The Turkey Book
AN INTRODUCTORY MANUAL FOR THE WARDS
AN INTRODUCTORY MANUAL FOR THE WARDS

For Medical Students,
By Medical Students
Prepared by UW chapter of AOA

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4th edition: Class of 1989
5th edition: Class of 1991
6th edition: Class of 1998
7th edition: Class of 2000
8th edition: Class of 2001
9th edition: Class of 2002
10th edition: Class of 2003
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http://uwmedicine.washington.edu/Education/MD-Program/Current-Students/Community/Alpha-Omega-Alpha/Pages/UWAOA.aspx

Introduction
This manual is a compilation of helpful hints, high yield facts, and introductions to many clinical topics that you will encounter in your clinical years of medical education. Use it as a starting point and supplement with other more exhaustive references as needed.

This is your time to learn from your team and from your patients, and to learn to enjoy medicine. Work hard, eat, sleep, enjoy yourself, and remember that you are not alone.

---------------------------------------------------------------------Disclaimer---------------------------------------------------------------------

Every effort was made to make this handbook as accurate as possible; however, the accuracy and completeness of the recommendations are not guaranteed. Don’t forget to think for yourself.

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If you have suggestions for improvements or changes in future editions of this manual, please e-mail them to Trish Zander in A-300 at uwaoa@uw.edu

Cover image courtesy of the National Library of Medicine.
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GENERAL INFORMATION FOR ALL CLERKSHIPS

ADMISSION ORDERS – ADC VAAN DIMLS (Post-op orders too)

Admit
Unit, team, attending, resident, intern, student

Diagnosis
Reason for admission, working diagnoses (pancreatitis, MI)

Condition
Satisfactory, serious, guarded, critical, etc.

Vitals
Frequency (per routine); specify telemetry, neuro checks, O2 sats

Allergies
Indicate drug and nature of allergic reaction

Activity
Ad lib or as tolerated, up in chair, ambulate TID, BR w/ BRP

Nursing
I&O’s, daily weights, bedside spirometry, wound care and dressing changes, drains (Foley to gravity). Call HO for T>38.5, HR>110, <60, SBP >160, < 90, RR> 30, < 8, UOP < 30 cc/hr

Diet
Regular, ADA, low salt, clear liquids, soft foods diet, NPO, advance as tolerated

IV Fluids
Indicate solution, additives, volume, and rate of flow, e.g. D51/2NS w/ 20 mEq KCl at 100cc/hr. Heplock with q shift flush, TKO = to keep open. If multiple lines or lumens are present, write a separate order for each

Meds
Name, dose, route, and frequency for all scheduled and PRNs

Labs
CBC, Chem 7, etc. Also include ECG and imaging.

Special
For orders that do not fall into above categories

ADMISSION NOTE

ID/CC
One sentence describing patient and reason for admission

Problem List

HPI
Don't forget pertinent + and -, relevant social and family hx

PMH
Medical and surgical history, diagnoses, and dates

Meds
Usually list outpatient meds

Allergies

Family Hx

Social Hx

Substance use

ROS

PE

Labs

Imaging

Assessment
Approach either by list of problems or by organ system. For each problem, include a differential diagnosis and brief explanation.

Plan
Again, by list of problems or by organ system. Consider further diagnostic measures and treatment. Include labs, meds, consults, procedures, etc.

DAILY PROGRESS NOTE

Date/Time, Service, MS3 Note

ID
Identify patient, include HD#, abx day#, POD#

E
Events (overnight pt had episodes of hypoxia)

S
Subjective - complaints, events, symptoms, pain

O
VS, weight, I&O, PE, labs, imaging

A
Assessment

P
Plan
DISCHARGE ORDERS
D/C To home, nursing home, SNF, etc.
Diagnoses #1_____, #2_____, #3________
D/C Meds Include length of continuation
Diet Include if special considerations (ADA, NPO before surgery)
Activity Note restrictions for work, sports, bathing or showering, weight-bearing
Follow-up Include MD, clinic, date, time for appts, dressing changes, labs

DISCHARGE SUMMARY
Date of admission
Date of discharge
Admission / Discharge diagnoses
H&P, procedures, consults
Admission medications
Hospital course List by diagnoses/system or by time course
D/C medications
Disposition Nursing home, SNF, rehab
Follow-up Appointments, instructions

PROCEDURE NOTE
Date/Time
Procedure: Paracentesis, chest tube placement, etc.
Permit: Explained to pt by ___, indications/risks discussed, questions answered
Indication: Ascites, concern for meningitis, etc.
Labs: Include INR, CBC, or creatinine if relevant
Physician(s):
Description: Area prepped and draped in a sterile fashion. (Describe the anesthetic used and how it was administered). Describe technique including instruments, body location, type of needles and suture used, etc. Also include any relevant findings.
Specimens: Cultures and/or labs sent, specimens sent to pathology, etc.
Complications:
Estimated Blood Loss: None, minimal, amount in cc
Disposition: Pt alert, oriented, and resting; breathing nonlabored; neurovascularly intact; incision clean, dry, and intact, etc.

WRITING PRESCRIPTIONS
Write neatly. Include patient's name, allergies, medication name, strength, and dosing.
Sig: Frequency of administration
Disp: Amount to dispense

<table>
<thead>
<tr>
<th>Date/Time</th>
<th>Patient Name</th>
<th>Allergies: NKDA</th>
<th>Medication: Furosemide 40 mg</th>
<th>Sig: 1 PO bid</th>
<th>Disp: #60</th>
<th>Refills__2___</th>
<th>Signature</th>
</tr>
</thead>
</table>

SIGNING NOTES
Give your name, MS3, pager number, and leave room for the co-signer.
PRESENTING A PATIENT (From Dr. McGee's guidelines)
1. Concisely present relevant information from the history, PE and labs/imaging to demonstrate your understanding of the patient’s condition and approach to initial management. Strive for 4-5 minutes (less on surgery) and a memorized presentation, except for lab values.
2. Include major presenting symptoms, pertinent positives/negatives, and work-up of complaint prior to hospital admission in HPI.
3. Avoid repetition, disorganization, and editorial comments. Practice before you present.

Format:
- ID/CC
- HPI
- Other active medical problems
- Meds/allergies/substance use/pertinent social history. Do not present ROS (pertinent +/– belong in HPI, rest of ROS is left out)
- PE, including vital signs. Can do relevant positives/negatives or full PE, depending on situation and team preference
- Labs, imaging. Compare to prior if available
- Assessment/plan/hospital course to date

ICU PRESENTATION
ID/CC, HD#
24-hour events Changes in management, big events
Subjective Rare because patients are usually intubated
Lines Know number of days each has been in
Drips and rate Pressors, diuetic, sedatives, etc.
Antibiotics Drug and day#
Vitals Tmax, Tcurrent, BP range, HR range, RR, SaO2
Ventilator settings Mode, TV, actual RR, set RR, FiO2, PEEP
Weaning parameters RR, TV, NIF, minute ventilation, RSBI
ABG pH / pCO2 / pO2, HCO3 / SaO2
Swan data CVP / PAP / Wedge / Cardiac Output & Index / SVR
I&O 24-hour volumes (have breakdown of each in case asked)
Drains Pertinent findings
PE
Labs / micro Follow trends and have info available if important
Imaging Usually CXR plus any other studies
Assessment & Plan by System: For each system, state problem and how you plan to treat or fix it. If there are no issues, state “Stable, no issues”.

ID
FEN
Prophylaxis
Endocrine
Disposition
Code Status

IMMUNIZATIONS
You must be current with all immunizations to participate in 3rd and 4th year clerkships. This includes an annual TB test and all immunizations required by the School of Medicine. Many
sites outside of Seattle require you to provide results of your most recent TB test, so it is wise to carry a copy with you. Contact myshots@uw.washington.edu for more information.

UNIVERSAL PRECAUTIONS

1. **Hand washing**: Essential. Wash hands before and after leaving a patient room, after performing procedures, after eating or using restroom, and after removing gloves.
2. **Gloves**: Use when contact with body fluids is anticipated. Change gloves and wash hands between patients.
3. **Gowns**: Used to cover skin or clothing that may become soiled with body fluids or substance during a procedure.
4. **Eye protection/mask**: Use whenever there is a risk of splashing or spraying of body fluids into mouth, nose, or eyes. Also use for airborne diseases (TB, pertussis).
5. **Isolation**: Used for patients with active TB, primary varicella, measles, mumps, rubella.
6. **Sharps**: Do not recap contaminated needles. Discard sharps in appropriate containers. Take care when passing sharps and inform others of sharp location to prevent accidental injury or needle sticks.

EXPOSURE PROCEDURES

If exposed to blood/body fluids, immediately:

1. Remove soiled clothing and wash exposed area with soap and water.
2. Notify attending physician, resident, or site coordinator.
3. Note the severity and type of exposure and likelihood that patient is at risk for HIV infection.
4. If you have questions about your exposure or believe you are a candidate for the antiretroviral post-exposure prophylaxis protocol, call these numbers:
   - In Seattle: UWMC Campus Health Services (206) 598-4848 7:30am to 4 pm M-F
     Or MEDCON 1-800-326-5300
     Or UWMC Emergency Department (206) 598-4000
   - Outside of Seattle: Call MEDCON or UWMC Emergency Department
5. Request venous blood from the source patient to be sent for HIV/HBV antibody testing. Follow rules/regulations of the hospital/clinic/state for HIV counseling and testing. Have a venous blood sample drawn from yourself and store. No specific storage procedures are necessary. If test result from source patient is positive for HIV, have your blood tested.
Nephrology

Anion Gap = Na - [Cl + HCO3]  Nml 12 +/- 2
Urine Anion Gap = UNa + UK - UCl
Delta Gap = (AG-12) + HCO3
>30 = metabolic alkalosis
<23 = non-AG metabolic acidosis

Creatinine Clearance = Urine creatinine mg/dL x urine volume mL/day
Plasma creatinine mg/dL x 1440 min/day
Creatinine Clearance = 140 – age(yrs) x weight kg (x 0.85 if woman)
Serum creatinine mg/dL x 72

FeNa = Urine Na x Plasma Cr x 100
Urine Cr x Plasma Na

Calculated serum osmolarity = (2 x Na) + BUN + Glu + EtOH
2.8 18 4.6

Osmolal Gap = measured osm – calculated osm
>10 think about methanol, ethylene glycol, sorbitol, mannitol

Total Body Water = lean body mass (kg) x 0.6 (male) or 0.5 (female)

Free Water Deficit = 0.4 x lean body weight x [plasma Na – 1]
140

Corrected Sodium (hyperglycemia) = measured Na + [(glu-100) x 1.6 / 100]

Corrected Calcium (hypoalbumin) = ↓ Ca 0.8mg/dL for every 1g/dL ↓ in Alb. below 4.0

Predicted Acid-Base Compensations
Acute Resp. Acidosis HCO3 up 1 for every 10 increase in pCO2
Chronic Resp. Acidosis HCO3 up 3-4 for every 10 increase in pCO2
Acute Resp. Alkalosis HCO3 down 2 for every 10 decrease in pCO2
Chronic Resp. Alkalosis HCO3 down 4-5 for every 10 decrease in pCO2
Metabolic Acidosis predicted CO2 = 1.5(HCO3) + 8 ± 2
Metabolic Alkalosis pCO2 = 0.6(HCO3) + 40

Cardiology

MAP = SBP + (2xDBP)
3
SVR = 80 x [MAP – RA pressure] PVR = 80 x [mean PA pressure – mean PCWP]
Cardiac output L/min Cardiac output L/min

Cardiac Output (CO) = HR x SV NI 4-8 L/min
Cardiac Index (CI) = CO NI 2.5-4.0 L/min; BSA= √ [ht in cm x wt in kg]
BSA 3600
\[ QT_c = \frac{QT}{\sqrt{RR}} \]

**Pulmonary**

\[ PAO_2 = (Pb-PH_2O) \times FiO_2 - (PaCO_2 \times 1.25) \]

At sea level, room air = 150 – (PaCO2 x 1.25)

\[(A - a) \text{ gradient} = PAO_2 - PaO_2\]

Tidal Volume (\(V_t\)) = \(V_d\) (dead space) + \(V_a\) (alveolar space)

\[ \text{Dead Space} = \frac{PaCO_2 - PeCO_2}{PaCO_2} \]

Minute ventilation (\(V_e\)) = \(0.863 \times V_{co2} \text{ (mL/min)} \)

\[ \frac{\text{Static compliance}}{\text{P}_{\text{plateau}} - \text{P}_{\text{end expiration}}} = \frac{V_t}{\text{PaCO}_2} \times (1 - \frac{V_d}{V_t}) \]

**Misc**

Corrected Reticulocyte count = (%retic / 2) x (Hgb / nml Hgb)

\[ \text{BMI} = \text{weight in kg} / \text{height (m2)} \]

<table>
<thead>
<tr>
<th>Test +</th>
<th>Disease +</th>
<th>Disease -</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>True Positive</td>
<td>B</td>
</tr>
<tr>
<td>Test -</td>
<td>C</td>
<td>False Negative</td>
</tr>
</tbody>
</table>

\[ \text{Sensitivity} = \frac{A}{A + C} \]
\[ \text{Specificity} = \frac{D}{B + D} \]
\[ \text{PPV} = \frac{A}{A + B} \]
\[ \text{NPV} = \frac{D}{C + D} \]

**CONVERSIONS**

<table>
<thead>
<tr>
<th>Celsius</th>
<th>Fahrenheit</th>
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<tbody>
<tr>
<td>34.0</td>
<td>93.2</td>
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<td>36.0</td>
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<td>37.0</td>
<td>98.6</td>
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<td>38.0</td>
<td>100.4</td>
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<tr>
<td>39.0</td>
<td>102.2</td>
</tr>
<tr>
<td>40.0</td>
<td>104.0</td>
</tr>
<tr>
<td>41.0</td>
<td>105.8</td>
</tr>
<tr>
<td>42.0</td>
<td>107.6</td>
</tr>
</tbody>
</table>

F = (1.8 x C) + 32
C = (F − 32) / 1.8

Unit Conversions

- 1 inch = 2.54 cm
- 1 cm = 0.394 inches
- 1 foot = 0.305 m
- 1 m = 3.291 ft
- 1 fl oz = 30 mL
- 1 mL = 0.0338 fl oz
- 1 gal = 3.79 L
- 1 L = 0.264 gal
- 1 tsp = 5 mL
- 1 Tbsp = 15 ml
- 1 oz = 30 g
- 1 gram = 0.0352 oz
- 1 lb = 0.45 kg
- 1 kg = 2.2 lb
PHYSICAL SIGNS AND EPONYMS

Argyll-Robertson pupil: Small, irregular, unequal pupils that do not react to light but react to accommodation

Babinski’s sign: Extension of big toe (toes upgoing) with stimulation of plantar surface

Battle’s sign: Ecchymosis behind ear, associated with basilar skull fractures

Bell’s palsy: Lower motor neuron lesion of the facial nerve affecting upper and lower face

Bisferiens pulse: Double-peaked pulse in severe aortic insufficiency

Bouchard’s nodes: Hard, non-tender nodules on dorsolateral aspects of PIP joints. Associated with osteoarthritis

Brudzinski’s sign: Flexion of the neck causes hips to flex. Seen in meningitis

Cheynes-Stokes respirations: Repeating cycles of gradual increase in depth of breathing followed by gradual decrease to apnea. Seen with CNS disorders, uremia.

Chvostek’s signs: Tapping of facial nerve causes facial spasm in hypocalcemia

Cullen’s sign: Ecchymosis around the umbilicus associated with retroperitoneal bleeding

Doll’s eyes: Conjugated movement of eyes in one direction as the head is briskly moved in the other direction. Tests oculocephalic reflex in comatose patients.

Dupuytren’s contracture: Contracture of the 4th and 5th digits due to proliferation of fibrous tissue of palmar fascia. Seen in alcoholics, individuals with seizure

Heberden’s nodes: Hard, non-tender nodules of the dorsolateral aspects of the DIP joints seen in osteoarthritis

Hegar’s sign: Softening of the distal uterus, seen in early pregnancy

Hoffman sign or reflex: Flicking of the volar surface of the distal phalanx causes fingers to flex. Associated with pyramidal tract disease.

Horner’s syndrome: Unilateral miosis, ptosis, anhidrosis. Due to destruction of the unilateral superior cervical ganglion (lung carcinoma)

Janeway lesion: Erythematous or hemorrhagic lesions on palms, soles in subacute bacterial endocarditis

Kernig’s sign: Extension of leg from flexed position elicits pain. Seen in meningitis

Kussmaul respirations: Deep, rapid respiratory pattern seen in DKA, coma

Levine’s sign: Clenched fist over chest describing chest pain. Assoc. with angina, acute MI.

Marcus-Gunn pupil: Dilation of the pupils with swinging flashlight test. Due to unilateral optic nerve disease.

McBurney’s point: Point located 1/3 of the distance between ASIS and umbilicus. Tenderness associated with acute appendicitis

Moro reflex: Abduction of the hips and extension of the arms when infant’s head/upper body are suddenly dropped several inches. Normal reflex in infancy.

Murphy’s sign: Severe pain and inspiratory arrest with RUQ palpation. Seen in cholecystitis.

Obturator sign: Flexion and lateral rotation of the thigh elicits pain. Seen in appendicitis and pelvic abscess.

Osler’s nodes: Tender, red, raised lesions on hands or feet. Seen in subacute bacterial endocarditis.

Pancoast’s syndrome: Carcinoma of the apex of lung resulting in arm or shoulder pain from involvement of brachial plexus and Horner’s syndrome from involvement of superior cervical ganglion.

Phalen test: Flexion of wrists by opposing dorsum of hands against each other. Positive test results in pain and tingling in the distribution of the median nerve.
Psoas sign: Extension and elevation of the right leg produces pain when psoas muscle is inflamed in appendicitis.

Pulsus alternans: Fluctuation of the pulse pressure with every other beat. Seen in aortic stenosis and congestive heart failure.

Quickne’s sign: Alternating blushing and blanching of the fingernail bed following light compression. Seen in chronic aortic insufficiency.

Rovsig’s sign: Pain in the right lower quadrant with deep LLQ palpation. Seen in appendicitis.

Traube's sign: Booming or pistol shot sounds heard over the femoral arteries in chronic aortic insufficiency.

Trousseau’s sign: Carpal spasm produced by inflating blood pressure cuff around arm above systolic pressure in hypocalcemia.

Turner’s sign: Ecchymosis at the flank associated with retroperitoneal bleeding.

Virchow node: Palpable left supraclavicular lymph node associated with GI neoplasm.

ABBREVIATIONS

A
A before
AAA abdominal aortic aneurysm
AAO alert and oriented
Ab abortion
Abd abdomen
ABG arterial blood gas
Abx antibiotics
AC before meals
ACE angiotensin converting enzyme
ACL anterior cruciate ligament
ACLS advanced cardiac life support
ACTH adrenocorticotropic hormone
AD right ear (auris dexter)
ADAT advance diet as tolerated
ADH antidiuretic hormone
Ad lib as desired
ADL activities of daily living
AFP alpha fetoprotein
A fib atrial fibrillation
Atx atelectasis
AKA above knee amputation
AOB alcohol on breath
AP anteroposterior
ARDs acute resp. distress synd
ALL acute lymphocytic leukemia
ALT alanine aminotransferase
AMA advance maternal age
against medical advice
AML acute myelogenous leukemia
APAP acetaminophen
APD afferent pupillary defect
AS left ear (auris sinistra)
Aortic stenosis
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASD</td>
<td>atrial septal defect</td>
</tr>
<tr>
<td>AST</td>
<td>aspartate aminotransferase</td>
</tr>
<tr>
<td>AT</td>
<td>atraumatic</td>
</tr>
<tr>
<td>AU</td>
<td>both ears (auris uterque)</td>
</tr>
<tr>
<td>AV</td>
<td>arteriovenous Atrioventricular</td>
</tr>
<tr>
<td>BAER</td>
<td>brainstem auditory evoked response</td>
</tr>
<tr>
<td>BAL</td>
<td>blood alcohol level brochioalveolar lavage</td>
</tr>
<tr>
<td>BBB</td>
<td>blood brain barrier</td>
</tr>
<tr>
<td>BCC</td>
<td>basal cell carcinoma</td>
</tr>
<tr>
<td>BE</td>
<td>barium enema</td>
</tr>
<tr>
<td>BID</td>
<td>twice a day (bis in die)</td>
</tr>
<tr>
<td>BiPAP</td>
<td>bilevel positive airway pressure</td>
</tr>
<tr>
<td>BKA</td>
<td>below knee amputation</td>
</tr>
<tr>
<td>BM</td>
<td>bowel movement</td>
</tr>
<tr>
<td>BMP</td>
<td>basic metabolic panel</td>
</tr>
<tr>
<td>BS</td>
<td>bowels sounds breath sounds</td>
</tr>
<tr>
<td>BSA</td>
<td>body surface area</td>
</tr>
<tr>
<td>BUN</td>
<td>blood urea nitrogen</td>
</tr>
<tr>
<td>Bx</td>
<td>biopsy</td>
</tr>
<tr>
<td>c</td>
<td>with</td>
</tr>
<tr>
<td>Ca</td>
<td>cancer</td>
</tr>
<tr>
<td>CABG</td>
<td>coronary artery bypass graft</td>
</tr>
<tr>
<td>CAD</td>
<td>coronary artery disease</td>
</tr>
<tr>
<td>CBC</td>
<td>complete blood count</td>
</tr>
<tr>
<td>CBD</td>
<td>common bile duct</td>
</tr>
<tr>
<td>CCU</td>
<td>coronary care unit</td>
</tr>
<tr>
<td>C/D/I</td>
<td>clean, dry, intact</td>
</tr>
<tr>
<td>CEA</td>
<td>carcinoembryonic antigen</td>
</tr>
<tr>
<td>CHD</td>
<td>common hepatic duct</td>
</tr>
<tr>
<td>CHF</td>
<td>congestive heart failure</td>
</tr>
<tr>
<td>CLL</td>
<td>chronic lymphocytic leukemia</td>
</tr>
<tr>
<td>CML</td>
<td>chronic myelogenous leukem</td>
</tr>
<tr>
<td>CN</td>
<td>cranial nerves</td>
</tr>
<tr>
<td>CNS</td>
<td>central nervous system</td>
</tr>
<tr>
<td>C/O</td>
<td>complaints of</td>
</tr>
<tr>
<td>COPD</td>
<td>chronic obstructive pulm dis</td>
</tr>
<tr>
<td>CP</td>
<td>cerebral palsy</td>
</tr>
<tr>
<td>CPAP</td>
<td>continuous positive airway pressure</td>
</tr>
<tr>
<td>CPS</td>
<td>child protective services</td>
</tr>
<tr>
<td>CRF</td>
<td>chronic renal failure</td>
</tr>
<tr>
<td>C/S</td>
<td>Caesarean section</td>
</tr>
<tr>
<td>CSF</td>
<td>cerebrospinal fluid</td>
</tr>
<tr>
<td>CT</td>
<td>computed tomography chest tube</td>
</tr>
</tbody>
</table>
HPF  high power field
HS  at bedtime (hora somni)
HTN  hypertension
Hx  history
ICU  intensive care unit
IMV  intermittent mandatory vent
INR  international normalized ratio
I&O  ins and outs (fluids)
IV  intravenous
IVC  inferior vena cava
IVDU  intravenous drug use
IVP  intravenous pyelogram
JP  Jackson-Pratt drain
JVD  jugulovenous distention
KUB  kidneys, ureter, bladder
LAD  left ant. descending (coronary)
Lap  laparoscopic, laparotomy
LCA  left coronary artery
LDH  lactate dehydrogenase
LDL  low density lipoprotein
LE  lower extremity
LFT  liver function test
LLL  left lower lobe
LLQ  left lower quadrant
LMP  last menstrual period
LPF  low power field
LP  lumbar puncture
LUL  left upper lobe
LUQ  left upper quadrant
Mb  myoglobin
MCC  motorcycle crash
MCH  mean cell hemoglobin
MCHC  mean cell hemoglobin conc.
MCL  medial collateral ligament
MCV  mean cell volume
MI  myocardial infarction
M&M  morbidity and mortality
MRI  magnetic resonance imaging
MVC  motor vehicle crash
NAD  no acute distress
NCAT  normocephalic, atraumatic
NGT  nasogastric tube
NIF  negative inspiratory force
NKDA  no known drug allergies
<table>
<thead>
<tr>
<th>Acronym</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>NOS</td>
<td>not otherwise specified</td>
</tr>
<tr>
<td>NPO</td>
<td>nothing by mouth (nulla per os)</td>
</tr>
<tr>
<td>NT</td>
<td>nontender</td>
</tr>
<tr>
<td>N/V</td>
<td>nausea, vomiting</td>
</tr>
<tr>
<td>O</td>
<td>right eye (oc dexter), overdose</td>
</tr>
<tr>
<td>OGT</td>
<td>orogastric tube</td>
</tr>
<tr>
<td>OR</td>
<td>operating room</td>
</tr>
<tr>
<td>ORIF</td>
<td>open reduction internal fixation</td>
</tr>
<tr>
<td>OS</td>
<td>left eye (ocular sinistre)</td>
</tr>
<tr>
<td>OT</td>
<td>occupational therapy</td>
</tr>
<tr>
<td>OU</td>
<td>both eyes (oculus uterque)</td>
</tr>
<tr>
<td>PA</td>
<td>posteroanterior</td>
</tr>
<tr>
<td>PAC</td>
<td>premature atrial contractions</td>
</tr>
<tr>
<td>PAT</td>
<td>paroxysmal atrial tachycardia</td>
</tr>
<tr>
<td>p</td>
<td>after (post)</td>
</tr>
<tr>
<td>PC</td>
<td>after meals (post cibum)</td>
</tr>
<tr>
<td>PCL</td>
<td>posterior cruciate ligament</td>
</tr>
<tr>
<td>PCP</td>
<td>primary care provider</td>
</tr>
<tr>
<td>PE</td>
<td>pulmonary embolus</td>
</tr>
<tr>
<td>PEA</td>
<td>pulseless electrical activity</td>
</tr>
<tr>
<td>PEEP</td>
<td>positive end-expiratory pressure</td>
</tr>
<tr>
<td>PID</td>
<td>pelvic inflammatory disease</td>
</tr>
<tr>
<td>PFT</td>
<td>pulmonary function test</td>
</tr>
<tr>
<td>POD</td>
<td>post-op day</td>
</tr>
<tr>
<td>PPD</td>
<td>purified protein derivative (Tb)</td>
</tr>
<tr>
<td>PPV</td>
<td>positive pressure ventilation</td>
</tr>
<tr>
<td>PRN</td>
<td>as required (pro re nata)</td>
</tr>
<tr>
<td>PSH</td>
<td>past surgical history</td>
</tr>
<tr>
<td>PT</td>
<td>prothrombin time</td>
</tr>
<tr>
<td>physical therapy</td>
<td></td>
</tr>
<tr>
<td>PTA</td>
<td>prior to admission</td>
</tr>
<tr>
<td>PTCA</td>
<td>percutaneous transluminal coronary angioplasty</td>
</tr>
<tr>
<td>PTT</td>
<td>partial thromboplastin time</td>
</tr>
<tr>
<td>PTX</td>
<td>pneumothorax</td>
</tr>
<tr>
<td>PVC</td>
<td>premature ventricular contraction</td>
</tr>
<tr>
<td>Q</td>
<td>every day (quaque die)</td>
</tr>
<tr>
<td>QH</td>
<td>every hour (quaque hora)</td>
</tr>
<tr>
<td>QHS</td>
<td>every bedtime (qh somni)</td>
</tr>
<tr>
<td>QID</td>
<td>four times a day (quarter in die)</td>
</tr>
<tr>
<td>QN</td>
<td>every night (quaque nox)</td>
</tr>
<tr>
<td>R</td>
<td>renal blood flow</td>
</tr>
<tr>
<td>RCA</td>
<td>right coronary artery</td>
</tr>
<tr>
<td>Rh</td>
<td>rhesus (Rh blood group)</td>
</tr>
<tr>
<td>RLL</td>
<td>right lower lobe</td>
</tr>
<tr>
<td>RLQ</td>
<td>right lower quadrant</td>
</tr>
</tbody>
</table>
RML  right middle lobe
R/O   rule out
RRR   regular rate and rhythm
RT    respiratory therapy
RUG   retrograde urethrogram
Rx    prescription
S     without (sine)
SA    sinoatrial
SAB   spontaneous abortion
SBE   subacute bacterial endocarditis
SBO   small bowel obstruction
SCC   squamous cell carcinoma
SLE   systemic lupus erythematosus
SNF   skilled nursing facility
SOB   shortness of breath
S/P   status post
SPEP  serum protein electrophoresis
SQ    subcutaneous
S/S   signs and symptoms
Stat  immediately (statim)
STH   said to have
STHB  said to have been
SVC   superior vena cava
SVT   supraventricular tachycardia
Sx    symptoms
Sz    seizure
T     tonsillectomy and adenoidectomy
TAH   total abdominal hysterectomy
TEE   transesophageal echocardiogram
TEN   toxic epidermal necrolysis
TIA   transient ischemic attack
TID   three times a day (ter in die)
TKO   to keep open
TNM   tumor, nodes, metastasis
TPA   tissue plasminogen activator
TPN   total parenteral nutrition
TSH   thyroid stimulating hormone
TTE   transthoracic echocardiogram
TURP  transurethral resection of prostate
TV    tidal volume
TVH   total vaginal hysterectomy
Tx    treatment
U     urinalysis
UOP   urine output
UPEP  urine protein electrophoresis
U/S   ultrasound
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>UTI</td>
<td>urinary tract infection</td>
</tr>
<tr>
<td><strong>VXWYZ</strong></td>
<td>visual fields</td>
</tr>
<tr>
<td>VA</td>
<td>visual acuity</td>
</tr>
<tr>
<td>VF</td>
<td>visual fields, v fib</td>
</tr>
<tr>
<td>VS</td>
<td>vital signs</td>
</tr>
<tr>
<td>VSS</td>
<td>vital signs stable</td>
</tr>
<tr>
<td>VSD</td>
<td>ventricular septal defect</td>
</tr>
<tr>
<td>VT</td>
<td>ventricular tachycardia</td>
</tr>
<tr>
<td>WNL</td>
<td>within normal limits</td>
</tr>
<tr>
<td>YO</td>
<td>year old</td>
</tr>
</tbody>
</table>
Clerkship director: Tom Greer, M.D.

