
artery – if this happens, remove the needle and hold pressure for 10 minutes on the site. Never dilate and place the line if you are unsure of where you are.

10) Insert the wire into the back of the needle and into the vein until there are 5-10 cm of wire sticking out the back. NEVER LET GO OF THE WIRE. Never force the wire if it doesn’t go in easily. If you can’t get it in easily attempt to tilt the needle more parallel to the patient to facilitate the direction it takes inside the vein. If this doesn’t work, you may have to start over with another stick in a slightly different location as there may be scarring or other obstruction within the vein itself at that site.

11) Remove the needle and make a small 0.5 cm skin nick where the wire is.

12) Slide the dilator over the wire and at least half way into the patient. A good deal of blood may now flow out as you have made a large hole in a large vein. Have some gauze at the ready on your sterile site. NEVER LET GO OF THE WIRE. Remove the dilator.

13) Slide your catheter over the wire. When you get the tip close to the skin you will have to push the wire back into it so that you don’t let go. The wire will come out the back of the shortest port. When it does, grab the wire and push the catheter in the rest of the way and remove the wire. For femoral lines, the catheter should go all the way in. For subclavian and IJ line the catheter should go to about 20 cm if on the left side and 16 cm if on the right side but you will have to judge by the size of your patient.

14) Now flush all ports with sterile saline to assure that they work easily. Place caps on their ends. Suture the line into place, remove your sterile drape and ask the nurse to secure the site with Tegaderm to assure that the line isn’t pulled out.

15) For all subclavian and IJ lines, FAILED OR SUCCESSFUL, order a CXR to assure no intrathoracic complications.

16) Write a procedure note for all lines, failed or successful.

Thoracentesis

Indications: therapeutic relief of dyspnea, diagnosis of unknown effusion

Complications: pain, bleeding, infection, hemothorax, pneumothorax, costal nerve damage, damage to liver or spleen

Equipment needed: thoracentesis kit, extra lidocaine, sterile gown, sterile gloves, glasses or mask, sterile cap

1) Obtain consent and place it in the chart.

2) Have patient sit with their legs over the side of the bed and have them lean over with their head resting on their arms on a bed side table – this is the ideal position and the one they will be in during the procedure.

3) Remember: as they can no longer see what you are doing and you plan to stick a large needle into them, explain what you are doing before it happens.

4) Tap out the effusions using your hands and determine a safe spot to insert your needle; a good choice is one-two ribs below the top of the effusion so long as this is above the 9th rib. Also, you can and should use ultrasound to visualize the effusion.

5) Prep and dress yourself in a sterile fashion.

6) Prep and drape the area in a sterile fashion as well.

7) Open the kit and anaesthetize the area you plan to invade– do this by making a small skin wheal of lidocaine with the little needle, then anaesthetizing deeper with the larger needle until you obtain the pleural fluid (unless the patient is very obese you should hit it with your lidocaine needle).

8) Make a small nick in the skin with the scalpel provided in the kit.
9) Connect the 60 cc syringe to the drainage needle/catheter set up and advance along the now-numb path you have made; always aim for the bone itself when entering with the large needle – when you hit it, move ever so slightly upward until you pass just over the bone, as the neurovascular bundle lies just below the rib.

10) When fluid is noted, gently slide the catheter forward over the needle while pulling the needle back ever so slowly – this will allow for passage of the soft, plastic catheter into the pleural space without allowing much of the needle in to cause complications.

11) Remove fluid as needed, first filling the syringe for studies, then connecting the large volume removal bag; take no more than 1.5L at a single time to avoid the risk of re-expansion pulmonary edema.

12) When you remove the catheter, have the patient hum the entire time you do so – this will avoid having them take a deep breath as the catheter is coming out and possibly sucking a large amount of air into the pleural space, causing a pneumothorax. A simple Band-Aid closes the hole you have made.

13) Write a procedure note.

Studies to be sent:
   All fluid: cell count and differential, glucose, albumin, total protein, gram stain, culture, LDH, pH. Cytology, AFB, ADA, amylase, cholesterol and triglycerides may be indicated if you are looking for special causes of effusion.

Paracentesis
Indications: diagnose cause of fluid, look for bacterial peritonitis, therapeutic relief
Complications: pain, bleeding, infection, bowel perforation, liver/spleen laceration, persistent leakage of fluid
Equipment needed: paracentesis kit, extra lidocaine, sterile gown, sterile gloves, glasses or mask, sterile cap, Vacutainers for fluid collection (1L per container)
   - you do not need a whole kit for a diagnostic-only tap, an 18 gauge needle and a 60 cc syringe will work fine

1) Obtain consent and place it in the chart.
2) Have the patient supine in the bed in a comfortable position.
3) There is no reason to perform a blind tap. Always use ultrasound to identify an optimal pocket. The standard location is in the RLQ, just superior to McBurney's point – if the liver is large, you can use the same spot on the other side which is just as safe. Before inserting the needle, assure that the area does not have any large, superficial veins running through it – with these patient's portal HTN and poor coagulation, these will bleed a lot if stuck so avoid them.
4) Prep and dress yourself in a sterile fashion.
5) Prep and drape the area in a sterile fashion as well.
6) Open the kit and anaesthetize the area you plan to invade with your needle – do this by making a small skin wheal of lidocaine with the little needle, then anaesthetizing deeper with the larger needle until you obtain ascetic fluid – note: the peritoneum is highly innervated, use a good deal of lido here.
7) Make a small nick in the skin with the scalpel provided in the kit.
8) Connect the 60 cc syringe to the drainage needle/catheter set up and advance along the now-numb path you have made; always hold the needle itself with your left hand, about 1
inch above the skin – this way when you pop through the peritoneum the needle doesn’t
drive deeply in.
9) When fluid is noted, gently slide the catheter forward over the needle while pulling the
needle back ever so slowly – this will allow for passage of the soft, plastic catheter into the
peritoneal space without allowing much of the needle in to cause complications.
10) Remove fluid as needed, first filling the syringe for studies, then connecting the
Vaccutainer to remove the large volume; for patient with cirrhosis or portal hypertension,
ever take more than 6L without administering 50g-100g of Albumin 25% soln. during the
procedure.
11) Close the wound with a Band-Aid or cover with an ostomy bag if continued leakage is a
problem.
12) Write a procedure note.

Studies to be sent:
   All Fluid:  cell count and diff, total protein, albumin, glucose, LDH, gram stain
   and culture.  AFB, cytology, fungal cultures may be needed depending on what you're looking
   for.

Lumbar Puncture
Indications:  infection, subarachnoid hemorrhage, assessing the etiology of a headache or fever
of unclear origin
Complications:  pain, bleeding, infection, post-procedure headache, brain herniation, persistent
leakage of fluid
Contraindications: Alteration in mental status or focal neuro deficits should get a non-con CT
of the head first to assure no elevated intracranial pressure; if this is the case, start the
antibiotics before the CT and do the procedure immediately afterward – never wait to start
antibiotics if you suspect bacterial meningitis for any reason!!
Equipment needed:  LP kit, extra lidocaine, sterile gown, sterile gloves, glasses or mask,
sterile cap

1) Obtain consent and place it in the chart.
2) Have the patient sit on the edge of the bed with upper body bent over a bedside table so the
spine is readily identifiable – always mark your intended point of entry in this position
before laying them on their side. This spot is midline in the spinal column, at the level of
the pelvic brim – both easily identifiable in the seated position. Mark it with the tip of a
retractable pen with the pen part in so a small circle is pressed into the skin (this won’t
wash away like ink when you clean the area).
3) Lay the patient on his side.
4) Prep and dress yourself in a sterile fashion.
5) Prep and drape the area in a sterile fashion as well.
6) Open the kit and anaesthetize the area you plan to invade with your needle – do this by
making a small skin wheal of lidocaine with the little needle, then anaesthetizing deeper
with the larger needle. You will need most or all of the lidocaine as the bony structures as
well as the ligamentum are all sensitive,
7) The key to this procedure is positioning: Get someone to help you keep the patient in as-
close to a fetal position as you can get them with their head tucked toward their knees.
Keep your needle parallel to the floor the whole time and aimed slightly rostrally (toward
the head).
8) Slowly advance the spinal needle along your anesthetized track, always parallel to the floor; you will feel several layers of 'popping' which are the ligaments, as you enter. The classic 'pop' of the dura mater as you enter the fluid itself is truly louder/more pronounced than the others and feels distinctly different. It is a feeling. Don't be surprised if you don't get it the first pass, the canal is often deeper than you think. If you miss, remove the needle almost all the way out but keep it under the skin and redirect inferiorly to your last pass – a common mistake is going too high.

9) When you feel the pop, remove the stylette from the needle and wait 2-3 seconds for fluid return. If clear fluid comes out, catch it. If an opening pressure is important, now is the time to connect the pressure column to the back of the needle and get a measurement before any fluid is removed. If you're not sure about needing an opening pressure, get one – you'd hate to have to do all this again. They have to be in the lateral decubitus position to check an opening pressure.

10) You'll need about 2cc per tube with 4-5 cc recommended in the 3rd one to be saved for later studies if needed.

11) Remove the needle when done and place a small Band-Aid over your hole. Instruct the patient not to sit up at all for at least 1-2 hours and not to walk around for 3-4 hours to decrease the chance of a headache.

12) Write a procedure note.

**Studies to be sent:**

- Tube #1: cell count and diff; Tube #2: glucose, protein, VDRL; Tube #3: culture and gram stain (any special studies should be added to this one including cytology, TB, viral panels, crypto latex agglutinin); Tube #4: cell count and diff (again)
THE EXPERIENCED APPROACH TO:

SUBSPECIALTY ISSUES

CARDIOLOGY

Basic Tips/Random Stuff

1. Keep K+ above 4 and Magnesium above 2 (don’t replace with ESRD or ARF unless cleared by UPPER LEVEL).
2. Always compare ECGs to old.
3. If on cardiology, make copies of all ECGs obtained and an old one and bring to rounds.
4. PO to IV furosemide is 2:1 (i.e. 40 mg of PO furosemide is 20 mg IV)
5. PO hydralazine to IV hydralazine is 4:1.
6. Toprol XL to metoprolol is 1.4 mg to 1 mg.
7. Remember you are not alone. Have a low threshold to call your resident and fellow.
8. Hold beta blockers for patients getting stressed with exercise or dobutamine at least 12 hours prior to procedure.
9. Generally, most patients admitted to the cardiology service with chest pain should be NPO after midnight and be on a no caffeine (in case persantine will be used), cardiac diet the day prior in anticipation of a stress test.
10. If a patient on the cardiology service has had a cath at an outside hospital, obtain cath report from the other hospital (can call the cath lab at outside hospital).

Chest Pain

Assess vital signs immediately, including O₂ sat. If unstable, see patient immediately.
- ECG immediately and always compare to old. Obtain new ECGs every time the pain changes or goes away - look to see if there are dynamic changes.
- Get a CXR (portable is O.K).

1. **DDX:** Acute coronary syndrome (unstable angina, NSTEMI, STEMI), Aortic dissection, Pneumothorax, PE, GERD, Musculoskeletal, pericarditis/myocarditis, pneumonia/pleurisy, PUD, esophageal spasm, esophageal rupture or tear (Mallory-
Weiss), candidiasis, herpes zoster, costochondritis (Tietze’s syndrome), anxiety (diagnosis of exclusion).

Directed History and Physical (rule out the bad stuff):
• ACS: typically pressure type of pain, associated with shortness of breath, nausea, vomiting, diaphoresis, radiation. Assess for risk factors including history of prior MI, prior stenting procedures, DM, HTN, tobacco use, FHx, hyperlipidemia. MI can present atypically in women and diabetics.
• Aortic dissection: “tearing” pain that usually radiates to the back. Associated with HTN, smoking.
• Pneumothorax: Associated with COPD, trauma, central lines. Decreased breath sounds, hyperresonance. Deviation of the trachea away from the side of the PTX, hypoxia.
• PE: dyspnea, tachycardia, tachypnea, pleuritic chest pain, hypoxia, A-a gradient, possible hemoptysis.

2. If angina is suspected: start Oxygen by NC, cardiac monitoring (i.e. telemetry), NTG (0.4 mg SL q 5 minutes X 3; hold for SBP <100; If has an RV infarct/inferior MI or has taken a 5 PGE antagonist- i.e. sildenafil- NTG may cause hypotension because pre-load is reduced). Nitropaste or nitroglycerine gtt, ASA 325 mg PO if no contraindications or has not yet been administered. Get a CXR (portable is O.K.) Labs: Troponins (if the chest pain just started, the test may not be positive yet, so cycle them more rapidly than q 6 hours; generally order troponins q6 hours X 3 or until they peak). Chem.-7, mg, phos, coags.

3. If PE is suspected: CT with PE protocol (generally contrast is not O.K. in a patient with ARF/AKI, CKD, contrast allergy) or V/Q scan. If have high level of suspicion, start anticoagulation right away as long as not contraindicated.

4. PTX: If tension, immediately place a 14 gauge angiocath into the 2nd intercostal space in the midclavicular line (Don’t wait for the CXR). Otherwise, obtain CXR and if PTX present and patient is stable call pulmonary fellow or attending (if on cardiology service, call the fellow) for chest tube placement (sometimes small ones can be treated with high flow oxygen).

5. If aortic dissection is suspected: transfer to ICU, give beta-blocker to reduce heart rate and BP. Get an emergent CT scan with dissection protocol or echo ASAP. If patient is not stable, don’t send down for a CT scan unless OK’d by someone more senior than yourself.

ACS

<table>
<thead>
<tr>
<th></th>
<th>Troponins</th>
<th>ECG changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>STEMI</td>
<td>Positive</td>
<td>ST elevations or new LBBB</td>
</tr>
<tr>
<td>NSTEMI</td>
<td>Positive</td>
<td>May have ST depressions, T wave inversions, or ST elevations that don’t meet criteria for STEMI.</td>
</tr>
<tr>
<td>Unstable angina</td>
<td>Negative</td>
<td>+/- May have ST depressions, T wave inversions, or ST ↑ that don’t meet STEMI.</td>
</tr>
</tbody>
</table>

Atrial Fibrillation

1. **If symptomatic or unstable** (CP/ischemia, SBP <90), proceed with cardioversion (ACLS).
2. **If relatively stable**, control rate, anticoagulate. Cardioversion may be considered further down the road.
3. **Causes:**
   - P (pulmonary, pericarditis/myocarditis, pain, post-op)
   - I (Ischemia)- rare unless large RCA infarct.
   - R (rheumatic heart disease/valvular heart disease)
   - A (alcohol)
   - T (thyroid)
   - E (emboli)
   - S (sepsis/sick sinus)

   Other things that don’t fit with the mnemonic: fever, CHF, HTN, cardiomyopathy, congenital heart disease, changes in myocardial wall stress, diabetes.
   *Hint: Almost anything you can think of that makes the heart go fast (drugs, alcohol withdrawal) or anything that causes the atria to be stretched out of shape (COPD, infiltrative disease) can cause atrial arrhythmias.

4. **Rate control:**
   - Be careful when using β-blockers and Calcium channel blockers together, as the combination may cause excessive AV nodal blockade.
   - β-blockers: don’t give if actively wheezing or if in decompensated heart failure.
     - Metoprolol PO if rate is relatively slow and patient is stable
     - Metoprolol 5 mg IV X 3
     - Esmolol gtt- good to consider in ICU patients/potentially unstable because half-life is about 9 minutes.
   - Calcium channel blockers:
- Contraindicated with VT, 2nd/3rd degree heart block without pacer, severe hypotension, cardiogenic shock, bypass tracts.
- Diltiazem: bolus administration and if does not work, then gtt.
  - Amiodarone: long term side effects. Consider in unstable patients or patients with CHF who need rhythm control as has short-term calcium channel blocking and sympatholytic effects.

5. Anticoagulation
   - Consider the use of unfractionated or LMWH in hospital. No LMWH for those with CrCl less than 30. Overlap with warfarin until INR 2 – 3 achieved.

6. Cardioversion
   - If in afib for longer than 48 hours or cannot document length of afib, patient will need to be anticoagulated for 4 weeks before cardioversion to decrease risk of embolization during procedure.
   - If TEE shows no atrial thrombus can proceed directly to cardioversion.
   - Following cardioversion, patients will need to be anticoagulated for at least 3 - 4 weeks.

Stress Testing

1. Who gets stress testing?
   - Intermediate probability for having had angina, or suspected arterial blockage

2. Stress testing can be divided into the stress and the modality used to look to see if there is ischemia.
   - Stress: modalities used to stress the patient.
     a) Treadmill:
        Requires a fair amount of work, increasing every 3 minutes in speed and grade. GOAL: 85% of MPHR (therefore no BB or rate-limiting agents).
        Everyone who can walk should be put on the treadmill, this will give you not only functional capacity, but also allow for assessment of ischemia.
        Contraindications: Unstable angina, ACS, inability to ambulate.
        Imaging: can be linked with ECG, echo, ECG with myoview.
     b) Dobutamine:
        Dobutamine either starts at 5 or 10 mcg/kg/min, increasing every 3 minutes like the Bruce in 10 mcg increments to a max of 50. At times atropine is used to further increase heart rate, so watch for urinary retention post procedure. Goal: 85% of MPHR (therefore no BB or rate limiting agents). Use in cases where persantine (RAD) is contraindicated and treadmill not possible.
        Imaging: used with either Myoview (cardiolyte) or echo. Caution in those at risk for dysrhythmia (electrolyte abnormalities, known dysrhythmias).
     c) Persantine:
Works by causing vasodilation, and a coronary steal phenomenon (i.e. blood goes to good areas and not to bad areas). You should see drop in BP with a small rise in HR. Generally, no ECG changes. Goal: No need to achieve HR here, therefore okay to give all the patients meds, including BB, except for theophylline. The methylxanthines negate the effect of Persantine, this is why you cannot have caffeine for 12 hours prior to the test. Imaging: always use myoview since you are not going for heart rate. Contraindicated in patients with bronchospasm. Reversed with 75 mg (3cc) of aminophylline which can be given via IV push.

d) Adenosine
Shorter acting than persantine. Can be used when you are concerned about bronchospasm. Same mechanism of action as persantine. Can be reversed with aminophylline.

- Imaging: pair with a stress above,
  a) ECG:
  Will be used with all stress tests to monitor the patient. Can be your definitive modality if the baseline ECG is normal. Cannot use ECG to assess ischemia when a) LBBB b) Dig effect c) LVH with repolarization or any other resting ST depression (can use it in RBBB). Goal: looking for ST depression which comes in 3 forms, upsloping (normal up to 2 mm), horizontal (abnormal + at 1mm), downsloping (abnormal again 1 mm); also need to look for ST segment elevation. Will stop the test if ST segment elevation, if ST depression with any sx, or electrical instability. Note J point elevation should come down with exercise, also there are some people who get rate-related RBBB and even LBBB.
  b) Echo:
  Can be paired with dobutamine or with exercise. Looking for inducible wall motion abnormalities. Need to know that the patient has good windows (i.e. if they are really obese, not a good idea). Great test for the possible false positive ECG.
  c) Myoview (cardiolyte):
Imaging agent for use with persantine/adenosine, also can be used with ETT when there are baseline ECG abnormalities. Also can be used with dobutamine when the patient hits the target heart rate

**Cardiac Catheterization**

1) **Indications:** Initial reperfusion therapy for acute STEMI or new LBBB MI. Or in NSTEMI with uncontrolled chest pain, hemodynamic instability or electrical instability.
2) If you suspect someone is going to get cathed, hold their metformin.
3) Types of stents placed: Bare metal stent (BMS) vs. drug eluting stent (Taxus®-paclitaxel or Cypher®-sirolimus or similar).

4) If cardiac catheterization is planned and pt has CKD or recent renal insult, consider NAC (600 mg PO BID X 4 doses- 2 before and 2 after procedure) before and after the procedure and IV fluid hydration 12 hours prior to the procedure and following the procedure.

5) Basic anatomy:
   • Left main gives off the Left circumflex (LCX) and the LAD. LAD branches include the septal arteries and the diagonal artery. Obtuse marginal artery branches off the LCX.
   • Branches of the right coronary artery include the SA node artery, conus artery, acute marginal artery, posterior left ventricular artery (PLV), posterior descending artery (PDA). However, if left dominant, the LCX may give off the PDA.

6) Post-cath care:
   • Check access site for oozing, bleeding, hematoma, or bruits after procedure.
   • Check distal pulses: If pulses are diminished or unequal consider acute limb ischemia. Remember the 5 P’s of acute limb ischemia: pain, pallor, pulselessness, paresthesia, paralysis. Call fellow immediately if suspected, as this is a medical emergency.
   • If a continuous bruit is present, an AV fistula may have formed. If a systolic bruit is present a pseudoaneurysm may have formed. Order US, call fellow.
   • If bleeding present, hold pressure cephalad to puncture site as ateriotomy is likely 1 – 2 cm above skin entrance. If bleeding does not stop contact fellow.
   • Oozing, hold pressure for at least 10 minutes.
   • Hematoma: check hct, platelets. Order type and cross. Follow size-outline border with pen.
   • If hct drops, consider retroperitoneal bleed and order non-contrast CT to rule this out.
   • If Cr increases within 48 to 72 hours of cath, consider contrast-induced nephropathy.

CHF
1. The most important thing to recognize is when a patient is cold and wet (see below) as they likely need inotropes- we usually give dobutamine. Do not start this without speaking to your resident and fellow.
   • Learn this chart and classify all your CHF patients into one of these four categories.

<table>
<thead>
<tr>
<th>Perfused? Yes</th>
<th>Congested? No</th>
<th>Congested? Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Warm and Dry</td>
<td>Warm and Wet</td>
</tr>
<tr>
<td>Perfused? No</td>
<td>Cold and Dry</td>
<td>Cold and Wet</td>
</tr>
</tbody>
</table>

*Signs/Symptoms of Congestion: orthopnea, high JVP, Increasing S3, Loud P2, edema, Ascites, rales, Hepatojugular reflux.
*Signs/Symptoms of Low Perfusion: Narrow pulse pressure, pulsus alternans, Cool forearms and legs, Sleepy/obtunded, low serum sodium, acute renal failure, symptomatic hypotension.

- Digoxin level in heart failure patients should be 0.8 to 1.1 and needs to be renally dosed.
- Hold beta blockers if patient is in cardiogenic shock, otherwise can the beta blocker continue unchanged in most situations
- Do not re-start beta blocker until patient is euvolemic.
- When adding an ACE inhibitor start with captopril, which is dosed q 8 hours. If tolerated increase dose at each interval (you can sign this out). See conversion table below to figure out how much lisinopril to discharge someone on. Never discharge someone on captopril, unless fellow/attending instruct you to do so because it is difficult for patients to take a drug TID.

<table>
<thead>
<tr>
<th>Captopril</th>
<th>Lisinopril</th>
</tr>
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<tbody>
<tr>
<td>6.25 mg PO TID</td>
<td>5 mg PO Daily</td>
</tr>
<tr>
<td>12.5 mg PO TID</td>
<td>10 mg PO Daily</td>
</tr>
<tr>
<td>25 mg PO TID</td>
<td>20 mg PO Daily</td>
</tr>
<tr>
<td>50 mg PO TID</td>
<td>40 mg PO Daily</td>
</tr>
</tbody>
</table>


**Heart Transplant Basics**

1. **Rejection**: 4 types.
   1. **Hyperacute**: minutes to hours. Secondary to preformed antibodies to HLA, ABO blood group antigens, endothelial antigens that cause immediate rejection. You will not see this unless you are on surgery.
   2. **Acute Cellular**: may occur at any time, but most common 3 to 6 months following transplant. T cell mediated. Dx made via endomyocardial biopsy where lymphocytic infiltrate is present along with macrophages with resultant myocytolysis. Patient may have no symptoms. Usual treatment is IV or oral steroids, monoclonal or polyclonal anti-lymphocyte antibodies.
   3. **Acute Humoral (vascular)**: days to weeks following transplant. Less common than acute cellular (7%). Mediated by antibodies against donor HLA or endothelial cell antigens. Highest risk are women, high panel reactive antibody screen, CMV +, sensitization to OKT3. DX on endomyocardial biopsy with immunoglobulin and complement in the vessels or by swollen endothelial cells. Commonly associated with severe LV dysfunction. Treat by intensifying immunosuppression and either by modulating antibody production or removing antibodies (cyclophosphamide, IVIG, plasmapheresis).
   4. **Chronic**: months to years. Likely a combination of humoral and cellular. Manifests as graft atherosclerosis with myointimal proliferation.
2. **The Immunosuppression Scheme:**
   - **Induction:** Intense perioperative immunosuppression X 7 to 14 days. Lympholytic agents (ATGAM, thymoglobulin). Increasingly, IL-2 receptor antagonists are being used.
   - **Maintenance:** Combination of an antiproliferative agent (mycophenolate or azathioprine), a calcineurin inhibitor (cyclosporine or tacrolimus) and steroids. Early on, higher cyclosporine levels (300 – 350) and TAC levels (10-15) are desired. Later (2 years out), Cyclosporine levels should be about 200 and TAC levels 5 – 10. Prednisone is generally tapered off in about 50% of transplant patients 6 to 12 months post-transplant.
   - **Rejection:** Intensity and type of regimen depends on severity of rejection and whether T or B cell mediated.

3. **Anti-lymphocyte Agents:** used in induction and steroid resistant rejection
   - ATGAM: polyclonal anti-lymphocyte Ab’s produced in horses.
   - Thymoglobulin: polyclonal anti-lymphocyte Ab’s produced in rabbits.
   - Muromonab CD3 (OKT3): murine antibody that recognizes epsilon chain of the CD 3 molecule on T cells, rendering T cells unable to respond to antigen challenge or bind to target cells.

4. **Anti-cytokine Receptor Antibodies:** bind to subunit of IL-2 receptor, preventing the binding of IL-2 to the IL-2 receptor, thus inhibiting the proliferation of T cells.
   - Daclizumab and Basiliximab are the two on the market.


**Advanced ECG reading**

1. **“LITANY OF CATEGORIZATION”**
   - The mean rate of the QRS complexes:
     - Slow rate
     - Normal rate
     - Fast rate
   - The duration of the dominant QRS complexes
     - Narrow (normal in adults 70-113 msec)
     - Wide (>120 msec)
   - The spacing of the R-R intervals
     - Regular
     - Irregular

2. **REGULAR NARROW-COMPLEX TACHYCARDIAS**
   - **Take home points**
     - Adenosine can be used to help differentiate narrow complex tachycardias. Do not use adenosine in wide-complex tachycardia that is irregular; use very carefully in transplant patients (if at all) and patients on dipyridamole.
     - Remember the differential: AVNRT is the most common and should break with adenosine. The relationship between the QRS and P-waves is important for narrowing the differential.
- The key to arrhythmia diagnosis is how it starts and how it ends, run a continuous rhythm strip when giving adenosine.

- **Step 1:** First make sure that the rhythm is narrow (QRS < 120 ms) or matches the patient’s previous baseline QRS morphology and that it is a tachycardia (rate > 100).

- **Step 2:** Is the dominant rhythm regular or irregular?

Regular narrow-complex tachycardia differential:
1. Sinus tachycardia
2. Atrial flutter
3. AV nodal reentrant tachycardia (AVNRT)
4. AV reentrant tachycardia (AVRT) (ORT)
5. Atrial tachycardia (AT)
6. Accelerated junctional tachycardia
7. Sinus node reentry tachycardia

**Note:** a very fast irregular rhythm (fast atrial fibrillation) can look like a regular narrow complex tachycardia.

- **Approach:**

  **What is the rate?**
  - 100-140: this will likely be sinus tachycardia, atrial tachycardia, or an accelerated junctional tachycardia. Remember that the upper limit of normal for sinus tachycardia is 220 – age. Also remember that an accelerated junctional tachycardia is usually at a rate of 100-120.
  - 140-160: think of atrial flutter with 2:1 block or atrial tachycardia, less likely AVNRT or AVRT.
  - 160-220: think of an AVNRT, AVRT, AT.

  **Find the P waves:**
  - No P waves seen: consider accelerated junctional tachycardia for slower rhythms and AVNRT for faster rhythms.
  - P waves present and PR < RP (= P wave just before the QRS): Is the P upright in lead I and the inferior leads (II, III, aVF). If the answer is yes, you’re probably dealing with a sinus tachycardia. If the answer is no, you’re probably looking at an atrial tachycardia. Remember that slow sinus rates come from inferior portion of the sinus node, and fast sinus rates more superiorly; hence, the P-wave becomes more peaked in the inferior leads at higher sinus rates.
  - P waves present and PR > RP (= P wave just after the QRS): if the rate is slower (100-120), think of an accelerated junctional tachycardia with retrograde P waves. If the rate is fast (> 160), think of AVRT.
  - P waves present and going at a rate of 300: think of atrial flutter.