REFERENCES AND HELPFUL RESOURCES
Centers for Disease Control: http://www.cdc.gov
Clinical Evidence from the British Medical Journal: http://www.clinicalevidence.com
Healthlinks: http://www.healthlinks.washington.edu

ROTATION TIPS
- Be proactive! Because of the wide variety of patients and medical conditions, this clerkship is a great opportunity to explore areas of interest. Ask for more involvement in deliveries, with kids, or with hospitalized patients if you are interested. This rotation offers a chance to manage patients both in the clinic and ER.
- Be aggressive about offering assessment and plans. Attendings can be quick to interject into your presentation what THEIR A/P is, but they notice if you try to come up with your own.

SELECTED FAMILY MEDICINE TOPICS
This section focuses mostly on outpatient topics. Please see other sections (Medicine, OB, Peds, etc.) for other relevant topics.

Diabetes (adapted from ACP Diabetes Care Guide, 2007)
Screening
Fasting plasma glucose level; encouraged in all >45 yo q3 yrs
< 100 mg/dL normal
100 – 125 mg/dL impaired glucose tolerance
≥ 126 mg/dL DM
Diagnosis
Fasting plasma glucose ≥ 126 (after ≥ 8 hours of fasting); symptoms of diabetes and casual plasma glucose of ≥ 200; OR plasma glucose ≥ 200mg/dL 2 hours after ingestion of 75 g of oral glucose.

Risk Factors
FHx, obesity, race, h/o GDM, HTN, PCOS, hypertriglyceridemia

Oral Medications by Class
Biguanides: Metformin (Glucophage, generic) is the only biguanide sold in the US.
Mechanism – mainly decreases hepatic glucose production; also increases peripheral glucose utilization. The most common side effects are GI-cramping and diarrhea. Rare lactic acidosis especially if decreased renal function.

Sulfonylureas: Glyburide (generic), glipizide (generic), glimepiride (Amaryl)
Mechanism – increase insulin secretion. Side effects include weight gain and hypoglycemia.

Thiazolidinediones: Pioglitazone (Actos), Rosiglitazone (Avandia)
Mechanism – decrease insulin resistance and increase insulin sensitivity of adipose tissue, skeletal muscle, and liver. Side effects of peripheral edema and weight gain.
Often used in combination with metformin and/or sulfonylureas

Non-sulfonylurea secretagogues: Nateglinide (Starlix), Repaglinide (Prandin)
  Mechanism – increase insulin secretion
  Used much less frequently than sulfonylureas
  Rapid onset – take before meal. Must eat afterward.

Alpha-glucosidase inhibitors: Acarbose (Precose), miglitol (Glyset)
  Mechanism – inhibit alpha-glucosidase enzyme at intestinal brush border. Must be taken with each meal. Many GI adverse effects.

Dipeptidyl Peptidase IV Inhibitors: Sitagliptin (Januvia)
  Mechanism – inhibit dipeptidyl peptidase IV preventing breakdown of GLP-1 which causes increased insulin release and suppression of glucagon release. Weight neutral.

Injectable Medications:

**Insulin**

<table>
<thead>
<tr>
<th>INSULIN TYPE</th>
<th>ONSET</th>
<th>PEAK</th>
<th>DURATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>SHORT-ACTING</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regular</td>
<td>30-60 min</td>
<td>2-4 hours</td>
<td>3-6 hours</td>
</tr>
<tr>
<td>Insulin lispro (Humalog)</td>
<td>&lt;15 min</td>
<td>1-2 hours</td>
<td>3-5 hours</td>
</tr>
<tr>
<td>Insulin aspart (Novolog)</td>
<td>&lt;15 min</td>
<td>1-2 hours</td>
<td>3-5 hours</td>
</tr>
<tr>
<td>Insulin glulisine (Apidra)</td>
<td>10-30 min</td>
<td>0.5-3 hours</td>
<td>3-5 hours</td>
</tr>
<tr>
<td>INTERMEDIATE-ACTING</td>
<td>2-4 hours</td>
<td>4-10 hours</td>
<td>10-16 hours</td>
</tr>
<tr>
<td>NPH/Lente</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LONG-ACTING</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glargine (Lantus)</td>
<td>2-4 hours</td>
<td>no peak</td>
<td>20-24 hours</td>
</tr>
<tr>
<td>Insulin Detemir (Levemir)</td>
<td>1-2 hours</td>
<td>Minimal/2-12 hours</td>
<td>Up to 24 hours</td>
</tr>
</tbody>
</table>

Exenatide (Byetta)
  Mechanism – GLP-1 analogue. Major effect of lowering post-prandial glucose. Causes weight loss, nausea/vomiting, and hypoglycemia if used in conjunction with sulfonylurea.

Pramlintide (Symlin)
  Mechanism – Amylin analogue potentiating the effects of insulin. Major effect is again lowering post-prandial glucose. Causes weight loss, hypoglycemia, and nausea/vomiting.

**Hyperlipidemia**

**Testing:**

Screening (nonfasting) – Total cholesterol (TC), HDL
Full lipid panel (fasting) – Total, LDL, HDL, triglycerides (TG)
• LDL is calculated: TC – HDL – TG/5 = LDL
• LDL can not be calculated if TG>400

**LDL Treatment Guidelines:**

<table>
<thead>
<tr>
<th>RISK CATEGORY</th>
<th>LDL GOAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD and CHD risk equivalents (see below)</td>
<td>&lt;70</td>
</tr>
<tr>
<td>Multiple (2+) risk factors (see below)</td>
<td>&lt;130</td>
</tr>
<tr>
<td>0-1 risk factor (see below)</td>
<td>&lt;160</td>
</tr>
</tbody>
</table>

**CHD risk equivalents:**
- Diabetes
- Other clinical forms of atherosclerotic disease (PVD, AAA, carotid disease w/sx)
- Multiple risk factors (see below) indicating a 10 year risk for CHD >20% based on Framingham risk scores (see NCEP report for details).

**Risk factors:**
- Cigarette smoking
- Hypertension (BP >140/90 or on antihypertensive medication)
- Low HDL (<40)
- FHx of premature CHD (1st degree relative: male <55 years, female <65 years)
- Age (men >45 years, women >55 years)
- NOTE: HDL >60 counts as a “negative” risk factor (subtract 1 from total number of risks)

**Treatment of Hyperlipidemia**

<table>
<thead>
<tr>
<th>DRUG CLASS</th>
<th>EFFECTS</th>
<th>SIDE EFFECTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Statins (HMG-CoA reductase inhibitors)</td>
<td>LDL ↓ 18%-55%</td>
<td>Myopathy, increased liver enzymes, expect lipid lowering effect within 6 weeks</td>
</tr>
<tr>
<td></td>
<td>HDL ↑ 5%-15%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TG ↓ 7%-30%</td>
<td></td>
</tr>
<tr>
<td>Bile acid sequestrants</td>
<td>LDL ↓ 15%-30%</td>
<td>GI distress, constipation, decreased absorption of other meds</td>
</tr>
<tr>
<td></td>
<td>HDL ↑ 3%-5%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TG No change or increase</td>
<td></td>
</tr>
<tr>
<td>Nicotinic acid (niacin)</td>
<td>LDL ↓ 5%-25%</td>
<td>Flushing (reduced with ASA therapy), GI distress, hepatotoxicity, worsened diabetes control</td>
</tr>
<tr>
<td></td>
<td>HDL ↑ 15%-35%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TG ↓ 20%-50%</td>
<td></td>
</tr>
<tr>
<td>Fibric acids</td>
<td>LDL ↓ 5%-20%</td>
<td>Dyspepsia, gallstones, myopathy</td>
</tr>
<tr>
<td></td>
<td>HDL ↑ 10%-20%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TG ↓ 20%-50%</td>
<td></td>
</tr>
<tr>
<td>Cholesterol absorption inhibitor (ezetimibe)</td>
<td>LDL ↓ 17%</td>
<td>? increased cancer risk; increased transaminase</td>
</tr>
</tbody>
</table>
levels when used in conjunction with statin

Omega-3 Fatty Acids | TG ↓ 50% + | May increase LDL, prolongation of bleeding time

Hypertension

<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>SYSTOLIC</th>
<th>DIASTOLIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>80-89</td>
</tr>
<tr>
<td>Stage 1 HTN</td>
<td>140-159</td>
<td>90-99</td>
</tr>
<tr>
<td>Stage 2 HTN</td>
<td>≥160</td>
<td>≥100</td>
</tr>
</tbody>
</table>

- Diagnosis is based on 2 or more readings at 2 or more visits. (Use the average.)
- If systolic and diastolic readings fall in different categories, the higher category is selected.

Etiology
Essential hypertension: 90% (aka primary or idiopathic)
Secondary hypertension: 10%
Causes: Acute or chronic renal failure, renal artery stenosis due to atherosclerotic disease (more common in older males), renal artery stenosis due to fibromuscular dysplasia (more common in younger females), sleep apnea, primary hyperaldosteronism, pheochromocytoma, Cushing’s syndrome, hypo/hyperthyroidism, hyperparathyroidism, aortic coarctation
Also
- Cigarettes, caffeine, pain, anxiety, drugs (amphetamines, cocaine), medications (cold medicine, NSAIDs, oral contraceptive pills, steroids, TCAs, cyclosporine), ephedra, ma huang, licorice (an aldosterone agonist, licorice root is used in many herbals)

Evaluation for all patients at initial diagnosis should include
- Careful lifestyle evaluation and assessment of cardiovascular risk factors
- Identify secondary causes of hypertension
- Assess presence or absence of target-organ damage (e.g., renal disease)

Relevant initial studies
- ECG – screen for LVH, ischemia
- BUN, Cr, Na, K – screen for renal disease, hyperaldosteronism
- Glucose – screen for diabetes and Cushing’s
- Hct – screen for anemia
- UA – screen for renal disease
- Lipid profile
When to suspect secondary hypertension

- Onset of hypertension prior to age 30
- Onset (not just diagnosis) after age 50
- Sudden onset hypertension
- Poor response to anti-hypertensives or increase in blood pressure after previous period of good response to anti-hypertensives

Possible work-up for secondary hypertension

- Renal artery duplex (renal artery stenosis)
- Renin/aldosterone ratio (primary hyperaldosteronism)
- 24 hour urine cortisol (Cushing’s)
- 24 hour VMA, metanephrines, catecholamines (pheo)
- Ankle/arm indices – >1 is normal (aortic coarctation)
- TSH
- PTH if patient has hypercalcemia
- Think of alcohol and sleep apnea

Blood pressure goals:

- For most: <140/90
- For patients with diabetes or renal disease: <130/80

Treatment

<table>
<thead>
<tr>
<th>Lifestyle modification</th>
<th>Weight reduction, dietary sodium reduction, DASH diet, exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiazide diuretics</td>
<td>Recommended initial therapy in uncomplicated patients</td>
</tr>
<tr>
<td>Beta blockers</td>
<td>Indicated for patients with stable CHF or prior MI. May also benefit those with migraine or essential tremor.</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>First or second line. Recommend for patients with CHF, prior MI, diabetic nephropathy, chronic renal failure</td>
</tr>
<tr>
<td>ARBs</td>
<td>Likely the same indications as ACE inhibitors but not fully proven, use when ACE inhibitors are not tolerated</td>
</tr>
<tr>
<td>Alpha blockers</td>
<td>Consider in patients with BPH, not usually a first-line HTN agent</td>
</tr>
<tr>
<td>CCBs</td>
<td>First or second line. Particular use when CAD, intermittent claudication, migraine, or Raynaud’s. Avoid non-dihydropyridine CCBs with beta blockers.</td>
</tr>
<tr>
<td>Aldosterone antag’s</td>
<td>Used for add-on therapy for resistant hypertension or in those with primary hyperaldosteronism</td>
</tr>
<tr>
<td>Cental alpha agonists</td>
<td>Used for add-on therapy. Watch for rebound hypertension with abrupt discontinuation.</td>
</tr>
</tbody>
</table>

Musculoskeletal

Shoulder Pain Differential Diagnosis

| Young adults            | Overuse injuries, subluxation, shoulder instability, fractures, AC separation |
| Older adults            | Rotator cuff tendonitis or impingement syndrome, rotator cuff tears, subacromial bursitis, adhesive capsulitis (frozen shoulder), bicipital tendonitis, osteoarthritis, fractures, AC separation, |
myofascial pain, cervical spine radiculopathy, polymyalgia rheumatica

**Low Back Pain Differential Diagnosis**

- Lumbar strain (70% of all adult primary care patients with back pain)
- Degenerative disk disease (DJD) (10%)
- Herniated disk (4%)
- Spinal stenosis (3%)
- Osteoporotic compression fracture (4%)
- Spondylolysis (2%)
- Others: traumatic fracture, congenital disease, spondylolysis, neoplastic disease, infection (including shingles), inflammatory arthritis, referred pain from visceral disease (prostatitis, chronic PID, nephrolithiasis, pyelonephritis, AAA, pancreatitis, cholecystitis)

**Knee Pain Differential Diagnosis**

- Medial knee pain Medial compartment osteoarthritis, pes anserine bursitis, MCL injury, medial meniscal tear, referred pain from hip
- Anterior knee pain Injury of quad mechanism (quad muscle, quad tendon, patella, patellar tendon), large knee effusions, patellofemoral syndrome, advanced osteoarthritis involving entire knee, prepatellar bursitis, patellar tendonitis (“jumper’s knee”), inflammatory arthritis, septic arthritis, Osgood Schlatter disease (epiphysitis occurring in patients under age 19)
- Lateral knee pain Iliotibial band syndrome, lateral compartment osteoarthritis, LCL injury, lateral meniscus tears

**Preventive Medicine / Screening**

**USPSTF = U.S. Preventive Services Task Force**

**NCEP = National Cholesterol Education Program**

**ACS = American Cancer Society**

1. **Hypertension**
   - USPSTSTF recommends periodic screening for all persons > 18 years old.
   - JNC VII: Recheck every 2 years if SBP<120 and DBP <80.
   - Recheck every year if SBP 120-139 DBP 80-89.
   - Check BP every visit.

2. **Hyperlipidemia**
   - USPSTF recommends screening for men 35 years and older. Include men age 20-35 and women age > 20 if other risk factors for heart disease are present.
   - USPSTF screening includes total cholesterol and HDL (both can be measured non-fasting). If elevated, order fasting lipid panel.
   - NCEP recommends full fasting lipid panel every 5 years for adults 20 or older.

3. **Type 2 diabetes**
   - USPSTF recommends screening in asymptomatic with sustained blood pressure greater than 135/80.

4. **Colorectal Cancer**
All individuals 50-75 years old should be screened. (Earlier for patients with family history of colon cancer, FAP, or HNPCC)

Screening options: FOBT (3 stool card samples) every year, combination of FOBT every 3 years and flex sig every 5 years, colonoscopy every 10 years.

5. Breast Cancer
- Consensus: women ages 50-69 should be screened annually with clinical breast exam and mammography.
- Recommendation varies for women ages 40-49.
- USPSTF recommends screening mammography with or without clinical breast exam every 1-2 years for women ages 40 and older.
- American Cancer Society, American College of Obstetricians and Gynecologists, American College of Radiology, and American Medical Association recommend screening beginning at age 40 (most recommend every 1-2 years between ages 40-49, then yearly).
- American College of Physicians and American Academy of Family Practice recommend screening for average risk females beginning at age 50.

6. Cervical Cancer
- Most groups (ACOG, AAFP, AMA, AAP) continue to recommend initiation of Pap test screening at age 18 or when sexually active. Screen annually until at least 3 normal Pap tests, then q 2-3 years.
- ACS now recommends initiation of screening 3 years after the onset of sexual activity but no later than age 21. Screen with annual Pap test or q 2 years if liquid-based cytology (ThinPrep) is used until age 30. After age 30, screen q 2-3 years if low risk and no history of abnormal results.
- Continue screening until age 65-70 (USPSTF recommends age 65) if repeated normal Pap tests or following total hysterectomy (including cervix) for benign disease.

7. Prostate Cancer
- Remains controversial.
- USPSTF does not recommend for or against PSA and digital rectal exam (DRE) for prostate cancer screening.
- ACS recommends that DRE and PSA be discussed and offered annually to men age 50 and over who have a life expectancy of at least 10 years. Consider earlier screening (age 45) for African-American men or those with a first-degree relatives with prostate cancer before 65 years of age.
- Counsel patients on an individual basis regarding these screening tests.

8. Osteoporosis
- USPSTF recommends women ages 65 and older be screened (bone density scan, i.e. DXA). Screen women ages 60 and older if risk factors are present.

9. Chlamydial Infection
- USPSTF recommends routine screening of all sexually active women age 24 and younger, and other asymptomatic women at increased risk.
Clerkship director: Douglas Paauw, M.D.
Web site: http://depts.washington.edu/medclerk/

REFERENCES & HELPFUL RESOURCES
1. UpToDate online
2. Pocket Medicine, Marc S. Sabatine editor, 2000
3. Internal Medicine Clerkship Guide, Douglas Paauw et al., 2007
4. Harrison’s Principles of Internal Medicine
5. Cecil’s Essentials of Medicine
6. Evidence-Based Physical Diagnosis, Steven McGee, 2007
7. Washington Manual of Internal Medicine
8. Sapira’s Art and Science of Bedside Diagnosis, 2005 (3rd edition)

WARD TIPS
1. Be on time!
2. Preround before morning rounds.
3. If you don’t understand something, ask.
4. Be sure to eat, drink enough fluids, and go to the bathroom. You can’t take good care of others if you don’t take care of yourself.
5. Try to get enough sleep.
6. Make every attempt to go to teaching conferences, including morning report.
7. Keep up on reading, patient write-ups, and studying for the final exam.

SELECTED TOPICS IN INTERNAL MEDICINE
Fluids
Total Body Water and Compartments:
TBW is approximately 60% of weight in males and 50-55% in females
- Value varies with age, sex and lean body mass
- Lowest in the elderly and obese; highest in the lean and young
Divided into two main compartments:
Intracellular = 2/3 of TBW (approximately 40% of body weight)
Extracellular = 1/3 of TBW (approximately 20% of body weight)
  a. Interstitial fluid = 3/4 of ECF (approximately 16% of body weight)
  b. Intravascular fluid = 1/4 of ECF (approximately 4% of body weight)
    Na is the main extracellular cation
    K is main intracellular cation

Signs of volume depletion
- Weight loss, postural hypotension, decreased skin turgor, dry mucous membranes, oliguria, tachycardia, increased BUN/ Cr ration

Signs of volume overload (often iatrogenic):
- Weight gain, jugular venous distension, edema, rales

Volume resuscitation in a volume depleted patient: Assess fluid status often
- If hypotensive: choose fluid that stays in the intravascular space (NS or Ringer’s)
- Ringer's has K+ so use with caution in renal failure/anuric patients
- Lactate is converted to HCO₃ in body, buffers acid.
- Use LR for large infusions, NS can → non-gap hyperchloremic metabolic acidosis.

### COMPARISON OF ECF TO CRYSTALLOID SOLUTIONS

<table>
<thead>
<tr>
<th></th>
<th>ECF</th>
<th>NS</th>
<th>1/2NS</th>
<th>LR</th>
<th>D5W</th>
<th>3%NS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺</td>
<td>142</td>
<td>154</td>
<td>77</td>
<td>130</td>
<td>0</td>
<td>513</td>
</tr>
<tr>
<td>Cl⁻</td>
<td>103</td>
<td>154</td>
<td>77</td>
<td>109</td>
<td>0</td>
<td>513</td>
</tr>
<tr>
<td>K⁺</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Ca++]</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>27</td>
<td>0</td>
<td>0</td>
<td>28</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>pH</td>
<td>7.4</td>
<td>4</td>
<td>4.9</td>
<td>6.7</td>
<td>5</td>
<td>5</td>
</tr>
</tbody>
</table>

Avg. Daily Water Losses
- Urinary 800-1500cc
- Intestinal 0-250
- Lungs, skin 600-900

Avg. Daily Electrolyte losses
- Na⁺ 100 mEq
- K⁺ 100 mEq
- Cl⁻ 150 mEq

### Maintenance needs for fluids and electrolytes
- Water 30-35 mL/kg
- Na⁺ 1 mEq/kg
- K⁺ 1 mEq/kg
- Cl⁻ 1.5 mEq/kg

Use D51/2NS for maintenance.

### Calculate Maintenance fluids: 4:2:1 rule
- For first 10 kg 4 mL/kg/hr
- For next 10 kg 2 mL/kg/hr
- For each kg over 20 1/mL/kg/hr

70 kg man = 40 + 20 + 50 = 110cc/hr
(may also use the 100:50:20 rule which gives maintenance for a 24 hr period)

### Electrolyte Abnormalities

#### Hypernatremia (Na > 145 mEq/L, deficit of water relative to Na)

**Causes** Reduced water intake, hypothalamic dysfunction (reduced thirst), inability to get to water (most common), increased water loss, insensible losses (burns, fever/heat, mechanical ventilation), GI loss (vomiting, NG tube, diarrhea), renal loss (central diabetes insipidus, nephrogenic diabetes insipidus, osmotic diuresis), hypertonic infusions, water shift out of extracellular fluid compartment, seizure, extreme exercise

**History/sxs** Lethargy, weakness, thirst, restlessness, oliguria/anuria, irritability that can progress to seizures, coma and death

**PE** Vital signs (BP, orthostatics, temperature), dry mouth and mucous membranes, flushed skin, lack of tears and decreased salivation, hyperreflexia

**Work-up** ASSESS VOLUME STATUS (vital signs, orthostatics, JVP, skin turgor, mucous membranes, edema, BUN/Cr, uric acid)

- If hypovolemic: get urine Na to determine if cause is extra/intrarenal
  - If intrarenal: urine Na > 20 mEq/L
  - If extrarenal: urine Na < 20 mEq/L
- If euvoletic: determine ADH activity with urine osms
  Uosm <300 and increased urine volume may be complete DI
  Uosm 300-600 and increased urine volume may be secondary to renal losses
  (diuretics, osmotic diuresis), partial DI, or reset osmostat
  Uosm >600 may be extrarenal H2O loss (GI or insensible)
- If hypervolemic: usually exogenous NaCl infusion/resuscitation or mineralocorticoid
  excess

Rx
  If hypovolemic, restore intravascular volume with isotonic fluid first, then replace
  free water. Calculate free water deficit:
  FWD = \{ 0.6 x ideal body weight x [(Na/140) - 1] \} (x 0.85 in women)
  Replace about 50% in the first 24 hours (too quickly ->cerebral edema)
  If patient is hypervolemic: loop diuretics + D5W
  With central DI, use DDAVP
  With nephrogenic DI, treat underlying cause, salt restriction, thiazide diuretics

Hyponatremia (Na < 130 mEq/L, excess of water relative to Na)

History/sxs
  Lethargy, disorientation, weakness, muscle cramps, anorexia,
  nausea/vomiting, agitation, stupor, seizures

PE
  Vital signs (BP, orthostatics, temperature), edema, ascites, lung crackles,
  decreased deep tendon reflexes, positive Babinski, Cheyne-Stokes respiration

Work-up/dx
  Determine tonicity
  Hypertonic hyponatremia: Presence of another effective osmole in excess (glucose,
  mannitol). Rule out pseudohyponatremia first!
  For every 100 mg/dL rise in glucose above 100 the Na will
  decrease by 1.6 mEq/L
  Isotonic hyponatremia: Secondary to hyperlipidemia/hyperproteinemia
  Hypotonic hyponatremia: True excess of water relative to Na

For hypotonic hyponatremia next determine volume status (vitals, orthostatics, JVP, etc)

If hypovolemic:
  Renal if Urine sodium is > 20. Causes include diuresis, hypoaldo,
  salt-wasting nephropathy
  Extrarenal if urine sodium is <20. Causes include GI losses,
  third-spacing, insensible losses

If hypervolemic:
  CHF, nephrotic syndrome, cirrhosis

If euvoletic:
  SIADH, psychogenic polydipsia, reset osmostat . Know the meds
  that cause hyponatremia- hydrochlorathiazide, SSRI's , carbamezepine.

Rx
  Hypovolemic: Correct Na deficit.
  Deficit=0.6 x body weight x(140 - measured Na)(x .85 in women)
  Overly rapid correction may lead to central pontine myelinosis
  Rate of correction should not exceed 0.5 mEq/L/hr
  Hypervolemic: sodium and water restriction, diuretics
  Euvolemic: water restrict
Hypokalemia (K < 3.5-3.7)

Causes
1) Diuretics
2) GI losses (vomiting, diarrhea)
3) Low magnesium (common in alcoholics). Other causes: Increased entry into cells, metabolic acidosis, increased beta-adrenergic activity, increased urinary loss, primary hyperaldosteronism, secondary hyperaldosteronism, renal tubular acidosis types I and II, very low intake

Signs/Sxs
Muscle cramps, ileus, weakness, nausea, vomiting, rhabdomyolysis, arrhythmias, hyperglycemia, polyuria, polydipsia

EKG Δs
Prominent U wave, flattened T wave, prolonged QT, AV block, ventricular ectopy

Work-up
Urine K+ to determine if loss is extra/intrarenal

<table>
<thead>
<tr>
<th>High Urinary K+ (renal loss)</th>
<th>Low Urinary K+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>GI loss</td>
</tr>
<tr>
<td>Renal tubular acidosis</td>
<td>Internal shifts</td>
</tr>
<tr>
<td>Drugs</td>
<td>Insulin, beta2-agonists, alkalosis</td>
</tr>
<tr>
<td>Acetazolamide</td>
<td>Dietary deficiency</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td></td>
</tr>
</tbody>
</table>

Rx
Administer K+: orally if possible, IV if necessary
For urinary wasting consider K+ sparing diuretic

Hyperkalemia (K > 5.0)

Causes
Increased intake, metabolic acidosis, insulin deficiency or hyperglycemia, beta adrenergic blockade, rhabdomyolysis, reduced K+ excretion, renal failure, hypoaldosteronism, drugs, (K+ sparing diuretics, ACE-I, TMP-SMX, succ, dig, beta-blockers), pseudohyperkalemia secondary to hemolyzed blood sample

Signs/Sxs
Ileus, constipation, weakness, hypotension, arrhythmias

EKG Δs
Peaked T waves, flattened P waves, widened QRS that can progress to sine wave pattern that is life-threatening

Rx
Administer Ca++ to stabilize membranes
Increase K+ entry into cells
Insulin and glucose (amp of D50)
Beta-adrenergic agonists
Sodium bicarb
Increase renal excretion by administering kaliuretic diuretics
Induce diarrhea, use K+-binding resin
Hemodialysis

Hypocalcemia (Ca < 8.5)

Causes
Decreased Mg, sepsis, alkalosis (increased Ca binding to albumin causing decreased ionized Ca), blood transfusion (Ca binds to citrate), renal failure (increased PO4 binds Ca), hypoparathyroidism, pancreatitis

Signs/Sxs
Tetany, hyperreflexia, Chvostek's and Trousseau's signs, ventricular ectopy, hypotension

Rx
CaCl: centrally for severe hypocalcemia
Calcium gluconate
Monitor for vasoconstrictive ischemia

Hypercalcemia (Ca > 10.5)
Causes
Hyperparathyroidism, malignancy, thiazides, vitamin D excess, sarcoid, TB, Milk alkali syndrome, Paget’s dz, Addison’s dz, acromegaly, Ca intake

Signs/Sxs
Hypovolemia, nausea, vomiting, ileus, shortened QT interval, coma

Rx
Correct hypovolemia and promote Ca clearance with NS
Lasix to get UOP >100 cc/hour
Pamidronate
Dialysis

Hypophosphatemia (PO₄ < 3.0)
Causes
Hormone alterations (hyperparathyroidism), alcohol, intracellular shifts (beta-agonists), decreased nutritional intake, GI disease, Vit D deficiency, glucose loading (PO₄ enters cells with glucose), respiratory alkalosis, sepsis, DKA (leads to osmotic diuresis and PO₄ loss)

Signs/Sxs
Reduced myocardial contractility, reduced ATP production, severe hemolytic anemia, impaired leukocyte function, platelet disorders, myopathy, metabolic encephalopathy

Rx
Replace with nutritional source (i.e. milk), Fleet enema orally
IV if levels are less than 1mg/dL

Hyperphosphatemia
Causes
Increased administration orally, intravenously, or rectally, hypoparathyroidism, pseudohyperparathyroidism, acromegaly, tumor cell lysis, rhabdomyolysis, renal insufficiency

Rx
Phosphate binders, dialysis

Hypomagnesemia (Mg < 1.5)
Causes
Decreased intake, GI or renal loss, malabsorption, redistribution out of ECF, chronic thiazide and loop diuretic use, primary hypoaldosteronism, chronic alcoholism or alcohol withdrawal, toxins (amp B, cyclosporine, aminoglycosides, pentamidine), complicated by hypocalcemia and hypokalemia, inherited renal tubular defects, serum levels may be normal despite total body depletion because most of the stores are in bone, muscle and soft tissue

Signs/Sxs
Tetany, lethargy, anorexia, convulsions, arrhythmias

Rx
Moderate deficiency can be replaced orally but Mg is poorly absorbed by the GI tract and large doses of magnesium can cause diarrhea
Severe deficits require parenteral replacement

Hypermagnesemia
Causes
Excessive exogenous load (IV infusion, oral salts, magnesium salt enemas), renal insufficiency

Signs/Sxs
Diminished deep tendon reflexes that may progress to flaccid paralysis, bradycardia, hypotension, heart block secondary to the calcium channel blocking effects of high magnesium

Rx
ECF expansion and loop diuretics, dialysis if severe

Acid-Base Disturbances
Definition
acidemia – arterial blood pH < 7.36  
alkalemia – arterial blood pH > 7.44  
acidosis – process that causes the accumulation of H⁺  
alkalosis – process that causes the accumulation of OH⁻

**General Approach**

1. Identify the primary process.
2. Identify the compensatory process.
3. Calculate the anion gap correcting for low albumin.
4. If the anion gap is elevated, calculate osmolar gap.
5. If the anion gap is elevated, use delta-delta to find simultaneous metabolic disorders.
6. Use clues from the history and physical exam (particularly, assess volume status by checking orthostatics) to determine specific conditions causing alterations.

(Courtesy of “Internal Medicine Clerkship Guide,” 2007; by Paauw, Burkholder and Migeon.)

**History**

Ingestion (ethylene glycol, paraldehyde, etc.), vomiting, diarrhea, blurry vision, fever, neurological status, alcohol use, H/O type 1 diabetes mellitus (precipitants include infection, lack of insulin, and new-onset diabetes), medication history

<table>
<thead>
<tr>
<th>PRIMARY DISORDERS*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary disorder</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
</tr>
</tbody>
</table>

(Adapted from “Pocket Medicine,” 2000; edited by Sabatine.)

*Numerous processes may occur simultaneously. (If three primary disorders co-exist, then it is known as the "triple ripple." Note that there cannot be two co-existent respiratory disorders.)

**Compensation**

- Occurs when the respiratory or renal system reacts to correct an altered pH
- It never fully corrects an altered pH; if the pH is normal, consider a mixed disorder

**Respiratory:** Hyper- or hypoventilation to alter the P_aCO₂ to counteract primary metabolic process (respiratory compensation takes minutes)

**Renal:** Excretion or retention of H⁺/HCO₃ by kidneys to counteract primary respiratory process (renal compensation take hours to days)
### RULES OF COMPENSATION

<table>
<thead>
<tr>
<th>Primary disorder</th>
<th>Mechanism</th>
<th>Mixed Disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>↓ $P_aCO_2 = 1.25 \times \Delta HCO_3$</td>
<td>If $P_aCO_2$ is too low $\rightarrow$ concomitant 1° respiratory alkalosis</td>
</tr>
<tr>
<td></td>
<td>($P_aCO_2 \sim$ last two digits of pH)</td>
<td></td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>↑ $P_aCO_2 = 0.75 \times \Delta HCO_3$ (through hypoventilation)</td>
<td>If $P_aCO_2$ is too high $\rightarrow$ concomitant 1° respiratory acidosis</td>
</tr>
<tr>
<td>Acute respiratory acidosis*</td>
<td>↑ $HCO_3 = 0.1 \times \Delta P_aCO_2$ (or ↓ pH = 0.008 \times \Delta P_aCO_2)</td>
<td>If $HCO_3$ is too high $\rightarrow$ concomitant 1° metabolic alkalosis</td>
</tr>
<tr>
<td>Chronic respiratory acidosis</td>
<td>↑ $HCO_3 = 0.4 \times \Delta P_aCO_2$ (or ↓ pH = 0.003 \times \Delta P_aCO_2)</td>
<td></td>
</tr>
<tr>
<td>Acute respiratory alkalosis</td>
<td>↓ $HCO_3 = 0.2 \times \Delta P_aCO_2$</td>
<td>If $HCO_3$ is too low $\rightarrow$ concomitant 1° metabolic acidosis</td>
</tr>
<tr>
<td>Chronic respiratory alkalosis</td>
<td>↓ $HCO_3 = 0.4 \times \Delta P_aCO_2$</td>
<td></td>
</tr>
</tbody>
</table>

*In acute (uncompensated) respiratory acidosis, the pH falls before the kidneys have time to compensate*

**Check anion gap**
The anion gap is the difference between the measured cations and measured anions

\[
\text{Anion Gap} = \text{Sodium} - (\text{Chloride} + \text{Bicarbonate})
\]

Normal AG = 8-12

Note: for each 1gm/dl decrease in albumin below 4, subtract 2.5 from the nml AG range.

**Calculate osmolar gap**

\[
\text{Osm gap} = (\text{measured serum Osm}) - (\text{calculated Osm})
\]

Where

\[
\text{Calculated Osm} = 2(\text{sodium}) + \text{BUN}/2.8 + \text{glucose}/18.
\]

(If the Osm gap is > 10, consider methanol or ethylene glycol ingestion.)

**Calculate the delta-delta**

In an isolated anion gap metabolic acidosis, the change in anion gap ($\Delta AG$) should rise by the same amount that the bicarbonate falls ($\Delta HCO_3 = 24 - HCO_3$). Use the delta-delta when an anion gap is present to determine simultaneous metabolic processes. There are 2 ways of calculating the delta-delta as detailed below.

A. Determine the change in anion gap ($\Delta AG$) which = measured anion gap - normal anion gap:

- If the $\Delta AG + HCO_3 < 22 \rightarrow$ AG met. acidosis + non-AG metabolic acidosis
- If the $\Delta AG + HCO_3 = 22-30 \rightarrow$ isolated AG metabolic acidosis
- If the $\Delta AG + HCO_3 > 30 \rightarrow$ AG metabolic acidosis + metabolic alkalosis

(Adapted from “Pocket Medicine,” 2000; edited by Sabatine.)
B. Divide the change in anion gap by the bicarbonate level (ΔAG / ΔHCO₃):  
- If ΔAG / ΔHCO₃ < 1 = AG metabolic acidosis + non-AG metabolic acidosis  
  (there is a loss of HCO₃ greater than expected)
- If ΔAG / ΔHCO₃ is between 1-2 = isolated metabolic acidosis  
  (there is the expected 1:1 relationship with an ↑ AG and ↓ HCO₃)
- If ΔAG / ΔHCO₃ > 2 = AG metabolic acidosis + metabolic alkalosis  
  (there is a loss of HCO₃ less than expected)

(Adapted from “Pocket Medicine,” 2000; edited by Sabatine and “Internal Medicine Clerkship Guide,” 2007; by Paauw, Burkholder, and Migeon.)

### Etiologies of Respiratory Acidosis and Alkalosis

<table>
<thead>
<tr>
<th>RESPIRATORY ACIDOSIS</th>
<th>RESPIRATORY ALKALOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category</td>
<td>Etiology</td>
</tr>
<tr>
<td>Upper airway</td>
<td>a. Acute airway</td>
</tr>
<tr>
<td>abnormalities</td>
<td>obstruction</td>
</tr>
<tr>
<td></td>
<td>b. Laryngospasm</td>
</tr>
<tr>
<td></td>
<td>c. Obstructive sleep</td>
</tr>
<tr>
<td></td>
<td>apnea</td>
</tr>
<tr>
<td>Lower airway</td>
<td>Asthma, COPD</td>
</tr>
<tr>
<td>abnormalities</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Lung parenchyma</td>
<td>a. Pneumonia</td>
</tr>
<tr>
<td>abnormalities*</td>
<td>b. Pulmonary edema</td>
</tr>
<tr>
<td></td>
<td>c. Restrictive lung</td>
</tr>
<tr>
<td></td>
<td>disease</td>
</tr>
<tr>
<td>Thoracic cage</td>
<td>a. Pneumothorax</td>
</tr>
<tr>
<td>abnormalities</td>
<td>b. Flail chest</td>
</tr>
<tr>
<td></td>
<td>c. Kyphoscoliosis</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>CNS depression,</td>
</tr>
<tr>
<td></td>
<td>neuromuscular</td>
</tr>
<tr>
<td></td>
<td>disorders</td>
</tr>
</tbody>
</table>

(Adapted from “Pocket Medicine,” 2000; edited by Sabatine.)

*Lung parenchyma abnormalities often cause hypoxia, leading to respiratory alkalosis and ultimately, respiratory muscle fatigue causing respiratory acidosis

### Metabolic Acidosis

Divided into anion gap and non-anion gap metabolic acidosis.

**ANION GAP** Metabolic Acidosis

- Methanol
- Uremia
- Lactic Acidosis
- Ethylene Glycol
- Paraldehyde
- Aspirin (metab acidosis + resp alkalosis: hyperventilation due to CNS effect)
Ketoacidosis

NON-ANION GAP Metabolic Acidosis
Loss of HCO₃ through the gut or kidney (see etiologies below)

<table>
<thead>
<tr>
<th>ANION GAP METABOLIC ACIDOSIS</th>
<th>NON-ANION GAP METABOLIC ACIDOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Category</strong></td>
<td><strong>Etiology</strong></td>
</tr>
<tr>
<td>Ketoacidosis</td>
<td>Diabetes mellitus, alcoholism, Starvation</td>
</tr>
<tr>
<td>Lactic acidosis</td>
<td>a. Circulatory or respiratory failure, sepsis</td>
</tr>
<tr>
<td></td>
<td>b. Ischemic bowel or limb, seizure, malignancy, liver failure, diabetes mellitus</td>
</tr>
<tr>
<td></td>
<td>c. Metformin, carbon monoxide or cyanide poisoning</td>
</tr>
<tr>
<td>Renal failure</td>
<td>Accumulation of organic anions (phosphates, sulfates, etc.)</td>
</tr>
<tr>
<td>Ingestions</td>
<td>a. Methanol (blurred vision)</td>
</tr>
<tr>
<td></td>
<td>b. Ethylene glycol (oxalate crystals in urine, ΔMS, renal or cardiopulmonary failure)</td>
</tr>
<tr>
<td></td>
<td>c. Paraldehyde</td>
</tr>
<tr>
<td></td>
<td>d. Salicylates</td>
</tr>
</tbody>
</table>

Medications Acetazolamide

(Adapted from “Pocket Medicine,” 2000; edited by Sabatine.)

Work-up of acidosis
Labs: ABG, electrolytes, CBC, Chem 7, LFTs
If elevated AG – check for ketonuria, assess renal function, uremia, lactate levels, toxin screen, osmolar gap, urinalysis
If normal AG – check urine anion gap ( = [U_{Na} + U_{K}] - U_{Cl}), which is an indirect assay for renal excretion of NH₄ and equals unmeasured anions – unmeasured cations
-Negative UAG – increased renal NH₄ secretion indicates GI causes, type I RTA
or exogenous acids
Positive UAG – failure of the kidneys to secrete NH₄ indicated type I or type IV RTA or early renal failure
Also check urine pH, serum K⁺ and FE_HCO₃ to further distinguish between types of RTA

**Metabolic Alkalosis**
Caused by
1. Loss of H⁺ from the GI tract or kidney
2. Exogenous alkali or contraction alkalosis (diuresis causes the excretion of HCO₃-poor fluid and extracellular fluid “contracts” around a relatively fixed amount of HCO₃)
3. Hypercapnia: respiratory acidosis causes renal compensation with HCO₃ retention
4. Volume depletion causes proximal reabsorption of NaHCO₃ and increased aldosterone
5. Hyperaldosteronism causes distal Na⁺ reabsorption in exchange for H⁺ and K⁺ excretion
6. Hypokalemia causes transcellular H⁺/K⁺ shift (hydrogen ions shift into cells as potassium moves from the cell into the extracellular space)

**ETIOLOGIES OF METABOLIC ALKALOSIS**

<table>
<thead>
<tr>
<th>Category</th>
<th>Etiologies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saline-responsive</td>
<td>GI loss of H⁺: vomiting, NGT drainage, villous adenoma</td>
</tr>
<tr>
<td></td>
<td>Diuretics</td>
</tr>
<tr>
<td></td>
<td>Prerenal azotemia (severe)</td>
</tr>
<tr>
<td></td>
<td>Post-hypercapnea</td>
</tr>
<tr>
<td>Saline-resistant</td>
<td>Hypertensive: mineralocorticoid excess – hyperaldosteronism, Cushing’s syndrome</td>
</tr>
<tr>
<td></td>
<td>Normotensive: severe hypokalemia, exogenous alkali load, Bartter’s syndrome, Gitelman’s syndrome</td>
</tr>
</tbody>
</table>

(Adapted from “Pocket Medicine,” 2000; edited by Sabatine.)

**Work-Up**
Check volume status and urine chloride
U_Cl < 20 mEq/L indicates saline-responsive
U_Cl > 20 mEq/L indicates saline-resistant (except concurrent diuretic use)

**Cardiology**

**ECG interpretation** (Use a systematic approach)

1. Rate
2. Rhythm
3. Axis
4. Intervals
5. Chamber enlargement (voltages)
6. QRST changes

**Rate**
Each little box = 0.04 seconds, each big box = 0.2 seconds
Rate = 300/x where x is no. of large boxes between each QRS complex

<table>
<thead>
<tr>
<th>Boxes b/t QRS complex</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>300</td>
<td>150</td>
<td>100</td>
<td>75</td>
<td>60</td>
</tr>
</tbody>
</table>
Rhythm  Normal sinus rhythm exists when “there is a P for every QRS and a QRS for every P” AND the p wave is upright in lead 2

Intervals  PR > 0.2 seconds = AV node block
QRS > 0.12 seconds = interventricular conduction delay (a BBB)
QT prolongation (varies, but > 450ms) can lead to torsades de pointes
?calculate QTc

Axis  Normal axis is between -30 and +90.
If QRS is upward in I and aVF, then axis is normal
LAD: axis > -30, or QRS is up in I and down in II
RAD: axis > +90, or QRS is down in I and up in II

QRS Axis Determination

Chamber  LVH: left axis deviation, S waves in V1-V2, large R waves in aVL, V5, V6
Enlargement RVH: large R waves in V1-V2
LAE: late negative deflection in biphasic P wave best seen in V1.
Negative portion of P wave should be > 1mm deep and 1 box wide.
RAE: large peaked P wave greater than 2.5mm high best seen in lead II

QRS/ T △'s  ST elevation: ACS, coronary spasm, pericarditis, normal early repolarization
ST depression: myocardial ischemia, digitalis, hypokalemia, LBBB or LVH
T-wave inversion: myocardial ischemia or infarct, pericarditis, cardiomyopathy, electrolyte abnormalities

Chest Pain Differential Diagnosis (From “Pocket Medicine,” 2000; edited by Sabatine)
Angina  Esophageal reflux
MI  Esophageal spasm

11
Pericarditis  Mallory-Weiss tear
**Aortic dissection**  Peptic ulcer disease
Pneumonia  Biliary disease
Pleuritis  Pancreatitis
Pneumothorax  Costochondritis
**PE**  Herpes Zoster
Pulmonary HTN  Anxiety

**Acute Myocardial Infarction / Unstable Angina**

5 Risk Factors:
1. Smoking
2. HTN
3. hyperlipidemia,
4. FH of premature CAD (1st degree female < 55, male < 45)
5. Age (female > 55, male > 45)

Coronary Risk Equivalent (chances = to someone w/previous MI):

**Causes**  Ruptured atherosclerotic plaque, coronary spasm, cocaine are most common

**Clinical**  Chest pain: typical is dull, squeezing, >30 min duration, not positional, not pleuritic, may radiate to jaw, neck, L arm. This may be ↓ in DM, women. Also nausea, lightheadedness, SOB.

**PE**  Diaphoresis, pallor. Severe: new MR murmur, findings of heart failure including ↑ JVP, crackles in lungs, S3, S4

**Labs**  Cardiac enzymes: Troponin, CK-MB, LDH; myoglobin is earliest marker, highly sensitive, but not specific.

**Studies**  ECG, CXR

**Rx**  Acutely:  Morphine (for pain management and decrease preload)
          Oxygen 4L NC or mask
          Nitroglycerin 0.4 mg SL q 5 min x 3, as limited by BP: (a. dilation ↓ preload, v. dil ↓ afterload)
          Aspirin, 325 mg PO (chewed)
          **B-Blocker**: metoprolol 25 mg po q 6hr, titrate up as tol.

Consider thrombolytics (Alteplase (tPA), Reteplase) or PCI (preferred)

<table>
<thead>
<tr>
<th><strong>Thrombolytic Therapy</strong></th>
<th><strong>Indications</strong></th>
<th><strong>Contraindications</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sxs c/w MI &gt; 30 min and &lt; 12 hr and ST ↑ ≥1mm in ≥2 contiguous leads or Presumably new LBBB (not on prior ECG, etc)</td>
<td>Absolute: Any prior ICH or non-hemorrhagic stroke w/in 1 year Intracranial neoplasm, aneurysm, or AVM Active internal bleeding Suspected aortic dissection</td>
<td>Relative: SBP &gt; 180 on presentation INR &gt; 2 or known bleeding diathesis Trauma or major surgery w/in 2-4 wks Prolonged CPR (&gt;10 min) Recent internal bleeding w/in 2-4 wks Noncompressible vascular punctures Prior streptokinase exposure</td>
</tr>
<tr>
<td>Age limits: in pts &gt; 75, thrombolysis is reasonable, but higher risk of ICH</td>
<td>Time limits: Benefits after 12 hrs are not clear, but if pt presents in 12-24 hrs and still has ST elevation, then consider thrombolytics</td>
<td></td>
</tr>
</tbody>
</table>
Inpt Mgmt:  Admit to CCU/monitored bed
ASA 325 mg PO qd
B-blocker: metoprolol 25 mg PO q6hr, titrate as tolerated for SBP
ACE inhibitor: lisinopril 5 mg qd, start >6hrs post onset
IV heparin 12 U/kg/hr infusion
Stress test and/or Echo after 5 days.
If stress test positive, do cardiac catheterization

D/C Meds:  ASA 325 mg PO qd
Continue beta-blockers
Continue ACE inhibitors
Add lipid-lowering agent and modify risk factors (smoking cessation, etc)

Meds that improve mortality post-MI
1. ASA  2. beta-blockers  3. Statins  4. ACEI (< than other 3)

### CONGESTIVE HEART FAILURE

<table>
<thead>
<tr>
<th>LV failure</th>
<th>RV failure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Causes</strong>  HTN and CAD account for 50-75% of LV failures. Other common causes include valvular disease, idiopathic dilated cardiomyopathy. Less common causes include chronic alcohol use, hypothyroidism, and toxins (i.e. chemotx)</td>
<td>Majority of RV failure due to LV failure. Also idiopathic pulm HTN, secondary pulm HTN (COPD, chronic PE, etc.), tricuspid valve disease, cardiomyopathy, RV infarct.</td>
</tr>
</tbody>
</table>
| **Symptoms**  **Pulmonary:** orthopnea, dyspnea on exertion, paroxysmal nocturnal dyspnea, cough, frothy hemoptysis  | increasing abdominal girth, RUQ pain, anorexia, LE edema  
With pulm HTN: SOB, exercise intolerance |
| **Signs**  Leg edema, crackles, S3, PMI >3 cm and laterally displaced, cool, mottled LE’s, abnormal abdominojugular reflex  | Leg edema, JVP > 8 cm, RV parasternal heave, S3, Ascites, abnormal abdominojugular reflex |
| **CXR findings**  Enlarged cardiac silhouette, cephalization of pulmonary blood flow, pulmonary edema, pleural effusion, Kerley B lines  | Depends on cause; similar to LV findings if LV is cause; if cor pulmonale, may see flattening of diaphragms, bullae consistent with COPD |

Work-up of new left-sided CHF:
- Chemistry panel, cholesterol, ECG, CXR, Echo.
- Echocardiogram: <40% EF is considered systolic dysfunction, but >40% does not rule out CHF. Focal wall motion abnormalities suggest ischemic injury.
- If unclear cause, consider EtOH, thyroid dz, hemochromatosis, amyloidosis, HIV

**Treatment:** goals are to ↓ symptoms, prevent complications, ↑ survival.
- 1st-line tx for LV systolic failure: ACEIs.
  - Diuretics usually needed for sodium/water overload.
  - B-blockers should be added after acute symptoms begin to resolve.
  - Add digoxin if pt remains symptomatic on full-dose ACEI and diuretics.
  - Sodium restriction.
First line tx for diastolic dysfunction: B-blocker and calcium channel blockers (increase cardiac output in these patients by increasing diastolic filling time)
- ACEI second line.

**Meds that improve mortality in CHF**
1. ACEIs  
2. Beta-blockers  
3. Spironolactone (in class IV HF)


CHF mortality predictor: www.SeattleHeartFailureModel.org

**Valvular Heart Disease**

**Aortic Stenosis**
- Causes: Bicuspid aortic valve, calcific stenosis, rheumatic heart disease
- Clinical: Angina, exertional syncope, heart failure, a. fib
- PE: Systolic crescendo-decrescendo murmur at right upper sternal border, radiates to carotids and apex
- Studies: ECG, CXR, Echo, cardiac cath for pressures
- Rx: Avoid exertion, diuretics for CHF, AVR surgery for symptomatic AS or asymptomatic AS with ↓ LV function

**Aortic Insufficiency**
- Causes: Rheumatic heart disease, bicuspid aortic valve, infective endocarditis, root disease (Marfan’s, syphilis, HTN, aortic dissection, RA, SLE)
- Clinical: Acute: pulmonary edema and hypotension
  Chronic: LV decompensation leads to CHF
- PE: Diastolic decrescendo murmur at left upper sternal border, wide pulse pressure, S3, laterally displaced and diffuse PMI
- Studies: ECG: look for LVH, LAD
  CXR: look for cardiomegaly, aortic dilatation
  Echo: assess LV size and function
- Rx: Reduce afterload: nifedipine, ACE inhibitors, hydralazine.
  Digoxin, diuretics for CHF.
  Surgery for acute or symptomatic AI

**Mitral Stenosis**
- Causes: Rheumatic heart disease, congenital, myxoma, SLE, amyloid, carcinoid
- Clinical: Dyspnea, pulm edema, atrial fibrillation, emboli, pulm HTN, hemoptysis
PE Low-pitched diastolic rumble at apex, opening snap, loud S1
Studies ECG shows LAE
        CXR shows dilated left atrium
        Echo to assess pressures and valve area; cardiac cath for pressure gradients
Rx Na restriction, diuresis, B-blockers, anticoagulation for atrial fibrillation,
        Surgery for symptomatic MS or pulmonary HTN

**Mitral Regurgitation**
Causes Myxomatous degeneration, endocarditis, rheumatic heart disease, collagen vascular disease, LV dilatation, ruptured chordae tendinae, papillary muscle dysfunction
Clinical Acute: pulmonary edema, hypotension
        Chronic: progressive dyspnea with exertion, fatigue, pulm HTN
PE High-pitched, blowing holosystolic murmur at apex, radiates to axilla
Studies ECG: look for LAE, LVH, atrial fibrillation
        CXR: look for dilated LA, dilated LV
        Echo to assess degree of MR
        Cardiac cath for pressures
Rx Reduce afterload: ACE inhibitors, hydralazine, nitrates
        Reduce preload: diuretics, nitrates
        Inotropy: digoxin
        Surgery for acute or symptomatic MR, or asymptomatic with ↓LV function

**Infectious Endocarditis** (Infection of endothelium of heart)
Acute (ABE) usu. involves normal valves with virulent organism (S. aureus)
Subacute (SBE) usu. involves abnormal valves with less virulent organism (S. viridans)

<table>
<thead>
<tr>
<th>Organisms</th>
<th>Prosthetic valve &lt; 6 months post-op</th>
<th>S. epidermidis, S. aureus</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prosthetic valve &gt; 6 months post-op</td>
<td>S. viridans, S. epidermidis</td>
</tr>
<tr>
<td></td>
<td>Native valve, IDVU</td>
<td>S. aureus</td>
</tr>
<tr>
<td></td>
<td>Native valve, non-IVDU</td>
<td>S. viridans, S. aureus</td>
</tr>
</tbody>
</table>

Risk Factors IVDU, indwelling venous catheters, rheumatic heart disease, prosthetic valve, prior history of IE
Clinical Sx Persistent fever, anorexia, weight loss, fatigue
PE Fever, weight loss, murmur, Janeway lesions, Osler’s nodes, Roth spots, petechiae, splinter hemorrhages, clubbing
Studies Blood cultures (3 sets), CBC w/ diff, ESR, RF, Chem 7, UA, Ucx, ECG – TTE first, then TEE if needed for diagnosis of valvular lesion

**Infectious Endocarditis** (Infection of endothelium of heart)
Acute (ABE) usu. involves normal valves with virulent organism (S. aureus)
Subacute (SBE) usu. involves abnormal valves with less virulent organism (S. viridans)

Organisms Prosthetic valve < 6 months post-op S. epidermidis, S. aureus
Prosthetic valve > 6 months post-op S. viridans, S. epidermidis
Native valve, IDVU S. aureus
Native valve, non-IVDU S. viridans, S. aureus

Risk Factors IVDU, indwelling venous catheters, rheumatic heart disease, prosthetic valve, prior history of IE
Clinical Sx Persistent fever, anorexia, weight loss, fatigue
PE Fever, weight loss, murmur, Janeway lesions, Osler’s nodes, Roth spots, petechiae, splinter hemorrhages, clubbing
Studies Blood cultures (3 sets), CBC w/ diff, ESR, RF, Chem 7, UA, Ucx, ECG – TTE first, then TEE if needed for diagnosis of valvular lesion

**Duke Criteria**

<table>
<thead>
<tr>
<th>Major</th>
<th>Minor</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Sustained bacteremia with organism known to cause endocarditis</td>
<td>1. Predisposing condition</td>
</tr>
<tr>
<td>2. Endocardial involvement documented by Echo or clearly established NEW valvular regurgitation</td>
<td>2. Fever</td>
</tr>
<tr>
<td></td>
<td>3. Vascular phenomena</td>
</tr>
<tr>
<td></td>
<td>4. Immune phenomena</td>
</tr>
<tr>
<td></td>
<td>5. + blood cultures not meeting major criteria</td>
</tr>
<tr>
<td></td>
<td>6. + echo not meeting major criteria</td>
</tr>
</tbody>
</table>

Highly probable diagnosis with 2 major, or 1 major plus 3 minor, or 5 minor criteria

Rx  
Get blood cultures first. Abx usually for 6 weeks
Native valve ABE: nafcillin + gentamicin or vancomycin + gentamicin
Native valve SBE: PCN/ampicillin + gentamicin
Prosthetic valve: vancomycin + gentamicin + rifampin

New endocarditis prevention guidelines: no need to prophylax most valvular lesions including AS and MR. Only prophylax artificial valves and previous endocarditis

Cardiac Tamponade

Causes  
Malignancy, uremia, proximal aortic dissection with rupture, myocardial rupture, idiopathic. Also see causes for pericardial effusion

Clinical  
Fatigue, dyspnea

PE  
Beck’s Triad: distant heart sounds, ↑ JVP, hypotension
Pulsus paradoxus seen in ~75% (↓ SBP >10mmHg during inspiration)

Studies  
ECG shows low voltage, electrical alternans
Echo will show effusion
Cardiac catheterization to get pressures

Rx  
Volume resuscitation, pericardiocentesis

Pericarditis / Pericardial Effusion

Causes  
Infection (coxsackie B, echovirus, adenovirus, EBV, VZV, HIV), idiopathic, uremia, neoplasm, collagen vascular disease, trauma, drug-induced, acute post MI

Clinical  
Pleuritic chest pain that decreases when leaning forward, fever

PE  
Pericardial friction rub, distant heart sounds if pericardial effusion present

Studies  
ECG shows diffuse ST elevations, PR depression
CXR shows cardiomegaly if effusion present
Pericardiocentesis: do cell counts, TP, LDH, glucose, gram stain, culture

Labs  
BUN, Cr, ANA, RF to rule out non-infection etiologies

Rx  
NSAIDs or ASA. If effusion is infected, may need pericardial drainage and antibiotics. If recurrent, consider pericardial window.