  **Clues for specific rhythms:**
  - Sinus tachycardia: upright P wave in I and the inferior leads and normal PR interval. The maximum rate is usually 220-age. In most cases, the rhythm speeds up and slows down gradually.
  - Atrial flutter: look for “saw-tooth” waves in the inferior leads (II, III, aVF). The rate is usually approximately 150 and there are P waves going at a rate of 300.
- AVNRT: look for the pseudo S wave in lead II and the pseudo R’ wave in lead V1.
- AVRT: the P-wave can have a variety of relationships with the QRS.
- AT: looks like a sinus tachycardia except that the P waves are not upright in both I and II and the PR interval can be shortened or prolonged.

Short RP, Long RP what are people talking about?

**Short RP tachycardias**
- most commonly AVNRT or Orthodromic Reciprocating Tachycardia (ORT)
- also can be atrial tachycardia with a long 1st AV block (usually at faster rates)

**Long RP tachycardias**
- most commonly atrial tachycardia
- can also be uncommon AVNRT, ORT, or junctional tachycardia (PJRT)
P in R Tachycardia
- most commonly AVNRT
- Two major presentations
  1) No discernable P waves (P wave buried in the QRS complex) (A)
  2) Pseudo S waves (B):
     a retrograde P wave blends with the end of the QRS complex in the
     inferior leads, producing a pseudo S wave –and– Pseudo R’
  3) (C): A P wave blends with the end of the QRS complex in lead V1
     producing a “pseudo” r’ wave.

3. IRREGULAR NARROW COMPLEX TACHYCARDIAS

Irregularly Irregular Rhythm (four main causes):

1. Atrial Fibrillation: Most common cause. Chaotic atrial depolarization (atrial rate
   ~600 bpm) likely from pulmonary veins. Because of decremental nature of AV node,
   ventricular rate 180s-200s
2. Atrial Flutter with variable block: Flutter waves in the inferior leads
3. Multi-focal atrial tachycardia: Often seen in patients with concomitant pulmonary
disease. Diagnostic criteria mandates 3 distinct P wave morphologies.
4. Frequent Premature atrial contractions

Regularly Irregular Rhythm: Grouped beating (clusters of mathematically-spaced QRS complexes, separated by pauses of identical duration). Wenckebach periodicity should always be considered either from AV block or during junctional tachycardia with exit block (Digoxin toxicity).

1. 2nd degree AV Block, type I (Wenckebach) or Type II (Mobitz II)
2. PVCs in a repetitive pattern (trigeminy, quadrigeminy)
3. PACs in a repetitive pattern

4. WIDE COMPLEX IRREGULAR TACHYCARDIAS

- Take home points:
  - These are rare, history of WPW or if patient has a known accessory
    pathway, think Afib with competing conduction down AP and some down
    HP network (varying degrees of pre-excitation).
  - Always note whether a pre-existing bundle is present, AFIB with a BBB
    is likely the most common cause of an irregular WCT
  - Principal: Wide complex supraventricular rhythms (excluding bundle
    branch blocks) result from ventricular activation either partly or
    completely from accessory pathway conduction. The electrical signal will
    propagate across the myocardium from cell to cell using gap junctions etc.
    This is in contrast to narrow complex QRS complexes which use the
    “slick” His Purkinje system and therefore depolarize efficiently across the
    myocardium in a fast manner. For example, a PVC has such a wide
    QRS because the initial depolarization takes more time to spread across
    the ventricle because it does not use the Purkinje system.
• Steps:
  1. Do all wide QRSs within any single lead have essentially identical morphology?
     If yes ➔ probably an existent RBBB or LBBB
     If no ➔ Exclude rate-related bundle branch block

5. REGULAR WIDE COMPLEX TACHYCARDIAS

• VT or not VT, this is the question
• Take Home Points:
  - Assume VT till proven otherwise, remember follow ACLS
  - Remember what a bundle branch block looks like.

Helpful hints:
  1. Remain calm, ensure what you are looking at it real, not motion artifact.
  2. If patient is hemodynamically unstable, follow ACLS guidelines (likely VT ➔ SHOCK, remember some sedation).
  3. If you shock a patient and the rhythm does not change, likely not VT.
  4. Try to get a baseline ECG to see what you were starting with.
  5. If the patient has a history of MI, likely VT.
  6. Is there a history of WPW?
  7. Remember what a bundle branch looks like, does the rhythm look like a bundle, if not then likely VT.
  8. Distinguish between a wide QRS and an elevated ST segment.
  9. Apply the Brugada criteria, you are trying to distinguish between SVT with aberrancy and VT.

Causes (Memorize this list):

  1. ST with BBB: Leading cause of regular WCT. Looks like a bundle and is fast and regular. Can see P waves with each QRS
  4. Antidromic reciprocating tachycardia: History of WPW with AP -should see retrograde P waves

References
Evans, Tom ECG Interpretation Cribsheets Ring Mountain Press, Corte Madera, CA available through the UCSF bookstore.
Post cardiac arrest hypothermia

Current ACLS guidelines recommend mild hypothermia for comatose patients resuscitated from prehospital cardiac arrest. This recommendation is based on two trials published in the NEJM in 2002. These studies found an increased number of survivors and improved neurologic outcomes with a number needed to treat of around 5.

All of our hospitals have protocols in place to help with this process. The general process is that the patient is sedated and paralyzed (to prevent shivering) and then cooled with cooling blankets to a core temperature between 32-34C. The temperature is maintained in this range until 24 hours after the start of cooling, at which point the patient is allowed to gradually rewarm over 6-10 hours.

Common complications include bradycardia, hyperkalemia, coagulopathy and overshoot to below target temperature. The bradycardia is usually well tolerated and does not require treatment. Electrolyte and coagulation abnormalities are monitored closely with frequent lab checks.
Diabetic Ketoacidosis (DKA)

1. **Signs/Symptoms:** polyuria, polydipsia, polyphagia, abdominal pain, nausea, vomiting, dizziness, and confusion. Kussmaul’s respirations (i.e. rapid, large-volume breathing), acetone or “fruity” breath. Tachycardia and orthostasis due to intravascular volume depletion. Coma, respiratory arrest, death.

2. **Potential Causes:** Infection is the most common precipitating factor, followed closely by inadequate use of insulin. Alcohol abuse, myocardial infarction, pancreatitis, eating disorders, trauma, hormonal changes (e.g., during pregnancy), undiagnosed diabetes, drugs (e.g. corticosteroids), medication noncompliance.

3. **Initial Evaluation:**
   - **History:** Look for precipitating factor(s)
   - **Exam:** Urgent evaluation with vital signs and orthostatics
   - **Lab:** BMP - calculate anion gap and corrected Na⁺, ABG, serum and/or urine ketones, CBC+diff. Consider ECG, CXR, UA, troponins, pregnancy test, lipase, blood and urine cultures.

4. **Treatment** (see Appendix A. DKA Treatment Protocol)
   - **Volume resuscitation**
     - An average of 6 L IVF is needed in the first 24 hr to replete intravascular volume
     - Initial bolus of NS 1-2 L followed by continuous infusion to achieve euvolemma
   - **Electrolyte repletion** -- Follow electrolytes and AG every 2 to 4 hr
     - Potassium - **may be elevated initially but will fall after initiating insulin**
       - If <3.3 mEq/L on presentation, replete prior to initiating insulin therapy. Otherwise supplementation should be initiated for K⁺ <5 mEq/L.
     - Sodium - **may be falsely low from hyperglycemia**
       - Calculate actual serum Na⁺ by adding 1.6 mmol/L for every 100 mg/dL glu >100 mg/dL.
     - Phosphorous - may be elevated initially. Insulin therapy will cause rapid intracellular shift; replete if <1 mg/dL.
   - **Glucose control**
     - Follow glucose level every hour
     - Initiate insulin drip (for bolus and initial drip rate see flowsheet in appendix or use the University of Colorado Hospital (UCH) standardized order set for DKA/HHS.)
Add dextrose (D5) infusion when glucose falls below 250 mg/dl to avoid hypoglycemia and to provide carbohydrate nutrition. Patient may still require continued volume resuscitation.

- Ketone resolution: follow ketones Q4hr initially and treat with fluids and insulin as above. Ketonemia takes longer to clear than hyperglycemia. Clearance of ketones should not be used as an indicator of response to therapy if the lab uses the nitroprusside method to measure urine or serum ketones since it does not measure the most prevalent acid in DKA (β-OH-butyrate).

5. Transition to subcutaneous insulin
   - Once DKA is resolved, time transition to coincide with reinstitution of PO intake
   - Administer long-acting insulin 1-2 hours PRIOR to stopping IV insulin infusion. Start rapid-acting insulin at the next meal.

   To calculate insulin doses
   - Use outpatient insulin regimen OR
   - Calculate Total Daily Dose (TDD) estimating from IV Insulin infusion as follows: TDD = add up insulin used in the last 6 hr of IV insulin gtt, multiply by 4 for 24 hr requirement, then take 80% of that calculation. 50% of TDD given as Lispro once a day and 50% administered as rapid-acting insulin (e.g. Lispro) split among 3 meals. OR
   - Estimate using body weight: If patient is overweight, initial TDD = 0.3-0.5 units/kg/day. If patient is thin, initial TDD = 0.1-0.3 units/kg/day.

   - UCH has standardized orders for subcutaneous insulin that can be used
   - Strongly consider Endocrine/Diabetes physician team consult to recommend dosing strategies or adjustments in insulin therapy.
   - Call inpatient Diabetes Clinical Educator as needed for patient education

### Hyperglycemic Hyperosmolar Syndrome (HHS)

1. **Signs/Symptoms**: altered mental status or coma. Elevated serum osmolality without ketosis. Often with h/o type 2 diabetes and insulin resistance. Serum glucose often much higher, with more severe volume depletion than in DKA, leading to higher mortality rate (15% versus <5%).

2. **Potential Causes**: similar to DKA. Often seen in elderly persons without easy access to PO fluids

3. **Initial Evaluation**: same as for initial DKA evaluation

4. **Treatment** (see Appendix B. HHS Treatment Protocol or UCH standardized order set for DKA/HHS
   - Volume resuscitation: Similar to DKA except that more IVF resuscitation is typically required
   - Electrolyte repletion: see DKA section 4.b.
   - **Carefully control rate of correction of serum osmolality and sodium**
Monitor closely for signs of cerebral edema (mortality 70%) and follow these guidelines to correct glucose and serum Osm levels:

- Keep serum glucose between 250-300 mg/dL until serum Osm <315 mOsm/kg
- Adding dextrose when glucose reaches 300 mg/dL may reduce risk of cerebral edema

5. **Transition to subcutaneous insulin** – see section I.A.5. above

### Hypoglycemia

1. **Signs/symptoms:** Anxiety, diaphoresis, confusion, tremor, fatigue, loss of consciousness

2. **Potential Causes:** Drugs (sulfonylureas, insulin, alcohol), infection/sepsis, hepatic or renal failure, adrenal insufficiency, malignancies (lymphomas/leukemias via IGF-2 secretion), insulinoma, prior gastric surgery, factitious.

3. **Initial Evaluation and Treatment:**
   - Confirm serum blood glucose <70 mg/dL
   - If patient is not diabetic, not on glucose-lowering diabetes agents or insulin and is asymptomatic, do not treat the hypoglycemia. Recheck blood glucose prn
   - For patients on glucose-lowering diabetes agents or insulin, treat immediately as follows:
     1. If patient can take PO, give 15 grams of carbohydrate (4 oz of fruit juice OR non-diet soda OR 15 grams glucose gel).
     2. If patient unable to take PO and has IV access, give 1/2 amp D50 IV
     3. If patient unable to take PO and has no IV access, give 1 mg (1 amp) glucagon IM
     4. Check BG in 15 minutes & repeat above until BG is ≥ 100mg/dL
     5. D10 IV infusion may be required for persistent hypoglycemia unresponsive to above
   - Search for underlying cause. Common causes of inpatient hypoglycemia are sudden changes in nutritional status (e.g. NPO) without adjustment in insulin dosing, and use of outdated insulin regimens such as Sliding Scale Regular Insulin.

4. Call Endocrine consult for recurrent/persistent hypoglycemic episode(s). Labs helpful for workup of recurrent hypoglycemia include glucose, C-peptide, insulin, sulfonylurea level (all while hypoglycemic)

### Hyperglycemia

*(with or without a prior diagnosis of diabetes)*

**Adjust insulin doses DAILY as needed, based on BG control and the amount of extra insulin given in the previous 24 hr for high BG**
1. The Basics

- **Total Daily Dose (TDD)** = sum total of all insulin scheduled to be given during a 24 hr period
- **Initial TDD for type 2 diabetes:** 0.3 – 0.4 units/kg/day. For type 1, use 0.3 – 0.8 units/kg/day
- **50% of TDD is given as basal insulin, 50% as rapid-acting insulin distributed across 3 meals**
  a) **Basal Insulin** = long acting insulin required to maintain normal BG overnight and while NPO
  b) **Mealtime Bolus Insulin** = rapid-acting insulin used to cover meal-induced rise in glucose
- Patients on intensive insulin therapy use a carbohydrate to insulin (C:I) ratio for meals. C:I ratio means that the patient takes 1 unit of rapid-acting insulin for every ‘X’ grams of carbohydrate eaten in a meal. Calculate C:I by dividing the correction factor (see below) by 3.
  - **Correction Factor** = rapid-acting insulin used pre-meal to correct hyperglycemia
  - **CF** means that 1 unit of rapid acting insulin will decrease the BG by ‘X’ mg/dl. CF is calculated by dividing the TDD into 1650.
  - For patients on standard insulin therapy, correction factors can be used in addition to meal dose insulin to customize hyperglycemia treatment scales.

2. Treatment

- If known diabetes, review outpatient diabetes management with patient and adjust if needed
- Fingerstick BG QAC and QHS (q4-6 hr if NPO); q1-2 hr on insulin drip. Goal <180 mg/dL
- **Goals:** avoid hypoglycemia, severe hyperglycemia, and electrolyte abnormalities
  - Critically ill patients: < 140 mg/dl, tight control 80-110 mg/dl may be associated with increased mortality
  - Non–critically ill patients: premeal BG levels between 90–130 mg/dl, all fasting BG < 180 mg/dl
- **Establish plan for treatment of hypoglycemia**
  - If patient is alert, treat with simple sugars (1/2 cup juice or 15 g glucose) and recheck BG
- **Adjust scheduled insulin regimen accordingly. Do not discontinue basal insulin.**
  - Oral medications: hold metformin and sulfonylureas upon admission in most cases

3. Injectable insulin

  *Strongly consider IV insulin drip in the ICU since critically ill patients do not absorb SQ insulin as well and require tighter glucose control*

- **History of outpatient insulin use?**
  1. Continue basal insulin (glargine/Lantus, NPH) at outpatient doses or cut in ½ if NPO
2. Continue bolus insulin for meals using rapid acting insulin (humalog/Lispro, novolog/Aspart, glulisine/Apidra)
3. If on insulin pump, continue in the hospital. Consult Diabetes Clinical Educator
4. Start CF based on equation in previous section

- No history of outpatient insulin use?
  1. Initiate scheduled-dose insulin (basal +/- mealtime bolus) based on previous day’s insulin requirements. May use weight to estimate TDD – see previous section
  2. Initiate CF using rapid acting insulin – see previous section

4. **Insulin Adjustment Pearls**
   - If BGs for past 24 hr were mostly >180, incorporate the extra insulin administered for hyperglycemia into the new scheduled dose insulin regimen
   - If there was a BG <80 in the past hr, reduce total daily dose by 10-20%
   - If AM fasting BG is elevated, check a 3 am FSBG to make sure this is not rebound hyperglycemia BEFORE increasing basal insulin dose
   - If AM fasting BG rises by >30 mg/dL above bedtime BG, increase basal insulin
   - If lunchtime BG is elevated, increase breakfast bolus. If dinnertime BG is elevated, increase lunch bolus. If bedtime BG is elevated, increase dinner bolus

5. **Call Diabetes Clinical Educator or Endocrine service with questions or consults**

6. **Discharge planning**
   - Diabetes education prior to discharge
   - Restart oral medications unless contraindicated
   - Schedule outpatient evaluation and follow-up
   - *Every patient admitted for DKA or HHS MUST be discharged on an insulin regimen*

**References**

Hirsch IB, Braithwaite SS, Verderese CA. Practical management of inpatient hyperglycemia. (monograph you received at beginning of residency)


**Thyroid Storm**

1. **Signs/Symptoms:** Fever, tachycardia, tachypnea, arrhythmias, CHF, abdominal pain, nausea, vomiting, diarrhea, hyperkinesis, psychosis, coma, goiter (not always present).

2. **Potential Causes:** Often in patients with unrecognized/inadequately treated thyrotoxicosis and a precipitating event: thyroid or nonthyroid surgery, infection, trauma, PE, MI, acute iodine load.

3. **Initial Evaluation:**
   - History: personal or family history of thyroid abnormalities, goiter, recent iodine contrast, agitation, recent weight loss

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- PE: goiter +/- bruit, stare, lid lag, proptosis, tachycardia, agitation, tremor, brisk relaxation phase of reflexes and hyperreflexia
- Lab: TSH (undetectable), free T4 (high) OR total T4 and T3 resin uptake (both high), total T3 (often high), CBC, BMP, LFT’s.
- Diagnosis: Labs alone are insufficient. If you have a high index of suspicion use the Burch score which takes into account body temperature, CNS changes, GI/hepatic dysfunction, and cardiovascular dysfunction (CHF, atrial-fib) to determine need for aggressive intervention (Appendix C). Consult Endocrine. Delayed/inappropriate treatment may result in death.

4. Treatment:
   - First, reduce the heart rate and stabilize patient:
     1. Esmolol drip - load with 250-500 μg then 50-300 μg/kg/minute
     2. Support the circulation – IVF
   - Once stable, decrease thyroid hormone synthesis:
     1. Propylthiouracil (PTU) up to 800 mg/day in divided doses (PO, PR, NG). Also blocks conversion of T4 to T3
     2. Methimazole (Tapazole) up to 80 mg/day QDor BID (PO, PR, NG)
   - Inhibit thyroid hormone release – give 1 hr after first dose of anti-thyroid drug
     1. SSKI 5 drops PO q8h OR
     2. Lugol’s solution 10 drops PO q8h
   - Block T4→T3 conversion with steroids (?relative adrenal insufficiency)
     – hydrocortisone 100 mg IV q8hr
   - Finally, search for and treat the precipitating condition

Myxedema Coma
1. Signs/Symptoms: altered mental state, hypothermia, precipitating event. Obvious physical signs of hypothyroidism, respiratory depression, bradycardia, hypotension, low-voltage non-specific ST changes on ECG, anorexia, abdominal pain, distention, constipation (may be severe causing ileus & megacolon; carries a 20-30% mortality rate)

2. Potential Causes: Complication of long-standing untreated or undertreated hypothyroidism with a precipitating event: infection, MI, CVA, sedatives, trauma, GI bleed

3. Initial Evaluation:
   - History: personal or family history of thyroid or pituitary disease, fatigue, constipation, depression, weight gain
   - PE: Hypothermia, bradycardia, edema, dry skin, delayed relaxation of reflexes, loss of lateral third of eye brows
   - Lab: TSH (high), Free T4 (low or undetectable) OR total T4 and T3 resin uptake (both low), total T3 (often low), BMP, CBC, Cort stim, CPK

4. Treatment:
• Rapid institution of parenteral thyroid replacement is necessary in true myxedema coma
• Proper dosing and form of replacement (T4 alone, T3 alone, T4 + T3) is controversial. **CALL ENDOCRINE**

5. Treatment of precipitating cause (e.g. broad-spectrum antibiotics)
   • Supportive care
   • Intensive care admission
   • Cautious IVF
   • Ventilatory support
   • Passive rewarming (active rewarming may produce peripheral vasodilation and could contribute to hypotension or shock)
   • Hydrocortisone (often a degree of impaired adrenal function) until you verify intact adrenal function with Cortisol Stimulation Test (see below)

**Euthyroid Sick Syndrome/Nonthyroidal Illness**

1. Defining the condition
   • Changes in thyroid function tests that occur in patients with a variety of nonthyroidal illnesses including infections, malignancies, MI, trauma, surgery, inflammatory conditions, and starvation
   • NOT a primary thyroid disorder. Results from changes in peripheral thyroid hormone metabolism and transport induced by the nonthyroidal illness

2. Management:
   • NO data demonstrating recovery or survival benefit from thyroid hormone treatment, so it is NOT recommended. Look for signs/symptoms of hypothyroidism, and if in doubt, consult Endocrine.

![Graph showing thyroid hormone levels](image)

*Ross D. Thyroid function in non thyroidal illness. Up-to-date 2006*

**Thyroid Pearls**

• Figuring out a dose for thyroid hormone replacement:
  - Full replacement dose for hypothyroidism: 1.6 mcg/kg PO QD (actual body weight)
  - Equivalent IV LT4 dose ~ 80% of PO dose

**Factors that affect interpretation of TSH**

• Glucocorticoids and dopamine IV gtt can lower TSH values
• TSH levels vary diurnally and are highest at night
Adrenal insufficiency

1. **Signs/symptoms** - fatigue, weakness, anorexia, nausea/vomiting, abdominal pain, orthostatic hypotension, cognitive: mild memory loss to psychosis, weight loss, hyperpigmentation (for Addison’s), hyponatremia, hyperkalemia

2. **Potential causes**

<table>
<thead>
<tr>
<th>Primary (adrenal)</th>
<th>Secondary/tertiary (pituitary/hypothalamic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autoimmune adrenalitis</td>
<td>Withdrawal from exogenous glucocorticoids</td>
</tr>
<tr>
<td>Infection – TB, fungal, CMV, HIV</td>
<td>Bilateral adrenal hemorrhage</td>
</tr>
<tr>
<td>Adrenomyeloneuropathy</td>
<td>Severe head trauma</td>
</tr>
<tr>
<td>Metastases (lung, breast, GI)</td>
<td>Sheehan’s syndrome</td>
</tr>
<tr>
<td>Meds – ketoconazole, rifampin</td>
<td>Pituitary apoplexy</td>
</tr>
</tbody>
</table>

3. **Initial evaluation**

*The usual diurnal variation of cortisol is lost in critical illness, so draw a random cortisol*

- A random cortisol of <5 mcg/dL in a sick patient
- 8 am cortisol of <5 mcg/dL in noncritically-ill patients is diagnostic for adrenal insufficiency
- A cortrosyn stimulation test (“cort stim”) should be performed for definitive diagnosis:
  - Draw a baseline cortisol. In sick patients, anytime; in non critically-ill patients, draw at 8 am
  - Administer 250 mcg Cortrosyn (synthetic ACTH) IV/IM
  - Re-draw cortisol at 30 and 60 minutes
  - A stimulated cortisol of >20 mcg/dL excludes adrenal insufficiency except in secondary or tertiary adrenal insufficiency developing within 2 weeks prior (usually from pituitary trauma). In septic shock, increment of <9 mcg/dL is consistent with “functional adrenal insufficiency”
  - Hydrocortisone is the synthetic version of cortisol, so a patient who has received hydrocortisone within 10 hr will have a cortisol level that may appear normal or elevated

*Suspect acute adrenal crisis in patients who are hypotensive despite aggressive fluid resuscitation, or in septic shock*

*Suspect adrenal insufficiency in any patient whose supraphysiologic regimen of glucocorticoids has been interrupted* (prednisone >5 mg QD or dexamethasone >0.5 mg QD >1 month)
4. **Treatment for adrenal crisis:** the 5 S’s (salt, sugar, steroids, support, search)

   1. Volume resuscitation with **D5NS**
   2. **Dexamethasone** 4 mg IV/PO if you want to give steroids before the cort stim test has been performed OR hydrocortisone 100 mg IV q8h if you have already drawn the baseline cortisol and/or performed the cort stim since hydrocortisone administration will “interfere” with testing
   3. **Steroids** can be tapered to maintenance doses after 1-3 days if the precipitating event or associated illness is under control
   4. ICU **supportive** care
   5. **Search** for precipitating cause

**References**

*Endocrine Secrets, 4th edition, 2005, chapter 31: Adrenal insufficiency*
*Harrison’s Principles of Internal Medicine online (http://hslibrary.ucdenver.edu/ebooks/, Part 14, chapter 321: Disorders of the adrenal cortex)*
*www.endotext.org Adrenal Disease and Function chapter 13: Adrenal insufficiency*

**Pituitary Apoplexy**

1. **Definition:** hemorrhage or infarction of the pituitary gland, often in a preexisting adenoma but may occur with head trauma, on anticoagulation, or immediately postpartum.

2. **Symptoms:** sudden and severe headache, visual and cranial nerve disturbances including bitemporal hemianopsia, confusion, symptoms of 2° adrenal insufficiency (N/V/hypotension).

3. **Lab/studies:** Head CT scan will usually show pituitary enlargement or hemorrhage. MRI is more sensitive for detecting adenoma and hemorrhage. Draw a pituitary panel – LH, FSH, estradiol or testosterone, alpha-subunit, IGF-1, cortisol, prolactin, TSH and free T4

4. **Management**
   - Medical: Dexamethasone 4 mg IV BID for cerebral edema and adrenal insufficiency
   - Surgical: If headache and visual disturbances do not improve on steroids, transsphenoidal pituitary decompression is indicated

**Severe Hypertriglyceridermia**

1. **Signs/Symptoms:** Often asymptomatic but may present with acute pancreatitis. May include symptoms of the chylomicronemia syndrome: abdominal pain (can be chronic), malaise, dyspnea, peripheral neuropathy, and memory loss. Abdominal tenderness, eruptive xanthoma (usu. upper back, buttocks, extensor surfaces of extremities), lipemia retinalis, hepatosplenomegaly.

2. **Potential Causes:** Usually an underlying genetic disorder of hypertriglyceridermia complicated by one or more secondary factors. Possible secondary factors (more common factors in **bold**):

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3. **Initial Evaluation** – Search for secondary factors
   • History: alcohol use, recent food intake, medications, pregnant?
   • Labs: glucose, TSH, UA (for proteinuria), LFTs, hCG (females), lipase (if suspect pancreatitis)

4. **Treatment**
   • NPO (if acute pancreatitis), or **zero-fat diet** (if asymptomatic/incidental diagnosis) – this is the most important treatment. Hypertriglyceridemia will not resolve if fat intake continues
   • Eliminate or treat any identified secondary factors; insulin treatment of poorly-controlled diabetes can be especially helpful.
   • Once pt. eating, initiate gemfibrozil 600mg BID or fenofibrate 160 mg QD (renal-dose if needed)
   • Follow daily TG until goal (TG < 400 mg/dL) is achieved. Diet can include fat (<20%) when TG <1000 mg/dL, and can be advanced to ≤30% when TG < 400 mg/dL

**Severe Hypercalcemia**

1. **Signs/Symptoms:** Confusion, Lethargy, Anorexia, Nausea, Constipation, Polyuria, Arthralgias, Weakness, HTN, renal insufficiency, RTA, shortened QT interval

2. **Potential Causes:** “VITAMINS TRAP

   - V = Vitamins
   - I = Immobilization
   - T = Thyrotoxicosis
   - A = Addison’s disease
   - M = Milk-alkali syndrome
I = Inflammatory disorders
N = Neoplastic-related disease
S = Sarcoidosis
T = Thiazide Diuretics (Drugs)
R = Rhabdomyolysis
A = AIDS
P = Paget’s disease, Parenteral nutrition, Pheochromocytoma, Parathyroid disease

3. Initial Evaluation
   • Serum BMP, calcium, albumin, phos, intact PTH, 25(OH) vitamin D, and possibly 1,25(OH)_2 vitamin D. If iPTH is low, draw PTH-related peptide
   • Follow Ca x Phos product Q2-4 hrs

4. Treatment
   • Saline infusion (250-1000 mL/h)
   • Bisphosphonate IV
     ▪ Zolendronic Acid 4 mg IV over 15 min OR Pamidronate 30-90 mg IV over 2 hrs
   • Consider dialysis if severe

Severe Hypocalcemia

1. Signs/Symptoms: numbness, tingling, muscle cramps, depression, psychosis, tetany and seizures, QT prolongation, papilledema
   • Chvostek’s sign – tap on facial nerve and look for upper lip twitch
   • Trousseau’s sign – inflate BP cuff and look for hand twitch

2. Potential Causes: hypoparathyroidism, hypoalbuminemia, hyperphosphotemia (rhabdo, tumor lysis, renal failure), hungry bone syndrome (recent treatment for hyperparathyroidism), chelation (citrate/transfusions), Mg depletion (decreased PTH secretion, PTH resistance), Vitamin D deficiency, sepsis/severe illness, chemo drugs

3. Initial Evaluation
   • Serum BMP, Ca++, albumin, phos, Mg++, intact PTH, 25(OH) vitamin D.
   • Ionized Ca++ (in severe illness)
   • Correct Ca++ (Ca_corrected = Ca_measured + [(4-albumin) x 0.8])
   • Urine Cr, Ca++ and Mg++

4. Treatment – Treat symptoms, NOT a number!! Pts. with chronic hypoparathyroidism often live at a much lower serum Ca level
   • Calcium carbonate PO (500 mg PO Q2-4 hr)
   • Active Vitamin D (calcitriol 0.25 – 0.5 micrograms PO BID)
   • Calcium gluconate IV bolus (1-2 gm IV bolus)
   • Calcium gluconate IV gtt – call Endocrine prior to starting gtt!!
   • Magnesium sulfate 2 gm IV (if Mg low; follow with oral Mg)

Hypernatremia/Diabetes Insipidus – See Renal Section

Key points:
   • Must have elevated serum Osm to be diagnosed with DI. If serum Osm is high normal and DI is suspected, confirm diagnosis with a water deprivation test; call Endocrine.
   • Differentiate between central and nephrogenic
Monitor I/Os carefully and ensure pt has access to free water

Hyponatremia/SIADH – See Renal Section, One Section

Key points:
- Patient should be euvolemic even though they have excessive Total Body Water
- Should have serum Osm < 280 mOsm/kg and urine Osm > 100 mOsm/kg
- Look for underlying cause of SIADH and exclude pituitary, adrenal and thyroid dysfunction before diagnosing

Appendix A. DKA Treatment Protocol

** Protocol for the management of adult patients with DKA. *DKA diagnostic criteria: serum glucose >250 mg/dl, arterial pH <7.3, serum bicarbonate <18 mEq/l, and moderate ketonuria or ketonemia. Normal laboratory values vary; check local lab normal ranges for all electrolytes.**

Complete blood count with differential, urinalysis, serum glucose, BUN, electrolytes, chemistry profile, and creatinine levels STAT. Obtain electrocardiogram, chest X-ray, and specimens for bacterial cultures, as needed. *Serum Na⁺ should be corrected for hyperglycemia (for each 100 mg/dl glucose >100 mg/dl, add 1.6 mEq to sodium value for corrected serum sodium value).