Aortic Dissection (Extravasation of blood into and along aortic media)

Acute < 2wks, Chronic > 2wks

Type A involves proximal aorta  
Type B involves distal aorta only

Risk Factors  
Age, hypertension, connective tissue disorder (Marfan’s for type A) congenital aortic anomaly, pregnancy, blunt trauma, cocaine cardiac/aortic surgery

Clinical  
Severe tearing chest pain radiating to back, syncope, CHF

Studies  
CT, aortic angiogram, TEE, MRI

Complication  
Aortic rupture, tamponade, obstruction of branching arteries leading to ischemia, aortic regurgitation

Prognosis  
For acute proximal dissection, 1% mortality per hour x 48 hours.

Rx  
Medical: IV B-blockers, then IV vasodilators, morphine for pain.
For chronic or Type B dissections, aim for long-term control of BP.
Surgical: for proximal dissection or distal with progression / complications.
# Arrhythmias

Tables adapted from Washington Manual of Medical Therapeutics, 30th ed., 2001

## AV Nodal Block

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
<th>Causes</th>
<th>Clinical</th>
<th>ECG</th>
<th>Rx</th>
</tr>
</thead>
<tbody>
<tr>
<td>First-degree AV block</td>
<td>Conduction delay within AV node</td>
<td>Increased vagal tone, drug effect, electrolyte abnml, ischemia</td>
<td>Usually asymptomatic</td>
<td>PR interval &gt; 0.2 seconds</td>
<td>No therapy needed usually. If symptomatic, consider pacing</td>
</tr>
<tr>
<td>Second-degree AV block: Mobitz Type I (Wenckebach)</td>
<td>Progressive PR interval prolongation until dropped beat occurs</td>
<td>Increased vagal tone, antiarrhythmics, electrolyte abnml, myocardial ischemia</td>
<td>Usually asymptomatic/benign</td>
<td></td>
<td>Stop drugs or correct cause. If symptomatic, can give atropine 0.5 mg IV q 2min to max of 0.04 mg/kg. If persistently symptomatic, consider pacing</td>
</tr>
<tr>
<td>Second-degree AV block: Mobitz Type II</td>
<td>Abrupt AV conduction block with no conduction delay or change in PR interval in preceding impulses.</td>
<td>Antiarrhythmics, myocardial ischemia, increased vagal tone, conduction system disease</td>
<td>Fatigue, palpitations, lightheadedness, syncope</td>
<td></td>
<td>Because of potential for progression to complete heart block, treat with permanent pacemaker</td>
</tr>
<tr>
<td>Third-degree AV block</td>
<td>All atrial impulses fail to conduct to ventricles</td>
<td>Ischemia, infarction, drug toxicity, amyloidosis, sarcoidosis, metastatic disease, polymyositis, scleroderma, Chagas disease</td>
<td>Dyspnea, CHF, lightheadedness, angina, syncope</td>
<td>Ventricular escape rhythm, no relationship between P waves and QRS</td>
<td>Permanent pacemaker</td>
</tr>
</tbody>
</table>
### Narrow-Complex (Supraventricular) Arrhythmias

#### Atrial Fibrillation
- **Causes:** Cardiac surgery, **hypertension**, acute alcohol ingestion, theophylline toxicity, pericarditis, MI, idiopathic or “lone” AF
- **Clinical:** (sx are poorly correlated)Palpitations, skipped beats, lightheadedness, breathlessness, CHF, angina, syncope
- **ECG:** Irregularly fluctuating baseline with irregular and sometimes rapid ventricular response
- **Rx:** Rate control, cardioversion (electric or pharm) post anticoag if duration >48h

#### Atrial Flutter
- **Causes:** Structural heart disease predisposes to development, CAD, CHF, valvular disease, pericarditis
- **Clinical:** Asymptomatic, or palpitations, lightheadedness, syncope
- **ECG:** Regular rhythm with “Sawtooth” appearance of P waves, atrial rate of 280-350 bpm
- **Rx:** See atrial fibrillation

#### Multifocal Atrial Tachycardia (MAT)
- **Causes:** COPD w/cor pulmonale, dig toxicity, rheumatic heart disease, ACS
- **Clinical:** Asymptomatic or palpitations, chest pain, lightheadedness, fatigue
- **ECG:** PR variable, 3 or more P wave morphologies
- **Rx:** β-blocker, diltizem, amiodarone. Do NOT cardiovert

#### Paroxysmal SVT
- **Causes:** Accessory conduction pathway; increased frequency in CAD, COPD, CHF
- **Clinical Palpitations at paryoxysmal onset, anxiety, low exercise tolerance**
- **ECG Rate seldom <150 bpm, regular, seldom see P waves**
- **Rx:** Vagal stimulation (carotid massage, Valsalva) AV blockade (β-blocker, diltizem, digoxin), amiodarone, cardioversion
Ventricular Tachycardia: a series of 3 or more ventricular complexes that occur at rate of 100-250 bpm where origin of activation is within the ventricle.
Causes: CAD, cardiomyopathy, infiltrative disease, SLE, RA, malignancy that involves the heart, congenital myocardial defects
Clinical: Palpitations, breathlessness, lightheadedness, angina, syncope, hemodynamic collapse, death
ECG: >3 Wide QRSs with T-wave polarity opposite of major QRS deflection.
Rx: DC cardioversion for pulseless VT, antiarrhythmics, ICD implant

Ventricular fibrillation: results from rapid, repetitive activation of ventricles from multiple areas of depolarization.
Causes: Ischemia, infarct, structural abnml, electrolyte abnml, drug toxicity
Clinical: Sudden hemodynamic collapse and death
ECG: Irregular, rapid oscillations, variably amplitudes, no identifiable QRS complexes or T waves
Rx: DC cardioversion, antiarrhythmic therapy. Long-term: implant ICD and prophylactic antiarrhythmics

Others include **SVT with aberrancy**, WPW syndrome, accelerated idioventricular rhythm

### Antiarrhythmic Agents

<p>| <strong>Class I agents:</strong> Inhibit fast sodium channels | Quinidine, Procainamide, Disopyramide |
| <strong>Class Ib:</strong> Can be proarrhythmic and ↑mortality | Lidocaine, Mexiletine, Tocainide, Phenytoin |
| <strong>Class Ic:</strong> Can be proarrhythmic and ↑mortality | Flecainide, Propafenone |
| <strong>Class II:</strong> B-adrenergic antagonists | Metoprolol, Atenolol, Propranolol |
| <strong>Class III:</strong> Prolong action potential duration and repolarization | Amiodarone, Sotalol, Bretylium, Ibutilide, Dofetilide |
| <strong>Class IV agents:</strong> Calcium-channel antagonists | Verapamil, Diltiazem |</p>
<table>
<thead>
<tr>
<th>Common Meds used in ACLS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Epinephrine</strong>: increases myocardial and cerebral blood flow. Recommended dose is 1 mg (10mL of 1:10,000 solution) every 3-5 minutes</td>
</tr>
<tr>
<td><strong>Vasopressin</strong>: at high doses acts as a peripheral vasoconstrictor. Give single dose of 40 units IV</td>
</tr>
<tr>
<td><strong>Atropine</strong>: used for symptomatic bradycardia and asystole. Give 0.5-1.0 mg IV; repeat every 3-5 minutes as necessary. For asystole or PEA give 1.0 mg every 3-5 minutes</td>
</tr>
<tr>
<td><strong>Amiodarone</strong>: give after defibrillation and vasopressors in persistent VT or VF. 300 mg rapid infusion diluted in 20-30mL of normal saline or dextrose in water. Subsequent doses are 150 mg by rapid infusion for persistent VT/VF. Then give 1mg/minute infusion for 6 hours, then 0.5 mg/minute to a max daily dose of 2 grams.</td>
</tr>
<tr>
<td><strong>Lidocaine</strong>: Used to treat VT/VF that persists after defibrillation and epinephrine. Give 1.0-1.5 mg/kg q5-10 minutes to max of 3 mg/kg</td>
</tr>
<tr>
<td><strong>Procainamide</strong>: used for VT when lidocaine fails or is contraindicated. Infuse 20-50 mg/minute to max of 17 mg/kg</td>
</tr>
<tr>
<td><strong>Magnesium sulfate</strong>: use for VT/VF/torsades de pointes. Give 1-2 grams IV over 1-2 minutes up to 4-6 grams</td>
</tr>
<tr>
<td><strong>Adenosine</strong>: used for SVT. Give 6 mg as rapid IV bolus over 1-3 seconds, followed by 20 cc saline flush.</td>
</tr>
<tr>
<td><strong>Diltiazem / verapamil</strong>: Use for atrial fibrillation, flutter, or multifocal atrial tachycardia. IV diltiazem bolus is 0.25 mg/kg. Second dose can be given after 15 minutes. Maintenance infusion is 5-15 mg/hr, titrated to control ventricular rate. Verapamil initial dose is 2.5-5.0 mg IV, followed by 5-10 mg IV up to max of 20 mg.</td>
</tr>
<tr>
<td><strong>Isoproterenol</strong>: may be useful for refractory torsades de pointes after magnesium and electrical pacing have failed. Give 2-10 microgram/minute</td>
</tr>
<tr>
<td><strong>Sodium bicarbonate</strong>: indicated for hyperkalemia, acidosis, tricyclic anti-depressant overdose, and to alkalinize urine in drug overdoses. Give 1.0 mEq/kg IV initially, then 0.5 mEq/kg q 10 minutes. Not useful for hypoxic lactic acidosis. In ACLS setting, acidosis is likely due to inadequate ventilation and this should be addressed first.</td>
</tr>
</tbody>
</table>
### Gastroenterology

#### COMMON ETIOLOGIES OF ABDOMINAL TENDERNESS

<table>
<thead>
<tr>
<th>Area of pain</th>
<th>Potential etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>RUQ</td>
<td>Liver, GB: Cholelithiasis, cholecystitis, choledocholithiasis, cholangitis, hepatitis, hepatic carcinoma, liver abscess (remember: pneumonia)</td>
</tr>
<tr>
<td>Midepigastric</td>
<td>Stomach, Pancreas, Aorta: Gastritis, peptic ulcer disease, pancreatitis, leaking AAA</td>
</tr>
<tr>
<td>LUQ</td>
<td>Spleen: Splenic rupture, splenomegaly, splenic infarct, splenic abscess</td>
</tr>
<tr>
<td>Umbilicus</td>
<td>Gastroenteritis, intestinal ischemia or infarct, obstruction or ileus, obstipation</td>
</tr>
<tr>
<td>RLQ</td>
<td>Appendicitis, inflammatory bowel disease (IBD), nephrolithiasis or ureteral stone, ovarian torsion, ectopic pregnancy, pelvic inflammatory disease (PID)</td>
</tr>
<tr>
<td>LLQ</td>
<td>Diverticulitis, IBD, toxic megacolon, nephrolithiasis or ureteral stone, ovarian torsion, ectopic pregnancy, PID</td>
</tr>
</tbody>
</table>

(Adapted from “Pocket Medicine,” Sabatine, 2000.)

Others to consider: Pre-herpetic neuralgia, hypercalcemia, acute intermittent porphyria

### Gastroesophageal Reflux Disease

**Pathophys**

Excessive transient relaxation of the LES or incompetent sphincter tone

Esophageal mucosal damage caused by prolonged contact with acid/ bile salts

Hiatal hernia can contribute to decreased LES tone and act as a reservoir for refluxed gastric contents

**Hx**

Heartburn, atypical “angina,” regurgitation of stomach contents, **cough**, asthma, **hoarseness** and warning symptoms: dysphagia, early satiety, weight loss or bleeding; precipitants: fatty foods, caffeine, colas, alcohol, cigarettes, supine positioning, large meals

**Dx**

Often diagnosed based upon history and trial of acid suppressive agent. EGD reserved for those with refractory symptoms or warning symptoms to detect Barrett’s esophagus, stricture, ulcer or esophagitis. 24-hour esophageal pH monitoring if the diagnosis is unclear

**Rx**

**CONSERVATIVE** - include elevating the head of the bed 6 inches, avoiding precipitants, avoiding late meals, avoiding calcium channel blockers, anticholinergics, sedatives and theophylline which can exacerbate symptoms

**MEDICAL** - Antacids; H₂-blockers; or proton-pump inhibitors which are more effective than standard-dose H₂-blockers (breakthrough nocturnal symptoms can be controlled by adding an H₂-blocker)

**SURGERY** - Fundoplication is an option for those who require continuous or increasing medical therapy or for whom continuous PPI therapy is undesirable

### Acute Liver Failure

**Definition**

Acute hepatic disease + coagulopathy + encephalopathy

Fulminant < 8 weeks; subfulminant between 8 weeks and 6 months

**Etiology**

Discussed under “ABNORMAL LIVER TESTS.”
Clinical manifestations

Neurologic: asterixis, encephalopathy, cerebral edema (Cushing’s reflex hypertension + bradycardia; papillary dilatation; decerebrate posturing; apnea)
Cardiovascular: hypotension with low SVR
Pulmonary: respiratory alkalosis, impaired peripheral O2 uptake, ARDS
Gastrointestinal: GI bleeding, pancreatitis
Renal: ATN, hepatorenal syndrome, hyponatremia, hypokalemia, hypophosphatemia
Hematology: coagulopathy (consider DIC)
Endocrine: hypoglycemia
Skin: jaundice, telangiectasias, palmar erythema, caput medusae, Dupuytren’s contractures, Terry’s nails (white proximal nail beds), gynecomastia
GU: testicular atrophy

Diagnostic studies

Labs CBC: anemia, neutropenia, thrombocytopenia; COAGs: increased PT, PTT, BT; decreased albumin, viral serologies, toxicology screen (APAP levels q1-2hr until peak determined) and others, as below

Imaging RUQ U/S, abdominal CT, doppler studies of hepatic and portal veins
Liver Bx CORRECT COAGULOPATHY with fresh frozen plasma prior to procedure

CHILD-TURCOTTE-PUGH Scoring System (severity of liver disease)

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumin (g/dl)</td>
<td>&gt;3.5</td>
<td>2.8-3.5</td>
<td>&lt;2.8</td>
</tr>
<tr>
<td>Bilirubin (mg/dl)</td>
<td>&lt; 2</td>
<td>2-3</td>
<td>&gt;3</td>
</tr>
<tr>
<td>INR</td>
<td>&lt;1.7</td>
<td>1.7-2.3</td>
<td>&gt;2.3</td>
</tr>
<tr>
<td>Ascites</td>
<td>Absent</td>
<td>Slight</td>
<td>Moderate</td>
</tr>
<tr>
<td>Encephalopathy (stage)*</td>
<td>None</td>
<td>Stage 1 or 2</td>
<td>Stage 3 or 4</td>
</tr>
<tr>
<td>Total Points</td>
<td>5-6</td>
<td>7-9</td>
<td>10-15</td>
</tr>
<tr>
<td>Classification</td>
<td>A</td>
<td>B</td>
<td>C</td>
</tr>
<tr>
<td>Survival</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 year</td>
<td>100%</td>
<td>80%</td>
<td>45%</td>
</tr>
<tr>
<td>2 year</td>
<td>85%</td>
<td>60%</td>
<td>35%</td>
</tr>
</tbody>
</table>

*Stage I - altered mental status (Brit J Surg 60:646;1973)
Stage II - lethargy, confusion (Hepatology 19:1513;1994)
Stage III - stupor
Stage IV - coma
Abnormal Liver Tests
Markers of hepatic functional status
- Albumin: general marker for liver protein synthesis
- Prothrombin time: depends on synthesis of coagulation factors I, II, V, VII and X
- Bilirubin: product of heme metabolism in the liver

Markers of hepatic injury
- Aminotransferases: intracellular enzymes
  - ALT (or SGPT) specific for liver
  - AST (or SGOT) found in liver, heart, skeletal muscle, kidney and brain
- Alkaline phosphatase: enzyme bound in hepatic canalicular membrane
  - Found not only in liver, but also in bone, intestines and placenta
  - CONFIRM liver origin with increased GGT

ABNORMAL LIVER TESTS IN DIFFERENT PATTERNS OF LIVER INJURY

<table>
<thead>
<tr>
<th>TYPE OF LIVER INJURY</th>
<th>Aminotransferase</th>
<th>Bilirubin</th>
<th>Alkaline phosphate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatocellular</td>
<td>↑↑</td>
<td>±↑</td>
<td>±↑</td>
</tr>
<tr>
<td>Cholestasis</td>
<td>±↑</td>
<td>↑↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>Isolated hyperbilirubinemia</td>
<td>Near normal</td>
<td>↑↑</td>
<td>Near normal</td>
</tr>
<tr>
<td>Infiltrative</td>
<td>±↑</td>
<td>±↑</td>
<td>↑</td>
</tr>
</tbody>
</table>

(Adapted from “Pocket Medicine,” 2000; edited by Sabatine.)

Patterns of Liver Injury
1. Hepatocellular injury
   - Viral hepatitis (~60%) - HAV, HBV, HCV, HDV, HEV, CMV, EBV, HSV, VSV. Test viral serologies. Aminotransferases significantly elevated (>1000) in acute viral hepatitis, with ALT > AST. Test viral serologies. Very high transaminases are not seen with hepatitis C.
   - Autoimmune -
     a. Type 1: anti-smooth muscle Ab (ASMA), ANA
     b. Type 2: anti-liver/kidney microsome type 1 (anti-LKM1)
     c. Type 3: anti-soluble liver antigen (anti-SLA)
   - Drugs and toxins (~20%) - Alcohol (AST:ALT > 2:1), medications: acetaminophen, phenytoin, INH, rifampin, sulfonamides, tetracycline, amiodarone, propylthiouracil, toxins (toxicology screen)
   - Vascular (hypotensive/CHF) - Ischemic, congestive, Budd-Chiari, veno-occlusive disease
   - Hereditary (systemic disease) - Hemochromatosis (elevated transferrin saturation and serum ferritin), α-1-antitrypsin deficiency (also, emphysema), Wilson’s disease (Kayser-Fleischer ring on slit lamp exam, elevated serum free copper and 24-hour urinary copper level; low serum ceruloplasmin)
   - Metabolic - Steatohepatitis, hepatic glycogenosis (Mauriac Syndrome in IDDM)
   - Idiopathic (20%)  

2. Cholestasis - Evaluate with RUQ ultrasound (also, ERCP, cholangiogram, cholescintigraphy)

| No biliary ductal dilatation on U/S | Biliary ductal dilatation on U/S |
Charcot’s triad = RUQ pain, jaundice and fever/chills
Reynaud’s pentad = above plus shock and altered mental status

3. Isolated hyperbilirubinemia
   Unconjugated (indirect)
   a. **Overproduction of bilirubin** - hemolysis, ineffective erythropoiesis, hematoma resorption
   b. **Defect in conjugation** - Gilbert’s (“zhil-bear”—benign) and Crigler-Najjar syndromes
   Conjugated (direct)
   **Defect in bile secretion** - Dubin-Johnson and Rotor syndromes

4. Infiltration
   a. Malignancy: HCC (↑AFP), metastatic disease (colon = ↑CEA), lymphoma
   b. Granulomas (TB, sarcoidosis, histoplasmosis)
   c. Abscess (amoebic, pyogenic)

Ascites
   Abnormal accumulation of fluid (> 25 cc) within the peritoneal cavity
PE
   Shifting dullness, fluid wave (positive LR 5.0), edema (negative LR 0.2), bulging flanks
Dx
   IMAGING - Abdominal U/S (if >100 cc), doppler studies of portal and hepatic veins, echocardiogram (if concerned about right-sided heart disease)

   PARACENTESIS - Obtain albumin, cell count with differential, total protein, gram stain and culture, LDH and glucose; amylase and triglyceride levels, cytology and mycobacterial smear/culture as indicated.

### SERUM TO ASCITES ALBUMIN GRADIENT (SAAG)

<table>
<thead>
<tr>
<th>Portal hypertension-related (SAAG &gt; 1.1)</th>
<th>Non-portal hypertension-related (SAAG &lt; 1.1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intrahepatic: cirrhosis, spontaneous bacterial peritonitis, hepatitis, HCC, liver metastases</td>
<td>Peritonitis: Tuberculosis, ruptured viscus, SBP Vasculitis Pancreatitis</td>
</tr>
<tr>
<td>Post-hepatic: constrictive pericarditis, right-sided CHF, Budd-Chiari</td>
<td></td>
</tr>
<tr>
<td>Pre-hepatic: portal or splenic vein thrombosis</td>
<td></td>
</tr>
</tbody>
</table>

(Adapted from "Pocket Medicine," 2000; edited by Sabatine.)
In portal HTN, expect ascitic fluid to have less albumin than serum—it has been “pushed” into the abdominal cavity by hydrostatic pressure, rather than leaked out through defects in the vasculature or a fluid derived from another source.

**Ascites fluid total protein (AFTP)**

May be used to distinguish intrahepatic and post-hepatic causes of ascites:

a. Cardiac ascites: SAAG > 1.1, but AFTP > 2.5
b. Cirrhotic ascites: SAAG > 1.1, but AFTP < 2.5
c. Spontaneous bacterial peritonitis: SAAG < 1.1, but AFTB < 2.5; >250 PMNs per μl

**Rx**

Decrease sodium intake (1-2gm/day), fluid restriction if hyponatremic diuretics: spironolactone (beware of hyperkalemia), loop therapeutic paracentesis-- if patient is dyspneic or uncomfortable (remove 4-6L; ±colloid replacement)

TIPS (Transjugular intrahepatic portosystemic shunt)

Liver transplantation if eligible (calculate MELD score), EtOH abstinence > 6 mos

**Gastrointestinal Bleeding**

Intraluminal blood loss anywhere from the oropharynx to the anus

*Upper = above the ligament of Treitz*

1. Oropharyngeal bleeding and epistaxis (swallowed blood)
2. Erosive esophagitis
   a. Immunocompetent patient: GERD/Barrett’s esophagus, XRT
   b. Immunocompromised patient: HSV, CMV, Candida
3. Varices (10% of cases)
4. Mallory-Weiss tear (7%; GE junction tear due to retching/vomiting against a closed glottis)
5. Gastritis/Gastropathy (23%)
6. Peptic ulcer disease (46%)
7. Vascular malformations (Dieulafoy’s lesion, Osler-Weber-Rendu)
8. Neoplastic disease
9. Other causes: hiatal hernia ulcerations, coagulopathy, amyloidosis, connective tissue disease

*Lower = below the ligament of Treitz*

1. Diverticular disease (more likely to be diverticulosis than diverticulitis; although diverticula are more common in the left colon, bleeding diverticula are more common in the right colon)
2. Angiodysplasia
3. Neoplastic disease
4. Colitis: infection, ischemic, radiation, inflammatory bowel disease (ulcerative colitis > Crohn’s disease)
5. Hemorrhoids

**Clinical manifestations**

UGIB > LGIB: nausea, vomiting, hematemesis, coffee-ground emesis, epigastric pain, vasovagal reactions, syncope, melena (some briskly bleeding ulcers can present as hematochezia)

LGIB > UGIB: diarrhea, tenesmus, BRBPR or maroon stools
Hx Use of aspirin, NSAIDs, anticoagulants; known anticoagulopathy; cirrhosis; alcohol abuse

PE Vital signs: tachycardia -- 10% volume loss
orthostatic hypotension -- 20%
shock -- 30%

HEENT: pale conjunctivae
Abdominal exam: Localized tenderness or peritoneal signs
Rectal exam (mandatory!): Appearance of stools, anal fissures, hemorrhoids
Skin: signs of chronic liver disease, pallor, delayed capillary refill

Dx Labs Hct, platelet count, PT, PTT, increased BUN/Cr, LFTs
NG tube can Dx UGIB (except if intermittent or duodenal bleed), remove GI contents (prior to EGD and to prevent aspiration); lavage "until clear" to evaluate rate/presence of continued bleeding

Imaging
UGIB: Esophagoduodenoscopy (EGD); potentially therapeutic.
LGIB: 1. Colonoscopy - if bleeding stops spontaneously; potentially therapeutic
2. Tagged RBC scan - uses $^{99m}$Tc-tagged RBC to detect bleeding rates of ≥0.1-1.0 ml/min in stable patients; localization difficult
3. Arteriography - detects bleeding rates of ≥0.5-1.0 ml/min in unstable patients; potentially therapeutic (intraarterial vasopressin infusion or embolization)
4. Exploratory laparotomy

Rx Acute treatment includes hemodynamic resuscitation:
1. Volume resuscitation with IV fluids (NS or lactated Ringer's)
2. Transfusion therapy (type and cross; may use O-negative blood if patient is exsanguinating). GI service generally wants a HCT > 25 to scope.
3. Identify and correct coagulopathies (FFP to normalize PT; keep platelets >50,000)
4. Nasogastric tube lavage
5. Airway management as needed
6. CONSULT GI and surgical services

<table>
<thead>
<tr>
<th>ETIOLOGY of GI BLEED</th>
<th>TREATMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Varices</td>
<td>Octreotide ± endoscopic sclerotherapy</td>
</tr>
<tr>
<td></td>
<td>Band ligation</td>
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<tr>
<td></td>
<td>Balloon tamponade</td>
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<tr>
<td></td>
<td>Embolization or TIPS if previous strategies fail</td>
</tr>
<tr>
<td></td>
<td>When HD stable - beta-blocker and nitrates</td>
</tr>
<tr>
<td>Peptic Ulcer Disease</td>
<td>PPIs</td>
</tr>
<tr>
<td></td>
<td>Endoscopic therapy (injection, thermal contact, laser)</td>
</tr>
<tr>
<td></td>
<td>Mesenteric arteriography with infusion of vasopressin or embolization</td>
</tr>
<tr>
<td>Mallory-Weiss</td>
<td>Usually stops spontaneously</td>
</tr>
<tr>
<td>Angiodysplasia</td>
<td>Arterial vasopressin, endoscopic therapy, surgery</td>
</tr>
<tr>
<td>Esophagitis, gastritis</td>
<td>PPIs, H$_2$-antagonists</td>
</tr>
</tbody>
</table>

(Adapted from "Pocket Medicine," 2000; edited by Sabatine.)
GASTROPATHY & GASTRITIS

<table>
<thead>
<tr>
<th>Classification</th>
<th>Etiology</th>
<th>Clinical manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute gastropathy</td>
<td>NSAIDs, alcohol, stress-related mucosal disease, glucocorticoids</td>
<td>Asymptomatic, anorexia, nausea, vomiting, epigastric pain, UGIB</td>
</tr>
<tr>
<td>Chronic fundal gastritis (“Type A”)</td>
<td>Autoantibodies directed against parietal cells, resulting in a lack of acid and intrinsic factor</td>
<td>Atrophic gastritis, achlorhydria, hypergastrinemia, pernicious anemia, gastric carcinoid tumors and adenoCA</td>
</tr>
<tr>
<td>Chronic antral gastritis (“Type B”)</td>
<td>H. pylori infection</td>
<td>Mostly asymptomatic; can progress to atrophic gastritis with increased risk of gastric adenoCA</td>
</tr>
</tbody>
</table>

Peptic Ulcer Disease

Etiologies H. pylori infection, NSAIDs and aspirin, malignant ulcers, Zollinger-Ellison syndrome (gastrinoma) and other hypersecretory states

Hx Epigastric abdominal pain: 2-5h postprandial in duodenal ulcers, soon after meals in gastric ulcers. Pain relieved by food, antacids in DU > than GU.

Dx Tests for H. pylori: serology (not in peds), urea breath test, EGD + rapid urease testing (CLOtest) or biopsy and histology

EGD or UGI series to detect ulcer

Rx Discontinue NSAIDs and smoking acid suppression with H₂-blockers or PPIs

H. pylori “triple therapy” (e.g. metronidazole 500 mg bid, clarithromycin 500 mg bid, omeprazole 20 mg bid for 10-14 days)

Surgery for intractable symptoms, GI bleeding, Zollinger-Ellison syndrome

Complications GI bleeding, gastric outlet obstruction, perforation, pancreatitis (erosion into the pancreas: seen with ulcers in the posterior wall of the duodenal bulb)

Diverticular Disease

Diverticulosis - Herniations of the colonic mucosa and submucosa through the colonic wall (L > R); may be a consequence of a low-fiber diet; affects 20-50% of patients > 50

Clinical Usually asymptomatic, but may be complicated by microperforations or bleeding

Rx Increase fiber content in diet; or as below (if bleeding occurs)

Diverticulitis - Undigested food and bacteria are retained within a diverticulum -> fecalith formation, obstruction, ischemia, infection and/or perforation. Abscess formation and/or peritonitis are severe presentations.

Clinical LLQ pain, fever, nausea, vomiting, constipation

PE LLQ tenderness, ± palpable mass, ± FOBT, diarrhea, fever, chills, anorexia, nausea, vomiting, dysuria, LLQ mass, Does not cause big lower GI bleeds

Severe – peritonitis, septic shock

Dx Labs: ↑ WBC

Imaging 1. Acute abdominal series to r/o obstruction, free air, ileus

2. Abdominal CT (with contrast for best visualization)
3. Colonoscopy or sigmoidoscopy are CONTRAINDICATED in an acute setting because of increased risk of overt perforation

Rx
Outpt Rx if mild; antibiotics (cipro, metronidazole)
Hospitalize for severe Sx; keep NPO, IV fluids, NGT
Broad-spectrum antibiotics to cover anaerobes and gram-negative rods
Surgery if medical therapy fails, for peritonitis, or to drain an abscess that is inaccessible to percutaneous drainage

Diarrhea (Stool output > 200cc/day)

Etiologies
1. Infectious
   a. Pre-formed toxins (S. aureus, C. perfringens, B. cereus)
   b. Viruses (Norwalk, Rotavirus)
   c. Non-invasive bacteria
      enterotoxin-producing (no fecal WBC or blood) – ETEC, Vibrio cholera
      cytotoxin-producing (+ fecal WBC or blood) – E.coli O157:H7, C. difficile
   d. Invasive bacteria (+ fecal WBC or blood) – enteroinvasive E. coli, Salmonella,
      Shigella, Campylobacter, Yersinia, V. parahelolyticus
   e. Parasites (Giardia, E. histolytica)
   f. Opportunistic (Cryptosporidia, Microsporidia)
   g. Chronic (Giardia, E. histolytica, C. difficile, opportunistic organisms)
2. Malabsorption
   Bile salt deficiency
   Pancreatic insufficiency
   Mucosal abnormalities
   Celiac sprue - check anti-tissue transglutaminase (Most Sensitive and Specific), anti-endomysial, anti-gliadin Abs, plus IgA (false neg in ped <2 yrs)
   Whipple's disease – caused by Trophryrema whippelii
   Tropical sprue
   Intestinal lymphoma
3. Osmotic
   Medications or Lactose intolerance
4. Inflammatory
   Inflammatory bowel disease
   Ischemic colitis
5. Secretory
   Hormonal - VIP (VIPoma); serotonin (carcinoid); calcitonin (medullary cancer of the thyroid); gastrin (Zollinger-Ellison);
   glucagons, substance P, thyroxine
   Villous adenoma
6. Motility
   Irritable bowel syndrome
   Scleroderma (pseudo-obstruction)
   Endocrinopathies

Acute diarrhea (< 3 weeks duration)
If severe dehydration, fever, duration > 5 days, mucus or pus in BMs, bloody diarrhea, abdominal pain, recent travel or recent antibiotic use are present consider:
Labs       fecal leukocytes, FOBT, C. difficile toxin, stool cultures, stool O & P X 3
Imaging flexible sigmoidoscopy/colonoscopy with biopsy

Chronic diarrhea (> 3 weeks duration)
Labs as for acute + consider 24-hour stool collection with evaluation of fecal fat, Giardia antigen, hormone levels, secretin test (pancreatic insufficiency), $^{14}$C-xylose breath test (bile salt deficiency), D-xylose test or small intestinal biopsy (mucosal abnormality), lactose test, ESR

**RESPONSE TO NPO**
- If diarrhea decreases with fasting = malabsorptive etiology
- If diarrhea does NOT change with fasting = secretory etiology

**STOOL OSMOTIC GAP** = $Osm_{stool} (290) - [2 X (Na_{stool} + K_{stool})]$
- If > 50, malabsorptive etiology
- If < 50, secretory etiology

**Inflammatory Bowel Disease**

**Ulcerative Colitis**
Idiopathic inflammation of the colonic mucosa; average age of onset 20-25 yo.

**Clinical**
Grossly bloody diarrhea, lower abdominal cramps, urgency, tenesmus, fulminant colitis, toxic megacolon, perforation

**EXTRACOLONIC**:
Erythema nodosum, pyoderma gangrenosum, aphthous ulcers, iritis, episcleritis, thromboembolic events, seronegative arthritis, chronic hepatitis, cirrhosis, sclerosing cholangitis, cholangiocarcinoma

**Complications**
Stricture; colon cancer (after 10 years, risk incr 1% each year)

**Crohn’s Disease**
Idiopathic inflammation; can **occur anywhere in the alimentary tract**, although often focal at terminal ileum. Bimodal age distribution with peaks in the 20s and from 50-70

**Clinical**
Smoldering disease with abdominal pain, mucus-containing, non-grossly bloody diarrhea; fevers, malaise, weight loss

**EXTRACOLONIC**:
same as UC and also, gallstones and kidney stones

**Complications**
Perianal fissures, perirectal abscesses, stricture, fistulas, cancer (small intestinal and colonic: risk similar to that of UC when the entire colon is involved)

**PATHOLOGY OF ULCERATIVE COLITIS VS. CROHN’S DISEASE**

<table>
<thead>
<tr>
<th>Pathology</th>
<th>Ulcerative Colitis</th>
<th>Crohn’s Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extent</td>
<td>involves the rectum/colon and extends contiguously</td>
<td>can affect any portion of the GI tract from the mouth to the anus. lesions with areas of sparing -- “skip lesions”</td>
</tr>
<tr>
<td>Appearance</td>
<td>granular, friable mucosa with small ulcerations; pseudopolyps</td>
<td>$\geq 1$ cm ulcerations, non-friable mucosa, cobblestoning, deep and lor fissures</td>
</tr>
<tr>
<td>Biopsy</td>
<td>superficial microulcerations, crypt abscesses (PMNs); no granulomas</td>
<td>transmural inflammation with mononuclear cell infiltrate, non-caseating granulomas, fissures</td>
</tr>
</tbody>
</table>
Rx of Inflammatory Bowel Disease
Fiber supplements (unless obstructive symptoms in CD)
No caffeine or gas-producing vegetables
Trial of lactose-free diet in CD
Antidiarrheals and antispasmodics
In acute flare:

MILD - 5-ASA compounds including sulfasalazine, mesalamine or olsalazine
MODERATE - Oral steroids (± azathioprine, methotrexate or 6-mercaptopurine
in CD)
SEVERE - Intravenous steroids (± cyclosporine ± Remicade for refractory CD);
bowel rest, TPN, antibiotics; serial abdominal exams and
radiographs/CT to rule out dilatation, perforation or abscess;
decompression in toxic megacolon.

TNF inhibitors: infliximab, etanercept (place PPD + controls prior to
administration); risk of granulomatous infection higher with
infliximab

Hematology
Anemia (Hematocrit <36/Hgb <13 in females and <41/Hgb <14 in males)
Sx Fatigue, headache, dyspnea, decreased exercise tolerance, chest pain,
pica, melena, BRBPR, hematemesis, dizziness; family history of anemia/heme
disorders, review meds (look for NSAIDS, ASA, coumadin)
PE Pallor, pale mucous membranes, HSM, jaundice, bone tenderness,
Orthostatic hypotension, tachycardia, koilonychia, numbness, paresthesias,
Labs Hgb, Hct, CBC w/ platelets, peripheral smear, retic count, stool guiac
Consider bilirubin, haptoglobin, LDH

Low corrected retic count (<100 billion/L ) & index (<2%) = hypoproliferation
High corrected retic count (>150 billion/L) & index (>2%) = %)=periph destruction, blood loss

Low corrected reticulocyte count/index (underproduction) -> look at MCV:
1. Low MCV (<80) = microcytic anemia
   I Iron-deficiency
      - chronic bleed / decreased supply / increased demand
      - special PE findings : angular cheilosis, atrophic glossitis,
koilonychia, pica
      - lab findings : low Fe, low ferritin, high TIBC, Fe/TIBC <1/6
   T Thalassemias
   S Sideroblastic anemia
2. Normal MCV (80-100) = normocytic anemia
   A Anemia of chronic disease/ inflammatory block (can also be microcytic)
      - lab findings: low Fe, low TIBC, +/- high ferritin, high ESR
      - tx: Fe supplementation unsuccessful, must tx underlying disorder
   N Nephrogenic - low erythropoietin levels
   E Endocrine - thyroid disease
   M myelophthisis - marrow replacement with tumor, fibrosis; teardrop cells
   I IVF- dilution
   A Aplastic anemia
   S sickle cell anemia
3. High MCV (>100) = macrocytic anemia
B12 deficiency (neuro Sx + hypersegmented PMNs)
Reticulocytosis (see in blood loss)
Alcohol
Nutritional - folate deficiency (no neuro findings, + hyperseg PMNs)
Drugs - AZT, MTX

(Thanks to Differential Diagnosis Mnemonics)

High corrected reticulocyte count/index (destruction or loss):
1. Blood loss
2. Hemolysis
   a. Hereditary spherocytosis
   b. Glucose-6-phosphate dehydrogenase deficiency
   c. Sickle cell anemia
   d. Autoimmune hemolytic anemia (AIHA)
   e. Drug-induced hemolytic anemia
   f. Microangiopathic hemolytic anemia (MAHA)
     - DIC, HUS, TTP, HELLP, malignant HTN
   g. Paroxysmal nocturnal hemoglobinuria (PNH)

Thrombocytopenia (Platelet count < 150,000/ mm³)
- at steady state, platelet production should equal destruction/removal
- causes include decreased production, increased destruction, or increased sequestration

<table>
<thead>
<tr>
<th>Platelet count</th>
<th>Risk of bleeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;100,000</td>
<td>No increased risk</td>
</tr>
<tr>
<td>50-100,000</td>
<td>Surgery is o.k., some increased risk with major trauma</td>
</tr>
<tr>
<td>20-50,000</td>
<td>Risk with surgery or trauma</td>
</tr>
<tr>
<td>&lt;10,000</td>
<td>Spontaneous bleeding</td>
</tr>
</tbody>
</table>

History/Sx: Mucocutaneous bleeding, epistaxis, small ecchymoses, petechiae, melena, BRBPR, menorrhagia, symptoms of infection. Review medication list, take thorough medical history (ask about HIV, bleeding disorders, etc)

PE: Look for hepatosplenomegaly, ecchymoses, petechiae, + stool guiac

Labs: CBC with diff and platelets, peripheral smear
Bone marrow aspirate and biopsy in some cases

Etiologies
a. Decreased production: aplastic anemia, myelodysplasia, drugs (EtOH, thiazide diuretics, sulfas), megaloblastic anemia, infiltrative marrow process
b. Increased destruction: immune-mediated (see below), drug-induced, infection, DIC, HUS/TTP, vasculitis, pre-eclampsia
c. Increased sequestration by the spleen

Idiopathic thrombocytopenic purpura (ITP) - autoimmune mediated
- Most common cause of immune thrombocytopenia
- Mediated by an auto-antibody against glycoproteins on platelet surface, platelets then destroyed by the liver, spleen, and marrow
- Often occurs following a viral illness and can be chronic in adults
- See large platelets on smear
- Rx: glucocorticoids, IV IG, platelet transfusion, splenectomy, Vincristine, immunosuppressants, Danazol

**Drug-induced thrombocytopenia**
- More than 100 drugs have been associated with thrombocytopenia
- Syndrome resembling ITP may be seen in those susceptible following ingestion
- Examples are quinine and quinidine derivatives
- Removal of drug is most effective therapy

**Heparin-induced thrombocytopenia (HIT)**
- Production of antibody that leads to destruction of platelets
- Can occur with trace amounts of heparin (IV flush)
- Can also cause platelet aggregation (less common)
- Rx: stop heparin

**Hemolytic-uremic syndrome (HUS) / Thrombotic thrombocytopenic purpura (TTP)**
- Normally von Willebrand factor is cleaved into multimers by a protease; in TTP/HUS an antibody against the protease leads to accumulation of larger multimers; results in platelet aggregation leading to thrombotic microangiopathy
- HUS: thrombocytopenia, renal failure and MAHA; syndrome has been associated with E. Coli O157H7
- TTP: HUS triad plus fever and neurologic abnormalities
- Rx: plasma exchange, immunosuppressants

**Hypercoaguability**
**Risk Factors**
- Virchow’s triad
  1. Endothelial cell injury (trauma, surgery)
  2. Stasis (e.g., immobility s/p hip replacement, long airplane ride)
  3. Hypercoaguable states
     - Acquired: Pregnancy
       - Surgery/trauma
       - Malignancy
       - Smoking
       - Oral contraceptives
       - Antiphospholipid antibody syndrome
       - DIC
     - Familial: Factor V Leiden
       - Antithrombin III deficiency
       - Protein S deficiency
       - Protein C deficiency

**Complications** DVT, PE, stroke
**Rx** anticoagulation—duration of treatment determined by etiology

<table>
<thead>
<tr>
<th>Leukemias</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AML</strong></td>
</tr>
<tr>
<td>Primarily occurs in adults</td>
</tr>
<tr>
<td>Dx: blasts, auer rods</td>
</tr>
<tr>
<td>Tx: chemo, BMT in 1st remission</td>
</tr>
<tr>
<td>---------------------------------</td>
</tr>
<tr>
<td>Complications: leukostasis -&gt; ARDS, intracerebral hemorrhage</td>
</tr>
<tr>
<td>M3 subtype (Promyelocytic): Tx w/transretinoic acid. DIC may occur w/tumor lysis.</td>
</tr>
</tbody>
</table>

**CML**
- One of the myeloproliferative d/o
- Chronic phase (median 3 yrs) -> accelerated phase -> blast crisis
- Dx: Philadelphia chromosome t(9;22), low LAP score
- Tx: gleevec, BMT during chronic phase

**CLL**
- Elderly patients
- Often present w/infection due to hypogammaglobulinemia
- Dx: Smudge cells on smear suggestive; CD5 Ag
- Tx: palliative chemo

**Multiple Myeloma**
- Proliferation of malignant plasma cells. Peak incidence in 70s.
- Sx: bone pain, pathologic fx, fever, epistaxis, subQ nodules (plasmacytomas), renal failure sx, peripheral neuropathy
- Dx: lytic bone lesions on XR, SPEP/UPEP
- Tx: observation for mild cases
- Bisphosphonates
- Palliative XRT, chemotherapy
- Early autologous BMT may increase disease-free survival

**Infectious Disease**

**Fever of Unknown Origin**
- Definition = Fever > 38.3C(101F) on multiple occasions, plus Duration of > 3 weeks, plus Uncertain diagnosis after 1 week of hospitalization*
- Etiology
  - Infectious (TB, abscess, osteo, endocarditis) ~30%
  - Malignancy (lymphoma, leukemia, RCC, HCC) ~30%
  - Collagen vascular disease (JRA, GCA, Stills, Wegener’s) ~30%
  - Other ~10% - drugs, thyroid, Familial Mediterraneann Fever, idiopathic
- Work-up
  - Detailed Hx: travel, pets, immunosuppression, meds, ROS
  - Labs: ESR, LDH, PPD, HIV, BCx x 3, RF, ANA, Heterophile Abs
  - Consider Bx bone marrow, liver, lymph node (TB, Bartonella)
  - Consider CT head/neck to rule out sinus disease, dental abscess, etc.
  - Consider CT abdomen
  - No empiric abx or steroids

**Catheter-related Bloodstream Infection** (Line infection)
- Definition = Isolation of same organism from catheter and peripheral blood
- No alternative source of infection
- Non-contaminated infusate
- Etiology
  - Incidence ~3%; Catheter colonized by flora from patient’s skin or hands of healthcare workers
  - Occasional hematogenous seeding of catheter, especially if thrombosed
- Risk
  - femoral > internal jugular > subclavian
- Prevention
  - Mask, gown, cap, gloves for insertion; chlorhexidine preferred over betadine
Bugs Coag negative staph and S. aureus, enterococci (50%), gram negatives (30%), candida (20%)

Work-up CBC, BCx (1 from line, 2 from separate peripheral sticks)
Examine insertion site for erythema, tenderness, or purulence
Empiric abx (vanco +[aminoglycoside or ceftaz/cefipime or cipro]) if sick
If positive BCx: pull line and culture tip
If non-septic and only coag negative staph are isolated, may change over wire and culture tip; remove if > 15 cfu from line
Adjust abx when sensitivities return

Duration of abx: S. aureus, 2-3 weeks; others, 10-14 days

Osteomyelitis

Definitions Infection of bone resulting in progressive inflammation, destruction, and reformation. Categories: hematogenous seeding, contiguous spread, vascular insufficiency.

Acute Days to weeks; usually painful, febrile; no necrotic bone on bx;
Chronic weeks to months; chronic pain, non-healing fx; elevated ESR, nl CBC

Bugs Hematogenous seeding: S. aureus (~70%); GNRs (~30%). Rarely candida, bartonella, C. immitis, P. acnes.
IVDU: pseudomonas, serratia, candida, TB
Contiguous spread: Staph, strep, enterococcus (75%); GNRs, anaerobes (25%); often polymictobial. Rarely, brucella or TB
Vascular insufficiency/diabetic foot: Polymicrobial staph, strep, enterococcus, proteus, pseudomonas, and/or anaerobes

Sx Fever, bone pain, warmth/swelling are classic presentation

Dx Elevated CBC, ESR; Sed rate >100 in diabetic foot highly specific for osteo.
Blood cultures positive in 50% of acute osteo
Plain film -> soft tissue swelling, bone destruction, periosteal reaction specific but not sensitive; poor yield in first 3 weeks of infection; positive culture and suggestive film obviate biopsy; empiric treatment recommended
CT -> with and w/o contrast very specific
MR preferred for spine/foot
Ultrasound in pediatrics to reduce exposure; fluid, periosteal elevation
Open bone biopsy if high suspicion and negative films or culture due to poor sensitivity of radiologic changes in acute infection

Treatment Hematogenous, Non-IVDU adult -> nafcillin, cefazolin, or vancomycin
Others: see Sanford Guide
Contiguous: Ceftazidime, Cefipime, or Cipro.
Post-ORIF -> Nafcillin + cipro.
Vascular insufficiency: Outpt -> amox/clavulanate or cipro + clindamycin
Inpt-> pip/taz, tic/clav, or amp/sublactam
Chronic: based on biopsy. Abx adjunct to surgery only. +rifampin if S. Aureus
Pneumonia

- Inflammation of the lung parenchyma. 6th leading cause of death in U.S.
- Organisms reach the lung by aspiration, inhalation, or hematogenous spread
- Contributing factors = Impaired glottic reflex or cough, immunocompromised, impaired ciliary function and smoking

PNEUMONIA ETIOLOGY

<table>
<thead>
<tr>
<th>Risk Group / Clinical Setting</th>
<th>Likely organisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Community-acquired</td>
<td>S. Pneumoniae, H. influenza, Mycoplasma, Chlamydia, Legionella, M. catarrhalis, Klebsiella, virus</td>
</tr>
<tr>
<td>Young healthy adults</td>
<td>Mycoplasma, Chlamydia, S. pneumo</td>
</tr>
<tr>
<td>Alcoholism / Aspiration</td>
<td>S. pneumo, anaerobes, H. flu, Klebsiella, TB</td>
</tr>
<tr>
<td>Hospital-acquired</td>
<td>S. aureus, S. pneumo. GNR including Pseudomonas Klebsiella, Enterobacter, Serratia, Acinetobacter</td>
</tr>
<tr>
<td>Nursing Home</td>
<td>Klebsiella, S. Aureus, TB (Consider same organisms as hospital-acquired pneumonia)</td>
</tr>
<tr>
<td>IDU</td>
<td>S. pneumo, anaerobes, S. aureus, TB</td>
</tr>
<tr>
<td>Smokers</td>
<td>S. pneumo, H. flu, M. catarrhalis, Legionella</td>
</tr>
<tr>
<td>HIV, immunocompromised</td>
<td>All of the above plus PCP, fungi, Nocardia, atypical mycobacteria, CMV, HSV</td>
</tr>
</tbody>
</table>

(Adapted in part from “Pocket Medicine”, 2000, edited by Sabatine)

Community-acquired Pneumonia

Symptoms Cough (90%), dyspnea (66%), sputum production (66%), +/- fever, rigors, sweats, myalgias, malaise

Diagnosis History, PE^(vitals, pulse ox)*, CXR (critical), Lab (CBC, ABG, Blood Cx, HIV at discretion), sputum gram stain and culture, consider ABG and blood cultures. Consider bronchoscopy in immunocompromised, critically ill or not responding to treatment
- Fremitus increased with consolidation, decreased with effusion, PTX, or obstructed bronchus.
- Percussion is dull over consolidation or effusion
- chest exam is neither sensitive nor specific in diagnosing CAP

Mgmt Inpatient vs. Outpatient therapy

Risk of Mortality, Pneumonia PORT prediction rules

Step 1: Assess baseline risk predictors—age < 50, normal vitals, no comorbidities, normal mental status
Step 2: Scoring system of the prediction rule:
### ASSIGNMENT TO RISK CLASSES II–V

<table>
<thead>
<tr>
<th>Patient Characteristics</th>
<th>Points assigned</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographic Factors</strong></td>
<td></td>
</tr>
<tr>
<td>Age (male)</td>
<td>No. of years old</td>
</tr>
<tr>
<td>Age (female)</td>
<td>No. of years old – 10</td>
</tr>
<tr>
<td>Nursing home resident</td>
<td>10</td>
</tr>
<tr>
<td><strong>Comorbid Illnesses</strong></td>
<td></td>
</tr>
<tr>
<td>Neoplasm</td>
<td>30</td>
</tr>
<tr>
<td>Liver disease</td>
<td>20</td>
</tr>
<tr>
<td>CHF</td>
<td>10</td>
</tr>
<tr>
<td>Cerebrovascular dz</td>
<td>10</td>
</tr>
<tr>
<td>Renal disease</td>
<td>10</td>
</tr>
<tr>
<td><strong>Physical Findings</strong></td>
<td></td>
</tr>
<tr>
<td>Altered mental status</td>
<td>20</td>
</tr>
<tr>
<td>Respiration &gt;30 breaths/min</td>
<td>20</td>
</tr>
<tr>
<td>Systolic BP &gt;90 mmHg</td>
<td>20</td>
</tr>
<tr>
<td>Temp &lt;35 C or &gt;40 C</td>
<td>15</td>
</tr>
<tr>
<td>Pulse &gt;125 beats/min</td>
<td>10</td>
</tr>
<tr>
<td><strong>Lab or Radiographic Findings</strong></td>
<td></td>
</tr>
<tr>
<td>Arterial pH &lt;7.35</td>
<td>30</td>
</tr>
<tr>
<td>BUN &gt;30 mg/dL</td>
<td>20</td>
</tr>
<tr>
<td>Na &lt;130 mEq/L</td>
<td>20</td>
</tr>
<tr>
<td>Glucose &gt;250 mg/dL</td>
<td>10</td>
</tr>
<tr>
<td>Hct &lt;30</td>
<td>10</td>
</tr>
<tr>
<td>PaO2 &lt;60 mmHg</td>
<td>10</td>
</tr>
<tr>
<td>Pleural effusion</td>
<td>10</td>
</tr>
</tbody>
</table>

A total point score is obtained by adding the patient’s age in years (age - 10, for females) and the points for each applicable patient characteristic.