Appendix B. HHS Treatment Protocol

** Protocol for the management of adult patients with HHS. HHS diagnostic criteria: serum glucose >600 mg/dl, arterial pH >7.3, serum bicarbonate >15 mEq/l, and minimal ketonuria and ketonemia. Normal laboratory values vary; check local lab normal ranges for all electrolytes. After history and physical exam, obtain capillary glucose and serum or urine ketones (nitroprusside method). Begin 1 liter of 0.9% NaCl over 1 h and draw arterial blood gases, complete blood count with differential, urinalysis, serum glucose, BUN, electrolytes, chemistry profile and creatinine levels STAT. Obtain electrocardiogram, chest X-ray, and specimens for bacterial cultures, as needed. Adapted from ref. 1. *Serum Na⁺ should be corrected for hyperglycemia (for each 100 mg/dl glucose >100 mg/dl, add 1.6 mEq to sodium value for corrected serum sodium value).

### Appendix C. Scoring system for thyroid storm. Only for use in hyperthyroidism

#### Diagnostic criteria for thyroid storm

<table>
<thead>
<tr>
<th>Diagnostic parameters</th>
<th>Scoring points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thermoregulatory dysfunction</td>
<td></td>
</tr>
<tr>
<td><strong>Temperature</strong></td>
<td></td>
</tr>
<tr>
<td>99.9–99.9</td>
<td>5</td>
</tr>
<tr>
<td>100–100.9</td>
<td>10</td>
</tr>
<tr>
<td>101–101.9</td>
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<tr>
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<td>20</td>
</tr>
<tr>
<td>103–103.9</td>
<td>25</td>
</tr>
<tr>
<td>≥ 104.0</td>
<td>30</td>
</tr>
<tr>
<td>Central nervous system effects</td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>0</td>
</tr>
<tr>
<td>Mild (agitation)</td>
<td>10</td>
</tr>
<tr>
<td>Moderate (delirium, psychosis, extreme lethargy)</td>
<td>20</td>
</tr>
<tr>
<td>Severe (seizures, coma)</td>
<td>30</td>
</tr>
<tr>
<td>Gastrointestinal-hepatic dysfunction</td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>0</td>
</tr>
<tr>
<td>Moderate (diarrhea, nausea/vomiting, abdominal pain)</td>
<td>10</td>
</tr>
<tr>
<td>Severe (unexplained jaundice)</td>
<td>20</td>
</tr>
<tr>
<td>Cardiovascular dysfunction</td>
<td></td>
</tr>
<tr>
<td><strong>Tachycardia (beats/minute)</strong></td>
<td></td>
</tr>
<tr>
<td>90–109</td>
<td>5</td>
</tr>
<tr>
<td>110–119</td>
<td>10</td>
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<tr>
<td>120–129</td>
<td>15</td>
</tr>
<tr>
<td>≥ 140</td>
<td>25</td>
</tr>
<tr>
<td><strong>Congestive heart failure</strong></td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>0</td>
</tr>
<tr>
<td>Mild (pedal edema)</td>
<td>5</td>
</tr>
<tr>
<td>Moderate (bibasilar rales)</td>
<td>10</td>
</tr>
<tr>
<td>Severe (pulmonary edema)</td>
<td>15</td>
</tr>
<tr>
<td><strong>Atrial fibrillation</strong></td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>0</td>
</tr>
<tr>
<td>Present</td>
<td>10</td>
</tr>
<tr>
<td>Precipitating event</td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>0</td>
</tr>
<tr>
<td>Present</td>
<td>10</td>
</tr>
</tbody>
</table>

Scoring system: A score of 45 or greater is highly suggestive of thyroid storm; a score of 25–44 is suggestive of impending storm, and a score below 25 is unlikely to represent thyroid storm.


GASTROENTEROLOGY

Gastrointestinal Bleed

**Ddx:** Upper: swallowed blood, gastritis/esophagitis, Mallory-Weiss tear, gastric or esophageal varices, portal hypertensive gastropathy, ulcer, mass, Dieulafoy’s lesion  
Lower: colitis, ischemic bowel, AVM, mass, hemorrhoids

**Key history points:** Acute vs. chronic, number of episodes, quality of emesis or bowel movements, prior bleeding history, risk factors such as liver disease, alcohol use, or NSAID/anticoagulant use

**Physical exam:** Vital signs (first to change, Hct may be normal for 8 to 24 hours), orthostasis ((+) with 20% loss), stigmata of chronic liver disease, abdominal pain/peritonitis, rectal exam

**Nasogastric lavage:** Negative lavage does not rule out upper bleed unless there is bilious return, G-tubes can be used instead if present, lavage is not contraindicated with varices, can be used to monitor for active bleeding. Consider in patients with significant rectal bleeding since a rapid UGIB can present as a LGIB as well.

**Initial orders:**
- Vital signs at least every 4 hours, initial orthostatic VS
- NPO
- Insert two large bore IV’s (at least 18 gauge)
- Hct: repeat on assessment and every 4 hours
- PTT/PT/INR
- BUN: suggestive of GIB if elevated in absence of renal disease
- Type and cross
- Platelet count
- ECG: especially if history of cardiac disease
- IVF: NS or LR
- Nasogastric tube: lavage until clear, low intermittent suction
- Consult gastroenterology

**Special Treatments:**

**Coagulopathy:** Correct with FFP or vitamin K for goal INR < 1.5, platelets for goal > 50

**Variceal bleed:** Octreotide 50 μg IV bolus followed by 50 μg/hr continuous infusion for 48-72 hr, endoscopy, balloon tamponade or TIPS if endoscopy fails

**PUD:** Start with high dose PPI (80 mg IV bolus then 8mg/hr), endoscopy

**Antibiotic prophylaxis:** Reduces mortality as well as bacteremia, pneumonia, SBP, and UTI’s in cirrhotics with UGIB. Start with IV therapy daily (e.g., ceftriaxone) and then transition to oral twice-daily norfloxacin or ciprofloxacin once GI bleeding is stabilized. Treat for a total of 7 days.

**Prokinetics:** Consider erythromycin 250 mg IV prior to endoscopy to improve visualization

**Evaluation of Ascites**

- 72 -
Ddx of ascites by serum-ascites albumin gradient (SAAG):
(SAAG = serum albumin – ascites albumin)

<table>
<thead>
<tr>
<th>High gradient/portal HTN (&gt; 1.1 g/dl)</th>
<th>Low gradient (&lt; 1.1 g/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous bacterial peritonitis</td>
<td>Peritonitis from TB or rupture</td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>Peritoneal carcinomatosis</td>
</tr>
<tr>
<td>Alcoholic hepatitis</td>
<td>Protein-losing enteropathy</td>
</tr>
<tr>
<td>Cardiac ascites</td>
<td>Nephrotic syndrome</td>
</tr>
<tr>
<td>“Mixed” ascites</td>
<td>Bowel obstruction or infarct</td>
</tr>
<tr>
<td>Fulminant hepatic failure</td>
<td>Pancreatic ascites</td>
</tr>
<tr>
<td>Budd-Chiari syndrome</td>
<td>Biliary ascites</td>
</tr>
<tr>
<td>Portal vein thrombosis</td>
<td>Postoperative lymphatic leak</td>
</tr>
<tr>
<td>Veno-occlusive disease</td>
<td>Serositis (connective tissue diseases)</td>
</tr>
<tr>
<td>Myxedema</td>
<td></td>
</tr>
<tr>
<td>Fatty liver of pregnancy</td>
<td></td>
</tr>
</tbody>
</table>

Paracentesis:

- Indications: Obtaining fluid for analysis
- Decreasing respiratory or abdominal distress
- Contraindications: Bowel obstruction or distension
- Significant coagulopathy

Please note that paracenteses can be performed in the vast majority of patients with ESLD despite elevated INR’s and thrombocytopenia; transfusion of blood product to reverse the coagulopathy before the procedure is not recommended in these patients.

Complications: secondary bacterial peritonitis, hemorrhage, abdominal wall hematoma, ascitic fluid leak, bowel perforation, hypotension, renal failure, bladder perforation

Orders:
- Gram stain
- Aerobic and anaerobic cultures
- Cell count and differential
- Albumin
- Total protein (If SAAG > 1.1, AFTP > 2.5 c/w cardiac ascites, > 2.5 c/w cirrhosis)
- +/- glucose, AFB, cytology, triglycerides, bilirubin

Adjustment for traumatic tap:
- Subtract 1 WBC per 750 RBC
- Subtract 1 PMN per 250 RBC

- Albumin for large volume paracentesis: none if <5 liters, if >5 liters is to be removed, give 50 gm (25% Solution)

Spontaneous Bacterial Peritonitis (SBP)
- Ascitic fluid PMN count > 250 cells/mm³:
  - Empiric antibiotic therapy (e.g., cefotaxime 2g IV q8h)
  - Albumin infusion 1.5gm/kg body weight initially and 1.0 gm/kg on day 3
- Ascitic fluid PMN count < 250 cells/mm³ with signs/symptoms of infection:
Empiric antibiotic therapy while awaiting cultures results
- Can hold on albumin reasonably in this circumstance
  - After treatment for SBP, patient will require life-long prophylaxis

---

**GERIATRICS**

1. **Guiding principles:**
   - Less is more (*i.e.*, risk/benefit ratio as you age)
   - Evaluate function first (ADLs and IADLs)
   - The problem is often a side effect of a medication
   - With delirium think of infection and medications as the most likely causes in hospitalized patients
   - Geriatric syndromes (falls, incontinence, weight loss) are generally multifactorial

2. **Medications to avoid in the elderly:**
   - Beers List Website: [http://www.dcri.duke.edu/ccge/curtis/beers.html](http://www.dcri.duke.edu/ccge/curtis/beers.html)
   - Benzodiazepines/high dose sleepers
   - Diphenhydramine (Benadryl)
   - Amitriptyline
   - Metoclopramide (Reglan)
   - Ketorolac (Toradol)/Indomethacin (and other NSAIDs)

3. **Sundowning and agitation:**
   - Reorientation is important!
   - Low dose haloperidol, 0.5-1 mg IV is a helpful adjunct
   - Think of alcohol (or BZD) withdrawal - don’t assume that old folks don’t drink
   - Look at the medication list and consider infection (especially UTI)

4. **Medicare:**
   - Eligibility: ≥65, ESRD, ALS, disabled, > 40 quarters of medical-eligible work by person or spouse, permanent resident
   - Part A (automatic, do not need to apply): Covers hospital, SNF, hospice (does not pay room and board; Medicare pays for care and meds), some home health care.
   - Part B (must apply, optional, costs extra): Covers 80% of doctor visits, home health care, other services.
   - Part C: Medicare HMOs (alternative to Parts A & B).
   - Part D: Prescription drug plan. Automatic, but benefits are only available through private companies at prices of $20 to $50 per month or through Medicare HMO.

5. **Nursing Homes:**
   - Two types of care: skilled/subacute rehab or custodial/long term care
   - Skilled care paid by Medicare and requires a 3 day hospital stay for them to pay
   - Custodial care is for patients who still need nursing care but are no longer able to rehab. Payer is predominantly self-pay or Medicaid

- 74 -
- If patient has assets, patient (or spouse) must pay until poor enough to qualify for Medicaid. This is termed “spending down.”
- If Medicaid pays, they take the resident’s social security check

“Cost of Aging ... Who Pays.” Susan T Bray-Hall, M.D.

HEMATOLOGY/ONCOLOGY

Anemia

Anemia is a common problem that you will be called with frequently. It is best to develop a systematic way of responding to and evaluating the causes of anemia.

One helpful mental image for anemia is to liken it to a child splashing around in a bathtub. The reason the tub isn’t full (anemia) could be one of three broad categories:

1) the faucet is off and no water is going in (decreased production)
2) the child is splashing all the water out (destruction/hemolysis)
3) the plug in the bottom of the tub has been pulled out (blood loss)

In all cases it is correct to consider each of these three categories when evaluating a low hematocrit, taking into account the historical information you know about the patient to help guide your thought process.

Common causes of anemia by Production/Destruction/Loss categories

<table>
<thead>
<tr>
<th>Cause to Consider</th>
<th>Associated Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Decreased Production</strong></td>
<td><strong>Substrate Deficiencies</strong></td>
</tr>
<tr>
<td>Cancers</td>
<td>Iron</td>
</tr>
<tr>
<td>Myeloproliferative Disorders</td>
<td>B12</td>
</tr>
<tr>
<td>Chronic Inflammatory Dis.</td>
<td>folate (rare but possible)</td>
</tr>
<tr>
<td>Acquired Intrinsic</td>
<td>Mechanical Trauma</td>
</tr>
<tr>
<td>PNH</td>
<td>TTP/HUS</td>
</tr>
<tr>
<td>liver disease</td>
<td>eclampsia</td>
</tr>
<tr>
<td>vitamin E deficiency</td>
<td>malignant HTN</td>
</tr>
<tr>
<td>Hereditary Intrinsic</td>
<td>cardiac valves</td>
</tr>
<tr>
<td>thalassemias</td>
<td>severe burn patients</td>
</tr>
<tr>
<td>spherocytosis</td>
<td>“march trauma”</td>
</tr>
<tr>
<td>G6PD</td>
<td>Other Infections</td>
</tr>
<tr>
<td>Warm Antibody Mediated</td>
<td>sepsis of any cause</td>
</tr>
<tr>
<td>HIV</td>
<td>acute viral infections</td>
</tr>
<tr>
<td>EBV</td>
<td>syphilis</td>
</tr>
<tr>
<td>SLE</td>
<td>malaria</td>
</tr>
<tr>
<td>idiopathic (50-70%)</td>
<td>babesiosis</td>
</tr>
<tr>
<td>Cold Antibody Mediated</td>
<td>clostridial infections</td>
</tr>
<tr>
<td>mycoplasma</td>
<td>Other</td>
</tr>
<tr>
<td>EBV</td>
<td>splenomegaly</td>
</tr>
<tr>
<td>lymphoproliferative d/o</td>
<td>hypophosphatemia</td>
</tr>
<tr>
<td>Drugs</td>
<td>transfusion reactions</td>
</tr>
<tr>
<td>PCN, cephalosporin, sulfa</td>
<td>snake/spider bite</td>
</tr>
<tr>
<td>NSAIDs</td>
<td></td>
</tr>
</tbody>
</table>

- 75 -
TCA’s, phenothiazines  
thiazides  
sulfonylureas

<table>
<thead>
<tr>
<th>Blood Loss</th>
<th>GI bleeding</th>
<th>repeated phlebotomy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GU bleeding</td>
<td>menstruation</td>
</tr>
<tr>
<td></td>
<td>trauma</td>
<td>pregnancy</td>
</tr>
<tr>
<td></td>
<td>surgery</td>
<td></td>
</tr>
</tbody>
</table>

**Common causes of anemia by MCV**

- **Microcytic (MCV < 80):** for w/u order ferritin, TIBC, smear, reticulocyte count.
  1. Iron Deficiency: ferritin is low (usually < 30), TIBC is high. But, ferritin is an acute phase reactant and may be elevated in inflammatory states. In early stages, MCV is also normal.
- **Normocytic (MCV 80 –100):** check reticulocyte count.
  1. Decreased reticulocyte count: aplastic anemia/red cell aplasia, myelophthisis, MDS, anemia of chronic disease, iron deficiency, chronic kidney disease, hypersplenism, hypothyroidism, multiple myeloma, HIV/AIDS, Mixed (e.g. iron deficiency with B12 deficiency).
  2. Increased reticulocyte count: acute blood loss, hemolysis.
- **Macrocytic (MCV >100)**
  2. Non-megaloblastic: alcohol, liver disease, hypothyroidism, reticulocytosis, aplastic anemia, MDS, drugs (AZT, anticonvulsants).

**Hint:** Hospitalized patients can drop their Hct by 1% per day by blood draws, so be aware.

**Pancytopenia**

**P-** Pernicious anemia/PNH
I- Infection (HCV, EBV, Parvo B19, HIV, leishmaniasis, brucellosis), Infiltrative (lymphoma, myeloma, carcinoma, hairy cell leukemia)
G- Granulomatous diseases (TB, Sarcoid)
H- Hypersplenism (CLL, lymphoma, ESLD)
A- Aplastic Anemia
M- Marrow infiltration/suppression (MDS, myelofibrosis, alcohol, toxins, medications).

**Thrombocytopenia**

1. Common in hospitalized patients and is defined as a platelet count less than 150,000/mcL.
2. The evaluation and management of thrombocytopenia should proceed in parallel when confronted with this problem, as some disorders that cause thrombocytopenia do not cause bleeding (HIT, some forms of DIC), and the transfusion of platelets in these disorders can worsen the clinical course (TTP/HUS/HIT/DIC).
3. Immediate action is necessary when a patient is bleeding, requires an invasive procedure, or the absolute platelet count falls to less than 10,000/mcL.
4. A peripheral smear is the first test that should be reviewed to rule out platelet clumping, or “pseudothrombocytopenia.” If platelet clumping is present, blood should be sent to the lab in a sodium citrate rather than an EDTA tube.
5. A focused physical examination should be directed at the vital signs, ocular fundi, subconjunctival areas, nasal and oral cavity, extremities, and rectum to look for evidence of retinal or subconjunctival hemorrhage, epistaxis, oral or cutaneous petechiae, raised ecchymotic areas at heparin injection sites, and occult blood in stool.
6. Although there is some conflicting data, the presence of fever, physical exam evidence of minor bleeding, and anemia appear to be risk factors for bleeding at a given level of thrombocytopenia. Wet purpura, or mucosal hemorrhage, appears to be a stronger risk factor for clinically serious bleeding than dry purpura, or petechiae and ecchymoses.
7. A brief differential should be considered: DIC, sepsis, HIT, TTP, massive blood transfusion coagulopathy, post-transfusion purpura, pulmonary embolism, and medications.
8. Concurrent anemia and thrombocytopenia as a result of microangiopathic hemolytic anemia occurs in DIC/TTP/HUS.
9. A review of clinical data should focus on the following: reason for DIC? (sepsis, pregnancy complication, trauma, malignancy), vital sign abnormalities/known site of infection to suggest sepsis, recent heparin administration (HIT development requires 5 days of continuous heparin therapy in any form or heparin administration in the prior 100 days with new heparin exposure), predisposing factors for TTP (recent organ transplant, transplant medications, clopidogrel), recent transfusion of any kind, and a scan of the medication list for chemotherapy, antimicrobials (e.g. piperacillin), H2 blockers, and GPIIb/IIIa antagonists.
10. Assessment of the temporal trend in platelet count and its relation to acute illness and medication initiation is helpful in prioritizing the differential diagnosis.
   - Discontinue medications that inhibit platelet function such as aspirin, NSAIDs, thienopyridines, GPIIb/IIIa antagonists. Ancillary laboratory studies that should be obtained include an INR, PTT, full CBC, fibrinogen level, peripheral smear if not done initially, and LDH.
11. A patient with HIT will require discontinuation of heparin and initiation of alternative anticoagulation (lepirudin, bivalirudin). This disorder can be difficult to diagnose definitively in the acute setting and typically hematologic consultation is necessary to assist in management.
12. Patients with TTP/HUS benefit from rapid institution of plasmapheresis.
13. There is no consensus regarding management of DIC, although treatment of the underlying disorder appears to be effective in most cases.
14. Drug-related thrombocytopenia requires discontinuation of the medication.
15. Thrombocytopenia related to sepsis typically resolves with treatment of the underlying infection.
16. A safe absolute platelet count has not been defined for many of the invasive procedures we perform on hospitalized patients including central venous catheterization, lumbar puncture, thoracentesis, paracentesis, and arthrocentesis. A good general dictum is to raise the platelet count to greater than 30,000/mL for medical procedures and to greater than 50,000/mL for surgical procedures.
17. Randomized clinical trials support the practice of withholding platelet transfusion in stable oncology patients receiving chemotherapy until the platelet count falls below 10,000/mcL.
18. When to transfuse a patient with ITP is controversial but most experts would use 5-10,000/mcL as the cutoff.
19. For more complicated patients, safe platelet count cutoffs and therefore transfusion triggers are less well defined. Expert opinion currently recommends a primary prophylactic threshold of 20,000/mcL and a threshold of 50,000/mcL for those with active bleeding or prior to invasion.
20. Recent studies show that only 50% of ICU patients with a variety of disorders will have an appropriate rise in the platelet count post transfusion, and therefore the risks and benefits in this population need to be weighed carefully in the absence of a clear evidence-based strategy.
21. Platelets can be given as pooled platelet packs from multiple donors or as apheresis units from a single donor. Some hospitals use only apheresis units while others use either six or ten unit platelet packs, with variable numbers of actual platelets administered.
22. Dosing of platelets is entirely empiric, and a reasonable practice is to give an apheresis unit or platelet pack and recheck the peripheral platelet count 1-4 hours later to determine whether more units are required to achieve the desired platelet count. In general, an appropriate response to platelet transfusion is an increase in the post transfusion count of between 10-50,000/mcL.

**Fever & Neutropenia “Hot ‘n Low”**

Fever in the neutropenic cancer patient is one of the most common oncologic emergencies. If left untreated, febrile neutropenia has a mortality rate as high as 70% at 48 hours. In the early days of chemotherapy use, infections were responsible for 75% of the treatment-related mortality. Aggressive cytotoxic therapy such as induction chemotherapy for leukemia has been made possible in large part by the availability of effective broad-spectrum antibiotics.

**Definitions**
- Neutropenia: Granulocyte count (polys + bands) < 500 or <1000 and expected to decline to <500
- Fever: single temperature >101°F or >/= 100.4 F for at least an hour
- 60% of patients with F+N are actually infected
- 16-20% with counts <100 are bacteremic

**Sources of infection**
- Endogenous: translocation from GI tract due to mucosal damage from chemotherapy
- Catheter-related
- Environmental: food, water, air
- Skin! Look everywhere! Check peri-rectal area but DO NOT DO rectal exam

**Evaluation**
History: S+Sx of infection - classic signs of infection may be absent or subtle
Afebrile patients with signs or symptoms compatible with infection should be treated empirically
Estimate length of neutropenia: what kind of chemotherapy and how long ago?
Most common sites: gums, pharynx, esophagus, lung, perineum and anus, skin, catheter sites
Avoid rectal examination, suppository use
Blood cultures from all catheter ports and one set from periphery, UA, culture other sites as indicated
CBC with diff, LFTs, lytes, CXR

Treatement

Begin therapy within 4 hours of fever spike
Empiric therapy: there are multiple effective regimens as seen below. Monotherapy is cheaper, safer and as effective as dual therapy in uncomplicated F + N. Dual therapy offers synergy against GNR and may be preferred in sicker patients or those with focal infections requiring broader coverage (pneumonia).
Regimen must always include anti-pseudomonal coverage
Consider need for gram + coverage (see criteria below)
Remember to consider allergies, renal function and dose adjust

Initial Empiric Therapy of Fever/Neutropenia (Clinical Infectious Diseases 2002; 34:730-751)

Commonly used regimens:
- Monotherapy: Cefepime, Imipenem, Meropenem. These all have excellent gram-negative coverage as well as coverage of S. viridans, S. pneumo and Staph. Ceftazidime can be used but has no gram-positive coverage.
- Dual therapy: Aminoglycoside or quinolone + one of the agents above.
- Add vancomycin if needed to any of the above combinations if criteria are met.
If Afebrile
If source found: adjust abx as needed
If no source found:
- if low risk switch to oral therapy (cipro + amox/clavulanate) and consider discharge
- if high risk, continue antibiotics
- All have excellent gram-negative coverage as well as coverage of S. viridans, S. pneumo and Staph. Ceftazidime can be used but has no gram-positive coverage.
- Dual therapy: Aminoglycoside or quinolone + one of the agents above.
- Add vancomycin if needed to any of the above combinations if criteria are met.

If Febrile
Continue initial antibiotics
- Panculture, CXR
- Examine lines, low threshold to change
- Consider adding vancomycin for empiric line coverage

If still febrile on day 4-5
- Repeat cultures, CXR
- If progressive disease, change antibiotics
- Consider adding empiric fungal coverage (particularly for high risk—see risk assessment above)

Duration of therapy
If afebrile by day 3:
- ANC >500 for 2 consecutive days stop abx 48 hours after afebrile and ANC > 500
- ANC <500 by day 7:
  - Low risk: stop abx when afebrile for 5-7 days
  - High risk: continue abx
If persistent fever:
- ANC >500 stop after 4-5 days and reassess
- ANC < 500: continue for 2 weeks and reassess. Stop if no disease sites identified.

(Clinical Infectious Diseases 2002; 34:730-751)
Empiric Therapy of Fever and Neutropenia

Initial Questions:
Is patient high risk?
- High risk: ANC < 100, evidence of focal infection, inpatient when F + N develops, outpatient with serious coexisting medical condition, clinically stable outpatient with uncontrolled cancer, neutropenia expected > 10 days
- Low risk: well appearing, no focal evidence of infection, no co morbidities, duration of neutropenia < 7 days, etc. Two recent RCT have shown oral cipro/amoxicillin-clavulanic acid is as safe and effective as IV antibiotics in selected low-risk inpatients. Outpatient therapy with vigilant observation may be an option in selected patients, but this is not yet widely practiced.

Is vancomycin needed?
- Indications include: cellulitis, severe mucositis, quinolone prophylaxis, colonized with MRSA, obvious catheter infection, hypotension/sepsis, pneumonia

Consider anaerobic coverage for:
- Gingivitis, rectal abscess, typhlitis

Use of GCSF
- Shortened duration of neutropenia and length of hospital stay and cost savings in some studies
- No effect on mortality
- Consider if:
  Worsening of course because of prolonged neutropenia expected: pneumonia, hypotension, severe cellulitis, sinusitis, systemic fungal infection, multi-organ dysfunction due to sepsis

Antibiotic prophylaxis
- Routine prophylaxis not recommended due to concern for resistance
- Consider if: pt expected to be profoundly neutropenic (<100), significant lesions to mucous membranes (periodontal disease, mucositis), catheters, etc.
- Bactrim and quinolone prophylaxis well studied: reduce infection rate but not necessarily mortality
- Goal: treat only high risk patients for as short a time as possible
- Antiviral prophylaxis with acyclovir or ganciclovir and antifungal prophylaxis with fluconazole are indicated in patients undergoing allogeneic BMT.
**Tumor Lysis Syndrome**

The tumor lysis syndrome is an acute metabolic emergency resulting from massive cell lysis caused by rapid cell turnover or rapid response to cytotoxic therapy. Cell lysis leads to hyperkalemia, hyperuricemia and hyperphosphatemia. Hormonal and renal regulatory mechanisms as well as phosphate binding cause serum calcium to fall. Hypoglycemia, hyponatremia, adrenal failure and lactic acidosis can also be seen. The syndrome can develop spontaneously in patients with acute leukemia or aggressive lymphoma (Burkitt’s), but is usually precipitated by treatment of hematologic malignancies in which there is a large tumor burden. It can occur during treatment of some solid tumors, although much less commonly.