### RISK MORTALITY RATES

<table>
<thead>
<tr>
<th>Risk Class</th>
<th>No. of points</th>
<th>Recommended site of care</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No predictors</td>
<td>Outpatient – mortality 0.1%</td>
</tr>
<tr>
<td>II</td>
<td>&lt; 70</td>
<td>Outpatient – mortality 0.6%</td>
</tr>
<tr>
<td>III</td>
<td>71-90</td>
<td>Outpatient vs close observation – mortality 0.9%</td>
</tr>
<tr>
<td>IV</td>
<td>91-130</td>
<td>Inpatient – mortality 9.3%</td>
</tr>
<tr>
<td>V</td>
<td>&gt; 130</td>
<td>Inpatient – mortality 27%</td>
</tr>
</tbody>
</table>

Adapted from Halm et al, NEJM 2002.
### Antibiotic Therapy

#### PNA Setting

<table>
<thead>
<tr>
<th>Treatment Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Outpatient</strong></td>
</tr>
<tr>
<td>Uncomplicated: macrolide, quinolone*, or doxycycline</td>
</tr>
<tr>
<td>If suspect resistant S.pneumo: quinolone*</td>
</tr>
<tr>
<td>If suspect aspiration: amoxicillin/clavulanate, or clindamycin, or PCN + metronidazole</td>
</tr>
<tr>
<td><strong>Inpatient</strong></td>
</tr>
<tr>
<td>Uncomplicated: 3rd generation cephalosporin + macrolide. OR quinolone*. OR β-lactam-β-lactamase inhibitor + macrolide.</td>
</tr>
<tr>
<td><strong>ICU</strong></td>
</tr>
<tr>
<td>3rd generation cephalosporin + macrolide, OR quinolone, OR imipenem OR Zosyn</td>
</tr>
<tr>
<td><strong>Aspiration</strong></td>
</tr>
<tr>
<td>Clindamycin, Timentin, Zosyn, or cefoxitin</td>
</tr>
<tr>
<td><strong>Hospital-acquired +/- mechanical ventilation</strong></td>
</tr>
<tr>
<td>Imipenem or meropenem + quinolone, OR Cefepime OR zosyn + tobramycin +/- quinolone*</td>
</tr>
<tr>
<td><strong>Immunocompromised</strong></td>
</tr>
<tr>
<td>Above plus TMP/Sulfa ± steroids for PCP (if pO2&lt;70 or A-a difference &gt;35mmHg)</td>
</tr>
</tbody>
</table>


*Use anti-pneumococcal quinolone (gati, gemi, levo, moxi, telithro)

### Urinary Tract Infection

#### Definitions

- **Cystitis**: Superficial infection of the bladder
- **Pyelonephritis**: Inflammation of the renal parenchyma
- **Lower UTI**: Urethritis, cystitis, prostatitis
- **Upper UTI**: Pyelonephritis, renal abscess
- **Uncomplicated**: Cystitis or pyelonephritis in nonpregnant woman; no underlying structural disease
- **Complicated**: Upper tract infection occurring in the setting of catheters, stones, obstruction; any UTI in a man or pregnant woman; UTI with underlying disease (resistant organisms are more common)

#### Etiology

- **Uncomplicated**: Commonly associated with E. coli, Staph saprophyticus
- **Complicated**: Can be catheter-associated. Commonly caused by E. coli, Pseudomonas, S. epi, GNR’s, Enterococcus
- **Urethritis**: Chlamydia trachomatis, Neisseria gonorrhea

#### History/Sxs

- **Dysuria**: urgency, increased frequency, change in urine color or smell, suprapubic pain, discharge with urethritis, fever, shaking chills, flank/back pain, vomiting, diarrhea with pyelo, symptoms of outlet obstruction w/ prostatitis

#### Work-up/dx

- **UA and dipstick**: +LE, nitrite, WBC, RBC
- **Urine culture (not for acute uncomplicated cystitis)**
- **DNA or culture for Neisseria or Chlamydia**
- **Abdominal CT to rule out abscess if necessary**
- **Consider IVP/VCUG to rule out structural problem in recurrent UTIs**
<table>
<thead>
<tr>
<th>Type of Infection</th>
<th>Treatment Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cystitis</td>
<td>TMP-SMX or quinolone for 3 days if uncomplicated. Rx 10-14 days if complicated.</td>
</tr>
<tr>
<td>Pyelonephritis</td>
<td>Quinolone, aminoglycoside, or cephalosporin for 14 d (E. coli resistance to amoxicillin is increasing) for outpatient. For inpatient, use IV x 24-48hr, then switch to Pos.</td>
</tr>
<tr>
<td>Urethritis</td>
<td>Ceftriaxone 125 mg IM x 1 (for Neisseria) and doxycycline 100 mg PO x 7 days or azithromycin 1 g PO x 1 (for Chlamydia)</td>
</tr>
<tr>
<td>Prostatitis</td>
<td>TMP-SMX or quinolone for 14-28 d if acute, up to 12 wks if chronic</td>
</tr>
<tr>
<td>Renal Abscess</td>
<td>Drain + Rx as for pyelonephritis above</td>
</tr>
</tbody>
</table>

(Adapted from "Pocket Medicine", 2000, edited by Sabatine)

**Nephrology**

**Acute Renal Failure**

**Definition**
Rapid deterioration of renal function: azotemia, elevated BUN and Cr
Decreased urine output
Increase in creatinine of > 0.5 mg/dL or increase in creatinine >50% or decrease GFR > 50%
Nonoliguria = UOP > 400 ml/24 h
Oliguria = UOP = 100-400 ml/24 h
Anuria = UOP < 100 ml/24 h

**Hx/sxs**
GI: anorexia, nausea, vomiting
Cardiopulmonary: chest pain (pericarditis), dyspnea (pulmonary edema)
Fatigue, confusion
Ask about recent procedures or hypotensive episodes, review med list, pertinent PMH

**PE**
Vital signs: may have hypertension from volume overload
Extra heart sounds
Ascites, abdominal masses
Skin changes, pallor, rash, breath odor

**Common Etiologies of ARF:**

1. **Prerenal**

**Causes**
Intravascular volume loss: vomiting, diarrhea, ↓ intake, diuresis, 3rd-spacing
Hypotensive episode: surgery, sepsis, drugs, blood loss
Decreased cardiac output: MI, CHF, renal artery stenosis, emboli, thrombosis, NSAIDs, ACEIs

**Work-up/dx**
History, PE (vitals, orthostatics, neck veins, CV, Resp) R/O bleeding
UA, BUN, creatinine, FeNa
BUN/Cr ratio >20 suggests prerenal ARF.
Renal ultrasound if RAS suspected

**UA**
Urinalysis is bland: few or no cells, +/- hyaline casts
Urine Na <20 mEq/L
(FeNa <1 suggests prerenal disease; FeNa >2 suggests intrinsic renal. FeNa >4 suggests post-renal etiology.)
2. Intrarenal

**Acute tubular necrosis (ATN)**

**Causes**  Drugs (aminoglycosides, amphotericin, cisplatin), toxins, ischemia, contrast dye, pigments, crystals, proteins  

**Work-up/dx**  History, PE, UA, BUN, Creatinine, FeNa  

**UA**  Renal tubular cells  
    - Muddy brown/pigmented casts  
    - Urine Na >20 mg/dL  

**Rx**  Stop offending medications or other cause  
    - Support BP  
    - Dialysis PRN

**Acute interstitial nephritis (AIN)**

**Causes**  Drugs (beta-lactam antibiotics, sulfas, NSAIDs), infections, toxins, autoimmune, infiltrative  

**Work-up/dx**  Same as for ATN, consider 24-hr urine, urine eosinophils, UPEP, renal bx  

**UA**  WBCs and WBC casts, eosinophils  
    - Urine Na usually low  

**Rx**  Remove cause  
    - Prednisone  
    - Dialysis PRN

**Glomerulonephritis**

**Causes**  Berger's disease (IgA nephropathy), post-strep GN, SLE, RPGN, hep C  

**Work-up/dx**  Same as above, consider renal bx  

**UA**  RBCs and RBC casts  
    - Urine Na usually low  

**Rx**  Post-strep: supportive  
    - If crescenteric (RPGN) then immunosuppression  
    - Dialysis PRN

**Vascular**

**Causes**  Wegener's, HUS/TTP, athero-embolic, scleroderma, hypertensive crisis, renal artery stenosis  

**Work-up/dx**  Same as above, consider renal bx  

**UA**  Few cells  
    - Urine Na usually low  

**Rx**  Immunosuppression  
    - Dialysis PRN

3. Post-renal (obstructive)

**Causes**  Stones, retroperitoneal fibrosis, intratubular (uric acid crystals, light chains, methotrexate, acyclovir crystals), prostate enlargement/cancer, bladder/ureteral disease, lymphadenopathy, anticholinergic meds

Rx  Stop offending medications  
Optimize volume status  
Correct underlying problem. Usually reversible
Work-up/dx  As above with attention to neuro exam, palpate and percuss bladder
    Post-void residual, catheterization
    Ultrasound, CT
UA  ± cells, crystals
Rx  Remove cause
    Dialysis PRN
Recovery is directly related to the amount of time spent obstructed

Chronic renal failure
Etiologies include any cause of intrinsic disease with chronic hypertension and diabetes
being the most common.

Consequences of CRF:
1. Uremia
   - secondary to Na, K, and water retention, metabolic acidosis, excess toxins
   - signs and symptoms: edema, HTN, (activated renin-angiotensin axis), nausea, fatigue,
anorexia, pericarditis, platelet dysfunction, encephalopathy, fluid volume disorder
     (early, can’t concentrate urine; late, can’t dilute)
   - treatment: diuretics, restrict Na and K, anti-hypertensive drugs, dialysis if severe acid-base or electrolyte disorders

2. Bone disease:
   - secondary to high phosphate levels, low 1,25-vitamin D, low Ca, High PTH
   - signs and sx: bone pain (fractures), pruritus, calcifications, carpal tunnel
   - treatment: lower phosphate levels, raise calcium, lower PTH

3. Anemia:
   - secondary to decreased production of EPO, fewer RBCs made, loss with dialysis
   - signs and symptoms: pallor, fatigue, anorexia
   - treatment: EPO, replace iron loss

Indications for Dialysis:
A  acid-base disturbance (acidemia)
E  electrolyte disorder (hyperkalemia)
I  intoxication (methanol, ethylene glycol)
O  overload of volume
U  uremic symptoms
Pulmonology
Pulmonary Function Testing
Interpretation of spirometry/lung volumes
1. Ensure acceptable and reproducible data
2. FEV1/FVC ratio
   a. Lower limit of normal (LLN) = predicted minus 8% men, 9% women
3. FEV1/FVC ≥ LLN - no airflow obstruction
4. FEV1/FVC < LLN:
   a. FEV1 65-100% predicted - mild airflow obstruction
   b. FEV1 50-64% predicted - moderate airflow obstruction
   c. FEV1 <50% predicted - severe airflow obstruction
5. FEV1/FVC ≥ LLN:
   a. TLC or VC 65-79% predicted - mild restriction
   b. TLC or VC 50-64% predicted - moderate restriction
   c. TLC or VC < 50% predicted - severe restriction
6. Bronchodilator response
   a. Increase ≥ 12% AND ≥ 200ml in FEV1 or FVC following administration of bronchodilator
7. If obstructed: RV > 120% - air trapping, TLC > 120% - hyperinflation
8. Other tests
   a. DLCO - estimates alveolar capillary surface area
      i. Decreased - interstitial lung disease, emphysema
      ii. Increased - erythrocytosis, increased pulmonary blood flow
   b. Maximal inspiratory/expiratory pressures - useful in neuromuscular disorders
   c. ABG (See general medicine section for ABG analysis)

Ventilator Mode Settings
1. AC or AMV: Preset tidal volume (8-12cc/kg) and rate. Patient triggers each breath but machine mandates minimum number of breaths.
2. SIMV: Preset tidal volume and rate. Preset machine breaths provide set tidal volume, and spontaneous breaths are allowed between machine breaths for patient to take spontaneous tidal volumes.
3. PC (pressure control): Set peak pressure, rate and inspiratory time. Tidal volume varies with airway resistance and compliance.
4. CPAP - Uses positive end expiratory pressure (PEEP) during spontaneous breathing through a mask.
5. BiPAP - Bilevel Positive Airway Pressure. Inspiratory and expiratory positive pressures delivered by mask to treat sleep apnea or to try to avoid intubation/mechanical ventilation.
6. Pressure support - Preset amount of pressure during inspiration, and patient controls inspiratory time, tidal volume and rate. Overcomes resistance in the breathing circuit and ETT. Basically, the ventilator does the initial part of the breath, and PS can be used in conjunction with SIMV to overcome resistance of airway/ETT. Caution: Pt. must have intact respiratory drive as tidal volume and minute ventilation are not set!
Pleural Effusions
- Effusions can be either exudative or transudative
- At least 150 mL of fluid is needed to be seen on upright PA films as blunting of costophrenic angle, and 25-50 mL on lateral films
- Obtain decubitus films on all patients prior to thoracentesis to decipher loculations and evaluate underlying parynchema. >1cm of fluid on decub film = tappable.
- Perform thoracentesis on all NEW effusions. Can observe effusions in pts with known CHF that have symmetrical effusions and no evidence of infection or other cause for effusion.
- Send fluid for cell count + differential, LDH, glucose, total protein, cholesterol, amylase, pH, Gram stain and culture. May also consider cytology, ANA, RF, AFB stains/cultures, or HCT depending on likely etiologies.

<table>
<thead>
<tr>
<th>Cause</th>
<th>Transudate</th>
<th>Exudate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive Heart Failure</td>
<td>YES</td>
<td>NO</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>NO</td>
<td>YES</td>
</tr>
<tr>
<td>Cancer</td>
<td>NO</td>
<td>YES</td>
</tr>
<tr>
<td>Pulmonary Embolism</td>
<td>Sometimes</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Viral Disease</td>
<td>NO</td>
<td>YES</td>
</tr>
<tr>
<td>CABG</td>
<td>NO</td>
<td>YES</td>
</tr>
<tr>
<td>Cirrhosis with Ascites</td>
<td>YES</td>
<td>NO</td>
</tr>
</tbody>
</table>

Listed in order of incidence, adapted from Light, NEJM 2002.

LIGHT’S Criteria - must satisfy at least one of the below criteria to be an exudate
- Ratio of pleural protein to serum protein > 0.5 = Exudate
- Ratio of pleural LDH to serum LDH > 0.6 = Exudate
- Pleural LDH > 2/3 upper limit of normal for serum LDH = Exudate

Can also use
- Pleural fluid protein > 2.9 mg/dL
- Pleural fluid cholesterol > 45 mg/dL
- Pleural fluid LDH > 45% upper limit of normal serum value

If an exudate is found, can use other laboratory values to decipher etiology
- Glucose < 60 seen in malignancy, Rheumatoid, TB, Lupus, empyema/parapneumonic
- pH < 7.3 seen in malignancy, Rheumatoid, TB, systemic acidosis, empyema
- If associated with bacterial pneumonia, abscess, or bronchiectasis it is a parapneumonic effusion:
  1. Uncomplicated parapneumonic = free flowing with pH > 7.2, gram stain negative, culture negative
  2. Complicated parapneumonic = loculated OR pH < 7.2 OR gram stain positive OR culture positive

- Needs chest tube if complicated parapneumonic, pH < 7.2, bacteria present, or pus
Pulmonary Embolism
- Most common signs/symptoms of PE include: tachycardia, tachypnea, SOB, and pleuritic chest pain
- Laboratory findings include elevated D-dimer (sensitive but not specific), hypoxemia with increased A-a difference. Can see leukocytosis, elevated LDH, and elevated cardiac enzymes due to right heart strain. D Dimer only useful when PE is not your top suspicion in the differential.
- EKG is nonspecific and is most commonly sinus tachycardia. Nonspecific ST-T changes can be seen, and rarely the classic S in I, Q in III, and inverted T in III due to elevated pressures in the right heart.
- CXR: Nonspecific abnormalities. Hampton’s Hump or wedge shaped peripheral opacities.
Gold standard is pulmonary angiogram. Typically, use spiral CT with IV contrast or V/Q scan.
Don’t forget that most (up to 90%) of PE’s come from lower extremity DVTs, so venous duplex is often indicated if high suspicion for PE. Treatment includes initiation of a heparin drip with a loading bolus of 80 units/kg followed by 18 units/kg/hr. The aPTT should be checked in 6hr and infusion rate adjusted to maintain the aPTT at 1.5-2.5 x control. (LMWH is as effective as unfractionated heparin) Warfarin should be initiated if no contraindications are found, to achieve an INR of 2-3. Once INR achieved for 24hrs, heparin can be discontinued.
Patients with their first DVT/PE should be maintained on warfarin for 3-6 months.

Lung Cancer (leading cause of cancer mortality in US)
Non-small cell 80%
- Adenocarcinoma 30-40%
- bronchoalveolar CA subtype
- SCC 20-30%
- Large cell 10%
Small cell 20%
- Hypercoagulable states
- ↑PTH, hypercalcemia
- ↑hCG
- SIADH, ↑ACTH, Eaton-Lambert

Sx
- Usually symptomatic at presentation; cough/change in chronic cough, dyspnea, hemoptysis, weight loss, SVC syndrome, pancoast syndrome

Dx
- CXR, CT chest, ± thoracentesis, VATS, mediastinoscopy

Non-small cell – TNM Staging (simplified)
- T1 tumor < 3 cm
- T2 > 3 cm or invades pleura
- T3 any size with chest wall/diaphragm invasion, or within 2cm carina
- T4 any size + mediastinal/heart/tracheal/esophageal invasion or malig pleural effusion
- N0 no node
- N1 ipsilateral hilar nodes
- N2 ipsilateral mediastinal/tracheal nodes
- N3 contralateral nodes
- M0 no mets
- M1 + mets
### Non-small cell

<table>
<thead>
<tr>
<th>Stage</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage I</td>
<td>T1 or T2N0M0 resection</td>
</tr>
<tr>
<td>Stage II</td>
<td>T1 or T2N1M0 or T3N0M0 resection ± chemo or rads</td>
</tr>
<tr>
<td>Stage IIIA</td>
<td>T3N1M0 or T1-T3N2M0 resection vs chemo/rads</td>
</tr>
<tr>
<td>IIIIB</td>
<td>anyTN3M0 or T4anyNM0 palliation vs chemo/rads</td>
</tr>
<tr>
<td>Stage IV</td>
<td>anyTanyNM1 palliation vs chemo</td>
</tr>
</tbody>
</table>

### Small cell

- Limited: disease limited to ipsilateral hemithorax (one radiation port), chemo (largely palliative)
- Extensive: metastatic disease beyond one hemithorax, palliation

---

### Rheumatology

#### Osteoarthritis (OA)

- Most common joint disorder in US and the world; major cause of disability and pain in the elderly; before age 50 males > females; after 50 females < males
- Overweight people are at greater risk. Injuries may increase risk of development

**History/Sx**

- Pain and stiffness in and around joints (knees, hips, hands esp. DIPS/PIPS, cervical/lumbar spine), characteristic pattern is axial and peripheral joints in an asymmetric pattern, mild to moderate pain often gradual or insidious in onset, relieved by rest, may have AM stiffness but usually less than 30 minutes

**PE**

- Bony enlargement is common and may lead to tenderness at joint margins ± decreased ROM, ± crepitus, locking of joints with ROM Heberden’s and Bouchard’s nodes

**Labs**

- Usually not necessary, may want to R/O inflammatory arthritis

**XR**

- Bony proliferation (osteophyte formation or spurs) Joint space narrowing

**Rx**

- Education, weight loss if necessary, acetaminophen, NSAIDs ± glucosamine/chondroitin
- Consider PT/OT, corticosteroid injections, surgery

---

#### Rheumatoid Arthritis (RA)

- Systemic inflammatory disease predominantly in the synovial membrane of diarthrodial joints, may have extra-articular manifestations
- Affects all ethnic groups, 2.5-3.1 to 1 female to male ratio
- Peaks in 4th-6th decades

Need 4 out of 7 criteria for diagnosis:

1. AM stiffness for > 1 hour
2. Arthritis of 3+ joint areas
3. Arthritis of hand joints
4. Symmetric joint involvement/arthritis
5. Rheumatoid nodules (common over bony prominences, extensor surfaces)
6. + serum rheumatoid factor (RF)
7. Radiographic changes consistent with RA (erosions, periarticular decalcification)

**Physical exam/clinical features:**

- Symmetric synovitis of joints, esp. PIPs, MCPs, wrists, ankles, knees, MTPs
- Joint deformities: ulnar deviation, swan neck deformity, Boutonniere deformity
- C1-C2 instability (check C-spine films and inform team prior to intubation)
- Nodules (subcutaneous, lung, pericardium, sclera)
- Malaise, fever, weight loss
- Eyes: episcleritis/scleritis
- Lungs: fibrosis, nodules, pleurisy
- Cardiac: pericardial effusion, pericarditis, aortitis, AI
- GI: rarely involved
- Renal: rarely involved
- Neuro: C-spine instability can lead to cervical myelopathy, nerve compressions, mononeuritis multiplex
- Heme: hypochromic microcytic anemia, leukopenia, thrombocytopenia

Labs
- + RF in 85% of people with RA. Higher levels may correlate with extra-articular features although RF is of little prognostic value (don't need to follow)
- anti CCP (citrulline containing peptide) antibodies—more specific but less sensitive than RF, often used on combination with RF for even greater specificity to r/o RA.
- Elevated ESR and CRP (nonspecific)
- Hypergammaglobulinemia, hypocomplementemia, thrombocytosis, eosinophilia

Treatment:
- Accurate dx
- Smoking cessation, immunizations, appropriate tx of infections, management of diabetes, HTN, osteoporosis
- NSAIDs are effective for treatment of pain, swelling and stiffness but don't alter the progression of disease
- DMARDs (disease-modifying antirheumatic drugs) are used to control disease and limit damage
- New class of biologics (e.g., infliximab, entanercept) that target inflammatory mediators (TNF-alpha, IL-20) have revolutionized RA treatment and resulted in 75% improvement of symptoms in many patients.
- Methotrexate and TNF-alpha inhibitors are a common combination therapy.
- Surgery may be needed to modify joint damage.

Systemic Lupus Erythematosus
General:
- Autoimmune disease consisting of multisystemic inflammation with broad spectrum of clinical manifestations
- Cluster of signs and symptoms classified as one entity
- Associated with production of antibodies to components of the cell nucleus (antinuclear antibodies)
- Peak between ages 15-40 with female to male ratio 6-10:1
- HLA linked (DR3)
- 11 diagnostic criteria that reflect the major clinical features (4+ needed for dx)

Clinical manifestations / diagnostic criteria (4+ for dx):
Cutaneous
1. malar rash
2. discoid rash
3. photosensitivity  
4. oral/nasopharyngeal ulcers

Musculoskeletal  
5. non-erosive arthritis

Cardiopulmonary  
6. serositis (pleuritis, pleural effusion, pericarditis, pericardial effusion)

Renal  
7. proteinuria ( > 500 mg/dL or urinary cellular casts)

Neurologic  
8. seizures or psychosis

Hematologic  
9. hemolytic anemia or leukopenia or lymphopenia, thrombocytopenia

Serologies  
10. +ANA  
11. +anti-dsDNA

Other systems involved:  
GI (serositis, vasculitis, abdominal pain, hepatitis, pancreatitis)
Ocular (cotton wool spots, corneal/conjunctival involvement)

Labs  
Each cellular element in the blood can be affected by SLE (get CBC and look for “penias”). ESR is commonly elevated but not reliable marker of clinical activity

### AUTOANTIBODIES

<table>
<thead>
<tr>
<th>Autoantibody</th>
<th>Frequency</th>
<th>Clinical association</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANA</td>
<td>90%</td>
<td>Any or all clinical findings</td>
</tr>
<tr>
<td>Anti-dsDNA</td>
<td>40-60%</td>
<td>Lupus nephritis, vasculitis</td>
</tr>
<tr>
<td>Anti-RNP</td>
<td>30-40%</td>
<td>Raynaud’s, musculoskeletal</td>
</tr>
<tr>
<td>Anti-Sm</td>
<td>30% (very specific)</td>
<td></td>
</tr>
<tr>
<td>Anti-Ro/SSA</td>
<td>30-45%</td>
<td>Dry eyes/mouth, neonatal lupus</td>
</tr>
<tr>
<td>Anti-La/SSB</td>
<td>10-15%</td>
<td>Dry eyes/mouth, neonatal lupus</td>
</tr>
<tr>
<td>Antiphospholipid</td>
<td>30%</td>
<td>Clotting diathesis</td>
</tr>
</tbody>
</table>

### Treatment
- Education, control fatigue, treat comorbidities (hypothyroidism, depression, etc), avoid sunlight
- NSAIDs: musculoskeletal complaints, pleuritis, pericarditis, HA monitor for side effects/contraindications (renal, GI, etc. …)
- Corticosteroids: topical for cutaneous lesions, oral for constitutional symptoms, arthritis, serositis. Be aware of side effects/toxicities
- Hydroxychloroquine/Plaquenil: constitutional symptoms, cutaneous, arthritis, serositis, musculoskeletal (steroid sparing)
- Azathioprine (inhibits nucleic acid synthesis/ affects cellular and humoral immunity): alternative for lupus nephritis, and steroid sparing for other nonrenal manifestations
- Cyclophosphamide (immunosuppressive): mainstay for lupus nephritis/severe organ disease
- Methotrexate: cutaneous and joint disease
Gout
- Disease resulting from monosodium urate (MSU) crystal deposition in tissues
- Predominantly disease of adult men with peak in 5th decade
- Rare in premenopausal women
- Classic gout passes through 3 stages
  1. Asymptomatic hyperuricemia
  2. Acute intermittent gout
  3. Chronic tophaceous gout

Precipitants
- Obesity; hypertriglyceridemia; diabetes; excessive EtOH intake; high purine diet; medications: cyclosporine, HCTZ, low-dose aspirin (high-dose is protective), allopurinol if given during an acute flare

History/sxs
- Intense, sudden onset of pain in joints commonly in 1st MTP (podagra), ankles, knees, typically monoarticular, warmth, swelling, redness of the area, common onset at night, fevers, chills, malaise may be present

PE
- Erythematous, swollen, very tender joints, overlying skin may be warm, tense, dusky, tophi in synovium, Heberden's nodes, Achilles tendon, gross joint deformities

X-rays
- Unremarkable in early stages
- Soft-tissue swelling
- Bony erosions with "overhanging edge"
- Joint space preserved until late

Labs/dx
- Elevated serum urate level
- Definitive diagnosis only by aspiration of synovial fluid and demonstration of characteristic crystals (needle/rod shaped, birefringent that are yellow when parallel to the axis of the polarized light)
- Synovial fluid findings are consistent with inflammation (WBC count 5-80,000, mostly PMNs)

Rx
- Acute attack: NSAIDs, colchicine, corticosteroids, DON'T INITIATE ALLOPURINOL DURING AN ACUTE ATTACK
- Prophylaxis: lifestyle modification, prevention of precipitants, weight loss, urate lowering agents (allopurinol, uricosuric agents)
REFERENCES & HELPFUL RESOURCES
1. Blueprints in Obstetrics and Gynecology (Callahan, et al.)
2. Obstetrics and Gynecology (Beckmann, Ling, Laube et al.)
3. Obstetrics: Normal and problem pregnancies (Gabbe, et al.)