Major dangers are:

- Acute renal failure, which can be caused by uric acid nephropathy or nephrocalcinosis
- Prior to allopurinol, 10% of patients treated for acute leukemia got uric acid nephropathy
- Now hyperphosphatemia causing calcium-phosphate uropathy is more common
- Cardiac arrhythmias from hyperkalemia or hypocalcemia

**Management:**

**Prophylaxis**

Pre-treat for at least 2 days before chemotherapy with:

- Allopurinol or rasburicase
- Aggressive hydration (with NS) to maintain urine output > 2.5L/day
- Alkalization is no longer recommended as it can enhance calcium/phosphate deposition
- Do not begin therapy until Cr <1.6 and UA < 8.0 (sometimes dialysis is needed before proceeding with therapy.)
- Do not treat hypocalcemia unless pt is symptomatic (Increased risk of CaPO4 ppt).

Once therapy has begun

- Check electrolytes, Ca, Mg, Po4 BID until stable
- Use PRN:
  - Phosphate binders: lower serum phos either with calcium carbonate (taken with meals) or aluminum based binders (amphogel, alternagel) if calcium-phosphorus product >60
  - Kayexalate
  - Dialysis

**Spinal Cord Compression**

- Prevalence: 5-10% of cancer patients and in 10% of these it is the presenting complaint
- Most common causes: lung, prostate, breast, multiple myeloma
- Most common sites: thoracic spine (70%), LS spine (20%), cervical (10%).
- Multiple sites common in breast, prostate; lung almost always a single site.
- Classification:
  - Epidural spinal cord compression (most common)
  - Vertebral body metastasis, paravertebral soft tissue mass, epidural mets
  - Epidural cauda equina compression
  - Leptomeningeal mets
  - Intramedullary mets
Presentation:
- 96% have pain as first symptom
- Loss of bowel/bladder function, motor weakness, sensory deficits are late findings
- Localized back pain worse with movement and also with lying supine (unlike disc disease)
- Usually present for months before neurologic symptoms appear
- Can have radicular pain but less common: unilateral or bilateral

Examination
- Palpate vertebrae, flex and extend cervical/lumbar spine
- Thorough neurologic examination including rectal tone and perineal sensation may localize lesion
- Can check post-void residual. If PVR > 150cc suspect bladder dysfunction.

Example algorithm for work-up of back pain in patients with cancer:
- Radicular pain only, normal neurologic exam → MRI within 24h
- Pain + radiculopathy → Admit, MRI within 24h then LP
- Brachial plexopathy → MRI, consider admission. If paraspinal mass, dexamethasone and XRT
- Pelvic plexopathy → MRI or CT, consider admission (if cannot walk), LP. If paraspinal mass, dexamethasone and XRT
- Myelopathy (upper motor neuron signs) → MRI, admit, dexamethasone, XRT

Treatment
- Pain control: narcotics plus NSAIDS, consider gabapentin/amitriptyline for neuropathic pain, pamidronate.
- Corticosteroids: help decrease cord edema, pain, preserve neurologic function, improve overall outcome.
- If high grade lesion and neurologic signs of compression consider high-dose therapy (one well-done RCT showed that 81% remain ambulatory vs., 63% with lower doses) 100 mg IV bolus then 24 mg QID x 3 days then taper over 10 days. Significant complication rate from steroids- discuss with an attending before using high-dose.
- If low grade lesion or not appropriate for high dose, use 10mg IV bolus then 4mg PO QID tapered over 14 days.
- Radiation therapy: equal success rate to surgery with similar duration of improvement.
- Indications for surgery (laminectomy): to establish diagnosis, progression despite radiation therapy, vertebral instability- recent data suggest early surgical intervention superior
- Breast, prostate, myeloma, lymphoma have 70-88% chance of responding to radiation alone with 70-85% resolution of back pain.

Prognosis
- Depends on primary tumor type
- If patients treated while still ambulatory, there is an 89-94% chance of remaining ambulatory
- If paraplegic, 10% chance of regaining ambulation
**Brain Metastasis**

- 25% of patients with cancer die with intracranial mets
- Lung, breast, melanoma most common
- Melanoma, germ cell, renal cell most likely to hemorrhage
- DDx of mass lesion: metastasis, intracerebral hemorrhage, brain abscess, hydrocephalus, subdural hematoma, radiation necrosis
- Location: multiple mets (50%) cerebral hemispheres (37%), frontal (12%) posterior fossa (14%)

**Presentation**

- Focal signs (hemiparesis, hemisensory deficit, aphasia, etc) 50%
- Headache 33%
- Mental status change 22%
- Seizure 20%
- Hemorrhage into tumor: sudden seizure or focal neurologic symptoms followed by headache
- Herniation syndromes

**Examination**

- Thorough neurologic exam including fundoscopy and gait evaluation.

**Work-up**

- CT and MRI are equally effective
- CT with contrast has higher yield
- If single metastasis or no lesions on CT, obtain MRI with gadolinium
- MRI more sensitive for small mets and better for visualizing posterior fossa

**Treatment**

- If symptomatic, treat with corticosteroids to lessen brain edema (see section on cord compression)
- If multiple mets: whole brain radiation
- If a single met and controlled extracranial disease can consider surgical excision prior to whole-brain radiation.
- Selected patients with hydrocephalus may benefit from a shunt
- If patient presents with seizure start phenytoin
- Patients with no history of seizure have a 10% chance of developing one. Prophylactic anticonvulsants considered for high-risk patients (frontal mets, focal hemispheric symptoms) but the data does not support prophylaxis.

**Hypercalcemia**

- Most common paraneoplastic syndrome. Prevalence is 10% in patients with advanced cancer.
- Responsible for 40% of all hypercalcemia
- Lung, breast, head and neck, renal and multiple myeloma most common causes

**Etiology**

- Humoral hypercalcemia of malignancy (HHM) 80% of cases
- Squamous cell lung ca, H+N ca, renal cell
- PTHrP produced by tumor has homology with parathyroid hormone.
Causes hypercalcemia by increased bone resorption, decreased bone formation, increased renal tubular resorption of Ca, increased excretion of phosphate, and increased urinary cyclic AMP.
- Lymphoma can rarely produce vitamin D
- Local osteolytic hypercalcemia (LOH) 20% of cases
- Myeloma, leukemia, lymphoma, breast and other solid tumors
- Caused by local production of hormones, cytokines which activate osteoclasts causing bone resorption
- In patients with breast cancer 50% have HHM and 50% have LOH

**Presentation**
- Malaise, fatigue, confusion, anorexia, bone pain, polyuria, polydipsia, weakness, constipation, nausea, vomiting
- Severe hypercalcemia: confusion, lethargy, coma, and death.

### Etiology

<table>
<thead>
<tr>
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<th>Calcium</th>
<th>Phos</th>
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</thead>
<tbody>
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<td>High or normal</td>
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<td>Low or normal</td>
<td>Low to normal</td>
</tr>
<tr>
<td>Local osteolytic Hypercalcemia</td>
<td>High</td>
<td>Normal</td>
<td>Low or normal</td>
<td>Low or normal</td>
</tr>
</tbody>
</table>

**Diagnosis**
- Check Ca, albumin, renal function, PO4, alk phos, EKG
- R/o bony mets
- Normal PTH and low PO4 suggest HHM
- Normal PTHrP, normal PO4 in presence of bone mets suggests LOH
- Check PTH prior to treatment (on admission).

**Treatment**
- Moderate (12-13.5 mg/dL)
  - IV NS for hydration-at least 2-4 liters/day
  - Once euvoletic begin furosemide to prevent volume overload- only if able to carefully match I/O’s
  - Pamidronate 60-90mg IV over 2 hours (or Zolendronate-more effective, more expensive)
  - This therapy reduces Ca to normal in 80% by 7 days
  - If LOH, glucocorticoids may inhibit bone resorption and cytokine production
  - Severe (>13.5 mg/dL)
  - In addition to above add calcitonin 4-8 U/kg SQ qid
  - If refractory, gallium 200mg/meter squared in 1 liter IV QD x 5 d-careful if AKI
  - Plicamycin can be considered (toxic to liver, kidney, BM)

**Prognosis**
- Median survival of patients with hypercalcemia only 1-3 months
- Treatment may not be indicated if patient has end-stage disease unless it will improve quality of life
SIADH

Definition
- Euvolemic hypotonic hyponatremia. Serum Na < 135, serum Osm < 280, urine Na > 20 urine Osm > 100

Etiology
- Production of ectopic ADH by tumor cells
- Associated with many tumors. 38% of patients with small cell lung ca (this group has a poor prognosis)
- Cytotoxic drugs: vincristine, cyclophosphamide, cisplatin, ifosfamide, levamisole, melphalan
- Other drugs: SSRIs, antineuroleptics, amitriptyline, carbamazepine etc.
- High dose cyclophosphamide is a common cause of SIADH because:
  - Drug enhances ADH release and effect.
  - Patients receive large amounts of IVF to prevent hemorrhagic cystitis
  - Nausea can also stimulate ADH release
- Other DDx: Renal, adrenal, thyroid insufficiency, CNS disturbance, major surgery

Presentation
- Most often asymptomatic
- Anorexia, depression, lethargy, irritability, confusion, muscle weakness, marked personality change.
- If Na < 110: seizure, areflexia, pseudobulbar palsy, coma, and death.

Treatment
- Treat underlying disease/remove offending drug
- Asymptomatic hyponatremia
- Fluid restriction alone may be enough (1-1.5L day)
- If patient requires IVF switch all IVF to NS and consider lasix to keep I = O
- Liberalize salt in diet
- Correct hypokalemia if present

If hyponatremia persists/worsens consider:
- Lasix and salt tablets
- Demeclocycline or lithium (decrease collecting tubule response to ADH)-watch for renal toxicity
- Urea 30g day to increase solute excretion (if above doesn’t work)

Symptomatic or severe hyponatremia
- If mental status change, NA < 115 in consider 3%NS, correcting ½ of total Na deficit over 24h no faster than 0.5meq/L/h. More rapid repletion occasionally indicated with severe cases.
- Patients with mild chronic SIADH who do not respond to therapy may have reset osmostat and do not need further treatment
Superior Vena Cava Syndrome

Definition
- SVC obstruction can result from compression, obstruction, or thrombosis of the SVC. Onset may be rapid. Patients may have a significant degree of discomfort, fear and distress.
- An increasingly common cause of SVC-like syndrome is thrombosis of the SVC in patients with central venous catheters.
- Other causes: granulomatous infections, goiter, aortic aneurysms, and fibrosing mediastinitis

Etiology
- Neoplastic causes include bronchogenic, lymphoma, leukemia, and germ cell tumors.
- 70% are due to lung cancers

Presentation
- Dyspnea, orthopnea, swelling of the face, limbs, and upper extremities, venous dilatation over the anterior chest wall, cough +/- hemoptysis, chest pain.
- Jugular venous distention, facial plethora, and cyanosis may be present.
- Hoarseness, syncope, dizziness, and confusion may also be present.

Evaluation
- CXR abnormal in >80% of cases. Widened superior mediastinum and pleural effusions.
- CT scan with IV contrast- shows reduced or absent opacification of central venous structures, with prominent collateral venous circulation.
- No advantage of MRI over CT
- Biopsy of the mass should be made if diagnosis or tumor type is unknown. Sputum cytology, biopsy of lymph nodes, bronchoscopy, and thoracentesis may also be helpful for diagnosis of tumor type.

Treatment
- Glucocorticoids- Dexamethasone 4mg q6h may be helpful, esp. if lymphoma or thymoma
- Elevate head of bed
- Oxygen
- Loop diuretics and low-salt diet
- Radiation therapy is useful for radiosensitive tumors, e.g. NSCLC
- Chemotherapy is useful for chemosensitive tumors, e.g. SCLC, Non-Hodgkin’s lymphoma
- Remove any indwelling central venous catheters that may be causing or contributing
- Other: Placement of intravascular stent, surgery

BLOOD PRODUCTS

PACKED RED BLOOD CELLS
Indications for transfusion:
- Symptomatic anemia in a normovolemic patient regardless of H/H
- Acute blood loss w/ evidence of inadequate O2 delivery
- Hemoglobin ≤7 in a hemodynamically stable hospitalized patient
- Post-operative hemoglobin of <8
- Consider transfusion for hemoglobin ≤8 in patients with CAD

Facts:
- Each unit pRBC has volume ~300ml (200ml of red cells) and should raise hemoglobin by 1g/dL and hematocrit by 3% unless active bleeding
- Each unit of pRBC contains ~200mg iron; hemosiderosis can produce organ damage when total iron load approaches 15-20grams (75-100 units)
- Loss of 1 unit of blood causes ~1.5 mEq K, but transfusion of 1 unit causes +10 mEq since K leaks out of red cells in stored blood; monitor in CKD and massive transfusion
- Citrate present in stored blood can result in metabolic alkalosis (citrate–bicarb). Ionized calcium may be decreased from calcium+citrate complexing

PLATELETS

Indications for transfusion of platelets:
- Platelet count of <10 in non-bleeding patient w/ marrow suppression; consider higher threshold (<30,000) for patients who are febrile/septic
- <50,000 if actively bleeding; <100,000 if CNS bleed
- <50,000 before surgery; <100K before ocular or CNS procedure
- <20,000 for most bedside procedures

Facts:
- One apheresis unit of platelets is equivalent to 4-6 pooled donor units; most hospitals use this type of product
- One apheresis unit should raise platelet count by 30K within 1 hour
- Platelets do not require ABO matching, but may have a more robust platelet response with matched units
- True platelet transfusion refractoriness is defined as increment of <10K on two or more occasions; HLA antibodies may contribute, so HLA matched units may be helpful

FRESH FROZEN PLASMA
Plasma containing all coagulation factors; frozen within 8 hours of collection

Indications for transfusion of FFP:
- Active bleeding in the setting of an INR >1.5 (includes in the setting of liver disease, DIC, warfarin overdose, vitamin K deficiency)
- INR>2 in non-bleeding patient scheduled for surgery or invasive procedure
- TTP (plasma exchange is preferred)

Facts:
- Dose is generally 10-15mL/kg (usually about 3-5 units)
- FFP can have INR as high as1.3, and generally doesn’t correct the INR to below 1.6
- One unit of FFP raises coagulation factor levels by ~8% and fibrinogen by ~13
CRYOPRECIPITATE
FFP that is thawed at 4 degrees C leading to separation of fibrinogen, Factor VIII, Factor XIII, and vWF.

□ Indications for transfusion of cryoprecipitate:
● Fibrinogen < 100 mg/dl in the setting of consumptive coagulopathy (DIC) or severe bleed

Facts:
● 10 units of cryo contain ~2g of fibrinogen and will raise the fibrinogen level by 1 point per kg of patient weight
● Cryo is no longer a standard therapy for Factor VIII deficiency hemophilia or VWD given availability of more concentrated factor products

DIFFERENT PREPARATIONS:

Leukoreduced blood components – Removal of leukocytes through filtration to <10⁶. Done to prevent complications of PRBC transfusion related to WBCs (transfusion reaction, CMV infection). Most hospitals automatically do this for PRBCs now.

□ Indications for leukoreduced product:
● Chronically transfused patients (HLA sensitization)
● Potential or previous transplant recipients
● Patients with previous febrile non-hemolytic transfusion reactions
● CMV seronegative at-risk patients (b/c CMV lives in white cells)

Irradiated blood components - Red cells that are exposed to at least 2500cGy of Gamma radiation to destroy the ability of T lymphocytes to divide. Done to avoid transfusion-associated GVHD.

□ Indications for irradiated product:
● Severe congenital immune deficiency
● Immunocompromised transplant recipients (especially BMT)
● Transfusions from family members
● CLL or other patients being treated with fludarabine chemotherapy
● Hodgkin lymphoma
● Consider in patients with solid tumors being treated with aggressive chemo

Washed blood components - Blood is washed to prevent infusion of proteins present in residual plasma in red cell concentrates that may cause allergic reaction.

□ Indications for washed product:
● Patients with severe or recurrent allergic reactions
● Documented IgA deficiency (pt’s can have anti IgA antibodies)
● Reduce plasma K levels in acute renal failure

CMV negative components - Includes leukoreduced and seronegative CMV products.

□ Indications for CMV negative components:
● CMV negative allogeneic BMT
● Immunosuppressed CMV negative patients, potential transplant candidates

PREMEDICATIONS FOR TRANSFUSION:
Goal is to prevent transfusion reactions

● Generally recommend APAP 650mg PO and benadryl 50mg PO
● May use dexamethasone in patients with history of severe reaction
● Be careful in patients at risk of complications from these therapies (ie liver disease, elderly)

Transfusion Reactions
Overall, 1-6% of all patients, and up to 10% of heme/onc patients who receive blood transfusions experience an adverse reaction.

Overview
Immuneologic Reactions:
▪ Febrile non-hemolytic transfusion reactions
▪ Acute hemolytic transfusion reactions
▪ Delayed hemolytic transfusion reactions
▪ Anaphylactic transfusion reactions
▪ Urticarial transfusion reactions
▪ TRALI
▪ Post transfusion purpura
▪ Graft-versus-host disease (GVHD)

Chemical/Physical Reactions:
▪ Volume overload
▪ Citrate toxicity
▪ Hyperkalemia, Hypokalemia/metabolic alkalosis

Febrile Non-hemolytic Reactions:
▪ Most common reaction (0.5-1% of all pRBC transfusions)
▪ Symptoms include fever, chills, mild dyspnea, and malaise 1-6 hours after transfusion
▪ Etiology is from cytokines that are generated and accumulate during the storage of blood components
▪ Benign and without any lasting sequelae, but cannot distinguish initially from acute hemolytic reactions so the initial treatment for both the same
▪ Treat by stopping the transfusion, IVF’s, draw appropriate labs, and antipyretics
▪ Prevented by using leukoreduced or washed products

Acute Hemolytic Reactions:
▪ 1 per 40,000 transfused units of pRBC’s
▪ Medical emergency from rapid destruction of donor RBC’s by preformed recipient antibodies
▪ Most commonly due to ABO incompatibility from clerical error…on occasion can have acquired alloantibodies like anti-Rh or anti-Jka
▪ Symptoms: The classic triad of fever, flank pain, and red/brown urine (hemoglobinuria) is actually rarely seen. Other symptoms include chills, flushing, nausea, chest tightness, malaise
▪ Treatment includes stopping the transfusion, initiating protocol for transfusion reactions (i.e. blood bank checks for clerical errors), maintain ABC’s, start IVF’s (Normal Saline), and check a direct antiglobulin (Coombs) test, Hemoglobin, and repeat T&C from the other arm.
• Maintain UOP of 100-200 ml/hr with IVF’s to avoid renal failure…urinary alkalinization doesn’t really do much
• Do not use Lactated ringers (calcium in this may initiate clotting of any remaining blood in the line) or dextrose containing IVF’s (dextrose may hemolyze any remaining RBC’s in the line)
• If massive hemolysis is present and there is concern for DIC, cautious early heparinization (10units/kg/hour) for 12-24 hours may be indicated.

Delayed Hemolytic Reactions:
• 1 per 7000 pRBC transfusions
• Anamnestic antibody response occurring after re-exposure to foreign red cell antigens previously encountered by transfusion, transplantation, or pregnancy
• Antibodies are often Kidd or Rh, and levels have fallen to undetectable levels on pre-transfusion testing, but increase rapidly in titer following the transfusion
• Time course is within 2 to 10 days after transfusion.
• Signs/Symptoms are falling hematocrit, slight fever, mild increase in indirect bilirubin, spherocytosis on smear
• Often actually diagnosed by blood bank when a new antibody screen is done when more blood is ordered.
• Hemolysis is usually extravascular, gradual and less severe than with acute reactions
• No treatment is required unless brisk hemolysis is present.

Anaphylactic Reactions:
• 1 per 20,000 to 50,000 transfused units
• Occurs within few seconds to minutes following initiation of transfusion that contains any plasma (often < 10 cc transfused)
• Almost always due to the presence of class-specific IgG, anti-IgA antibodies in patients who are IgA deficient
• Selective IgA deficiency occurs in 1 in 300-500 people, but not all have developed antibodies. Usually not a problem in patients with acquired IgA deficiency
• Symptoms: Anaphylaxis (tachycardia, urticaria, flushing, laryngeal edema, hypotension, respiratory distress)
• Treatment: stop transfusion, Epinephrine (0.3 ml of 1:1000 solution IM), possible IV epinephrine gtt, Airway, IVF’s, occasionally pressors
• Prevented by using washed blood products

Urticarial Transfusion Reaction:
• 1-5% of all reactions
• Occurs when soluble allergic substance in plasma of donated blood product react with preexisting IgE antibodies. Cause mast cells and basophils to release histamine leading to hives
• Treatment: stop transfusion, give diphenhydramine. If hives resolving, can actually resume transfusion

Transfusion-Related Acute Lung Injury (TRALI):
• Actually under reported…one series found 1 in 2000 transfusions at a university hospital
• ARDS type of picture 30 minutes to 6 hours post transfusion…not related to volume overload
• Pathogenesis still unknown…you’re never wrong if you blame cytokine
  - elevated risk with increasing units of FFP
• Treatment is supportive
Post Transfusion Purpura:
- Uncommon (~250 reported cases), female: male ratio 26:1
- Severe thrombocytopenia 5-10 days following platelet containing product.
- Prior sensitization to a foreign antigen by pregnancy or prior transfusion, most commonly implicated is human platelet antigen 1a (HPA-1a)
- Treatment is high dose IVIG

Graft-versus-host disease (GVHD):
- Rare, primarily immunocompromised patients when viable donor lymphocytes attack recipient tissues (prevented by using irradiated blood products in this patients)
- Can also happen if recipient receives blood products from a relative who is a partial HLA match (also prevented by irradiation)
- Very poor prognosis

HOSPITALIST

Pre-Op Workup/Surgical Clearance

The first rule is never say in your note, “The patient is OK to go to surgery.” The goal of a “clearance” is not to say whether a patient can or cannot go to surgery but rather what the risk level is in taking them and what needs to be done immediately before, during, and after. All patients are “OK” to go to surgery if the setting is right and the benefits outweigh the risks. Conversely, no patient should go unless these criteria are met. Here are the issues that need to be addressed in every medical evaluation prior to surgery.

Cardiac Risk Assessment for Non-Cardiac Surgery

<table>
<thead>
<tr>
<th>Emergent surgery?</th>
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<tbody>
<tr>
<td>No</td>
<td>Active cardiac condition?</td>
</tr>
<tr>
<td></td>
<td>Yes → Evaluate and treat, then consider OR</td>
</tr>
<tr>
<td></td>
<td>No</td>
</tr>
<tr>
<td></td>
<td>Low-risk surgery?</td>
</tr>
<tr>
<td></td>
<td>Yes → OR</td>
</tr>
<tr>
<td></td>
<td>No</td>
</tr>
<tr>
<td></td>
<td>METs &gt; 4 No symptoms</td>
</tr>
<tr>
<td></td>
<td>Yes → OR</td>
</tr>
</tbody>
</table>

Active cardiac conditions:
- Unstable coronary syndromes
- MI within 30 days
- Decompensated HF
- Significant arrhythmia
- Severe valvular disease
- AS with valve area <1.0cm²
- Symptomatic mitral stenosis

Surgery-specific risk:
- High
  - Vascular, prolonged surgery with anticipated large volume shifts
  - Intraperitoneal, intrathoracic
  - Head and neck, CEA
Peri-operative cardiac risk should be calculated with the Revised Cardiac Risk Index (RCRI): (Circulation 1999;100:1043-1049)

**RCRI Score (1 pt for each risk factor):**
- High-risk procedure
- Ischemic heart disease (h/o MI, Q waves on ECG, angina, +stress, NTG use)
- Congestive heart failure
- Cerebrovascular disease
- Insulin-dependent diabetes
- Creatinine > 2

The risk of major perioperative cardiac complications is 0.4% for 0 pts, 0.9% for 1 pt, 6.6% for 2 pts, and 11% for ≥3 pts. Include the RCRI score in your note.

Alternatively, you can use the new NSQIP risk calculator developed in 2011:
[http://www.surgicalriskcalculator.com/miocardiacarrest](http://www.surgicalriskcalculator.com/miocardiacarrest)

In general, risk <1% is considered low, 1-5% is considered intermediate, and >5% is considered high.

Now that you know the patient’s baseline cardiac risk, you need to make recommendations about whether the patient needs any additional diagnostic studies or medical management prior to surgery.

**Perioperative Beta-Blockade**
- Continue BB if patient already on BB
- If RCRI < 2, no need for BB unless patient has CAD
- If RCRI ≥ 2, start a BB if there is time to safely titrate to goal HR 50-60 pre-operatively.
  - Goal HR 60-80 post-op. Continue for 30 days post-procedure.

**Perioperative Statin Therapy**
- Continue statin if patient already on statin
- Consider statin if vascular surgery or if should be on one as outpatient

**Perioperative Stress-Testing**
- Consider for RCRI ≥ 1 only if will change management (i.e., if it will help you decide about more aggressive medical management; little reason for preoperative revascularization even if + stress therefore this is rarely done anymore)

**Pre-Op ECG's**
- Obtain in patients with any RCRI points unless undergoing low-risk surgery

**How should you manage patients with a previous PCI?**
- **Bare Metal Stents**
  - <30-45 days → delay surgery if not urgent; continue dual antiplatelet therapy if urgent
  - >30-45 days → OR with aspirin
- **Drug-Eluting Stents**
  - <12 months → delay surgery if not urgent; continue dual antiplatelet therapy if urgent
  - >12 months → OR with aspirin

**Pulmonary Risk:** Suggest things that can be done in the acute setting to lower surgical risk. *(NEJM 1999: 340:937-944)*
1. Treat restrictive disease adequately if present
2. Obtain a CXR only if infection is suspected and treat if present
3. If severe pulmonary disease, suggest spinal or regional anesthesia
4. Encourage incentive spirometry use immediately after
5. Recommend CPAP and the settings if patient uses this at home
**Remember, while quitting smoking is good, it only helps with surgery if the patient has quit 6-8 weeks prior to procedure. Recommend nicotine patch.**

**Diabetes Management:** If diabetic, use the following guidelines. *(Arch. Int. Med. 1999: 159:2405-2411)*
1. Hold all oral agents on the day of surgery
2. In theory, patients should be able to continue their normal long-acting (i.e., glargine) insulin dose even while NPO since it covers their basal requirements. However, to be conservative, consider giving patients 1/2 to 2/3 their usual morning insulin dose if they are dosed daily, and 1/3 to 1/2 their morning dose if they are dosed 2-3 times per day.
3. Recommend QAC + QHS fingersticks for all diabetics
4. Recommend short-acting insulin at mealtimes + CF for all diabetics and adjust basal insulin according to these needs
5. If a patient needs to go to surgery emergently and they already took a dose of long acting insulin, using a dextrose drip of 50 cc/hr of 10% solution with frequent monitoring is wise
6. Consider continuous insulin drip if long, complex procedure

**Perioperative Management of Patients on Chronic Steroids**
Patients who have taken any dose of glucocorticoids for <3 weeks, who are on chronic alternate day therapy, or who are on < 5 mg/day of prednisone are unlikely to have a suppressed HPA axis and can continue their usual dose of steroids.

Patients taking prednisone doses >20mg/day for ≥ 3 weeks or who have Cushingoid appearance should be assumed to have HPA axis suppression. Patients on 5-20 mg prednisone/day may or
may not have adrenal insufficiency. Stress-dosing has been suggested according to surgical intervention, though adrenal insufficiency without increased dosing is relatively rare. If high-dose therapy is employed, replacement dose should be resumed within 48 hours.

If on baseline steroids ≥ 5 mg/day, supplement with: *(JAMA 2002: 287 No.2:236-240)*

1. Minor surgical stress → Hydrocortisone 25 mg IV q8h on day of procedure only
2. Moderate surgical stress → Hydrocortisone 50-75 mg q8h on day of procedure, taper to baseline dose over 1-2 days
3. Severe surgical stress → Hydrocortisone 100-150 mg IV q8h on day of procedure, taper to baseline dose over 1-2 days
4. Critically ill patients get 50 mg IV hydrocortisone q8h + 50 μg IV fludrocortisone qday until shock resolves, then slow taper

**Perioperative Medication Management**
- Consider holding ACEI or ARB the night before or morning of surgery to minimize the risk of perioperative hypotension
- Hold NSAIDs preoperatively

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**INFECTION DISEASE**

**General Considerations:**
- Consider what organisms the patient is at risk for; do they have a history of resistant organisms, were they recently hospitalized, what is the source of infection (different drugs for different bugs)
- Whenever possible, narrow your antibiotics. If you have sensitivities, use them.
- If the patient is not improving, do you have the wrong drug, the wrong bug, or is the bug resistant?