SAMPLE NOTES
OB Admit Note
ID/CC: ___ yo G__ P____@ ___weeks by LMP/US (date performed and whether consistent w/ LMP) presents w/ (onset of ctx, SROM, vaginal bleeding, decreased fetal movement, abdominal pain, etc)
HPI:
PNC: Labs: ABO type, Ab +/-, Rubella status, RPR, HbsAg +/-, HIV, GC/CT Hct, Pap, UA, GBS status, GCT (1 hr), GTT (3 hr), Quad screen
1st visit @__ weeks, total wt gain, 1st trimester BP
Imaging/fetal assessment: list U/S, NST, CST, BPP, amniocentesis, previous L/D admits (dates, gestational age, results)
Important issues: twins, PTL, infections, drug abuse, comorbidities, etc
Past OB hx: List each previous pregnancy w/ delivery date, delivery route, wt, sex, complications (including miscarriages/abortions)
Past Gyn hx: Menarche, cycles (reg or irreg), abnormal Paps, cervical operations/procedures, contraceptive hx
PMH:
PSH:
Medications:
Allergies:
Social hx: Include tobacco, ETOH, IVDU
Family hx: Metabolic/genetic disorders, diabetes, HTN, twins, etc
PE: Vitals, FHT (doppler rate or baseline/variability/accels/decels on monitor)
general appearance, lungs, CV, ext (signs of DVT, edema), DTR (hyperreflexia, clonus), abd (gravid, +/- tender, position by Leopold's, EFW), cx exam (dilation, effacement, station, position, consistency)
Assessment: 1. IUP @__ weeks in active labor/early labor/r/o PTL, etc
2. Pregnancy complicated by…
3. Intact membranes/SROM or AROM @__ w/ ___fluid…
Plan: Admit to L/D, Expectant management, Analgesia, Anticipate NSVD
Delivery Note
IUP @__wks, delivered by (CS, NSVD, vacuum, LTCS, VBAC, etc)
Labor: Spontaneous / induced / augmented (max rate of pitocin, etc)
ROM: Spontaneous vs artificial, +/- meconium, date/time, rupture length
Anesthesia: Epidural, pudendal, none (include amounts)
Infant: Wt, sex, position, APGAR scores, time of birth, +/- nuchal cord, bulb/DeLee
suction at perineum/delivery
Placenta: Spontaneous vs. manual extraction, time of delivery, intactness, # vessels (3),
+/- pitocin
Complications: Episiotomy (nth degree laceration, (where: cervical / vaginal vault / perineum,
type of suture)
EBL: Duration of labor: 1st stage, 2nd stage, 3rd stage, total
Postpartum condition: Mother and baby
In attendance: Attending, resident, med student

Postpartum Progress Note
S: Pain control, calf pain, breast tenderness, vaginal bleeding/lochia,
bowel/bladder fxn, ambulation
O: Vitals, lungs, CV, abd (fundal height/consistency, incision/episiotomy),
ext (edema, reflexes)
Labs: CBC, Rh status
A/P: PPD #1 uncomplicated NSVD, doing well (discuss complications if any)
Need for teaching (breast feeding), F/U, contraception, +/- RhoGAM,
circumcision for male infants

NORMAL OB
G/P Notation
Gravida = # of pregnancies the woman has had in her lifetime including
current one (not affected by multiple gestations)
Para = results of the above pregnancies divided into 4 categories: TPAL—Term
/ Preterm / Abortions (spontaneous or elective) / Living children

Pregnancy Dating Information
Nagele’s Rule: EDD = 1st day of LMP – 3 months + 1 week
Date of 1st positive pregnancy test: (usually 4 weeks gestation)
Date of pregnancy symptoms: 5-6 weeks
Doppler U/S of fetal heart tones: 7-12 weeks (closer to 12)
Fetoscope of heart tones: 19-20 weeks
Quickening: 20 weeks for primips, and 16-17 weeks for multips
Fundal height: 20 weeks at umbilicus, + 1 cm/week thereafter
Ultrasound accuracy best in 1st trimester (+/- 1 wk), 2nd (+/- 2 wks) and 3rd (+/- 3 wks)
ROUTINE PRENATAL TESTING

Initial Visit/ First Trimester
- Hct, blood type & screen, RPR, Rubella antibody, Hep B, GC/CT, PPD, Pap smear, UA w/ culture, HIV, VZV titer in pts w/ no h/o exposure

Second Trimester
- Quad screen (MSAFP, unconj estradiol, β-HCG, inhibin AHC, alpha subunit), screening ultrasound (18-20 wks), amnio if indicated

Third Trimester
- 50 g OGTT, Hct, GBS culture (36 wks), RPR, GC/CT if indicated; administer RhoGAM(D) IG for Rh negative at 28 wks; cervical checks at visits after 37 wks

Normal Lab Values In Pregnancy

<table>
<thead>
<tr>
<th>Lab</th>
<th>Normal Pregnancy Range</th>
<th>Change from Non-Pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>5-15 X 10^3/mm^3</td>
<td>↑</td>
</tr>
<tr>
<td>Hgb</td>
<td>11-14 g/dL</td>
<td>↓</td>
</tr>
<tr>
<td>Hct</td>
<td>33-42%</td>
<td>↓</td>
</tr>
<tr>
<td>Arterial pH</td>
<td>7.4-7.45</td>
<td>↑</td>
</tr>
<tr>
<td>PCO2</td>
<td>27-32 mmHg</td>
<td>↓</td>
</tr>
<tr>
<td>HCO3</td>
<td>19-25 mEq/L</td>
<td>↓</td>
</tr>
<tr>
<td>Creatinine</td>
<td>&lt;1.0 mg/dL</td>
<td>↓</td>
</tr>
<tr>
<td>BUN</td>
<td>4-12 mg/dL</td>
<td>↓</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>400-500 mg/dL</td>
<td>↑</td>
</tr>
<tr>
<td>Thyroid Functions</td>
<td>↑ TBG, T4, ↓ T₃ uptake, normal TSH, FT₄, and FTI</td>
<td></td>
</tr>
<tr>
<td>ECG</td>
<td>May have flat or inverted T-waves or Q-waves in inferior leads</td>
<td></td>
</tr>
</tbody>
</table>

Labor induction: Attempt to begin labor in a nonlaboring patient, usually done with prostoglandins, pitocin, mechanical dilation of the cervix, and/or amniotomy.

Cervical ripening:
Necessary before induction of labor when the cervix is unfavorable
- Nonpharmacologic methods: membrane stripping, amniotomy, mechanical dilators (e.g. balloon catheter), hygroscopic dilators (laminaria, Dilapan, laminicel)
- Pharmacologic methods: PGE2 (dinoprostone gel, Cervidil), PGE1 (Cytotec), pitocin

Bishop Scoring: Used to assess if the cervix is favorable for labor induction; total score >7-8 = favorable for induction

<table>
<thead>
<tr>
<th>BISHOP SCORE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parameter</td>
</tr>
<tr>
<td>Dilation (cm)</td>
</tr>
<tr>
<td>Effacement</td>
</tr>
<tr>
<td>Station</td>
</tr>
<tr>
<td>Consistency</td>
</tr>
<tr>
<td>Position</td>
</tr>
</tbody>
</table>

Labor augmentation: Intervening to increase already present contractions when contractions are inadequate or labor is prolonged; done with pitocin &/or amniotomy. Excess pitocin can cause uterine hyperstimulation, and rarely uterine rupture or water intoxication
Stages of Labor
Stage I
- Latent phase: closed cervix to 3-4 cm dilation
- Active phase: 3-4 cm dilation until fully dilated cervix (10 cm)
Stage II
- Fully dilated cervix to delivery of the infant
Stage III
- Delivery of the infant to delivery of the placenta

Abnormal labor patterns
- Prolonged latent phase = 20+ hours in nullips, 14+ hours in multips; doesn’t necessarily mean active phase will be abnormal or adversely affect perinatal outcome
- Protraction disorders: prolonged active phase = cervical dilation < 1.2 cm/hr in nullips, <1.5 cm/hr in multips; or descent of presenting part <1 cm/hr in nullips, <1.5 cm/hr in multips
- Arrest disorders: secondary arrest = cessation of previously normal active phase cervical dilatation for a period of 2+ hrs in nullip or multip; arrest of descent = no descent of the presenting part in > 1 hr in 2nd stage of labor
- 3rd stage of labor should be ≤ 30 minutes for both nullips and multips, after 30 minutes intervention is indicated to expedite delivery of placenta

APGAR SCORE (for newborn assessment; done at 1 and 5 min of life)

<table>
<thead>
<tr>
<th>Sign</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance (color)</td>
<td>Blue, pale</td>
<td>Body pink, extremities blue</td>
<td>Pink</td>
</tr>
<tr>
<td>Pulse</td>
<td>Absent</td>
<td>&lt;100 bpm</td>
<td>&gt;100 bpm</td>
</tr>
<tr>
<td>Grimace (reflex irritability)</td>
<td>No response</td>
<td>Some response</td>
<td>Facial grimace, sneeze, cough</td>
</tr>
<tr>
<td>Activity (muscle tone)</td>
<td>Flaccid</td>
<td>Some flexion</td>
<td>Good flexion of arms and legs</td>
</tr>
<tr>
<td>Respiratory effort</td>
<td>Apneic</td>
<td>Weak, irregular, gasping</td>
<td>Regular, good cry</td>
</tr>
</tbody>
</table>

OB COMPLICATIONS
1st Trimester Bleeding
Obstetric Causes: spontaneous abortion, ectopic pregnancy, extrusion of molar pregnancy
Nonobstetric Causes: 1) Cervical = severe cervicitis, polyps, benign/malignant neoplasms, 2) Vaginal = lacerations, varices, benign/malignant neoplasms, 3) Other = postcoital bleeding hemorrhoids, bleeding disorder, abd/pelvic trauma

3rd Trimester Bleeding
Obstetric Causes: 1) Placental = placenta previa, placental abruption, circumvallate placenta; 2) Maternal = uterine rupture, clotting disorders; 3) Fetal = fetal vessel rupture
Nonobstetric Causes: 1) Cervical = severe cervicitis, polyps, benign/malignant neoplasms, 2) Vaginal = lacerations, varices, benign/malignant neoplasms, 3) Other = hemorrhoids, bleeding disorder, abd/pelvic trauma

Placental Abruption (30% of 3rd trimester hemorrhages)
Definition: premature separation of normally implanted placenta from uterine wall resulting in hemorrhage between uterine wall and placenta
Presentation: painful bleeding (not always!), abd pain, uterine tenderness, ctx
Risk Factors: HTN, h/o abruption, trauma, AMA, cig/cocaine, rapid decompression of overextended uterus
Work-up: CBC, coag panel, fibrinogen, FDP, type and cross-match, Apt test, U/S (r/o placenta previa), speculum exam (after r/o previa), FHT, monitor contractions
Treatment: stabilize pt; prepare for future hemorrhage and preterm delivery; deliver if baby is mature or bleeding is life-threatening, can do vaginally if pt is stable & fetal testing reassuring
Complications: DIC, hypovolemic shock, preterm delivery

**Placenta Previa** (20% of 3rd trimester hemorrhage)
Definition: abnormal implantation of placenta over internal cervical os; can be complete, partial or marginal
Presentation: painless vaginal bleeding, usually in third trimester
Risk factors: prior placenta previa, uterine scars, multiple gestations, multiparity, prior C/S, cigs, AMA
Work-up: transabdominal/labial U/S – no vaginal exam!, CBC, type & cross, coag panel
Treatment: deliver by immediate C/S if unstoppable preterm labor, large hemorrhage, non-reassuring FHT, or at >36 w with mature lung testing; otherwise stabilize pt, prepare for hemorrhage and preterm delivery; 70% of pts will require delivery before 36 weeks
Complications: preterm delivery, PPROM, IUGR, antepartum hemorrhage

**Postpartum Hemorrhage** blood loss > 500 mL in vaginal delivery, > 1000 mL in C/S
Risk factors: 4 T’s
- **Tissue** Retained POCs, placenta accreta, cord avulsion
- **Trauma** Genital tract laceration, pelvic hematoma, uterine inversion, uterine rupture
- **Thrombin** Coagulopathy, DIC
- **Tone** Uterine atony (#1 cause, can be due to grand multiparity, multiple gestation, macrosomic fetus, prolonged labor w/ pitocin, chorioamnionitis, rapid labor, Mg tx)

Treatment:
- Fluid resuscitation, prepare for blood transfusion
- Laceration repair, removal of placental products/POCs
- For uterine atony: manual uterine massage, pitocin, Methergen or prostaglandin. If pharmacologic methods fail, to OR for D&C, if this fails then laparotomy.

**Placenta Accreta**
Definition: placenta forms an abnormally firm attachment to the uterine wall
- **Accreta** attached to the myometrium
- **Increta** invades the myometrium
- **Percreta** penetrates the myometrium (may invade bladder, etc)
Risk factors: prior C/S, other uterine surgery

**Premature rupture of membranes (PROM)**
Definition: spontaneous rupture of fetal membranes before the onset of labor regardless of gestational age
Presentation: gush of fluid from the vagina followed by persistent, uncontrolled leakage
Work-up: use sterile speculum exam (avoid digital exam), look for pooling of fluid in vaginal vault and perform Nitrazine test (amniotic fluid is alkaline), look for ferning under microscope, may also do U/S for amniotic fluid volume, and fetal fibronectin
Preterm PROM = PROM that occurs before 37 weeks gestation
Risk factors: STDs, smoking, prior PROM, short cervical length, prior preterm delivery, hydramnios, multiple gestations
Complications: chorioamnionitis, neonatal sepsis/pna/meningitis, placental abruption cord prolapse, pulmonary hypoplasia secondary to oligohydramnios
Treatment: expectant management with fetal and maternal monitoring; time to onset of labor inversely correlated with gestational age; immediately deliver for fetal distress or maternal infection; if PPROM use prophylactic antibiotics (reduce risk of neonatal GBS infection and prolong latency to labor onset) and antenatal corticosteroids as necessary

Pre-eclampsia
Definition: Preeclampsia = hypertension (BP>140/90 or 30/15 elevation over pts baseline), proteinuria (0.3 g/24 hr or >+1 on dipstick), and nondependent edema
Severe preeclampsia = SBP >160 and/or DBP >110 (on 2 occasions, 6hrs apart, at rest), proteinuria >5g/24 hr or 3-4+ on dipstick, nondependent edema; or any of the following in mild preeclamptic pt: oliguria, pulmonary edema, RUQ pain, headache/scomata, altered LFTs, thrombocytopenia, IUGR
Risk factors: Nulliparity, extremes of maternal age, multiple gestations, underlying chronic HTN, family h/o preeclampsia
Labs: CBC, LFTs, UA, creatinine, BUN, uric acid
Treatment: Delivery is the only cure; in mild pts deliver if at term or unstable, otherwise control blood pressure and manage expectantly; for severe pts deliver immediately

Eclampsia
• Grand mal seizure in preeclamptic patient not attributed to other cause
• Can occur before labor (25% of pts), during labor (50%) or after delivery (25%)
• Prophylaxis with MgSO4; treat seizure w/ MgSO4 and HTN management, deliver once pt stable

HELLP
A variant of severe preeclampsia: Hemolysis, Elevated Liver enzymes, Low Platelets
• Plt count <100K most consistent finding
• Microangiopathic hemolytic anemia
• Liver enzymes AST >72, LDH >600
• Treat with immediate delivery!

Pre-Term Labor
Definition: contractions resulting in cervical change before 37 weeks
Risk factors: infection, uterine malformations, antepartum hemorrhage, smoking, cocaine, h/o PTL, cervical incompetence, PROM, congenital anomalies, HTN, DM
Complications: preterm delivery can result in RDS, hypothermia, hypoglycemia, jaundice, bronchopulmonary dysplasia, patent ductus arteriosus, necrotizing enterocolitis, sepsis
Work-up: sterile speculum exam to r/o PROM, U/S, FHT, amnio for FLM if clinically indicated
Treatment: hydration, bed rest, fetal monitoring, treat infections, steroids, tocolytics

Tocolytic Drugs
Magnesium sulfate: may compete w/ calcium to reduce excitation or reduce calcium influx into the cell
• Maternal side effects: flushing, nausea, vomiting, headache, generalized muscle weakness, diplopia, SOB, pulmonary edema
• Neonatal side effects: (crosses placenta), serious complications uncommon; clinically insignificant decreased baseline heart rate and variability
• Monitor I/Os, DTRs (Mg toxicity causes hyporeflexia), vital signs q hr

*Indomethacin*: COX inhibitor (prostaglandins are mediators of the final pathway of uterine muscle contractions)
• Maternal side effects: GI sx, GI bleed, platelet dysfunction
• Neonatal side effects: oligohydramnios, ductal constriction, primary pulmonary hypertension w/ prolonged tx

*Terbutaline*: beta agonist (beta-2-receptor stimulation leads to uterine smooth muscle relaxation)
• Maternal side effects: tachycardia, palpitations, hypotension, chest discomfort, myocardial ischemia (rare!), SOB, tremor, pulmonary edema (rare!), hypok, hyperglycemia
• Neonatal side effects: tachycardia, hyperglycemia

*Nifedipine*: calcium channel blocker
• Maternal side effects: hypotension, tachycardia, headache, flushing, dizziness, nausea

*Contraindications to Tocolysis*
IUDR, lethal fetal anomaly, nonreassuring fetal assessment, severe IUGR, chorioamnionitis, maternal hemorrhage with hemodynamic instability and severe preeclampsia/eclampsia

**Ectopic Pregnancy**
Definition: pregnancy outside uterine cavity (99% are in the fallopian tube – 78% in ampulla, 12% in isthmus)
Risk factors: h/o PID/STD, prior tubal surgery, IUD use, prior ectopic
Presentation: amenorrhea, abnormal vaginal bleeding, unilateral abd/pelvic pain, tender adnexal mass
Ddx: salpingitis, threatened abortion, appendicitis, ovarian torsion
Work-up: serial quantitative β-HCG, Hct, U/S, culdocentesies if concern for rupture
Treatment: surgical or medical (methotrexate); if ruptured, immediate surgery!

**Gestational Diabetes**
Screening: 28 wk 50 g OGTT - value > 140 mg/dL at 1 hour is abnl. If abnl, then do fasting 3 hour 100 g OGTT - values controversial, but fasting > 95, 1 hr > 180, 2 hr > 155 or 3 hr > 140 considered abnl (+ if fasting or 2+ postprandial values ↑)
Comps: Fetal: macrosomia, traumatic delivery, shoulder dystocia, delayed organ maturity, congenital malformations, IUGR
Maternal: polyhydramnios, preeclampsia, infection, diabetic emergencies (hypoglycemia, ketoacidosis, diabetic coma), vascular or end organ damage, peripheral neuropathy, GI disturbance
Treatment: Diet/exercise and insulin or oral agents as needed to keep fasting levels < 100. Requires more frequent visits, possible referral to high-risk clinic. Delivery between 38-40 wk
Postpartum: Monitor blood sugars postpartum, and screen for DM as at increased risk (40% in 15 years)
White Classification for diabetes during pregnancy

- \(A_1\) - gestational diabetes, diet controlled
- \(A_2\) - gestational diabetes, insulin controlled
- \(B\) - onset age >20 or less than 10 years duration
- \(C\) - onset age 10-19 or 10-19 yrs duration
- \(D\) - onset at <10 yrs, or >20 yrs duration
- \(F\) - with nephropathy
- \(R\) - with proliferative retinopathy
- \(H\) - with ischemic heart disease
- \(T\) - with renal transplant

**FETAL HEART RATE MONITORING: DECELERATIONS**

<table>
<thead>
<tr>
<th>Type</th>
<th>Significance</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
<td>Head compression</td>
<td>Begins with and mirrors ctx, uniform shape</td>
</tr>
<tr>
<td>Variable</td>
<td>Cord compression</td>
<td>Variable in relation to ctx, abrupt onset</td>
</tr>
<tr>
<td>Late</td>
<td>Placental insufficiency</td>
<td>Symmetric, begins after ctx onset and returns after ctx completion</td>
</tr>
</tbody>
</table>

**ASSESSMENT OF FETAL WELL-BEING**

<table>
<thead>
<tr>
<th>Test</th>
<th>Reassuring</th>
<th>Non-reassuring</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal kick counts</td>
<td>(\geq 4) kicks per hour</td>
<td>(\leq 3) kicks per hour</td>
</tr>
<tr>
<td>Nonstress Test (NST) (50% of strips are NR at &lt; 28 wks, 28W, by 32w, 15% NR at 32 wks)</td>
<td>Reactive: 2 accelerations (&gt;15 bpm above baseline for (&gt;15) sec in (20) min)</td>
<td>Non-reactive: not having 2 accels/20min, even after acoustic or glucose stim</td>
</tr>
<tr>
<td>Contraction Stress Test / Oxytocin Challenge Test</td>
<td>No late or significant variable decels with contractions. Must have at least 3 contractions in 10 minutes.</td>
<td>Persistent late or significant variable decels after (&gt;50)% of contractions without hyperstimulation.</td>
</tr>
<tr>
<td>Amniotic Fluid Index: U/S measurement of amniotic fluid in 4 quadrants</td>
<td>(&gt;8), although does vary with gestational age</td>
<td>(&lt;5): oligohydramnios (&gt;20): polyhydramnios</td>
</tr>
<tr>
<td>Biophysical Profile (BPP): U/S used to assign score (0-2) in 5 parameters: fetal tone, breathing, movement, AFI, and NST</td>
<td>Score 8-10</td>
<td>(&lt;8), consider delivery, repeat BPP in 24 hours, or perinatology consult</td>
</tr>
<tr>
<td>Umbilical artery Doppler U/S velocimetry</td>
<td>Depends on gestational age</td>
<td>Elevations – IUGR, fetal hypoxia, acidosis Absent or reversed end-diastolic flow – IUGR</td>
</tr>
</tbody>
</table>
**BIOPHYSICAL PROFILE (BPP)**

<table>
<thead>
<tr>
<th>Category</th>
<th>Score = 2</th>
<th>Score = 0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-stress test</td>
<td>Reactive</td>
<td>Non-reactive</td>
</tr>
<tr>
<td>AFI</td>
<td>1 pocket &gt; 1 cm in two planes</td>
<td>Largest pocket &lt;1cm</td>
</tr>
<tr>
<td>Fetal tone</td>
<td>At least 1 extremity flex/ext/flex</td>
<td>Extended with slow or /no return to flex or no movement</td>
</tr>
<tr>
<td>Fetal movement</td>
<td>≥ 3 gross movements</td>
<td>&lt; 3 gross movements</td>
</tr>
<tr>
<td>Fetal breathing</td>
<td>30 secs of sustained effort (can have 5 sec pauses)</td>
<td>&lt; 30 seconds of fetal breathing</td>
</tr>
</tbody>
</table>

**BENIGN GYN**

**Polycystic Ovary Syndrome (PCOS)**

**Syndrome:** Most common cause of androgen excess and hirsutism, characterized by oligo/amenorrhea, related to obesity

**Diagnosis:** 2 of 3 required: 1) oligo/amenorrhea and/or anovulation, 2) clinical and/or biochemical signs of hyperandrogenism, 3) polycystic ovaries by US; must also exclude other etiologies

**Labs:** Prolactin, testosterone, DHEA-S, thyroid fxn

**Treatment:** Non-medical – weight loss, diet, exercise, hair removal techniques

Surgical – ovarian wedge resection, lap ovarian laser electrocautery

Pharmacological – if patient NOT wishing to be pregnant: OCPs, antiandrogen (i.e. spironolactone), metformin; if desiring pregnancy: clomid and metformin

**Comps:** Increased risk of PIH, GDM, endometrial & ovarian cancer, metabolic syndrome (DM, HTN, CVD, dyslipidemia), sleep apnea

**Pap smears**

Significantly reduced the incidence of cervical cancer. Recommended annually beginning 3 years after the onset of sexual activity, but no later than age 21. May be repeated less often at the discretion of the physician if the patient has three normal pap smears in a row. An adequate sample must contain some endocervical cells from the squamocolumnar junction since this is where 95% of cervical cancers occur. The Bethesda system is the current classification used.

**VAGINITIS**

<table>
<thead>
<tr>
<th>Organism</th>
<th>Discharge</th>
<th>Wet Prep</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trichomonas</td>
<td>Thin, yellow-green</td>
<td>Motile, flagellated organisms</td>
<td>Metronidazole</td>
</tr>
<tr>
<td>Bacterial Vaginosis</td>
<td>Thin, gray-white</td>
<td>Clue cells, + KOH “whiff test”</td>
<td>Metronidazole</td>
</tr>
<tr>
<td>Candida</td>
<td>Thick, clumpy, cottage cheese-like</td>
<td>Spores and pseudohyphae</td>
<td>Miconazole, diflucan, clotrimazole</td>
</tr>
</tbody>
</table>

**Pelvic Inflammatory Disease (PID)**

**Symptoms:** Abd/pelvic pain, increased vag discharge, abnormal odor, abnormal bleeding, GI or urinary disturbances

**Diagnosis:** Made clinically by cervical motion tenderness, elevated WBC, and pelvic pain

**Organisms:** C. trachomatis, N. gonnorhea, occasionally Bacteroides, E. coli, and Strep
Risk factors: Multiple sexual partners, teen, prior PID, current or past IUD, vaginal douching, cervical instrumentation
Complics: Adhesions, ectopic pregnancy, infertility, chronic pelvic pain.
Outpatient treatment:
- Ofloxacin 400 mg PO BID or Levofloxacin 500 mg PO QD with or without Metronidazole 500 mg PO BID for 14 days
- Ceftriaxone 250 mg IM x 1 plus Doxycycline 100 mg PO BID for 14 days
Inpatient treatment:
- Cefotetan 2 g IV q12° or Cefoxitin 2 g IV q6° plus Doxycycline 100 mg IV or PO q 12°
- Clindamycin 900 mg IV q8° plus Gentamicin 2 mg/kg loading dose, then 1.5 mg/kg q8° or 4.5 mg/kg QD, then Doxycycline 100 mg PO BID for 14 days

Sexually Transmitted Diseases
Chlamydia trachomatis
Symptoms: Often asymptomatic in women. May present with cervical discharge.
Diagnosis: Ligase chain reaction (LCR) of swab or urine (>95% sensitive)
- Antigen detection, direct florescent antibody of swab from cervix (sensitivity 80-95%)
Treatment: Azithromycin 1g PO as single dose or doxycycline 100mg PO BID for 7 days

Neisseria Gonorrhoea
Symptoms: 50% of infected women are asymptomatic, when symptoms occur they often include vaginal discharge or vaginal pruritus; infection can involve any portion of the genital tract or oropharynx
Diagnosis: Culture may only be 60-85% sensitive in asymptomatic women
- Gram stain in symptomatic women is only 60% sensitive (compared to almost 100% in men), so in women you must also send a culture Urine LCR, wide range of reported sensitivity (50-95%)
Treatment: Cefixime 400 mg PO X 1
- Ceftriaxone 125 mg IM X 1
- Ciprofloxacin 500 mg PO x 1
With any of the above also give Azithromycin 1g PO X1 or Doxycycline 100 mg PO BID x 7 days to cover for possible concomitant chlamydia

Herpes Simplex virus: types 1 and 2 can cause genital lesions
Symptoms: Highly variable and include painful or itchy genital ulcers (characteristic “dew drops on a rose petal” appearance), dysuria, fever, inguinal lymphadenopathy
Diagnosis: HSV PCR of swab, most sensitive if a blister is unroofed and swab is placed on fluid
- Viral culture
- Serology provides evidence of previous infection
Treatment: Acyclovir may shorten duration of outbreak if started within 24 hours of symptom onset, 200 mg PO five times per day for 10 days or 400 mg PO TID
- Suppressive therapy can be given to those with recurrent outbreaks (Acyclovir 400 mg PO BID).
Infertility
Definition: Inability to conceive after one year of unprotected intercourse; 15% of all couples will have infertility (40% of infertility is due to male factor i.e., abnormal morphology or motility, 40% due to female factor i.e., endometriosis, adhesions of fallopian tubes, 20% unexplained)
Work-up: Semen analysis, post-coital test, basal body temperature charts to identify ovulation, endometrial biopsy, hysterosalpingogram, U/S, laparotomy, midluteal progesterone, endocrine w/u. Appropriate tests determined by history.