**Evaluation and Management of Staphylococcus aureus Bacteremia (SAB)**

1. Background
   - *Staphylococcus aureus* is one of the most common causes of bacteremia and is the most serious
   - About 1/3 have definite or possible endocarditis (modified Duke criteria¹)
   - About 1/3 have deep-tissue infections due to hematogenous seeding (e.g., vertebral osteomyelitis, septic arthritis, psoas abscess)
   - Mortality of SAB: 9% to 47%

**Things to remember during the initial management of S. aureus bacteremia**
- *S. aureus* should not be considered a blood culture contaminant, unless discussed with the Infectious Diseases (ID) consult service
- Central venous catheters (if removable) should be removed and the catheter tip sent for culture as soon as possible after the identification of *S. aureus*
  - Risk of relapse is increased when catheters are not removed²

- 95 -
use peripheral venous access until bacteremia has cleared whenever possible

- *All* patients should have a follow-up blood culture drawn 2-3 days after the initial positive culture, even if signs or symptoms of infection have resolved
  - a positive follow-up blood culture is a strong predictor of endocarditis and deep-tissue infections

- Echocardiography should be performed routinely
- Localizing signs or symptoms should be evaluated with the appropriate imaging study (e.g., MRI of spine to evaluate for osteomyelitis or epidural abscess in a patient with SAB and back pain)
- We recommend consultation of the ID service *early in the inpatient course of all cases of SAB* for assistance with evaluation and management

**Identifying patients at risk for complications**

- The following are predictors of endocarditis and/or deep-tissue infections:
  - Positive follow-up blood culture
  - Failure to remove intravascular catheter
  - Persistent fever >3 days after initial culture
  - Prior endocarditis or underlying valvular disease
  - Prosthetic valve, pacemaker, implantable defibrillator, prosthetic joint, or other prosthetic devices
  - Time to blood culture positivity of ≤14 hours
  - Community-acquisition of infection
  - Unknown source of infection

**Treatment of *S. aureus* bacteremia**

- Optimal treatment requires intravenous therapy
- Oral therapy such as fluoroquinolones, linezolid, clindamycin, and beta-lactams should NOT be used
- Empiric coverage should be directed at methicillin-resistant *S. aureus* (MRSA) until susceptibilities are known (≥33% of isolates are methicillin-resistant)
- Antistaphylococcal penicillins (nafcillin/oxacillin) and cefazolin are superior to vancomycin and daptomycin for the treatment of methicillin-sensitive *S. aureus* (MSSA) bacteremia
  - consider cefazolin for patients with MSSA bacteremia who have a history of penicillin allergy that does not include hives, symptoms of airway compromise, or anaphylaxis
- Adjunctive aminoglycoside use for 3-5 days in suspected endocarditis decreases time to clearance of bacteremia but has not been shown to improve cure rates and increases the risk of nephrotoxicity

**Methicillin-sensitive *S. aureus* (MSSA) bacteremia treatment options**

<table>
<thead>
<tr>
<th>Nafcillin</th>
<th>Usual Dose</th>
<th>Considerations</th>
<th>Formulary</th>
<th>Approx. Cost</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2gm Q4h OR 10-12gm cont infusion (Adjust hepatic*)</td>
<td>Standard therapy for MSSA bacteremia/endocarditis. <strong>Monitor:</strong> CBC, LFTs, Creatinine</td>
<td>UCH DHMC</td>
<td>$57/10gm</td>
</tr>
<tr>
<td><strong>Oxacillin</strong></td>
<td>2gm Q4h (Adjust hepatic*)</td>
<td>Standard therapy for MSSA bacteremia/endocarditis. <strong>Monitor:</strong> CBC, LFTs, Creatinine</td>
<td>VA</td>
<td>$60/10gm</td>
</tr>
<tr>
<td>--------------</td>
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<td>------------------------------------------------------------------------------------------------</td>
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</tr>
</tbody>
</table>

| **Cefazolin** | 2gm Q8h (Adjust renal) | Consider for patients with concomitant gram negative infections **Monitor:** CBC, Creatinine | All | $6/3gm $10/5gm |

*discuss hepatic insufficiency dose adjustments with ID and Pharmacy services*
### Methicillin-resistant *S. aureus* (MRSA) bacteremia treatment options

<table>
<thead>
<tr>
<th>Drug</th>
<th>Usual Dose</th>
<th>Considerations</th>
<th>Formulary</th>
<th>Approx. Cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vancomycin, IV</td>
<td>See section below</td>
<td>Standard therapy for MRSA bacteremia/endocarditis. <strong>Monitor:</strong> CBC, Creatinine</td>
<td>All</td>
<td>$10-15/day</td>
</tr>
<tr>
<td>Daptomycin, IV (Cubicin®)</td>
<td>6mg/kg Qday (Adjust hepatic*)</td>
<td>FDA-approved for MRSA bacteremia/endocarditis. <strong>NOT</strong> for use in cases with pulmonary involvement. <strong>Monitor:</strong> LFTs, CK</td>
<td>Requires ID approval-All</td>
<td>$170/day</td>
</tr>
<tr>
<td>Linezolid, IV/PO (Zyvox®)</td>
<td>600mg Q12h (Adjust hepatic*)</td>
<td><strong>NOT FDA-approved</strong> for MRSA bacteremia, use only in consultation with ID service <strong>Monitor:</strong> CBC, LFTs</td>
<td>Requires ID approval-All</td>
<td>$150/day</td>
</tr>
</tbody>
</table>

*discuss hepatic insufficiency dose adjustments with ID and Pharmacy services

### Dosing recommendations for vancomycin

**I. Initial Dose**
- All patients, irrespective of renal function, should receive an initial dose of 15mg/kg* once
  - *Based on actual body weight unless patient is obese (>130% of ideal body weight (IBW))
  - IBW (kg) = [50 (male) OR 45 (female)] + 2.3(patient height >60inches)
  - If patient >130% of IBW, then use Adjusted body weight (AdjBW (kg) = IBW + 0.4(Actual-IBW))
- Please round all doses to the nearest 500mg dose

**II. Maintenance Dosing**
The following are empirical maintenance regimens based on creatinine clearance (CrCl) by the Cockcroft-Gault equation:

<table>
<thead>
<tr>
<th>Renal function (ml/min)</th>
<th>Empiric Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>ClCr&gt;60</td>
<td>15mg/kg* Q8-12h</td>
</tr>
<tr>
<td>Ccr 40-60</td>
<td>1000mg Q12-24h</td>
</tr>
<tr>
<td>ClCr 10-40</td>
<td>1000mg Qday</td>
</tr>
<tr>
<td>CrCl&lt;10 or intermittent HD</td>
<td>**</td>
</tr>
<tr>
<td>CVVH</td>
<td>1000mg Qday</td>
</tr>
</tbody>
</table>

*See weight-based dosing for obese patients above
**Serum TRough levels should guide therapy in patients with severely impaired or rapidly changing renal function. Please refer to specific institution recommendations for maintenance dosing.
III. Pharmacokinetic (PK) Monitoring

In general, many acute care patients will be appropriate for PK monitoring of vancomycin serum trough concentrations.

- A trough level should be drawn within 30 minutes of the next dose, after a patient has reached steady state (after approximately 3-5 doses have been administered).
- Vancomycin serum concentrations may be increased by altering dose or administration interval. Adjusting the interval is the preferred method for increasing serum trough concentration (e.g., 1gm Q12hrs → 1gm Q8hrs)
- Goal serum trough concentration: 10-20mg/dl (15-20mg/dl for bacteremia associated with MRSA pneumonia)
  - Note: each institution has specific policies for PK monitoring

Duration of therapy

- The optimal duration of therapy for SAB remains unclear; consultation with the ID service is recommended
- Generally, a duration of therapy of least 14 days for uncomplicated infections and at least 28 days for complicated infections (positive follow-up blood cultures, endocarditis, deep-tissue infections) can be expected

Patient follow-up

- All patients discharged on intravenous antimicrobial therapy need weekly laboratory and clinical monitoring
- Contact the ID service to arrange appropriate follow-up at least 48 hours prior to discharge

References


Contributions by members of the Divisions of Infectious Diseases and Pharmacy
Endocarditis

1. **Infective Endocarditis** - infection of the lining of the heart, including the heart valves

   - If you suspect someone has endocarditis, they should be evaluated using the modified Duke criteria:
     - **Definite endocarditis:** Presence of 2 major criteria or 1 major criterion and 3 minor or 5 minor criteria.
     - **Possible endocarditis:** Presence of 1 major criterion and 1 minor or 3 minor criteria.

2. **Major Criteria:**
   - **Positive Blood Cultures:** Positive blood cultures for microorganisms that typically cause endocarditis (*Strep. viridans*, *Strep. bovis*, HACEK organisms, *Staph. aureus* or community acquired enterococci with no other primary focus). Persistently positive blood cultures of one of the above organisms in blood cultures drawn more than twelve hours apart or positive blood cultures in three out of four sets drawn at the same time or single positive blood culture or serology test for *Coxiella burnetii*.
   - **Evidence of Endocardial Involvement:** Positive echocardiogram (oscillating intracardiac mass on valve or supporting structures, abscess or dehiscence of prosthetic valve). New valvular regurgitation (does not include change in pre-existing murmur).

3. **Minor Criteria:**
   - **Predisposing heart condition** or history of intravenous drug use.
   - **Fever** $\geq 38.0^\circ$ C
   - **Vascular phenomena** (septic infarcts, Janeway lesions, mycotic aneurysms).
   - **Immunologic phenomena** (Osler’s nodes, Roth spots, glomerulonephritis).
   - **Positive blood culture** not meeting major criteria and also excluding single positive set for coagulase negative staphylococci and organisms that do not cause endocarditis.

4. **Workup should include:**
   - **At least three sets of blood cultures:** Depending on the severity of the illness, it may be reasonable to initially withhold antibiotic therapy until sufficient cultures are obtained or until diagnosis is made.
   - **Electrocardiogram:** Presence of a new conduction abnormality (i.e. first degree AV block) may indicate a complication such as a perivalvular abscess.
   - **Chest x-ray:** Evidence of new heart failure or septic emboli may be detected.
   - **Echocardiogram:** This should be performed in all cases of suspected endocarditis. TTE can be a reasonable first test, but TEE should be pursued if TTE is negative and clinical suspicion is high. TEE should be considered as the first test if the likelihood of obtaining good windows with TTE is low (morbid obesity, previous thoracic surgery, COPD) or if clinical suspicion of endocarditis and its complications is fairly high (prosthetic valve or new conduction block on EKG). However, if TEE needs to be delayed because of the clinical scenario, then TTE should be performed without delay.
5. Common Organisms:
- **Native valve, non-IV drug user:** *Strep. viridans > Staph. aureus > Enterococcus*
- **Native valve, IV drug user:** *Staph. aureus > Strep. viridans > Enterococcus*
- **Prosthetic valve, within 6 months:** *Staph. epidermidis > Staph. Aureus > gram negative rods*
- **Prosthetic valve, after 6 months:** *Strep. Viridans > Staph. Epidermidis > Staph. Aureus and enterococcus*

6. Initial Therapy:
- **Acute endocarditis with native valve:** Typically start vancomycin and gentamicin and then tailor based on culture results. Nafcillin is a superior agent to vancomycin if MRSA has been ruled out because it is bactericidal instead of static (vancomycin).
- **Acute endocarditis with prosthetic valve:** Typically start vancomycin, gentamicin, and rifampin if within one year of valve replacement and then tailor based on culture data.

7. Continued Care:
- Tailor antibiotic therapy as soon as culture data is available
- Obtain two sets of blood cultures every 24-48 hours until cultures are negative
- Duration of therapy should be based on counting the first day that blood cultures are negative as the start of therapy
- Duration of therapy is typically 4-6 weeks and can often be completed in the outpatient setting once the patient is clinically stable
- Repeat echocardiography should be obtained at the end of therapy to establish a new baseline for the patient

8. Surgical Intervention:
- This is often a complicated decision, made in conjunction with cardiothoracic surgery, but should be considered in certain scenarios:
  1. Fungal infection.
  2. Heart failure from valvular dysfunction that is unresponsive to medical therapy
  3. Persistent infection despite appropriate medical therapy (positive blood cultures after one week or embolic events during the first two weeks of therapy).
  4. Prosthetic valve dehiscence or perivalvular abscess.


**Osteomyelitis**

1. Three Types:
   1. Due to local spread from a contiguous contaminated source of infection. Usually secondary to trauma, bone surgery, or joint replacement.
   2. Secondary to vascular insufficiency. Predominantly in diabetics. Most cases start with a soft tissue infection.

2. Acute vs. Chronic
   - Acute: evolves over several days or weeks.
• Chronic: evolves over weeks to months to years. Characterized by persistence of microorganisms, low-grade inflammation, presence of dead bone, and fistulous tracts. Hard to treat and usually requires surgical intervention.

3. Microbiology

<table>
<thead>
<tr>
<th>Clinical Scenario</th>
<th>Microorganism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Most common in any type</td>
<td><strong>Staphylococcus aureus</strong></td>
</tr>
<tr>
<td>Foreign-body-associated</td>
<td>Coagulase-negative Staph or <em>propionibacterium spp.</em></td>
</tr>
<tr>
<td>Associated with bite, diabetic feet, and decubitus ulcers</td>
<td>Streptococci and/or anaerobic bacteria</td>
</tr>
<tr>
<td>Sickle-cell disease</td>
<td><em>Salmonella spp</em> or <em>Streptococcus pneumoniae</em></td>
</tr>
<tr>
<td>HIV</td>
<td><em>Bartonella henselae</em> or <em>B. quintana</em></td>
</tr>
<tr>
<td>Human or animal bites</td>
<td><em>Pasteurella multocida</em>, <em>Eikenella corrodens</em></td>
</tr>
<tr>
<td>Immunocompromised</td>
<td><em>Aspergillus spp.</em>, <em>Candida albicans</em>, or <em>Mycobacteria spp.</em></td>
</tr>
<tr>
<td>Populations with high TB prevalence</td>
<td><strong>Mycobacterium tuberculosis</strong></td>
</tr>
<tr>
<td>Populations in which these organisms are endemic</td>
<td><em>Brucella spp.</em>, <em>Coxiella burnetii</em>, cocci, blasto, histo.</td>
</tr>
</tbody>
</table>

4. Signs and Symptoms: range from open wounds exposing fractured bone, a draining fistula, local swelling and bone pain tenderness without skin lesions. +/- fever. Joint instability or painful joint in patients with prosthetic joints.

5. Diagnosis:
   - ESR and CRP will often be high. CRP goes up first and is more reliable to assess treatment response.
   - WBC count may be normal.
   - Start with plain films (often normal early on) ➔ if they do not show anything, order an MRI.
   - If you can probe down to bone, the patient has osteomyelitis.
   - Getting tissue is the key: Bone biopsy/aspiration whether under CT guidance or during surgery is critical as this will guide treatment.

6. Treatment:
   - Anything but acute or hematogenous osteomyelitis is almost impossible to treat without surgery. Consult Ortho.
   - Antibiotics should be based on culture and sensitivity results. Hold therapy until after biopsy or specimens from I&D obtained.
   - Antibiotic courses are generally long (4-6 weeks), so get ID involved early to facilitate disposition, need for vascular access, and outpatient antibiotic coordination.

HIV / AIDS

1. Diagnosis:
   - ELISA is positive one month after infection in 80% of patients.
   - If considering acute HIV infection, check viral load.
   - Review risk factors and screen anyone between ages of 13 and 64.
   - Obtain accurate contact information for the patient who will be tested for HIV in case discharged prior to test result.
   - AIDS: HIV + and CD4 < 200 or evidence of opportunistic infection (OI)

2. If diagnose new HIV infection:
   - Call and notify ID service.
   - Vaccinate against pneumonia, flu, Hep A, Hep B and place ppd.
   - Talk to patient about diagnosis!

3. Treatment recommendations:

   **Symptomatic disease**: Start treatment

   **Asymptomatic disease**
   - CD4 > 500…………..defer rx
   - CD4 350 – 500…………generally not recommended
   - CD4 200 – 350……….therapy considered and treatment individualized
   - CD4 < 200…………..therapy recommended

### Acute HIV Symptoms

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>Asymptomatic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(majority)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rash</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fever</td>
<td></td>
<td></td>
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<tr>
<td>Fatigue</td>
<td></td>
<td></td>
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<tr>
<td>Rash</td>
<td></td>
<td></td>
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<tr>
<td>Headache</td>
<td></td>
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<tr>
<td>Pharyngitis</td>
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<tr>
<td>LAD</td>
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<tr>
<td>Aseptic Meningitis</td>
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<tr>
<td>Myalgias</td>
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<td></td>
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<tr>
<td>Night Sweats</td>
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<td></td>
</tr>
</tbody>
</table>

### Drugs: Generally use 2NRTI’s and 1 NNRTI or 2NRTI’s and 1 PI.

<table>
<thead>
<tr>
<th>NRTI</th>
<th>NNRTI</th>
<th>PI*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emtricitabine (Emtriva, FTC)</td>
<td>efavirenz (Sustiva, EFV)</td>
<td>atazanavir (Reyataz, ATV)</td>
</tr>
<tr>
<td>lamivudine (Epivir, 3TC)</td>
<td>nevirapine (Viramune, NVP)</td>
<td>lopinavir/ritonavir (Kaletra, LPV/r)</td>
</tr>
<tr>
<td>zidovudine (Retrovir, AZT, ZDV)</td>
<td></td>
<td>fosamprenavir (Lexiva, FPV)</td>
</tr>
<tr>
<td>didanosine (Videx EC, ddi)</td>
<td></td>
<td>tipranavir (Aptivus, TPV)</td>
</tr>
<tr>
<td>tenofovir (Viread, TDF)</td>
<td></td>
<td>indinavir (Crixivan, IDV)</td>
</tr>
<tr>
<td>stavudine (Zerit, d4T)</td>
<td></td>
<td>saquinavir (Invirase, SQV)</td>
</tr>
<tr>
<td>abacavir (Ziagen, ABC)</td>
<td></td>
<td>ritonavir (Norvir, RTV)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>darunavir (Prezista, DRV)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>nelfinavir (Virecept, NFV)</td>
</tr>
</tbody>
</table>
Combination formulations:
Atripla: TDF + FTC + EFV
Truvada: TDF + FTC
Combivir: AZT + 3TC
Epzicom: ABC + 3TC
Trizivir: AZT + 3TC + ABC
*Ritonovir boosted PI's: ritonovir will elevate other PI drug levels due to its interaction with the CYP 3A450 system.

4. When pts on HAART are admitted:
   - Notify the ID service, regardless of whether you want them to consult (not necessary to call them in the middle of the night, but a courtesy call in the morning is appropriate).
   - If tolerating PO, may continue treatment unless you think their admission problem is directly related to their medication.
   - If unable to take PO, stop ALL the HAART meds.
   - If unsure, stop all HAART meds and speak with ID.
   - Don’t add a PPI until you check it does not interfere with HAART absorption.

5. Opportunistic Infections:

<table>
<thead>
<tr>
<th>CD4 Count</th>
<th>Possible OIs</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;500</td>
<td>Bacterial Infections, seborrheic derm, KS, oral/pharyngeal Candida, HSV, TB</td>
</tr>
<tr>
<td>&lt;200</td>
<td>Esophageal candidiasis, Histoplasmosis, Blastomycosis, Cryptococcosis, Coccidioidomycosis, PCP</td>
</tr>
<tr>
<td>&lt;100</td>
<td>CMV, MAC, invasive Aspergillosis, PML, CNS lymphoma, Toxoplasma</td>
</tr>
</tbody>
</table>

Prophylaxis:

<table>
<thead>
<tr>
<th>OI</th>
<th>When to start (CD4)</th>
<th>1” Line</th>
<th>2” Line</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCP</td>
<td>&lt;200</td>
<td>Bactrim DS one daily (or SS daily or DS 3x week)</td>
<td>Dapsone, aerosolized pentamidine, or atovaquone</td>
</tr>
<tr>
<td>Toxo</td>
<td>&lt;100</td>
<td>Bactrim DS one daily</td>
<td>Dapsone-pyrimethamine</td>
</tr>
<tr>
<td>MAC</td>
<td>&lt;50</td>
<td>Azithro 1250mg qWeek</td>
<td>Clarithromycin, Rifabutin</td>
</tr>
<tr>
<td>Histo</td>
<td>&lt;150 (Must live in endemic area)</td>
<td>Itraconazole 200mg daily</td>
<td></td>
</tr>
<tr>
<td>TB</td>
<td>At diagnosis check PPD</td>
<td>9 months INH</td>
<td>RIF + PZA x 2 months</td>
</tr>
</tbody>
</table>

6. PCP:
   - Consider PCP if CD4 <250, questionable compliance with prophylaxis, sub-acute course, dry cough, exertional hypoxia, night sweats, etc.
   - Just because the patient has taken prophylaxis does not mean they cannot have PCP! Especially if prophylaxis is not TMP/SMX.
   - CXR can be normal or with bilateral interstitial infiltrates.
   - DX: Have RT induce sputum to check PCP DFA. If suspicion high and sputums are negative, bronchoscopy with BAL has sensitivity of 95-99%. Elevated LDH is also suggestive.
   - RX: TMP/SMX for 21 days.
If suspected, get an ABG! If PaO2 > 70: consider oral TMP/SMX 2 DS tabs PO TID. If PaO2 < 70: Steroids first (Prednisone 40 mg BID x 5d, then 40mg daily x 5d, then 20mg daily x 11d), then IV TMP/SMX.

Alternative therapies: clindamycin + primaquine PO (check for G6PD first) OR pentamidine IV (watch glucose, blood pressure, electrolytes) are usually best alternatives. Use atovaquone when other therapies impossible.

7. Other organisms to keep in differential of pulmonary infections: TB, CAP pathogens, Nocardia, fungal pneumonia (histo, blasto, coccidio, aspergillosis, etc), viral pneumonia (HSV, CMV), KS, lymphoma. Consider all that you would in a patient without HIV (do not limit differential to just infectious causes, i.e. PE, CHF, pericarditis, etc).

8. AIDS and Diarrhea:
   - Protozoal: Cyclospora/Microsporidium/Isospora, Cryptosporidium, Giardia, Entamoeba.
   - Bacterial: Salmonella/Shigella/Campylobacter, MAC, TB, C. diff.
   - Viral: CMV, HSV, HIV.
   - Fungal: Histoplasmosis, Coccidioidomycosis, Candida albicans.

Work up:
1. Stool: Culture, C. diff toxin, O and P, modified acid fast smear (crypto, cyclospora, isospora), trichrome for microsporidium if CD4 <100, stool antigen for Giardia and Cryptosporidium.
2. Blood: MAC blood cultures if suspected.

9. AIDS and Esophagitis: HSV, CMV, Pill induced, Candida.


11. AIDS and Altered Mental Status:
   - Always perform a thorough H & P/neuro exam, lumbar puncture, and some form of neuro imaging (preferably with contrast).
   - If CT negative, think meningitis: bacterial (Listeria, plus common meningitis organisms), viral (CMV, HSV), Cryptococcus, TB.
   - If CT shows mass lesions: Toxo, Lymphoma, pyogenic abscess, PML.
   - Labs to check: RPR, serum crypto antigen, and typical cultures.
   - DDX:
     3. Other: metabolic, CVA, drugs, systemic infection, psychiatric.
TUBERCULOSIS

1. Consider with high risk populations
   - Contact with known TB case
   - Homeless
   - Prisons
   - Chronically ill (ESRD, DM, Alcoholics, cancer)
   - HIV/AIDS
   - Healthcare workers
   - Foreign born or immigrant

2. Transmission via airborne droplets….ISOLATE ALL PATIENTS THAT YOU ARE GOING TO RULE OUT WITH SPUTUMS! If you are suspicious enough to check sputums, isolate the patient.

3. Diagnosis: Induced sputum for AFB. Typically, need three negative AFB’s and/or alternative diagnosis to discontinue isolation. They have to be collected on different days, so don’t delay in ordering them. Make sure the lab has the specimen. Sometimes they will not tell you if they were unable to collect the sample or if the sample is insufficient. Avoid use of fluoroquinolones for suspected CAP when TB is considered as these drugs can cloud dx due to some activity against MTB.

4. Interpretation of PPD test: to detect Latent infection. 2-12 weeks is needed after an acute infection for the PPD to be positive.

<table>
<thead>
<tr>
<th>Size of PPD</th>
<th>Considered positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;5mm*</td>
<td>HIV +, immunosuppressed (pts on greater than 15mg/day of prednisone), close contact with known active pulmonary TB case, CXR c/w prior TB.</td>
</tr>
<tr>
<td>&gt;10mm**</td>
<td>IVDA, employees/residents of high risk congregate settings, chronic medical condition</td>
</tr>
<tr>
<td>&gt;15mm</td>
<td>Everyone else</td>
</tr>
<tr>
<td>Previous BCG</td>
<td>Interpret as if they never had the BCG vaccine.</td>
</tr>
</tbody>
</table>

* highest risk for developing active TB if they become infected with M. tuberculosis

** increased probability of recent infection or who have conditions that increase the risk for TB

5. +PPD with active disease ruled out:
   - NON HIV: 5% develop active disease in 2years
   - HIV +: 5% develop active disease in their lifetime
   - 90% will be asymptomatic
   - 5-12% will develop active disease PER YEAR!

6. Treatment of Latent TB: Many drug regimens available. Most commonly used is: INH daily x 9months

7. What is MDR TB: TB that is resistant to at least INH and RIF. Risk Factors for having MDR-TB:
   - History of previous TB treatment
   - Direct contact with MDR-TB patient
   - HIV+
   - Persistent positive cultures after 3 months of therapy
   - People from areas with high MDR-TB prevalence

- 107 -
8. **What is XDR TB:** MDR TB that is also resistant to any fluoroquinolone and any of the injectable drugs.

9. **Clinical manifestations of TB are protean:**
   - Primary pulmonary TB: this is asymptomatic in most (HIV pts have higher risk of symptomatic primary disease).
   - Reactivation pulmonary TB: most commonly seen in apical segments of lungs, but can be atypical, especially in HIV pts.
   - TB pleurisy: can cause pleural effusion. Typically lymphocyte predominant, high LDL, exudative, and elevated ADA levels. Usually need to get pleural biopsy to diagnose.
   - Miliary TB: pulmonary disease secondary to hematogenous spread throughout the lung.
   - Extrapulmonary TB: lymphadenitis, peritonitis, pericarditis, meningitis, osteomyelitis, enteritis, adrenal, etc. Don’t be fooled, higher rates of extrapulmonary TB in HIV patients.

10. **Treatment:** This will be done in consultation with an ID specialist.
   - TB (known susceptible to standard agents):
     1. **1st line Therapy:** INH, RIF, ETH, PZA….varying dosing schedules available.
     2. **2nd line agents:** Ciprofloxacin, Ofloxacin, Cycloserine, PAS, Ethionamide.
     3. **Injectables:** Streptomycin, Amikacin, Kanamycin, Capreomycin
   - MDR TB: Need 4 active drugs in regiment. At least one 2nd line agent and one injectable.

---

**Nephrology**

**Acid-Base**

1. A simple approach to acid-base disorders:
   - Rule 1: Look at the pH. Whichever side of 7.4 the pH is on, the process that caused it to shift to that side is the primary disorder (i.e. acidosis or alkalosis). This is because the body never fully compensates for the primary acid-base disorder.
   - Rule 2: Calculate the anion gap. Look at the compensation. For a metabolic process the \( pCO_2 \) should increase (metabolic alkalosis) or decrease (metabolic acidosis). For a respiratory process, the bicarbonate should increase (respiratory acidosis) or decrease (respiratory alkalosis).
   - Rule 3: Calculate the delta gap (if there is an anion gap present). Take the patient’s anion gap and subtract it from a normal anion gap (12 or 3 X albumin) and add this to the measured bicarbonate concentration. If sum is greater than 30, then there is also an underlying metabolic alkalosis. If the sum is less than 23, then there is also an underlying non-anion gap metabolic acidosis.

2. Most complicated acid-base disorder is a “triple ripple”.

3. Anion gap and protein: to get a patient’s normal anion gap, multiply their albumin by 3 (rough estimate).

4. Compensation Formulas:
   - Metabolic Acidosis (can use either):
     1. \( pCO_2 \) decreases 1.2 for each mmol/L change in bicarbonate.
     2. \( pCO_2 \) should roughly equal last two digits of the pH.
   - Metabolic Alkalosis:
     1. \( pCO_2 \) increases 0.6 for each mmol/L change in bicarbonate.
   - Respiratory Acidosis:
1. Acute: Bicarbonate increases 0.1 for every mmHg change in pCO₂.
2. Chronic: Bicarbonate increases 0.35 for every mmHg change in pCO₂.

- Respiratory Alkalosis:
  1. Acute: Bicarbonate decreases 0.22 for every mmHg change in pCO₂.
  2. Chronic: Bicarbonate decreases 0.5 for every mmHg change in pCO₂.

5. Causes of Respiratory Alkalosis: anxiety, hypoxia, lung disease with or without hypoxia causing pulmonary receptor stimulation, CNS disease, Drugs (salicylates, catecholamines, progesterone), pregnancy, sepsis, hepatic encephalopathy, liver disease, mechanical ventilation.

6. Causes of Respiratory Acidosis: CNS depression (drugs, CNS event), neuromuscular disorders (myopathies, neuropathies), acute airway obstruction, severe pneumonia or pulmonary edema, hemothorax/pneumothorax, thoracic cage injury, chest wall disorders, central hypoventilation, COPD.

7. Causes of Metabolic Alkalosis:
   - Urine Chloride <10: emesis, NG suction, diuretics, post-hypercapneic, CF, villous adenoma, congenital chloride diarrhea.
   - Urine Chloride >20: hypokalemia, primary aldosteronism, secondary aldosteronism (CHF, cirrhosis, ascites), Cushings’s, Gitelman’s, Liddle’s, licorice.
   - Misc: refeeding alkalosis, excess alkali administration, massive transfusions, milk-alkali.

8. Causes of Non-anion Gap Metabolic Acidosis:
   - GI Bicarbonate Losses: Diarrhea, urinary tract diversions, ileus, fistulas, villous adenoma.
   - Renal Bicarbonate Losses: RTA, renal failure, carbonic anhydrase inhibitors, aldosterone inhibitors.
   - Misc: post-hypocapnea, HCl administration, TPN, urinary tract diversions.
   - Calculate the urine anion gap to differentiate between GI and renal causes. UAG: \[ \text{Urine}^+_{\text{Na}} + \text{Urine}^+_{\text{K}} - \text{Urine}^-_{\text{Cl}}. \] If positive, then GI cause most likely. If negative, then renal cause.

9. Causes of Anion Gap Metabolic Acidosis:
   - M-Methanol
   - U-Uremia
   - D-DKA/AKA/Starvation ketosis
   - P-Propylene glycol(lorazepam gtt), paraglutamic acid(APAP toxicity)
   - I-INH/Iron
   - L-Lactic Acidosis
   - E-Ethylene Glycol
   - R-Rhabdomyolysis
   - S-Salicylates


**Hyponatremia**

1. Classify total body sodium status of the patient first
   - Edema, elevated neck veins, etc=total body sodium is elevated
   - Dry mucous membranes, orthostatic VS, etc=total body sodium is decreased

2. Then classify the patient’s total body water status in relation to sodium by the beaker principle
   - If the patient is hyponatremic and total body sodium is elevated (ie edema, elevated neck veins), their total body water is even greater
• If the patient is hyponatremic and total body sodium is decreased, (ie dry, tachy) their total body water is relatively higher but may be normal or slightly low.

3. Check serum osm, urine osm, urine sodium.

4. Hypertonic hyponatremia is due to the gain of impermeable solute other than sodium (i.e. glucose-no osmolar gap) or IV mannitol or glycine (will have osmolar gap).

5. Hypovolemic Hyponatremia: ADH is being secreted in order to maintain intravascular volume.
   • Renal sodium loss: Diuretics, osmotic diuresis (mannitol), adrenal insufficiency, salt wasting nephropathy, bicarbonaturia (RTA’s, emesis), ketonuria, cerebral salt wasting syndromes. Serum osm < 280. Urine sodium > 20.
   • Extrarenal sodium loss: GI (vomiting, diarrhea), blood loss, surgical drains, excessive sweating (marathon runners), fluid sequestration (bowel obstruction, pancreatitis, peritonitis, burns, muscle trauma). Serum osm > 280, urine sodium < 10. These patients will have to be getting free water in order to lower their sodium.

6. Euvolemic Hyponatremia:
   • Hypothyroid
   • Adrenal Insufficiency
   • Psychogenic polydipsia (need to drink at least 10 to 15 L/day, low urine osm), beer potomania (low urine osm)
   • SIADH
     1. Pulmonary: infection (PNA, TB), acute respiratory failure
     2. CNS: mass lesions, stroke, inflammatory and demyelinating disorders, meningitis, hemorrhage, trauma, acute psychosis.
     4. Drugs: carbamazepine, vincristine, cyclophosphamide, opiate derivatives, desmopressin, SSRIs, TCAs.
     5. Other: post-op state, pain, severe nausea, HIV.

   • Cirrhosis, CHF, nephritic syndrome. Urine sodium < 20.

8. Treatment
   • **Fire Water:** Only give 3% NaCl (513 meq sodium per liter) if patient is seizing, in a coma, or serum sodium is less than 110 to 115 meq/L (don’t give if they are asymptomatic, as this is likely chronic). Call renal fellow before initiating 3% NS! Initial goal is to correct at 1 to 2 meq/L per hour until sx resolve or sodium is 118 meq/L. Then, stop the 3% NaCl, call renal fellow and discuss plan. From here the goal will be to correct no more than 8 to 10 meq/L over the first 24 hours from the initial sodium.
   • Correcting the serum sodium too quickly can result in central pontine myelinolysis (CPM) manifested as flaccid paralysis, dysphagia, dysarthria.
   • General guideline for correction is 0.5 meq/hour. Goal is 8 to 10 meq/L over the first 24 hours. Monitor electrolytes q 4 hours at least. Based upon result may need to adjust fluid rate or switch fluid altogether.
   • Hypovolemic Hyponatremia: use 0.9% NaCl or normal saline (154 meq/L). See above for 3% NaCl indications.
   • Euvolemic Hyponatremia: If asymptomatic, initiate fluid restriction to less than 1 Liter per day. Watch free water in gits and meds. See above for 3% NaCl indications.
   • Hypervolemic Hyponatremia: Fluid restriction. Diuresis. If unable to get volume off with diuresis (i.e. diuretic resistance, tenuous hemodynamics, may need to call renal fellow to discuss ultrafiltration or SCUF).
   • To calculate the change in serum sodium per liter of fluid (i.e. NS, ½ NS etc):
Infusate Na+ - Serum Na+ / TBW + 1


Hypernatremia
1. The majority of the time is due to free water loss, but can also be caused by hypertonic sodium gain. In order to maintain the hypernatremia, requires impairment of thirst or inadequate access to free water. Therefore, most commonly occurs in hospitalized patients such as those in the ICU, AMS, and the elderly.
4. Net Pure Water Loss as a cause
   - Unreplaced insensible losses (septic and febrile)
   - Not drinking due to impaired thirst or no access to water (on ventilator)
   - Diabetes Insipidus: patient is hypovolemic, urine output much greater than 1 Liter per day. Urine osm < 250. Use DDAVP to differentiate central from nephrogenic.
1. Central DI: urine osm should increase with DDAVP. Causes include CNS trauma or infection, brain tumors, cerebral aneurysms, granulomatous diseases (TB, sarcoid, histiocytosis X).
2. Nephrogenic DI: no change in urine osm with DDAVP. Causes include lithium, demeclocycline, amphotericin B, V2 receptor antagonists, foscarnet, hypercalcemia, hypokalemia, renal disease.
5. Net Hypotonic Fluid Loss as a cause
   - Renal causes: loop diuretics, osmotic diuresis (e.g. glucose), post-obstructive diuresis, polyuric phase of ATN, intrinsic renal disease. Generally, these patients are hypovolemic, urine osm > 500, urine output > 1 L/day, urine Na+ > 20.
   - GI causes: emesis, diarrhea, NG suction, enterocutaneous fistula, use of osmotic cathartic agents (lactulose etc.).
   - Cutaneous causes: burns, excessive perspiration
6. Net Hypertonic Sodium Gain as a cause
   - Hypertonic sodium bicarbonate infusion, hypertonic feeding preparations, ingestion of a lot of sodium chloride (salt lick?), ingestion of sea water, primary hyperaldosteronism, Cushing’s syndrome, hypertonic dialysis, infusion of hypertonic saline, sodium chloride rich emetics. Patient is usually euvoletic to hypervolemic, urine osm > 500, urine Na+ > 100. It is hard to get this if patient has normal renal function!
7. Treatment
   - If sodium is corrected too rapidly, patient may develop cerebral edema.
   - Maximum correction per day is 10 meq/L.
   - Calculate the Water deficit and replace the deficit according to the above goal. May want to check lytes q6 hours to make sure you are not over correcting or that you are correcting fast enough. Also, add about 30 cc per hour of insensible losses.
   - Water Deficit: TBW X (1 – 140/serum sodium). TBW is weight in kg X 0.4 in women and 0.6 in men.
   - If patient is hypovolemic, fill the tank first with NS (sodium cannot get higher than 154 meq) and then work on the rest of the free water deficit with ½ NS.
   - If patient is hypervolemic, D5W is usually the best IVF to use.
   - Remember to use NG/OG to give free water. As an example you can write: “250 cc of free water NG q 6 hours.”
   - With diabetes insipidus, it is a different story and you should call the renal fellow.
Hypokalemia

1. Usually, patients with mild hypokalemia are asymptomatic. Non-specific symptoms include: lassitude, constipation, generalized weakness. With levels less than 2.5, muscle necrosis can occur and with levels less than 2, ascending paralysis is possible.
2. EKG changes: T wave flattening, ST segment depression, U waves, QT prolongation, arrhythmias.
3. Causes:
   - Decreased intake (i.e. alcoholics)
   - GI losses: infectious diarrhea, tumors (VIPoma, villous adenoma, ZE syndrome), jejunoileal bypass, malabsorption, chemotherapy, radiation enteropathy.
   - Renal losses: Diuretics, mineralocorticoids (fludrocortisone), high dose glucocorticoids, drugs associated with magnesium depletion (aminoglycosides, cisplatin, amphotericin), high dose abx, mineralocorticoid excess (primary hyperaldosteronism, congenital adrenal hyperplasia, rennin secreting tumor, Cushing’s syndrome, renovascular/malignant hypertension, vasculitis), apparent mineralocorticoid excess (Liddle’s syndrome), impaired chloride associated transport (Gitelman’s and Bartter’s syndrome).
   - Transcellular Shift: β2 agonists (epinephrine, pseudoephedrine), bronchodilators, tocolytics, theophylline, caffeine, insulin overdose, verapamil overdose, alkalosis.
4. Treatment:
   - Make sure you check the magnesium, because you need to replete that too- via IV.
   - Check the creatinine! Give K+ carefully to those with ARF/CKD. Call renal fellow if you have any questions. Generally, you do not correct the K+ and Magnesium of patients with ESRD.
   - PO and IV are equivalent. In general, 10 meq of KCl will raise the serum K+ by 0.1 meq/L. If the K+ is low enough, you will often want to combine PO and IV.
   - You can give 10 meq per hour of KCl through a peripheral IV and 20 meq per hour through a central line.

Hyperkalemia

1. ECG changes: peaked T waves, shortened QT → PR and QRS lengthening → disappearance of P waves → sine wave.
3. Treatment: “C BIG K DROP”
   - C- 1 to 2 Amps of Calcium Chloride to stabilize myocardium if ECG changes
   - B- Beta agonists: albuterol given as 3 continuous nebs; Bicarb: 2 Amps NaHCO3
   - I- Insulin: 10 units regular insulin IV
   - G- Glucose: 1 to 2 AMPs of D50.
   - K- kayexalate: 30 to 60 grams PO- need to make em poop to work- overall controversial tx
   - D- Diuretics (lasix), Dialysis
   - R- Repeat the lab draw to make sure value is correct.
   - O- Observation via telemetry at least.
   - P-
3. Caution in ESRD!
   - They can tolerate higher levels of K as it is a chronic issue - Check the ECG!
   - Check Phos and Ca and calculate Ca/Phos product (multiply the 2 values) prior to giving calcium - if higher than approximately 70 high risk of precipitation and ectopic calcification

**Hypomagnesemia**

1. ECG changes: PR and QT prolongation
2. Symptoms: tremor, fasciculations, ataxia, tetany, seizure
3. Often associated with hypocalcemia, hypokalemia
4. Replete - if in the hospital use IV as PO is rarely effective - causes diarrhea.
5. If patient has ESRD, do not replete the magnesium unless cleared by fellow.
6. General Sliding Scale:

<table>
<thead>
<tr>
<th>Serum Magnesium</th>
<th>Magnesium Sulfate Dose IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.8 to 1.9</td>
<td>1 gram</td>
</tr>
<tr>
<td>1.5 to 1.7</td>
<td>2 grams</td>
</tr>
<tr>
<td>1.2 to 1.4</td>
<td>3 grams</td>
</tr>
<tr>
<td>&lt; 1.2</td>
<td>4 grams and recheck</td>
</tr>
</tbody>
</table>

**Hypophosphatemia**

1. Again, watch repletion in ARF/CKD and generally do not replete in someone with ESRD unless cleared by renal fellow.
2. Follow Ca, K, and Mg levels as you are repleting.
3. IV repletion: 15 mmol of Kphos (has 22 meq of K+), Sodium phos.
4. PO repletion: Kphos or Neutraphos: one to two tabs TID to QID; can also give skim milk with meals also.

**Hyperphosphatemia**

1. You will usually see this in patients with ESRD.
2. If really high, calculate the Calcium-Phos product by multiplying serum calcium and serum phos. If > 70, worry about ectopic calcifications.
3. Treatment usually proceeds by giving a medication which binds phosphorus in the gut. If calcium is high, do not give calcium-containing compounds. Give with meals. What you give will also depend what is on formulary at the specific hospital.
   - Calcium carbonate
   - Calcium acetate (PhosLo)
   - Aluminum Hydroxide (Amphogel)
   - Renagel.

**Hyper- and Hypocalcemia - see endocrine section.**

**Acute Kidney Injury (A.K.A. Acute Renal Failure)**

1. Definition: Increase in Cr of 0.5 mg/dL, within 1 week or less, if baseline is less than 2.5 mg/dL or an increase in serum Cr by more than 20% if baseline is greater than 2.5.
2. Associated with a 20% mortality rate.
3. Three Causes:
   - **Pre-renal:** conditions leading to decreased renal perfusion.
        Inadequate cardiac output.
     2. Hypotension: sepsis, anesthesia and medication induced, hepatorenal syndrome, relative
        hypotension.
     3. Pharmacologic: NSAIDs, ACE inhibitors, contrast dye.
     4. Large vessel: thrombosis, embolus, dissection, RAS.
   - **Intra-renal:** diseases or states that affect the nephron (e.g. glomeruli, tubules, vessels, or
     interstitium). Most common is ATN.
     1. Small vessel: atheroembolism, malignant hypertension, scleroderma, TTP/HUS, DIC.
     2. Glomeruli: acute or rapidly progressive glomerulonephritis, vasculitis.
     3. Tubules: ATN (causes include hypovolemia, hypotension, sepsis, IV contrast,
        aminoglycosides, amphotericin B, cisplatin, myoglobin, hemoglobin), obstruction (uric acid,
        calcium oxalate, acyclovir, indinavir, light chains).
     4. Interstitium: AIN (NSAIDS, antibiotics, pyelonephritis, infiltration by lymphoma/sarcoid).
   - **Post-renal:** obstruction from renal pelvis to urethra.
     1. Ureteral: tumors, calculi, clot, sloughed papillae, retroperitoneal fibrosis, lymphadenopathy.
     2. Bladder neck: tumor, thromboemboli, calculi, prostatic hypertrophy or carcinoma,
        neurogenic.

**Diagnosis:**

<table>
<thead>
<tr>
<th></th>
<th>Pre-renal</th>
<th>Post-renal</th>
<th>ATN</th>
<th>Acute GN</th>
<th>AIN</th>
</tr>
</thead>
<tbody>
<tr>
<td>BUN:Cr</td>
<td>&gt; 20:1</td>
<td>&gt; 20:1</td>
<td>&lt; 20:1</td>
<td>&gt; 20:1</td>
<td>&lt; 20:1</td>
</tr>
<tr>
<td>Urine Na</td>
<td>&gt; 20:1</td>
<td>Varies</td>
<td>&gt; 40</td>
<td>&lt; 20</td>
<td>&lt; 20 (acute), &gt; 40 (few days)</td>
</tr>
<tr>
<td>Urine Osm</td>
<td>&gt; 500</td>
<td>&lt; 400</td>
<td>&lt;400</td>
<td>&gt; 400</td>
<td>Variable</td>
</tr>
<tr>
<td>FeNa (%)</td>
<td>&lt; 1</td>
<td>Varies</td>
<td>&gt; 2</td>
<td>&lt; 1</td>
<td>&lt; 1 (acute), &gt; 1 (few days)</td>
</tr>
<tr>
<td>FeUrea (%)</td>
<td>&lt; 35%</td>
<td>Varies</td>
<td>&gt; 35%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urinary Sediment</td>
<td>Benign or hyaline casts</td>
<td>Normal or red cells, white cells, or crystals</td>
<td>Renal tubular epithelial cells, granular and muddy brown casts.</td>
<td>Dysmorphic red cells and red cell casts.</td>
<td>White cells, white cell casts, with or without eosinophils. + Hansel’s stain.</td>
</tr>
</tbody>
</table>

4. Further Diagnostic Studies to Consider:
   - Renal Ultrasound.
   - Spot urine protein and creatinine to calculate the urine protein:Cr. Good estimate for 24 hour
     protein excretion.
   - Place foley especially if anuric or worried about obstruction.
   - Follow urine output closely.
CK if worried about rhabdomyolysis.


CKD Stages
- Stage 1: Kidney damage with normal or increased GFR. GFR $\geq 90$ mL/min/1.73m$^2$.
- Stage 2: GFR 60 – 89 mL/min/1.73m$^2$
- Stage 3: GFR 30 – 59 mL/min/1.73m$^2$
- Stage 4: GFR 15 – 29 mL/min/1.73m$^2$. Preparation for dialysis (i.e. access planning).
- Stage 5: GFR < 15 mL/min/1.73m$^2$ or on dialysis.

Renal Replacement Therapies (RRT)
1. Indications for emergent dialysis:
   - Acidosis (esp. if pH < 7.2)
   - Electrolytes (e.g. hyperkalemia- with EKG changes and/or level > 6.5)
   - Ingestion (such as ethylene glycol, lithium)
   - Volume overload
   - Uremia

2. RRT Modalities
   - There are two main types of dialysis, intermittent and continuous. The difference between the two is duration. Intermittent is usually done 4-6 hours per day and continuous is done 24 hours per day.

3. Indications
   - All RRT therapies have the same indications: severe acidemia, electrolyte abnormalities (hyperkalemia), volume overload, uremia, and drug overdoses.
   - Continuous renal replacement therapies (CRRT) are primarily used in the ICU setting in hemodynamically unstable patients. Intermittent hemodialysis (IHD) is used in hemodynamically stable patients.
   - Both modalities are effective at removing solutes, but IHD allows for faster removal of solutes. CRRT allows for slower removal of volume and solutes resulting in less fluctuation of solutes and continuous volume control.
   - The disadvantages of CRRT include 1) immobilization, as patients are connected to a machine 24 hours per day; 2) cloting, therefore, there is a need for anticoagulation with heparin; 3) higher costs; 4) higher demands on nursing time.
   - The disadvantage of IHD is more significant volume shifts which can result in hemodynamic instability.

4. Three types of “dialysis”
   - 1. Hemodialysis: Solute removal by diffusion. Solute passively diffuse down their concentration gradient. Dialysate is run countercurrent to the patient’s blood flow to increase the gradient. Urea, creatinine, and potassium move from the blood to the dialysate. Calcium and bicarbonate move from the dialysate to the blood. No volume is removed.
   - 2. Hemofiltration or Ultrafiltration: Solute and volume removal, although the main purpose is to remove volume. Solute removal is by convection (solute drag) and solutes are only significantly removed if it is done for a prolonged period of time. Transmembrane pressure
gradient drives water across a semi permeable membrane and “drags” solutes with it. Replacement fluid is needed, as high ultrafiltration causes volume contraction and can lead to hypotension.

3. Hemodiafiltration: Combination of both hemodialysis and hemofiltration. Solutes are removed by both diffusion and convection. Volume is also removed and, therefore, a replacement fluid is needed. This is typical of outpatient dialysis.

5. IHD
   - Standard IHD: Uses one of the above 3 types of dialysis, done 4-6 hours per day. This is the type of dialysis mainly used in patients with ESRD in an outpatient setting.
   - Sustained Low Efficiency Dialysis (SLED): Is slower IHD done for a longer period of time (6-12 hours). Allows for hemodynamic stability as has slower removal of solutes and volume. This is becoming more popular and used frequently at Rose Hospital.

6. CRRT (done 24 hours/day)
   - Slow continuous ultrafiltration (SCUF): Dehydrating procedure used only for volume overload. No intention of removing solutes. Transmembrane pressure gradient slowly drives water across a semi permeable membrane. No replacement fluid is needed as goal is to remove fluid. 6-7 L of fluid can be removed daily.
   - Continuous Venovenous Hemofiltration (CVVH): Hemofiltration only, no hemodialysis. Solutes removed by convection only and are efficiently removed over 24 hours. Replacement fluid is needed as a large amount of volume is being removed.
   - Continuous Venovenous Hemodialysis (CVVHD): Solutes removed by diffusion. Allows for greater solute removal than CVVH. There is no volume removal as no hemofiltration is done and therefore there is no replacement fluid.
   - Continuous Venovenous Hemodiafiltration (CVVHDF): Combination of CVVH and CVVHD. Solutes removed by convection and diffusion. Volume can be removed and replacement fluid is needed.

7. The choice of RRT needs to be individualized. Identify the primary goal for the patient, i.e. fluid removal, solute removal, or both. Choice will also depend on what machines, nursing staff, etc are available. Use the method with less side effects and greatest benefit in a given case. Treatment of the underlying condition outweighs the choice of RRT.
Seizures are a source of great excitement and drama for most nurses and housestaff, usually unnecessarily so. No doubt they are difficult to watch, but if you can lend a little structure to the inevitable chaos of the seizing patient, it will be easier on everyone. The first step is always the same—ensure the safety of the patient, then once the dust has settled you can figure out what triggered the seizure.

The approach to the seizing patient:
1. Remain calm, and try to keep everyone around you calm as well.
2. Note the time of onset and duration of the seizure—this means looking at your watch, because it will always seem longer than you think if you’re just estimating.
3. Check a STAT finger-stick blood glucose.
4. Try to determine if the seizure was focal or generalized in its onset. This is often difficult information to come by.
5. There usually isn’t any reason to call a COR.
6. Keep the patient safe. Place them in the left lateral decubitus position to prevent aspiration, and try to keep them from banging their head and limbs against bedrails, the floor, etc.
7. Don’t put anything in the seizing patient’s mouth.
8. Most seizures are self-limited and brief, with most lasting less than one minute. When you get called about a seizure, have the nurse have lorazepam 4 mg IV ready at the bedside, but you DO NOT need to give it unless the seizure has not stopped by two minutes. If it hasn’t self-terminated by two minutes, the patient is at relatively high risk for ongoing seizures and even status, so must be treated.
9. If the seizure is still going at two minutes, give 2 mg of lorazepam IV. Repeat lorazepam 1-2 mg every one to two minutes as needed until seizures have stopped. Watch for respiratory depression with higher doses of lorazepam. Consider transfer to higher level of care if needed. Consider phenytoin load if long-lasting seizure.
10. Evaluate for underlying cause of seizure once seizure has terminated.

New-onset seizures in the hospitalized patient are usually provoked, meaning that there is some underlying identifiable cause such as a metabolic abnormality, infection, etc. that will explain the seizure.

Differential Diagnosis:
- *Metabolic*: hypo- or hypernatremia, hypo- or hyperglycemia, hypo- or hypercalcemia, hypomagnesemia, hyperosmolality, uremia, hyperammonemia, hyperthyroidism
- *Toxic*: PCP, cocaine, or amphetamine intoxication; ETOH withdrawal
- Drugs: haloperidol, TCA’s, levofloxacin, imipenem, penicillins, cephalosporins, bupropion, amantadine, meperidine, fentanyl, anticholinergics, lidocaine, procaine, methotrexate, vincristine
- Trauma: subdural or epidural hematoma, subarachnoid hemorrhage
- Vascular: ischemic stroke, cerebral hemorrhage
- Infection: meningitis, encephalitis
- Tumor: metastatic disease, primary brain tumor

History:
- Obtain all available history regarding seizure from witnesses
- Review chart
- Any prior history of seizures or epilepsy?
- What is the admitting diagnosis?
- Any current infections? Any history of HIV?
- What are the medical comorbidities, i.e. renal failure, hepatic failure, hypoxia?
- Any h/o ETOH or drug abuse or withdrawal?
- What are the recent labs?
- Review the patient’s medications carefully

Physical Examination:
- Vitals: look for interictal arrhythmias, fever, hypertension, hypoxia
- Check lateral aspect of tongue for laceration after seizure
- Check for any evidence of head injury
- Check for papilledema
- Check for meningismus
- Perform careful pulmonary exam, listening for decreased breath sounds or rales that may suggest aspiration during seizure
- Look for urinary or fecal incontinence
- Briefly assess level of alertness after seizure—orientation, simple commands, counting backwards, etc.
- Look for any signs of hemiparesis, reflex asymmetry, etc.

Diagnostic Work-up:
- CBC, CMP, Mg
- Consider utox, lumbar puncture, TSH, alcohol level if clinically indicated
- If on any antiepileptic medications, check appropriate levels
- Check routine bedside EEG
- If the patient has a focal deficit on neurological exam and/or no other cause is identified on the above studies, obtain CT or MRI of brain with and without contrast
- Serum prolactin levels may be elevated for up to one hour following a seizure
- Seizures also result in mild peripheral leukocytosis due to demargination of neutrophils, elevated CPK, anion gap metabolic acidosis, and elevated serum lactate; these are all transient and resolve within a few hours

Treatment:
- Make sure the patient has adequate IV access, i.e. two working peripheral IV’s.
- Treat any underlying metabolic, infectious, or other cause identified on the above evaluation.
For a single first-time seizure that has stopped on its own, no treatment is warranted beyond treating any provoking factors.

Antiepileptic drugs are reserved for patients with status epilepticus, those with multiple seizures, and those with risk factors that make them more likely to continue to experience seizures.

If the patient is in status, treat as per the status epilepticus protocol provided in a separate section.

If a structural lesion is found or if there is a metabolic insult that cannot be rapidly treated, the patient should be loaded with phenytoin 20 mg/kg IV or fosphenytoin 20 PE/kg IV.

Place the patient on seizure precautions.

**Headache Emergencies**

Headache is one of the most common complaints among patients in the clinic, in the ED, and on the wards, so it’s important to know when a headache indicates a life-threatening process. This is sometimes easier said than done.

When someone says they have the “worst headache of my life,” what does that really mean? That’s going to happen to everyone at some point—if you’ve never had a headache before, any headache will be the worst headache of your life. Most of the time it’s nothing, and you don’t need to proceed with imaging, LP, labs, or any other workup.

However, there are the minority of headache cases that are something more major, with potentially lethal underlying etiologies that must be diagnosed early and treated appropriately. So, what signs or symptoms should you worry about?

There are several “red flags” in headaches that should push you to undertake a more thorough evaluation of the headache patient. These include:

- Focal neurological deficits
- Fever
- Meningismus
- Papilledema
- Associated seizures
- Abrupt onset with pain maximal in intensity at onset (thunderclap headache)
- Onset of frequent headaches after age 50 without prior history of headaches
- Altered level of consciousness
- Headache worsening with valsalva, bending over, cough
- Headache awakening patient from sleep
- Change from prior headache pattern

**Differential Diagnosis:**

- Subarachnoid hemorrhage
- Intraparenchymal cerebral hemorrhage
- Meningitis or encephalitis
- Cerebral venous sinus thrombosis
- Pituitary apoplexy
- Brain tumor
- Brain abscess
• Spontaneous intracranial hypotension
• Subdural or epidural hematoma
• Extracranial vascular dissection (carotid or vertebral arteries)
• Complicated migraine

**Careful History:**
- Ask about any prior headaches (character, location, quality, duration, associated symptoms) for comparison to current headache
- Ask about current headache (rate of onset, intensity, quality, location, associated symptoms, alleviating factors, exacerbating factors)
- Is it worse with Valsalva? Cough? Sneeze?
- Are headaches worse in the morning or do they awaken the patient from sleep?
- Are they associated with nausea and/or vomiting?
- Is there any associated photophobia or phonophobia?
- Is there any history of head trauma, either recent or remote?
- Is there any seizure activity noted?
- Is there any fever or other evidence of systemic illness or infection?

**Careful Physical Examination:**
- Check the vitals: fever, tachycardia, Cushing’s reflex (hypertension with bradycardia, associated with increased intracranial pressure)
- Look for any focal neurological deficits on exam
- Cranial nerve VI palsies are common false localizing signs with increased ICP

**Diagnostic Work-up:**
- CT head without contrast is usually the first step, since intracranial hemorrhage is a chief concern in abrupt-onset severe headaches.
  - Sensitivity of CT for acute intracranial bleeds approaches 100% in the first 12-24 hours, but drops after 24 hours as well as with small volumes of blood and anemia (hematocrit <30).
- If CT of the head is negative and clinical suspicion for subarachnoid hemorrhage remains high, you MUST do a lumbar puncture to rule out this diagnosis.
  - CSF will show elevated RBC’s that do NOT clear from tube #1 to tube #4 and elevated protein.
  - Xanthochromia (yellowing of the CSF) may also be seen, but remember that this takes 8-12 hours to occur as it is the result of the breakdown of the RBC’s within the CSF, which takes time.
- LP if evidence of infection.
- Consider MRI if CT has not provided definitive diagnosis.
- Use your neurology consultants early and often.

**Notes:**
- In general, migraines, cluster headaches, and tension headaches tend to be fairly stereotypical within the same patient.
- Seizures are decidedly uncommon as a feature of any of the primary headache syndromes (i.e. migraine, cluster, tension) and suggest an underlying structural CNS lesion.
• Papilledema takes at least 24-48 hours to form and so will not be present in the acute setting. If it is seen, it implies possible increased intracranial pressure.
• Migraines, cluster HA’s, and tension HA’s are gradual in onset, building in intensity over several minutes to even hours. True “thunderclap” headaches from subarachnoid bleeds, etc. are dramatic and abrupt in onset, with pain that is maximal from the beginning.
• Onset of primary headache syndromes after the age of 50 years is uncommon, so the older patient with new-onset frequent headaches must be evaluated for potential sources such as tumor or chronic subdural hematoma.
• None of the primary headache syndromes should produce a frank alteration in consciousness.
• While focal neurological deficits may be part of a patient’s migraine aura or even a complicated migraine, remember that the patient will often have a history of similar events in the past to help clarify this question. When in doubt, get a scan and work them up.

CNS Infections

Clinical Presentation:
• Bacterial meningitis typically presents with fever, stiff neck, and headache with progressive deterioration in level of consciousness. Seizures and focal neurological deficits can also occur.
• Viral meningitis typically presents with fever, headache, stiff neck, and constitutional signs of viral infection such as GI symptoms or URI. Significant alteration in consciousness does not occur and should suggest another etiology.
• Encephalitis typically presents with fever, headache, altered mental status, and focal neurological signs.
• Presence of a diffuse maculopapular rash suggests either enterovirus or meningococcus as the causative organism.
  o The rash of *Neisseria meningitides* begins as a maculopapular rash on the trunk and lower extremities that rapidly becomes petechial or purpuric and spreads to involve the conjunctivae and mucous membranes.
  o The rash of enterovirus starts on the face and trunk then spreads to the extremities.
• Rashes due to antibiotics usually start on the face, spread to the chest and trunk, and then to the lower extremities. By the time it reaches the lower extremities, the rash on the face is often nearly gone.

Epidemiology:
• In adults 15-50 years, *Streptococcus pneumoniae* and *N. meningitides* are the most common organisms causing community-acquired bacterial meningitis.
• Pneumonia, acute sinusitis, and acute otitis media are common preceding illnesses for pneumococcal meningitis.
• Risk factors for pneumococcal meningitis: complement deficiency, hypogammaglobulinemia, splenectomy, head trauma with basilar skull fracture, ETOHism, diabetes, sickle cell disease, thalassemia major, multiple myeloma.
• Patients with defects in cell-mediated immunity must be covered against *Listeria monocytogenes*. (This includes those with HIV, pregnancy, hematologic malignancies, chronic steroid use, cancer, chemotherapy, organ transplantation, alcoholism, the elderly, and infants.)
• In patients who have undergone neurosurgical procedures, excluding shunts and Ommaya reservoirs, the most common organisms causing meningitis are gram-negative bacilli and staphylococci.
• In patients with CSF shunts and Ommaya reservoirs, coagulase negative staphylococci and *Staphylococcus aureus* are the most common organisms.
Differential Diagnosis:
- DDx for fever, headache, stiff neck: bacterial meningitis, viral meningitis, fungal meningitis, tuberculous meningitis, drug-induced hypersensitivity meningitis, carcinomatous meningitis, meningitis due to inflammatory disorders (SLE, sarcoidosis, Sjögren syndrome), subarachnoid hemorrhage.
- DDx for fever, headache, stiff neck plus altered level of consciousness, seizures, or focal neurological deficit: bacterial meningitis, viral encephalitis, Rocky Mountain Spotted Fever, fungal meningitis, brain abscess, epidural abscess, subdural empyema, venous sinus thrombosis, subarachnoid hemorrhage.

Evaluation:
- Immediate blood cultures and empiric antibiotic and adjunctive therapy should be initiated prior to lumbar puncture and before head CT.
- You do NOT need a CT prior to lumbar puncture in every patient!
- In patients with suspected bacterial meningitis, you should get a CT before LP if one or more of the following is present: (1) focal neurological deficit; (2) new-onset seizure; (3) papilledema; (4) altered level of consciousness; or (5) immunocompromised state.
- CT cannot reliably predict whether a patient will or will not herniate after LP in bacterial meningitis in the absence of focal asymmetric parenchymal lesions!

Perform lumbar puncture if safe:
- Never defer antibiotics until after the LP is done.
- CSF gram stain and culture results may be affected if antibiotics are given for several hours prior to LP, but CSF cell count and diff, glucose, and protein will not be affected enough that a diagnosis of bacterial meningitis cannot be inferred.
- Always obtain opening pressure on the LP.
- Send CSF for cell count and differential on tubes 1 and 4, glucose, protein, Gram stain, and culture to start. You can add viral PCRs, fungal cultures, VDRL, and other studies depending on the initial CSF profile.
- Typical CSF profile in bacterial meningitis: opening pressure greater than 18 cm H$_2$O; polymorphonuclear (PMN) leukocytosis; elevated protein; low glucose (CSF-blood glucose ratio less than 0.6).
- Typical CSF profile in viral meningitis: opening pressure usually normal (less than 18 cm H$_2$O); lymphocytic leukocytosis (may be PMN predominant in first 24 hours); normal to slightly elevated protein; normal glucose.

Remember, LP is never a life-saving procedure!
1) If concerned about increased intracranial pressure, consider a bolus of IV mannitol 20 mg/kg, then LP 20 minutes later with careful measurement of opening pressure. If opening pressure is markedly elevated (greater than 35-40 cm H$_2$O), stop the LP and collect the CSF in the manometer; this will usually give you enough fluid for basic CSF studies to make a diagnosis.
2) If there is significant concern about herniation with LP, the procedure can be deferred while the patient is empirically treated with antibiotics until the patient stabilizes enough that LP is deemed safe or the organism if identified by blood culture.

Treatment:
Empiric antibiotic selection based on age, associated premorbid conditions, and possibility that a penicillin- and cephalosporin-resistant strain of *S. pneumoniae* is responsible for the patient’s meningitis.

- For patients 15-50 years: ceftriaxone 2 grams IV every 12 hours, vancomycin one gram IV every 12 hours, and acyclovir 10 mg/kg IV every 8 hours.
- For patients over the age of 50 and in immunocompromised patients, add ampicillin 2 grams IV every 4 hours to the above regimen to cover against *Listeria*.
- Dexamethasone 10 mg IV every 6 hours should be given for a total of four days, with the first dose administered with or 15 to 20 minutes prior to the first dose of antibiotics.
- If initial CSF profile is consistent with bacterial meningitis, acyclovir can be discontinued.
- Antibiotic regimen can be refined once Gram stain and culture results with sensitivities return.
- Duration of therapy: *S. pneumoniae* or *H. influenzae* 10-14 days IV abx, *N. meningitides* 5-7 days of IV abx followed by 2 days of oral rifampin, *Listeria* 3-4 weeks of IV abx.
- Patients with suspected meningococcal meningitis must be placed in isolation for the first 24 hours after initiation of antibiotic therapy.
- Anyone who has come into contact with a patient with meningococcal meningitis must receive prophylactic antibiotics with rifampin 600 mg bid for 2 days, a single dose of ceftriaxone 250 mg IM, or a single dose of ciprofloxacin 500 mg PO.
- Contacts of patients with *H. influenzae* meningitis must receive prophylactic treatment with rifampin 600 mg daily for four days
- Viral meningitis requires supportive care only.
- Headache in viral meningitis may persist for months and is best managed with NSAIDs and amitriptyline.

**Prevention:**

- Those over age 65, those with asplenia, and those over age 2 years who are at risk for pneumococcal disease as a result of chronic illness should receive the pneumococcal vaccine, which confers protective antibody levels for 5 years in most adults.
- The meningococcal conjugate vaccine is recommended prior to entry into high school.

**Notes:**

- Mortality in bacterial meningitis remains 25% despite optimal therapy.
- 50% of patients develop complications such as cerebral edema, hydrocephalus, septic venous sinus thrombosis, arteritis, seizures, cranial nerve palsies, septic shock, DIC, renal failure, SIADH, cerebral salt wasting syndrome, and ARDS.
- In HIV patients, opportunistic agents to consider include *Toxoplasma*, CMV, EBV, VZV, JC virus (causes progressive multifocal leukoencephalopathy, PML), TB, syphilis, *Nocardia*, *Cryptococcus*, *Candida*, *Aspergillus*, *Coccidioides*, and *Histoplasma*.
- In transplant patients, consider fungi, molds, toxo, CMV, VZV, EBV, HHV-6.
- A brief word about herpes encephalitis:
  - 90% of cases are due to reactivation of HSV-1, usually in previously healthy patients.
    - Fever, headache, and altered mental status are invariably present.
    - Other common findings include seizures, aphasia, weakness, impaired memory, and hallucinations.
    - MRI reveals hyperintensities in one or both frontotemporal regions.
    - LP shows increased opening pressure, lymphocytic pleocytosis, elevated protein, and normal glucose.
• CSF HSV PCR is 95% sensitive and 100% specific, but can be falsely negative within the first 72 hours of symptoms. If clinical suspicion is high with a negative HSV PCR, the patient should be treated empirically with acyclovir and the LP and PCR repeated in 2-3 days.
• Treatment is acyclovir 10 mg/kg IV every 8 hours for 14-21 days. Mortality is 70% without treatment, but drops to 19% with therapy. If the patient survives the initial infection, morbidity is nearly 100% and varies from mild cognitive impairments to more significant deficits such as hemiparesis or epilepsy.

- A bloody CSF can alter some results of the LP:
- WBC count is increased by 1 WBC/mm³ for every 700 RBC’s/mm³.
- CSF protein concentration will be increased by 1 mg/dL for every 1000 RBC’s /mm³.
- If CSF is contaminated by blood, a false-positive CSF EBV PCR and a false-positive CSF VDRL may occur.
- If CSF is contaminated by blood, a false-negative CSF HSV PCR may result.

Anoxic Brain Injury

Causes:
Cardiopulmonary arrest and other causes of decreased cardiac output, prolonged hypotension, prolonged hypoxia, prolonged hyperventilation. It only takes 2-5 minutes of ANOXIA to cause irreversible damage, but longer periods of hypoxia or decreased cerebral perfusion will cause permanent injury as well.

Clinical Presentation:
Coma, lack of purposeful responsiveness to stimulation and pain, myoclonus, decorticate (flexor) or decerebrate (extensor) posturing, loss of some or all brain stem reflexes (i.e. blown pupil, decreased blink reflex, loss of gag/respiratory drive)

Initial Workup after patient stabilized:
• Imaging: Head CT: r/o acute bleed, severe cerebral edema, or mass lesion as explanation.
• Labs: LFT’s, Chem 7, Mg, Phos, Ca, ABG. Important to r/o severe metabolic disturbances as they confound the neurologic exam, but may be reversible

Initial Management:
• Cooling Protocol, DON’T WAIT. Goal: hypothermia initiated within 90 minutes, for patients who have no ongoing cardiac dysrhythmia. Hypothermia to 33°C with external cooling blankets (or per hospital protocol) with use of paralytics. Hypothermia to be maintained for 24 hours followed by passive rewarming.
• Avoid neuromuscular blockers, barbiturates, narcotics, and benzo’s OUTSIDE of the cooling protocol. These medications can suppress brainstem reflexes and interfere with the neurological exam and prognostication. If there truly is an anoxic injury, there should not be a significant need to “sedate” patient.
• If cerebral edema is present, elevate head of bed, hyperventilate, and avoid excess IVF. Serial Head CT’s q12-48 hours depending on severity. No evidence that mannitol or steroids improve outcome if etiology is anoxia, but may be used in dire situations. If signs of impending herniation (third nerve palsy or dropping brain stem reflexes) or head CT demonstrates severe edema, neurosurgical consult for intervention.
• Neurology consult for prognosis. They will want the patient to be OFF cooling protocol and OFF all sedating medications to perform meaningful examination.
Document type of arrest or arrhythmia if known, duration of CPR, and time to return to normal vital signs. These things provide important information about prognosis.

**Prognostic Hints (Evidence Level in parentheses):**

1. Serum NSE (neuron specific enolase) levels >33 μg/L at days one to three post-CPR accurately predict poor outcome (Level B).
2. The assessment of poor prognosis can be guided by the presence of bilaterally absent cortical SSEPs (N20 response) within one to three days (Level B).
3. The prognosis is invariably poor in comatose patients with absent pupillary or corneal reflexes, or absent or extensor motor responses three days after cardiac arrest (Level A).
4. Patients with myoclonus status epilepticus within the first day after a primary circulatory arrest have a poor prognosis (Level B).
5. There are inadequate data to support or refute whether neuroimaging is indicative of poor outcome (Level U).

**Acute Spinal Cord Injury/Compression**

Acute Spinal Cord Injury is most often due to trauma, with MVA’s as the most common cause, and will be recognized/managed in the ED, but acute nontraumatic compression can occur in the inpatient setting.

Causes of Inpatient Acute Spinal Cord Injury or Compression: Epidural Abscess (think of those with Diabetes, ETOH, IVDA or multiple comorbidities with prolonged hospital stay), SC ischemia (operative complication, embolic infarct, or decreased perfusion), Metastatic tumor, Primary tumor, or Epidural Hematoma (post procedure or coagulopathy)

**Clinical Manifestation of Acute Cord Compression:**

- Can have symptoms stemming from nerve roots or the cord, depending on site of compression
- Epidural abscess presents as neck or back pain that is relentless when patient lies in bed. It often progresses to paresthesias, radiating pain that has radicular nature. Paralysis is late finding.
- If cord compressed before roots, usually see sensory loss, weakness, impairment of cutaneous and proprioceptive sensation below the lesion. May have incomplete compression, so can have crossed findings if Brown-Sequard syndrome (hemi-cord).
- Poor control of bladder usually proceeds bowel dysfunction acutely.
- In cauda equina or conus compression, pain in lower back, rectum, and legs with early loss of bladder function and impotence. Saddle anesthesia.
- In the first 24-48 hours of compression, expect a lower motor neuron pattern with flaccid tone and hyporeflexia that transitions to spasticity and hyperreflexia. If left untreated, will progress to wasting and atrophy at level of lesion and spastic paraplegia or quadriplegia below the lesion.

**Initial Evaluation:**

- EMERGENT MRI with and without contrast of region of spine of interest is GOLD STANDARD. If spinal cord compromise is suspected, cannot wait until next day!! CT of spine will not show soft tissue or abscess compression of cord unless bones are displaced and is an inadequate study to r/o compression.
- DO NOT LP if epidural abscess is suspected. LP can seed the CSF if needle transects pus pocket. Wait for imaging and neurology consult to guide.
• If abscess suspected, try to isolate etiology. Blood Cultures, Urine Cultures, Chest X-ray. ESR (often elevated in epidural abscess).
• Check Post Void Residual

Management:
• **Urgent Neurology and/or Neurosurgery Consult if appropriate.**
• Emergent Decompression indicated if cord compromise due to surgical lesion
• Urgent Radiation therapy if non-resectable metastatic disease
• If Epidural Abscess, start empiric antibiotics with MRSA coverage until bug isolated
• In acute, noninfectious, SC injury, current recommendation is to treat with steroids within 8 hours of injury: starting methylprednisolone 30mg/kg bolus over 15 minutes followed by 5.4 mg/kg/h for 23 hours
• Recommendations vary for subacute cord compression (i.e. epidural abscess, tumor), but most use dexamethasone 4-20 mg q6h.

**Status Epilepticus**

**Status Status epilepticus (SE) should be considered for:**
• Generalized tonic clonic (GTC) seizure lasting longer than 5 minutes. Look for stiffening with or without rhythmic jerking, often bowel/bladder incontinence and/or tongue biting.
• Any 2 or more GTC seizures without clearing of consciousness between them.
• A GTC seizure followed by prolonged unconsciousness >15 minutes even after treatment.

**Be suspicious of ongoing seizure activity for:**
• Unconsciousness with subtle rhythmic movements (eye or limb twitching) or fluctuating vital signs.
• Unconsciousness with a history of epilepsy AND history of noncompliance or NPO.

**DON'T PANIC:**
• Stay calm, near the head of the bed. Think of it like a code. Assess the situation, time the seizure, and get assistance from the nurse immediately.
• Do not try to restrain the patient. You (and the patient) will only end up hurt.
• Err on the side of treating – better to treat a non-seizure than to wait to treat SE.
• Establish IV access ASAP if not already done.
• Be prepared to intubate if necessary. Place oxygen once patient stable.
• Notify the neurology resident for help with management.
• EEG is not necessary unless inducing chemical coma.
• Don’t stop until the seizures stop!

**Treatment protocol:**

1. **Benzos first! Choose one.**
   - Ativan 0.1mg/kg IV: load 4mg, then 2mg IV q2 min up to 8mg; onset 6-7 min, off 12 hrs
   - Valium 0.2mg/kg IV: load 10mg, then 5mg IV q2 min up to 30mg; onset 2-3 min, off 15 min

2. **Load Anti-Epileptic Drug (AED) at the same time! Choose one.**
   - Dilantin (phenytoin/PHT): 20mg/kg IV load at 50mg/min (1500mg for 70kg)
   - Cerebyx (fosphenytoin/PPT): 20 PE/kg IV load at 150mg/min
   - Depakote (valproic acid/VPA): 25mg/kg IV load at 20mg/min
   - Phenobarbital (PB): 20mg/kg IV load at 75mg/min
3. **Give more AED if still seizing! Use the same one. Check levels.**
   - PHT 10mg/kg IV, goal serum level 25-30
   - fPT 10 PE/kg IV, goal serum level 25-30 (check phenytoin level)
   - VPA 10-25mg/kg IV, goal serum level 100-150
   - PB 10mg/kg IV, goal serum level 40-45
   - LEV 1500mg IV push over 5 minutes (no level)

4. **Add a second AED (ask neurology resident for recs) if still seizing!**
5. **Give anesthetic if still seizing! EEG and intubate if not already done.**
   - Versed (midazolam) 200mcg (microgram)/kg IV load, then 0.75-10mcg/min gtt
   - Propofol 1-2mg/kg load, then 2-10mg/kg/hr gtt
   - Pentobarbital 5-20mg/kg load, then 1-4mg/kg/hr gtt

**Notes:**
- If no IV access, give Valium 10mg IM or PR to stop seizure and establish access
- PHT/fPT/PB associated with arrhythmia and respiratory suppression – **stop infusion and reduce rate**
- Phenobarbital can take up to 10 days to clear
- VPA associated with platelet dysfunction, thrombocytopenia, and increased peri-surgical bleeding risk
- Beware of “subclinical status” – uncover the patient and look for any rhythmic movement
Basics of Chest X-ray Interpretation
Remember to be systematic every time. Avoid tunnel vision and look at the whole picture.

Step 1: Is the film correct?
- Correct patient and exam?
- Correct technique?
- Check rotation – The medial ends of the clavicles should be centered over the spinous processes.
- Penetration – If correct, the vertebral bodies should be barely seen in the lower cardiac silhouette, and intervertebral spaces are just visible. Over penetrated films look darker.
- Check inspiration - The right diaphragm at full inspiration should be between the 5th and 7th rib. If there are more than 10 ribs, then the lungs are hyperinflated.
- Correct line placement (NG, central lines, pacemakers, swan-ganz, ETT)

Step 2: THE BONES – (ribs, vertebrae, scapulae, and clavicles)
- Compare symmetry and look for changes in opacity
- ? fractures, masses, arthritis

Step 3: THE SOFT TISSUES
- Compare symmetry and look for changes in density
- ? edema, subcutaneous air, breasts, masses

Step 4: THE DIAPHRAGM
- The right should be higher and both should look smooth.
- Check the costophrenic angles for sharpness.
- ? air under the diaphragm

Step 5: THE HEART AND MIDLINE STRUCTURES
- The cardiac silhouette should be less than half the width of the thoracic cage.
- ? widened mediastinum, trachea midline

radiology.creighton.edu/basic/cxray/image84.gif
Step 6: THE LUNG FIELDS

- Compare for symmetry and density
- ? lung markings to the chest wall. If not, think pneumothorax.
- Identify horizontal fissure - ? fluid

Common chest x-ray findings

- **Pneumonia** – Consolidation: ill-defined opacity without loss of volume; look for air bronchograms (a black tube within the white opacity); identify which lobes involved

- **Pulmonary edema** - looks like consolidation, but usually bilateral and near hilum; cephalization (larger lung markings in the upper fields)
- **Pneumothorax** – no lung markings and black at the periphery, tracheal shift with tension
- **Atelectasis** – area of collapse or volume loss with increased opacity
- **COPD** – hyperinflated lung fields, flattened diaphragms, apical decreased opacities with lung markings (bullae)
- **Pleural effusions** - presence of a fluid level (may just be a gradual haze), blunted costophrenic angles, fluid in the fissures

http://www.usfca.edu/fac_staff/ritter/chestxra.htm

**HYPOXIA**

Think of 5 etiologies:

1. Alveolar Hypoventilation: will be hypercarbic and have a normal A-a gradient (Age / 4).
4. Decreased FiO₂: does not happen in Denver.
5. Decreased Diffusion
Hypercarbia
Remember that PCO₂ = VCO₂ (CO₂ Production) / (minute ventilation - dead space ventilation)

Etiologies:
1) Decreased Minute Ventilation (RR X TV)
   a. Can’t Breath: Airway Obstruction (COPD, Asthma), Stiff Lungs/Pleura/Chest Wall
      (Pulmonary Edema, ARDS, PNA, effusions), Weakness (neuromuscular disorders, Hypophosphatemia)
   b. Won’t Breath: Narcotics, Benzos, Hypothyroidism, CNS Disease…
2) Increased Dead Space: COPD, PEEP, PE without ability to increase minute ventilation
3) Increased CO₂ production: Carbs or increased metabolic production

Pleural Effusions
Transudative effusions form when hydrostatic pressures favor fluid formation exceeding clearance.
Exudative effusions form through increased vascular permeability. Pleural effusions usually aren’t
visible on CXR until there is at least 300ml present. Exam is notable for decrease breath sounds,
dullness to percussion, decreased tactile fremitus, egophony, and possible friction rub.

When to tap:
• When diagnosis is unknown, symptomatic relief, new effusion (max 1500mL) or unexplained effusion
• IF A PATIENT HAS A PNEUMONIA WITH EFFUSION THAT LAYERS OUT >1cm ON LATERAL DECUB CXR, YOU HAVE TO TAP IT TO EVALUATE FOR EMPYEMA

What to order:
• serum protein and LDH (if you have one within the past 24 hours, no need to repeat), pleural fluid
  protein, LDH, glucose, gram stain, culture, pH, cytology if indicated
• if you suspect TB, order adenosine deaminase (>40), IFN Gamma (>140) or PCR
• if it’s a bloody effusion, order a hematocrit on the effusion to evaluate for hemothorax
• pH needs to go to lab in an ABG syringe that is put on ice IMMEDIATELY
• check pleural cholesterol or TG if suspect chylothorax (>110 is diagnostic)
• pleural amylase if you suspect esophageal perforation

Light’s Criteria: (exudate if any of 1 of the 3 is positive: 98% sensitive, 83% specific)
1. Pleural fluid (PF) to serum total protein ratio of >0.5
2. PF to serum LDH ratio of >0.6
3. PF LDH value of >2/3 of the upper limit of normal for serum LDH
   -can also use serum albumin-pleural albumin >1.2 g/dL: more specific

Differential Diagnosis:
Transudative Effusion: CHF, nephrotic syndrome, hypoalbuminemia, liver dx/cirrhosis, myxedema, constrictive pericarditis
Exudative Effusion: Malignancy, PE, parapneumonic effusion, TB, RA, lupus, asbestosis, pancreatitis, trauma, esophageal perf (high salivary amylase), radiation, drugs, chylothorax, yellow nail syndrome, sarcoid

Other Effusion Tips/Pearls:
• Hematocrit >50% of serum hematocrit = hemothorax
• Hematocrit of 1-20% suggests cancer, PE or trauma
Anchovy paste effusion = amoebiasis
Stinks = empyema
Low glucose: complicated parapneumonic effusion, RA, CA, TB, hemothorax
Lymphocyte-predominant: lymphoma, sarcoïd, TB, yellow nail, chylothorax
Eosinophilia: pneumothorax, asbestosis, TB, PE, fungal infxn, parasites, drugs

Management of Parapneumonic Effusions

<table>
<thead>
<tr>
<th>Description</th>
<th>Insignificant parapneumonic effusion</th>
<th>Typical parapneumonic effusion</th>
<th>Borderline complicated parapneumonic effusion</th>
<th>Complicated parapneumonic effusion</th>
<th>empyema</th>
</tr>
</thead>
<tbody>
<tr>
<td>Features</td>
<td>&lt;1 cm</td>
<td>&gt;1cm, glucose &gt;40, pH&gt;7.2</td>
<td>pH 7.0-7.2 and/or LDH&gt;1000; glucose&gt;40</td>
<td>pH&lt;7.0 and/or glucose &lt;40</td>
<td>pus</td>
</tr>
<tr>
<td></td>
<td>gram stain and cx negative</td>
<td></td>
<td>Gs and cx negative</td>
<td>and/or positive GS or cx</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>no frank pus</td>
<td>no frank pus</td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>Antibiotics only</td>
<td>Antibiotics only</td>
<td>Abx plus serial thoracentesis</td>
<td>Chest tube plus antibiotic</td>
<td>Abx plus large tube +/- decortication</td>
</tr>
</tbody>
</table>


COPD EXACERBATIONS

1. Signs and Symptoms: change in amount or character of sputum production, +/- fever, viral syndrome. Most common causes are viral, bronchospasm, and consider PE if you cannot explain the cause.
2. Labs and tests: CXR, CBC, chem.-7, CXR, ABG.
3. Treatment:
   a. Duonebs q 6 hours with prn albuterol nebs in between (can do continuous, Q 1 hour, Q 2 hour etc.)
   b. Azithromycin: IV if in the ICU or start a Z-pack.
   c. Steroids: Start with prednisone 60 mg with a taper depending upon improvement. If admitted to the ICU, give solumedrol 60 mg IV q 6 hours and then convert to oral prednisone.
   d. Non-invasive positive pressure ventilation: BiPaP if respiratory distress/ hypercapnic. This won’t work if the patient is not conscious or obtunded.
   e. Heliox is an adjunct which increases laminar flow in the airways.
   f. Smoking cessation
1. Target oxygen therapy to achieve an O2 sat of 90 – 92%

ARDS

Definition:
Acute hypoxic respiratory failure meeting the following criteria:

- Bilateral infiltrates
- No evidence of heart failure (ie, wedge pressure ≤18)
- PaO2/FiO2 is ≤200 mmHg
SEPSIS

Definitions:
Systemic Inflammatory Response Syndrome Criteria (must have 2 to have SIRS):
1) $36^\circ C \geq T \geq 38^\circ C$
2) $RR \geq 20$ breaths per minute, $PCO_2 < 32$ mm Hg
3) $HR \geq 90$ beats/min,
4) $4,000 \text{ cells/mm}^3 \geq WBC \geq 12,000 \text{ cells/mm}^3$ or $> 10\%$ bands

Sepsis: Two SIRS Criteria + Infection (+ UA, Infiltrate on CXR, etc)
Severe Sepsis: Sepsis with evidence of Organ Dysfunction, Hypoperfusion, Hypotension
Septic Shock: Severe Sepsis not responsive to fluids. Vasopressors Required.

Vasopressors and Sepsis (for dosing see table below):

Surviving Sepsis 2008 Recommendations (CCM, 2008)
a. MAP should be maintained greater than or equal to 65 mm Hg.
b. Norepinephrine as the first pressor of choice, can consider dopamine (Remember that dopamine has more chronotropic effects).
c. Vasopressin can be added to norepinephrine in anticipation that its effects are equivalent to norepinephrine alone
d. Epinephrine is the next pressor of choice if norepinephrine or dopamine are not sufficient
e. Then think about phenylephrine, methylene blue … At this point the pulmonary fellow should be by your side.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Bolus:</th>
<th>Dose</th>
<th>Infusion Concentration</th>
<th>Set Pump to</th>
<th>Dose delivered</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone</td>
<td>100 mg</td>
<td>5-15 mg/min</td>
<td>450 mg/250 mL DSW</td>
<td>33.3 mL/hr x 6 hours then 16.7 mL/hr x 18 hours</td>
<td>Bolus: IV push if pulseless otherwise over 10 min Drip: 1 mg/min x 6 hours, then 0.5 mg/min x 18 hours</td>
<td>After bolus dose, obtain continuous infusion from PharmacyMaximum dose of 22 g/24 hr</td>
</tr>
<tr>
<td>Cisatracurium</td>
<td>0.2 mg/kg</td>
<td>0.1-5 mg/kg/mi n</td>
<td>200 mg/100 mL DSW</td>
<td>Weight based</td>
<td>Drip: 0.5-10 mcg/kg/min</td>
<td>Train of four monitoring required. Goal 1-2 twitches.</td>
</tr>
<tr>
<td>Dexmedetomidine</td>
<td>0.5-1 mcg/kg</td>
<td>0.2-0.7 mcg/kg/min</td>
<td>200 mcg/50 mL</td>
<td>Weight based</td>
<td>Drip: 0.2-0.7 mcg/kg/hr</td>
<td>Titrate to desired Riker sedation score. Bolus dose may cause hemodynamic instability and may be omitted. Max 1.5 mcg/kg/hr</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>0.1-0.25 mg/kg</td>
<td>1 mcg/kg/min</td>
<td>100 mg/100 mL D5W</td>
<td>Start: 0.15 mg/kg/min</td>
<td>Drip: 5-15 mg/hr</td>
<td>Max may cause bradycardia and hypotension. Monitor HR and BP.</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>2.5-20 mcg/kg/min</td>
<td>10 mg/kg/min</td>
<td>500 mg/250 mL or 1000 mg/250 mL D5W, 10 mg/20 mL for pump setting</td>
<td>Weight based</td>
<td>Start: 2.5-5 mcg/kg/min</td>
<td>Suggested dose 2.5-20 mcg/kg/min. Max 40 mcg/kg/min. May cause tachycardia, PvcvS, hypotension.</td>
</tr>
<tr>
<td>Dopamine</td>
<td>2.5-20 mcg/kg/min</td>
<td>10 mg/kg/min</td>
<td>400 mg/250 mL or 800 mg/250 mL D5W, 1600 or 3200 mcg/mL for pump setting</td>
<td>Weight based</td>
<td>Start: 2.5-5 mcg/kg/min</td>
<td>Suggested dose 2.5-20 mcg/kg/min. Max 50 mcg/kg/min. May cause tachycardia, arrhythmias, and tissue ischemia.</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>0.01-0.5 mcg/kg/min</td>
<td>1 mcg/kg/min</td>
<td>1-10 mg/250 mL D5W</td>
<td>Start: 0.5 mcg/kg/min</td>
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</tr>
</tbody>
</table>

**Comments:***

- **Octreotide**: Suggested dose is 50 mcg bolus, then 50 mcg/hr for variceal bleed.
Phenytoin (IV): Drip: 0.2-2.5 mcg/kg/min. Max: 5 mcg/kg/min. 30 mg or 100 mg/250 mL D5W or 200 or 400 mcg/mL for pump setting. Weight based. Start 0.2 mcg/kg/min. Suggested dose: 0.2-2.5 mcg/kg/min. Central line only. May cause arrhythmias and tissue ischemia.

Procaainamide: Bolus: 17 mg/kg over 30 min. Max infusion rate 50 mg/min. Drip: 1-6 mg/min. 2000 mg/500 mL or 2000 mg/250 mL D5W or NS. 4 or 8 mg/mL for pump setting. 1 mg/m²=15 or 7.5 mL/hr 2 mg/m²= 30 or 15 mL/hr 3 mg/m²= 45 or 22.5 mL/hr. Drip: 1-6 mg/min. Renal dose adjustment necessary. Monitor procaainamide and NAPA metabolite levels.

Propofol: Bolus: 0.5 mg/kg. Drip: 5-50 mcg/kg/min. 1000 mg/100 mL NS or pump setting. Weight based. 10 mg/mL for pump setting. Drip: 5-50 mcg/kg/min Max: 150 mcg/kg/min. May cause hypotension and increased triglycerides. Contains 1.1 kcal/mL. Use sedation scale to titrate.

Vasopressin: Bolus: 40 units only for asystole or VF/pulseless VT). Drip: 0.03 units/min. 100 units/250 mL NS or 0.8 units/mL for pump setting. 0.04 units/min= 3 mL/hr Sipas/IV: 0.01-0.04 units/min. O2 Bleed: 0.2-0.4 units/min. HR: 0.04-0.8 units/min. Suggested dose: 0.01-0.04 units/min. ***Mesenteric ischemia possible with doses >0.04 units/min.*** Serum lactate monitoring may be indicated in doses >0.04 units/min.****

Vecuronium: Bolus: 0.1-0.2 mg/kg. Drip: 0.5-2 mg/kg/min. 100 mg/250 mL D5W or NS. Weight based. Drip: 0.5-2 mg/kg/min. Train of four monitoring required. Goal 2-2 twitches.

### Code Cart and Common Intensive Care Unit IV Push Medications

<table>
<thead>
<tr>
<th>Drug</th>
<th>Suggested Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine</td>
<td>6 mg initial dose; after 1-2 min 12 mg (May repeat 12 mg x1) Due to short half-life, must administer by rapid IV push (1-3 seconds) followed by 10-20 mL saline or sterile water flush.</td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Pulsless VT/VF: Pulseless VT/VF: 300 mg IV push unfiltered/undiluted—may repeat with 150 mg if needed. Stable VT: 150 mg diluted in 25-50 mL NS or D5W infused over 10 minutes. Max 2.2g/24h</td>
<td></td>
</tr>
<tr>
<td>Aminophylline</td>
<td>Asystole: 250 mg 6 mg/kg over 30 min Blocks adenosine receptors. Give 250mg IV push for atropine resistant asystole or 6 mg/kg IV over 80 minutes (Max infusion rate 25 mg/min) for atropine resistant bradycardia.</td>
<td></td>
</tr>
<tr>
<td>Atropine</td>
<td>0.5-1.0 mg Can be administered every 1-5 minutes to a maximum dose of 0.04 mg/kg.</td>
<td></td>
</tr>
<tr>
<td>Calcium Chloride</td>
<td>1 gm 10 mL (1 amp=10mL) Given for known hyperkalemia, hypocalcemia, hypomagnesemia, or toxic effects of calcium channel blockers. May repeat as needed. Vescicant, use extreme caution when administering; incompatible with phosphate solutions. Central line preferred.</td>
<td></td>
</tr>
<tr>
<td>Calcium Gluconate</td>
<td>1 gm/10 mL (1 amp = 10 mL) Preferred over calcium chloride for non-emergent calcium supplementation. Dilute in 50 mL of NS or D5W and administer over 1 hour.</td>
<td></td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>50 mg Administer over 1-2 minutes.</td>
<td></td>
</tr>
<tr>
<td>Epinephrine</td>
<td>1 mg (repeated every 3-5 minutes during arrest) Available in 1:10,000 (1mg in 10 mL abbojet) or 1:1,000 (1mg/mL 30 cc vials).</td>
<td></td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>100 mg IV push over 30 seconds to 1 minute.</td>
<td></td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>Bradycardia: 0.2mcg 20-60mcg Torasdes de Pointes: 0.5mcg 10-40mg</td>
<td>Given for atropine resistant bradycardia or torsade de Pointes. May repeat or start continuous infusion. May induce tachycardia and worsen cardiac ischemia.</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>1-1.5 mg/kg. (May repeat dose in 3-5 min) Typical dose is 100 mg. Total maximum dose is 3 mg/kg.</td>
<td></td>
</tr>
<tr>
<td>Magnesium Sulfate</td>
<td>1-2 grn Dilute to 10 mL with D5W or NS; administer over 1-2 minutes. For general magnesium supplementation administer each 2 grams over 1 hr. Given for known hypomagnesemia and/or fordsde of Pointes.</td>
<td></td>
</tr>
<tr>
<td>Methypradinol</td>
<td>25 mg IV push over 30 seconds to 1 minute.</td>
<td></td>
</tr>
<tr>
<td>Metoprolol</td>
<td>5 mg slow IV push May repeat every 5 minutes to total of 15 mg.</td>
<td></td>
</tr>
<tr>
<td>Naloxone (Narcan)</td>
<td>0.4-2 mg every 2-3 min Dilute to 10 mL with NS or sterile water.</td>
<td></td>
</tr>
<tr>
<td>Phenytoin (loading dose)</td>
<td>15-20 mg/kg Careful when administering IV Push (max rate 50 mg/min). Max concentration 10 mg/mL. Must be mixed in NS. Watch vital signs carefully.</td>
<td></td>
</tr>
<tr>
<td>Sodium Bicarbonate</td>
<td>1 ml/kg (may repeat) dose every 10 min or per ABG’s Typical dose is 50 mlq. Incompatible with epinephrine and other sympathomimetic drugs.</td>
<td></td>
</tr>
<tr>
<td>Vasopressin</td>
<td>40 Units IV push May Repeat in 10-20 min Use in Pulsless VT/VF or asystole. May resume epinephrine dosing in 5-10 minutes.</td>
<td></td>
</tr>
</tbody>
</table>

**NARYEL:** Narcan, atropine, valium, vasopressin, epinephrine and lidocaine may be administered via the ETT (endotracheal tube) if an IV is not available. Dose is usually doubled and given in at least 10 mL of NS or sterile water (vasopressin dose is 40 Units via the ETT – same as the IV dose). Patient is ventilated aggressively after administration to facilitate absorption. (University of Colorado Hospital. Updated January 2008. Ty Kiser, Pharm.D., BCPS)
RHEUMATOLOGY

Indications: Arthrocentesis is both diagnostic and therapeutic. Any new mono- or polyarthritis unrelated to trauma should be tapped for diagnosis, especially when considering the possibility of a bacterial arthritis. Irreversible damage to cartilage occurs within the first day of infection, so timely diagnosis is critical.

Contraindications: Relative contraindications include anticoagulation, bleeding diathesis, overlying cellulitis, and active bacteremia. Discuss with ortho prior to tapping a joint with known hardware in it.

Differential:
1. Noninflammatory: OA, RA, trauma, osteochondritis dissecans, sickle cell, lupus, scleroderma, amyloidosis, hypothyroid, Milwaukee shoulder
2. Inflammatory: RA, CPPD, gout, psoriatic arthritis, arthritis of IBD, ankylosing spondylitis, viral arthritis, rheumatic fever, Behcet’s, vasculitis, Lyme dx, infection
3. Septic: Bacteria, Fungus, Mycobacterium
4. Hemorrhagic: trauma, bleeding disorder, crystalline arthropathy, tumor, prostatic joint, sickle cell, Charcot’s arthropathy

Synovial Fluid Examination: Normal fluid is clear/colorless to straw colored. Fluid is cloudy because of the presence of increased cells/fibrin/lipids/crystals/etc. Off-white fluid is often PMNs while very white fluid is usually gout (monosodium urate crystals). Greenish fluid is often RA. Orange-brown fluid is associated with pigmented villonodular synovitis, a rare disorder.

Labs: Send the fluid gram stain, culture, cell count and microscopic examination under polarized filter. Monosodium urate crystals (gout) are needle shaped and yellow when the filter is parallel with the crystal=negative birefringence. Calcium pyrophosphate dehydrate (CPPD) crystals are rhomboid shaped and blue when the filter is parallel with the crystal=positive birefringence. Pneumonic: ABC=align, blue, calcium pyrophosphate. Unfortunately, the presence of crystals does not exclude infection: the two can co-exist. Gram stain and culture are very helpful in this situation.

<table>
<thead>
<tr>
<th>Type of Fluid</th>
<th>Special Features</th>
<th>Leukocytes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Clear, colorless, viscous</td>
<td>&lt;200(&lt;25%PMNS)</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>Clear, yellow, viscous</td>
<td>200-100,000 can be&lt;50% PMNs</td>
</tr>
<tr>
<td>Septic</td>
<td>Purulent</td>
<td>&gt;50,000 (&gt;75%PMNS) although 33% will be lower than 50,000</td>
</tr>
</tbody>
</table>

If WBC >50,000, strong suspicion for septic joint, but Reiter’s, gout and RA pseudosepsis can all look like this. When in doubt, treat but trust your gram stain and cx. Gram stain is + in 80-85% of staph infections. It’s 50% positive in gram negative infections. Culture is 100% specific for all infections except gonococcus, TB and fungal.

The NEJM website has a helpful PDF and video on arthrocentesis of the knee. Use it through the website hslibrary.ucdenver.edu for all arthrocentesis. A great picture of crystals is found there. A video on approach to the shoulder is at http://webmedia.unmc.edu/intmed/general/demo/Arthro/shov1001.htm
-Use ultrasound!
Medical Toxicology/Poison Center Consults

Both Denver Health and UCH have active inpatient Medical Toxicology Consulting services. We are here to assist you with the evaluation and management of poisoned patients or patients who have symptoms that may be due to poisoning. Our service provides real-time bedside consultation and we hope to be consulted on all admitted overdose patients. As poisoning is a dynamic process, please contact us as soon as possible so we can provide any assistance early. We are particularly interested in patients who have undifferentiated presentations that may be due to poisoning (such as altered mental status, hypotension or metabolic acidosis without other explanations). Please call the Poison Center at 1-800-222-1222 and ask to speak with the Medical Toxicology Fellow on call for all suspected ingestions.

General Hint: The recognition of all toxicities requires a high level of suspicion. Look for them in all patients with unexplained symptoms, those with active or historical depression (especially in young, otherwise healthy patients) and in those with access to certain meds.

Aspirin Overdose

What to look for:
- Early/mild: nausea, vomiting, hyperventilation, tinnitus, lethargy, delirium
- Severe: coma, seizures, fever, hypoglycemia, unexplained pulm edema

Lab Abnormalities:
- mixed respiratory alkalosis with metabolic acidosis
- toxic levels generally greater than 30-40 (though levels are very inexact)

Therapy:
- fluids to euovolemia as overcorrection will cause pulmonary edema; forced diuresis not helpful
- bicarbonate (via boluses with ABG to follow serum pH, goal 7.45-7.5) to help with urinary excretion, keep CNS penetration to a minimum
- check labs including lactate, lytes, salicylate levels, ABG’s q4h till stable
- dialysis if renal failure, pulm edema, coma, seizures, levels greater than 100 or failure of conservative techniques with worsening clinical course

(Adapted from Goldfrank’s Toxicologic Emergencies, 7th ed., 2002)

Tylenol Overdose

What to look for:
- Stage I (1-24h): asymptomatic or nausea/vomiting, anorexia
- Stage II (24-72h): all of the above + RUQ pain, LFT rise
- Stage III (72-96h): jaundice, renal failure, coagulopathy, encephalopathy

Lab abnormalities:
- initially will be normal but look for LFT, INR, PTT, creatinine changes
- ABG may show metabolic acidosis although this can be variable
• Tylenol levels need to be drawn at 4h after ingestion then every four hours till < 10; if time cannot be established or patient shows up after 4 hours then the Rumack-Matthews nomogram not helpful (otherwise very sensitive)

• **Remember:** Chronic toxicity occurs at much lower APAP levels!

**Therapy:**
- **When in doubt, treat**
- N-acetyl cysteine 140 mg/kg PO load followed by 70 mg/kg every 4 hours x 17 doses or until INR < 2 and encephalopathy resolved (whichever comes last)

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(Adapted from Goldfrank’s Toxicologic Emergencies, 7th ed., 2002)

**Lithium Overdose**

**What to look for:**
- Mild/moderate: asymptomatic, tremor, slurred speech, weakness, ataxia lightheadedness, hyperreflexia, nausea/vomiting, dry mouth, TWI on EKG
- Severe: confusion, clonus, coma, seizures, renal failure

**Lab abnormalities:**
- look or renal insufficiency, unexplained leukocytosis, hypothyroidism
- Li+ levels for mild/moderate toxicity occur at 1.5 – 3.5 mEq/L, severe toxicity occurs at levels greater than 3.5 mEq/L

**Therapy:**
aggressive volume replacement (watch for diabetes insipidus complicating this) with lytes replacement as needed; forced diuresis not helpful

dialysis indicated for all ingestions with a level above 4.0, when level 2.5 with moderate neurologic symptoms or if renal failure

(Adapted from Goldfrank’s Toxicologic Emergencies, 7th ed., 2002)

TCA Overdose

What to look for:

- ECG: sinus tach at 120-160 BPM, prolonged QRS duration; R-wave in aVr 3mm a good early predictor of ingestion
- altered mental status, hyperthermia, ileus, delirium, coma, seizures, unexplained hypotension, ventricular arrhythmias
- Expect a variable, very unpredictable course for about 24 hours
- 70% of cases will have a co-ingestion complicating things…look for this!!

Lab abnormalities:

- TCA levels take a long time to come back and are very unreliable when used to predict toxicity, unlikely to help your management of the patient
- check ABG’s q4h if giving bicarb

Therapy:

- most patients will require supportive care only (fluids, lytes, monitoring) which they should receive in a step down or ICU
- any ECG changes, hypotension or neurologic signs should get sodium bicarb:
  - bolus 1-2 mEq/kg x 1 followed by NaBicarb drip (3 amps in 1L D5W) at 150 cc/hr till symptoms resolve (goal pH 7.5 – 7.55)
- intubation and hyperventilation (with same goal pH) for all critically unstable patients
- treat seizures as above plus aggressive benzos to stop seizing
- treat hypotension with aggressive fluids, norepinephrine if a pressor is needed
- no role for dialysis

(Adapted from Goldfrank’s Toxicologic Emergencies, 7th ed., 2002)
WITHDRAWAL SYNDROMES

Alcohol Withdrawal

What to look for:
- consider this diagnosis in all patients who admit to consuming alcohol in any significant quantity or are known to historically do so
- mild withdrawal: irritability, agitation, hypervigilance, sleep disturbance, tremor, elevated HR, elevated BP, elevated temperature without other known cause
- hallucinosis: sensorium intact with above symptoms + auditory or visual hallucinations
- seizures: always generalized tonic/clonic, often with a history of this during times of alcohol abstinence
- DT’s: all the above + delirium and clouded consciousness, very hyperadrenergic; approx. 5% of these patients will die as a result so treat this as a medical emergency

Lab abnormalities:
- alcohol levels not helpful as these patients are always chronic, heavy drinkers and can withdraw at high what would be a high BAL for a non-drinker
- follow lytes carefully as malnutrition with resultant hypoK, hypoMg, hypoPhos can complicate; hyponatremia is common and must be considered prior to aggressive fluid resuscitation

Therapy:
- most mild withdrawal can be treated as an outpatient with PO benzos and abstinence
- more severe cases can be treated on the floor with careful observation; any patients with a history of seizures or DT’s or those requiring hourly benzo doses in the ER should be admitted to the step down or ICU services for at least the first 48-72 hours
- the mainstay of treatment is benzodiazepines, either via PO or IV depending on the severity of symptoms and doses required; early symptom-based therapy (using a CIWA scale, downloadable from UpToDate) has been proven to be the most effective method of keeping people from entering full-on DT’s
- when benzodiazepine doses reach 15-20 mg lorazepam IV per hour it is good to consider adjunctive therapy with clonidine 0.3 mg po q6h or halol 0.5 – 1.0 mg IV q4h if delirium and psychotic features are present
- severe cases will often be associated with respiratory compromise and mechanical ventilation should be considered as symptoms worsen and benzodiazepine needs increase
- all patients admitted with an alcohol history must receive thiamine 100 mg/day, folate 1 mg/day and MVI 1 tab/day either PO or IV starting immediately on admission and continuing for the length of their stay
How To Think About Differential Diagnoses

The approach to a differential diagnosis is where it all comes together for the internist. It is the blending of the ‘art’ and the ‘science’ of medicine. The art comes from your pretest probabilities and the science from the tests you choose to discern which of your possible diagnoses is the actual culprit.

When you are approaching an unknown condition, it is best to think of the top 5 things it is most likely to be in your mind…the art. How you come to these five possible conclusions comes from your history taking, physical exam and clinical sense. While five is a random number, it is workable (and you have to start somewhere). Once you have your most likely causative conditions, you can apply the science in the form of tests, imaging and other studies. Here is the approach to the art and science of medicine using chest pain as an example:

1) Select your five possible diagnoses and assign them a probability of being the correct one. Make sure that all of the probabilities, when taken together add up to 100% (because it has to be something). This is the art, in that the skills you will acquire over the next few years will refine your ability to make this list accurately every time…you’ll see why this is so important as tests are ordered and therapies considered.

- myocardial infarction – 35%
- GERD – 20%
- pneumonia – 10%
- anxiety – 15%
- pulmonary embolus – 20%

2) Perform a series of tests to rule in or rule out each diagnosis. Evaluate one condition at a time. Take your pre-test probability and convert them to odds. This is done in order to make use of the likelihood ratio associated with every test that you can order which will tell you if your condition is more or less likely based on either a positive or negative test result. (Ex. for pneumonia with positive CXR).

<table>
<thead>
<tr>
<th>Pre-test Prob</th>
<th>Pre Test Odds</th>
<th>x Pos LR</th>
<th>Post Test Odds</th>
<th>Post test Prob</th>
</tr>
</thead>
<tbody>
<tr>
<td>10%</td>
<td>10/90</td>
<td>(5.5) or 5.5/1</td>
<td>55/90</td>
<td>55/145 or 38%</td>
</tr>
</tbody>
</table>

3) Now that you have a new post-test probability, your list of five will no longer add up to 100%. You have to reassign probabilities to make sure that it does (more art). If a test has made something so low on your list as to no longer be worthy of consideration, drop it from the list. Now you are down to the four most likely things and are making progress. If new information has come to light and something needs to be added to the list, feel free to do so, making sure to keep everything adding up to 100%.

- myocardial infarction – 27%
- GERD – 15%
- pneumonia – 38%
- anxiety – 0% (drop from list)
- pulmonary embolus – 20%

4) Once something has become the dominant diagnosis, you have to decide if you are going to treat or not. A treatment threshold (the percent probably something needs to be for you to feel justified and correct in treating it) must then be considered. If you came up with GERD as your most likely explanation of the chest pain but it was still only at 20% probability, you will feel OK treating it as the therapy is very non-toxic and non-invasive (TUMS). If however your highest probability on your list is myocardial infarction at 35%, you will need to do more testing to raise it above your treatment threshold (likely more like 80-90% to perform cardiac catheterization). Pneumonia, as in the example above, likely falls somewhere in between as antibiotic therapy is more toxic than TUMS but not nearly
as risky and invasive as a catheterization. 38% probable may be enough to start antibiotics; if not, getting an negative ECG and negative d-dimer may lower other possibilities, allowing you to add their probabilities to the diagnosis of pneumonia in order to raise it above your treatment threshold.

5) Continue this process until you have successfully diagnosed and treated the offending condition. Usually there will still be other diagnoses on your list that you do not think are likely enough to treat but you also cannot entirely rule out…this is OK. These are the things you are going to keep in the back of your mind as "other possible causes" in case the treatment for your first consideration does not produce the therapeutic results you had hoped for.

   It sounds complicated but gets much easier with time. It’s what good doctors do in their heads every time they see a patient and it will become second nature for you, too.
The Patient-Centered Approach

The patient must always be at the forefront of our thoughts. Without patients there would be no doctors, and caring for our patients in a compassionate, professional and intelligent manner is our ultimate goal. While the rest of this book has been about this implicitly, we wanted to take one last opportunity to mention it explicitly.

Your patients will come to you scared and looking for answers. Your mission over the next three years is to learn to help with this fear and uncertainty. Diagnosis and treatment can be tricky and time-consuming, without always being fruitful. Sometimes you will find the diagnosis and realize there is no adequate treatment you can offer your patient. This is why the most important thing you can do for your patients is to help alleviate the fear that comes with being ill. When approached correctly, this goal can be accomplished almost every single time, in almost every single patient.

Make an effort to see your patients for who they really are: People, just like you, with families, goals, dreams and desires who see all those things being thrown into disarray and peril by a gnawing pain in the gut or a sudden flash of pain across the chest. Every “interesting” or “cool” case for you is a potential threat to their existence. While we have come to residency to learn and expand our knowledge, it is only because of the patients and their ordeals that this experience is possible.

So please be respectful of patients and their families. Understand that anger or irritation on their part is not directed at you, and do your best not to take it personally. When you take the time to understand that a patient’s anger might actually just be one of the stages of loss or dying, this becomes much easier to accept. Learning to step outside of yourself and think from the patient’s point of view will help bring all these things into perspective.

As we have said before, your time in the hospital over the next few years is going to be an incredible, life-changing time for you. Understanding that it is the same for your patients will make it all the richer, and will make you a better doctor.
Chief's Pagers

There is always a CMR who can be reached, 24 hours a day, 7 days a week. During the day it is best to call or page the chief assigned to the hospital you are working at that month, though all of us will have our pagers on during the day if there is someone specifically with whom you would like to address an issue or question.

At night there is a CMR on call. The call schedule is posted on the message board of the main medical floor on which you are working or can be found on amion.com (logon: UCO). Feel free to call with any question at any time.

Bryan Brimhall  303-266-3912
Julia Clemons  303-266-3914
Elsbeth Jensen-Otsu  303-266-3936
Adrienne Mann  303-266-3951
Allison Nitsch  303-266-3958

Housestaff Office Contact Info

Your housestaff office is a central resource for all your questions about the program. It is staffed by some of the hardest working people in the department: Jennifer Weber is the Program Administrator, and Allison Claybrook, Heidi Eckhoff and Melanie Murray are the administrative assistants. Make it a point to introduce yourself to Jennifer, Alli, Heidi and Melanie early in the year; they are fantastic and will be an incredible resource for you across your three years. They work very hard for you, so be especially nice to them always. Danaa Kennedy is the Med/Peds Coordinator and Akemi Iwanabe is the Primary Care contact. You can contact them at:

Alli Claybrook  303-724-1792
Heidi Eckhoff  303-724-1791
Akemi Iwanabe  303-724-2264
Danaa Kennedy  303-724-6595
Melanie Murray  303-724-1784 (main number)
Jennifer Weber  303-724-1788
Library information:

This comes from the pocket librarian guide that the medical librarians at Anschutz Medical Campus put out.

POCKET LIBRARIAN, 2008

Health Sciences Library
University of Colorado
Anschutz Medical Campus
(303) 724-2152
http://hslibrary.ucdenver.edu/

Find these resources with a keyword search on the Library’s webpage

- Residents Guide  Keyword search: resident
- Ask a Librarian  http://hslibrary.ucdenver.edu/aal/
- Online Journals  Type the journal title into the search box and click on Journal Titles
- Patient Education  Keyword search: patient education
- Handouts and Tutorials  Keyword search: handouts or tutorials
- PDA Resources  Keyword: pda

Databases Page  http://hslibrary.ucdenver.edu/databases/  (Choose Clinical Medicine or Find Databases by Title or type the resource name into the webpage search box)
- Differential Diagnosis: First Consult or Micromedex
- Disease Reviews, Dx & Tx: ACP PIER, Dynamed, or FirstConsult
- Calculators, Rules, Labs, Tables:
  Micromedex - Main tab/Calculators & Labs
  Essential Evidence Plus - Clinical Rules and Calculators
- ACP PIER - disease chapters include dx & tx tables
- Tests and Procedures - ACP PIER & FirstConsult-Procedures
- Herbs and Supplements - Natural Medicines Comprehensive Database

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- 144 -
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Enter type 2 diabetes into the search box
The MeSH database suggests the preferred term:
  Diabetes Mellitus, Type 2
Click the checkbox next to the term & click the Send to menu, then click Search Box with AND
Type exercise into the search box
The MeSH database provides a list of possible matches
Click the checkbox next to Exercise and click the Send to menu, then click
  Search Box with AND
Click the Search PubMed button
Display Abstracts Plus Change from summary to abstract plus, & click the ARTICLE LINKER (green & white) button for full text

“Each morning sees some task begun, each evening sees it close;
Something attempted, something done, Has earned a night’s repose”
*Henry Wadsworth Longfellow*