Abnormal Uterine Bleeding (premenopausal)
Definition: Any irregularity in menstrual cycle from the norm
Dysfunctional uterine bleeding: Idiopathic abnormal bleeding.
Menorrhagia: Heavy or prolonged menstrual bleeding (>80 mL/cycle)
  Etiologies include fibroids, adenomyosis, endometrial hyperplasia, endometrial polyps, endometrial/cervical cancer, primary bleeding disorders.
Metorrhagia: Bleeding between periods
  Etiologies include endometrial polyps, endometrial/cervical cancer
Hypomenorrhea: Periods with unusually light flow
  Etiologies include hypogonadotropin hypogonadism in anorexics or athletes; can also be caused by Asherman’s syndrome, intrauterine adhesions, OCPs, Depo-Provera, outlet obstruction
Polymenorrhea: Frequent periods (<21 days between similar bleeding episodes)
  Usually caused by anovulation
Oligomenorrhea: Periods >35 days apart
  Most common cause is pregnancy, can also be due to any disruption of pituitary-gonadal axis or systemic disease

Postmenopausal Bleeding
Definition: Vaginal bleeding > 12 months after menopause; always abnormal and should always be investigated!
Work-up: 1) Perform pelvic exam and determine site of bleeding (bladder, rectum, vagina, cervix, vulva), note suspicious lesions and assess uterine size
  2) Biopsy any suspicious lesion and perform a Pap smear
  3) Transvaginal ultrasound: in postmenopausal bleeding this is 96% sensitive for detecting endometrial cancer when the stripe is > 4-5mm.
  4) Proceed to endometrial biopsy if:
      - endometrial lining is thicker than 5 mm
      - endometrium shows diffuse or focal increased echogenicity (heterogeneity)
      - bleeding persists
      - endometrium is not adequately visualized
      (Note: EMB samples 5-15% of the endometrial lining and up to 70% are not diagnostic)
  5) Saline infusion sonography
  6) Hysteroscopy
Ddx: Hormone replacement therapy (#1 cause), endometrial or cervical cancer, endometrial polyp, endometrial or vaginal atrophy, endometrial hyperplasia, fibroids, adenomyosis, infection, anticoagulant therapy or bleeding disorder, post radiation
Amenorrhea

**Primary** (Absence of menarche by age 16 or 4 years after thelarche)

Etiologies: (after excluding pregnancy)

- Outflow tract abnormalities: imperforate hymen, transverse vaginal septum, vaginal agenesis or atresia, testicular feminization, uterine agenesis
- End-organ disorders: ovarian agenesis, ovarian failure (e.g. Turner’s Syndrome)
- Central disorders: hypothalamic (local tumor compression, trauma, sarciodosis, Kallmann’s syndrome), pituitary (damage from surgery or radiation)

Work-up: History (including family history, diet, exercise, drug use)
- Physical exam (presence of breasts, uterus, vagina)
- Labs sent will depend on findings of physical exam

**Secondary** (Absence of menses for > 6 months or 3 normal cycle lengths in a woman who previously had menstrual cycles)

Etiologies: Pregnancy, anatomic abnormalities (Asherman’s syndrome, cervical stenosis), ovarian failure (from surgery, infection, radiation, chemo or idiopathic), polycystic ovarian syndrome, hyperprolactinemia, disruption of hypothalamic-pituitary axis (stress, anorexia)

Work-up: History and physical
- Pregnancy test
- Prolactin – if normal, proceed with progesterone challenge. If fail
- Progestin challenge, check FSH

Contraception

1. Hormonal methods
   - Oral contraceptives
   - Injectable contraceptives (depot medroxyprogesterone acetate, medroxyprogesterone acetate/estradiol cypionate)
   - Contraceptive implant
   - Contraceptive vaginal ring (NuvaRing)
   - Transdermal contraceptive patch (Ortho Evra)

2. Barrier methods
   - Male or female condom
   - Diaphragm
   - Cervical cap
   - Spermicides

3. Other
   - IUD (copper or levonorgestrel)
   - Sterilization (tubal ligation, vasectomy)
   - Withdrawal (18-20% failure rate)
   - Periodic abstinence

Calendar method: Find first day of the fertile period by subtracting 18 days from the length of their shortest menstrual cycle. The last day of the fertile period is determined by subtracting 11 days from the length of the longest cycle. Thus, a woman whose period varies from 28 to 30 days should abstain from intercourse on days 10 to 19 (with day 1 being the first day of their menstrual period). Not effective in women with irregular cycles

Ovulatory method: Measure daily basal body temperatures and abstain after cessation of menses until three days after a rise in temperature of 0.5 degrees has been detected. Cervical mucus: more watery around ovulation; abstain until
3-4 days after peak mucus production. Failure rates with perfect use 3-9% (up to 86% with incorrect use)

4. Emergency Contraception
Given within 72 hours of intercourse to prevent ovulation or if ovulation has occurred to prevent implantation. WILL NOT DISRUPT ALREADY IMPLANTED EMBRYO, i.e. THESE ARE NOT ABORTIFACIENTS
- Estrogen plus progesterone = Ethinyl estradiol 100 $\mu$g + levonorgestrel 0.5 mg x 2 Q12° (75-80% of pregnancy prevented)
- Progesterone only = Levonorgestrel 0.75 mg x 2 Q12° (equally or more effective than above regimen): known as Plan B and in Washington state some pharmacists can give this to women without a prescription
- Mifepristone 600 mg x 1 (100% effective); this is an abortifacient
- Copper IUD inserted within 120 hours after intercourse (>90% effective)

Gynecologic Oncology

Risk Factors for Cervical Cancer
1. Early onset of sexual activity
2. Multiple sexual partners
3. High risk partner: sexual exposure to partner with known HPV
4. History of STDs
5. Smoking
6. High parity
7. Immunosuppression
8. Low socioeconomic status
9. Previous history of vulvar or vaginal squamous dysplasia
10. HPV infection strongly associated

Risk Factors for Endometrial Cancer
1. Type I endometrial carcinoma is estrogen-related, tends to be associated with hyperplasia, and typically presents as a low-grade endometrioid tumor.

Risk Factors known for Type I (not type II) [Relative risk]
Increasing age
Unopposed estrogen [2-10]
Late menopause (after age 55) [2]
Tamoxifen therapy [2]
Nulliparity [2]
Polycystic Ovary Syndrome [3]
Obesity [2-4]
Diabetes mellitus [2]
Hereditary nonpolyposis colorectal cancer [22-50% lifetime risk]

2. Type II endometrial carcinoma appears unrelated to estrogen or hyperplasia, and tends to present with higher-grade tumors or poor prognostic cell types such as papillary serous or clear cell tumors.
Staging of Cervical Cancer (greatly simplified)

**TNM FIGO Definition**

TX  Primary tumor cannot be assessed
T0  No evidence of primary tumor
Tis  Carcinoma in situ
T1  Cervical carcinoma confined to uterus/cervix; subtypes classify extent of measured stromal invasion
T2  Cervical carcinoma invades beyond uterus but not to pelvic wall or to the lower third of vagina
T3  Tumor extends to the pelvic wall, and/or invades the lower third of vagina and/or causes hydronephrosis or nonfunctioning kidney
T4  Spread to adjacent organs (bladder, rectum or both)
M1  Distant metastasis

NX  Cannot assess regional lymph nodes
N0  No regional lymph node metastasis
N1  Regional lymph node metastasis

**AJCC stage grouping**

<table>
<thead>
<tr>
<th>Stage</th>
<th>TNM</th>
<th>FIGO stage</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 0</td>
<td>TisN0M0</td>
<td>Stage IIIB</td>
<td>T1-3aN1M0 or T3b any NM0</td>
</tr>
<tr>
<td>Stage I</td>
<td>T1N0M0</td>
<td>Stage IVA</td>
<td>T4 any N M0</td>
</tr>
<tr>
<td>Stage II</td>
<td>T2N0M0</td>
<td>Stage IVB</td>
<td>Any T any N M1</td>
</tr>
<tr>
<td>Stage IIIA</td>
<td>T3aN0M0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Staging of Endometrial Cancer (greatly simplified)

**TNM FIGO stage Definition**

TX  Primary tumor cannot be assessed
T0  No evidence of primary tumor
Tis  Carcinoma in situ
T1N0  Tumor confined endo/myometrium
T2N0  Tumor invades cervix, but does not extend beyond uterus
T3N0  Local and/or regional spread as to serorosa/adnexa (IIla); vagina
N1  with spread to pelvic/pre-aortic nodes
T4  Tumor invades bladder mucosa and/or bowel mucosa
M1  Distant metastasis Including metastasis to intra-abdominal lymph nodes other than para-aortic, and/oringuinal lymph nodes

Staging of Ovarian Cancer (greatly simplified)

**Stage I** Growth limited to the ovaries
**Stage II** Growth involving one or both ovaries with pelvic extension
**Stage III** Tumor involving one or both ovaries with peritoneal implants outside the pelvis and/or positive retroperitoneal or inguinal nodes. Superficial liver metastasis equals stage III. Tumor is limited to the true pelvis, but with histologically proven malignant extension to small bowel or omentum.
**Stage IV** Growth involving one or both ovaries with distant metastasis. If pleural effusion is present, there must be positive cytologic test results in order to classify as stage IV. Parenchymal liver metastasis equals stage IV.
Clerkship director: Curt Bennett, MD, fbennett@u.washington.edu
Web site: http://depts.washington.edu/peds/students/

HELPFUL RESOURCES
Seattle Children’s Hospital Survival Guide, latest ed. – the Bible, handed out at orientation.
Harriet Lane Handbook, latest ed. – particularly useful for pediatric dosing and NICU.
Pediatrics course textbook – good overview, basis for final exam, but not sufficiently detailed
for clinical practice.

WARD TIPS
Seattle: Ward team - attending, 2 senior residents, 4 interns, 1 sub-I, 1-2 MS3’s
3 weeks inpatient, 3 weeks outpatient clinic w/2 days in nursery & some shifts
in the ER. Call q4 until 11pm, off weekends when not on call.

WWAMI sites: ± Residents, spend more of your time with attendings.
Time is typically divided equally between wards and clinic.

- Pay attention to diet, prenatal hx, immunization hx, development, social hx
- Pediatrics is not just about the child; you must interact and build trust with the parents
  and extended family members as well.
- Introduce yourself to the patient and parents, take the time to say what you are doing
  and why, at a level appropriate for your patient.
- White coats are allegedly scary to children; consider leaving your coat in the hall.
- For infants, toddlers, and small children who appear frightened or nervous, try to keep
  the child on the parent’s lap during the PE. Do cardiac and pulmonary exams first,
  while the patient is still quiet. Look into ears and throat last.
- Smile and speak softly. Turn the exam into a game (“You listen to my heart, then I’ll
  listen to yours!”) or demonstrate on parents first.
- When possible, speak to the patient’s primary care physician.
- Tarascon Pharmacopoeia has pediatric drug dosing schedules.
- Read about your patients as soon as you have a chance.
- Practice your presentation. Ask your senior resident for feedback as soon as possible.
- Have fun! Even as an MS, you can make a difference in your patients’ care.

SAMPLE NOTES
Pediatrics MS3 Admission Note
ID/CC: The usual - include age, primary complaint, duration
HPI: Include pertinent positives and negatives ROS, exposure hx, and sick contacts.
Birth History: (for infants) _____week EGA (M/F) born by (vag/C-section) to a ___year old
G_P_A_. If C-section, indication? Prenatal infections? Was mom given antibiotics?
Birth weight: Apgars:
Complications: # of days in hospital:
Development: Milestones - gross and fine motor, social, and language. For older children:
school performance, attendance, favorite subjects.
Immunizations: UTD (up-to-date). Ask to see the immunization record.
Diet/Nutrition: Formula vs. breast; amount per day. Type of formula. Any special diet?
Frequency, solids, appetite.
Allergies: NKDA
Medications: Include vitamins and any herbal supplements.
Past Medical & Surgical History:
Social History: Patient lives with _______ in _______. Recent stressors (moves, parents divorcing), home environment (smoking, pets, environmental hazards), the primary caregiver, attendance in day care or school, performance in school, friends, sexual activity, and alcohol, tobacco, and other drug use.
Sexual history (older children): Menarche, problems with menstruation, sexual activity, contraception, STDs. Use discretion here (would you have enjoyed having this conversation in front of your parents when you were 15?)
Family History: Inherited diseases, miscarriages, sudden deaths, congenital anomalies, developmental delay, mental retardation, consanguinity, asthma, epilepsy, atopy, and cardiovascular disorders.
Review of Systems:
Physical Exam:
General: Note alertness, playfulness, consolability, hydration status, social interaction, responsiveness, nutritional status.
Vital Signs: Temperature (and method by which it was obtained: axillary, rectal, oral). Weight (kg and percentile) Height (cm and percentile) OFC - occipito-frontal circumference (head circumference (cm and percentile)). Plot these values on a graph with old values if available. BP, HR, RR, O₂ saturation (if available) Input (cc/kg/hr) and Output (stool + urine in cc/kg/hr), when available.
Skin: Note jaundice, cyanosis, mottling, birthmarks (location, color, size, number), rashes, capillary refill.
Hair: Lanugo and Tanner stages
Head: Circumference, shape, sutures, fontanelles
Eyes: Red reflex (newborn), strabismus, scleral icterus, EOMI, PERRLA, fundoscopic
Ears: In infants, gently pull the auricle posteriorly and inferiorly, insert speculum. In an older child, pull the external ear posteriorly and superiorly
Nose: Nares patent. Watch for nasal flaring (respiratory distress).
Throat: Teeth, palate (cleft?), thrush. Oropharynx (injection/exudate), adenoids
Neck: Range of motion. Supple vs. rigid. Thyroid exam.
Chest: Longer period of expiration than in adults. Comment on regularity of respirations, presence of skin retractions between ribs, grunting, stridor (inspiration), wheezing (expiration).
Cardiac: Rate, rhythm, murmurs. Check radial and femoral pulses. Note cyanosis.
Abdomen: Inspect, auscultate, then palpate. Note hepatosplenomegaly, masses. Check umbilicus in newborns
Back: Check for defects along spine, abnormal curvature (scoliosis)
MSK: Barlow/Ortolani maneuvers in newborns. Check ROM, effusions
Genitalia: Males: Circumcision, testes (undescended/descended), Tanner stage
Females: Tanner stage, labia (adhesion)
Neuro: Mental status (alertness, orientation, language); CN II-XII; Motor: Tone, strength, atrophy, fasciculations; Sensory: temperature, light touch, vibration; DTRs; primitive reflexes (root, grasp, Moro, etc); Cerebellar function; Gait
Assessment/Plan: Include summary statement that incorporates relevant history, physical, and studies. Break down assessment by system or by problem, and address all issues, no matter how small.
In pediatrics, always address FEN (fluids, electrolytes, and nutrition).

**Daily Progress Note**

MS3 PN, Pediatrics

**ID:** 2mo girl w/ fever without localizing signs.

**S:** Include overnight events, parents’ and nurses’ observations.

Check medications and dosages given overnight.

**O:** Report vital signs as a range of values over the past 18-24 hours.

24 I&Os, daily weight (your patient should be growing!)

**PE:** Gen, HEENT, resp, CV, abd, ext

Labs / Imaging

**A/P:** Summarize, and address issues by system (FEN, Pulm, CV, GI, GU, MSK, Neuro, Skin, Endo, ID/Heme).

**SELECTED TOPICS IN PEDIATRICS**

**VITAL SIGNS BY AGE GROUP**

<table>
<thead>
<tr>
<th>Age</th>
<th>Wt (kg)</th>
<th>HR</th>
<th>RR</th>
<th>SBP</th>
<th>DBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>27-30w</td>
<td>1</td>
<td>100-160</td>
<td>30-60</td>
<td>32-52</td>
<td>13-29</td>
</tr>
<tr>
<td>30-34w</td>
<td>2</td>
<td>100-160</td>
<td>30-60</td>
<td>40-60</td>
<td>20-36</td>
</tr>
<tr>
<td>Newborn</td>
<td>3</td>
<td>100-160</td>
<td>30-60</td>
<td>50-70</td>
<td>29-45</td>
</tr>
<tr>
<td>1 month</td>
<td>4</td>
<td>120-180</td>
<td>30-60</td>
<td>70-95</td>
<td>30-62</td>
</tr>
<tr>
<td>6 mo</td>
<td>7</td>
<td>110-170</td>
<td>25-40</td>
<td>80-100</td>
<td>50-70</td>
</tr>
<tr>
<td>1-2 y</td>
<td>12</td>
<td>90-150</td>
<td>20-30</td>
<td>80-100</td>
<td>51-90</td>
</tr>
<tr>
<td>3-4 y</td>
<td>16</td>
<td>70-140</td>
<td>20-30</td>
<td>80-110</td>
<td>39-89</td>
</tr>
<tr>
<td>5-6 y</td>
<td>20</td>
<td>65-130</td>
<td>20-30</td>
<td>85-115</td>
<td>45-85</td>
</tr>
<tr>
<td>7-8 y</td>
<td>26</td>
<td>60-130</td>
<td>18-25</td>
<td>85-115</td>
<td>50-70</td>
</tr>
<tr>
<td>9 y</td>
<td>30</td>
<td>60-130</td>
<td>15-20</td>
<td>90-120</td>
<td>50-70</td>
</tr>
<tr>
<td>10-12 y</td>
<td>36</td>
<td>60-130</td>
<td>15-20</td>
<td>90-120</td>
<td>50-70</td>
</tr>
<tr>
<td>Adolescent</td>
<td>50</td>
<td>60-120</td>
<td>15-20</td>
<td>90-120</td>
<td>50-70</td>
</tr>
</tbody>
</table>


**Immunizations** (See also www.cdc.gov)

**Birth:** HBV #1

2 months: HBV #2 DtaP #1 Hib #1 IPV #1 PCV #1

4 months: DtaP #2 Hib #2 IPV #2 PCV #2

6 months: DtaP #3 Hib #3 PCV #3

6-18 months: HBV #3 IPV #3

12-15 months: Hib #4 PCV #4 MMR #1

12-18 months: Varicella

15-18 months: DtaP #4

4-6 years: Varicella DtaP #5 IPV #4 MMR #2

11-18 years: MCV4 Td/Tdap HPV
<table>
<thead>
<tr>
<th>Age</th>
<th>Gross motor</th>
<th>Visual/fine motor</th>
<th>Language</th>
<th>Social</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mo</td>
<td>Raises head</td>
<td>Tight grasp, follows to midline</td>
<td>Alerts to sound</td>
<td>Looks at faces</td>
</tr>
<tr>
<td>2 mo</td>
<td>Lifts chest</td>
<td>Fists no longer clenched, follows past midline</td>
<td>Social smile</td>
<td>Recognizes parent</td>
</tr>
<tr>
<td>3 mo</td>
<td>Supports on forearms</td>
<td>Responds to visual threat</td>
<td>Coos</td>
<td>Reaches for familiar people</td>
</tr>
<tr>
<td>4 mo</td>
<td>Rolls over</td>
<td>Reaches with both arms in unison</td>
<td>Laughs, orients to voice</td>
<td>Enjoys looking around</td>
</tr>
<tr>
<td>6 mo</td>
<td>Sits unsupported</td>
<td>Unilateral reach, transfers objects</td>
<td>Babbles, razzes</td>
<td>Recognizes that a person is a stranger</td>
</tr>
<tr>
<td>9 mo</td>
<td>Crawls, pulls to stand, cruises</td>
<td>Immature pincer grasp, throws objects</td>
<td>“mama” and “dada” indiscriminately</td>
<td>Pat-a-cake, explores</td>
</tr>
<tr>
<td>12 mo</td>
<td>Walks</td>
<td>Mature pincer grasp</td>
<td>2 words besides “mama/dada”</td>
<td>Imitates, comes when called</td>
</tr>
<tr>
<td>15 mo</td>
<td>Creeps up stairs, walks backward</td>
<td>2-block tower, scribbles</td>
<td>4-6 words</td>
<td>15-18 mo: uses spoon and cup</td>
</tr>
<tr>
<td>18 mo</td>
<td>Runs, throws objects from stand</td>
<td>3-block tower</td>
<td>7-10 words, knows 5 body parts</td>
<td>Plays in group</td>
</tr>
<tr>
<td>24 mo</td>
<td>Up and down stairs</td>
<td>7-block tower, undresses</td>
<td>50 words, 2-word sentences</td>
<td>Parallel play</td>
</tr>
<tr>
<td>3 yrs</td>
<td>Alternates feet going up stairs, rides tricycle</td>
<td>Draws circle, partial dressing</td>
<td>250 words, 3-word sentences</td>
<td>Shares toys</td>
</tr>
<tr>
<td>4 yrs</td>
<td>Hops, skips</td>
<td>Draws square, dresses self, catches</td>
<td>Knows colors, asks questions</td>
<td>Plays cooperatively</td>
</tr>
<tr>
<td>5 yrs</td>
<td>Jumps over low obstacles</td>
<td>Draws triangle, ties shoes</td>
<td>Prints 1st name, asks what words mean</td>
<td>Competitive games</td>
</tr>
</tbody>
</table>

(Adapted from Harriet Lane Handbook, 16th ed, 2002)
Fluids and Electrolytes

Daily water needs are estimated based on energy expenditure:
1 kcal expended/day = 1 mL H2O required

“4-2-1” method for pediatric IV fluids:
- First 10 kg: 4 mL/kg/hr H2O*
- Second 10 kg: 2 mL/kg/hr H2O
- Weight over 20 kg: 1 mL/kg/hr H2O

So, for a 25 kg child, IV fluids based on this method would be:
- 40 mL/hr for the 1st 10 kg
- 20 mL/hr for the 2nd 10 kg
- 5 mL/hr for the 5 kg over 20 kg

TOTAL: 65 mL/hr for a 25 kg child, or 1560 ml/day

Daily electrolyte requirements:
- Sodium: 2-3 mEq/100 mL fluid/day
  - If weight <12 kg, D5 1/4NS+10 mEq KCl
- Potassium: 1-2 mEq/100 mL fluid/day
  - If weight >12 kg, D5 1/4NS+20 mEq KCl
- Chloride: 2-3 mEq/100 mL fluid/day

Nutrition

Formulas: often confusing with many brand names; talk to the nutritionists
- Breast Milk, Standard Infant Formula: 20 kcal/oz
- Regular infant formulas (eg Enfamil, from cow’s milk) and breast milk contain lactose
- Pediasure: 30 kcal/oz. Not for children under one year of age.
- Standard soy-based formulas: ProSobee, Isomil
- Hydrolysate formulas: Nutramigen, Pregestimil, Alimentum. The protein is enzymatically hydrolyzed and less allergenic.
- Renal formulas: (for fluid restriction) Nepro, Novasource (60 kcal/oz)
- Elemental formulas: Contain proteins broken down completely to amino acids; used in children with gastroschisis/short gut syndrome – Tolorex (low fat, for children with pancreatitis), Vivonex pediatric, Neocate.
- Premature formulas: Enfamil Premature, SMA Premie, Neocare 24 kcal/oz

Mixing formulas:
- 20 kcal/oz (regular): 1 scoop powdered formula with 2 oz water
- 24 kcal/oz: 3 scoops powdered formula with 5 oz water
- Thickened feeds: 1 tsp rice cereal per 4 oz formula

Volume conversion: 1 fl oz = 30 cc. So, 20 kcal/oz = 0.67 kcal/cc.

Normal weight gain:
- Week 1: May lose up to 10% of birth weight
- Week 2: Regain up to birth weight and establish weight gain pattern
- Small premies: 15 g/day
  - 0-3 months: 25-35 g/day
  - 3-6 months: 15-21 g/day
  - 6-12 months: 10-13 g/day
- 1-6 years: 5-8 g/day
  - 7-10 years: 5-11 g/day
Calorie Requirements by age

<table>
<thead>
<tr>
<th>Age</th>
<th>Kcal/kg/day</th>
<th>Grams protein/kg/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-6 months</td>
<td>108</td>
<td>2.2</td>
</tr>
<tr>
<td>6-12 months</td>
<td>98</td>
<td>2.0</td>
</tr>
<tr>
<td>1-3 years</td>
<td>102</td>
<td>1.2</td>
</tr>
<tr>
<td>4-6 years</td>
<td>90</td>
<td>1.1</td>
</tr>
<tr>
<td>7-10 years</td>
<td>70</td>
<td>1.0</td>
</tr>
</tbody>
</table>

BMI = weight (kg)/ height$^2$(m$^2$)


In healthy infants, children, and adolescents, a vitamin D intake of at least 400 IU/day is recommended.

1. Breastfed and partially breastfed infants should be supplemented with 400 IU/day of vitamin D beginning in the first few days of life. Supplementation should be continued unless the infant is weaned to at least 1 L/day or 1 qt/day of vitamin D–fortified formula or whole milk. Whole milk should not be used until after 12 months of age.

2. All nonbreastfed infants, as well as older children who are ingesting 1000 mL/day of vitamin D–fortified formula or milk, should receive a vitamin D supplement of 400 IU/day.

3. Adolescents who do not obtain 400 IU of vitamin D per day through vitamin D–fortified milk (100 IU per 8-oz serving) and vitamin D–fortified foods (such as fortified cereals and eggs [yolks]) should receive a vitamin D supplement of 400 IU/day.

4. On the basis of the available evidence, serum 25-OH-D concentrations in infants and children should be 50 nmol/L (20 ng/mL).

5. Children with increased risk of vitamin D deficiency, such as those with chronic fat malabsorption and those chronically taking antiseizure medications, may continue to be vitamin D deficient despite an intake of 400 IU/day. If a vitamin D supplement is prescribed, 25-OH-D levels should be repeated at 3-month intervals until normal levels have been achieved. PTH and bone-mineral status should be monitored every 6 months until normal.

**Causes of abdominal pain in children**

**Intussusception**
- telescoping of a portion of gut into an adjacent segment; peak 5-9 mo
- Lead points: HSP, meckel’s, neurofibroma, hemangioma, lymph node (post viral, NHL)
- Sx: Sudden onset of severe, paroxysmal pain (child may cry, draw knees to chest) interspersed with near-normal periods; BRBPR
- Dx/Tx: air/barium enema, surgery

**Malrotation w/volvulus**
- rule of 2’s: 2% of population, 2” long, 1st 2 years of life, 2’ from ileocecal valve, 2 types of epithelium (gastric, pancreatic)
- 1st year of life. Bilious vomiting, distension, fever
- Usually 2-5 yo, h/o preceding URI

**Meckel’s diverticulitis**
- triad of abdominal pain, LE palpable purpura and GI bleeding
- rotavirus most common in <2 yo. Antibiotics for Shigella, invasive E. coli (not EHEC -> HUS), amebiasis. Anticholinergics contraindicated

**HSP**
- triad of abdominal pain, LE palpable purpura and GI bleeding.
- Usually 2-5 yo, h/o preceding URI

**Gastroenteritis**
- rotavirus most common in <2 yo. Antibiotics for Shigella, invasive E. coli (not EHEC -> HUS), amebiasis. Anticholinergics contraindicated

**Other causes**
- appendicitis, DKA, testicular/ovarian torsion, UTI, IBD, Juvenile RA

**Congenital abdominal pathology**

**Duodenal atresia**
- bilious vomiting following feeding in 1st days of life; associated w/
prematurity, polyhydramnios, down syndrome
idiopathic hypertrophy of pylorus, onset 2-3\textsuperscript{rd} week of life.
Nonbilious vomiting, progresses to projectile. Surgical treatment.

**Hirschsprung’s**
Absence of parasympathetic myenteric ganglion cells in rectum/sigmoid. Abd distension, constipation, pass stools after digital exam

**Abdominal neoplasms**

**Neuroblastoma**
most common abd neoplasm in children, <5 yo. From neural crest cells. GI obstruction, diarrhea, opsoclonus-myoclonus, raccoon eyes

**Wilm’s**
derived from embryonal renal cells; 4 mo – 6 yrs, median 3 yrs, 10% bilateral. Presents w/flank mass, hematuria.
WAGR: Wilsms, aniridia, ambiguous genitalia, mental retardation

**Work-up of a fever in a neonate** (<3 mo)
Admit to hospital, complete PE (look in ears to r/o OM)
CBC with diff, BCx
UA with Cx
LP
CXR if any pulmonary Sx

**Febrile seizures**
2-4\% of all kids; 6 mo – 6 yrs old. 1/3 recur
Associated w/rapid rise in temp, T>38; commonly see w/HHV-6
Simple: <15 min, no focal features
Complex: >15 min, +focal features/post ictal paralysis, or series of sz >30 min
Tx if last > 5 min – lorazepam, APAP to lower fever, dilantin if persists
Does increase risk of developing epilepsy, especially w/complex

**Kawasaki’s disease**
acute febrile vasculitis, likely immune-mediated. <8 yo.
Sx:
high fever 5+ days, bilateral conjunctivitis, red lips/mouth, red palms/soles, maculopapular rash
15-25\% develop coronary artery aneurysms
Tx:
IVIG, high dose ASA until defervesces (80-100 mg/kg/d ÷ qid)

**Ddx limp**

**Infectious**
septic arthritis, osteomyelitis

**Legg-Calve-Perthes**
idiopathic avascular necrosis of femoral head, 3-12 yo. Pain may refer to inner thigh

**SCFE**
typically, overweight teen boys; proximal metaphysis displaces anteriorly. Pain may refer to medial knee

**Osgood-Schlatter**
teens, due to overuse; pain over tibial tuberosities

**Neoplasms**
Osteosarcoma, Ewing’s sarcoma – primarily distal femur, proximal tib/fib.
Leukemia - ALL

**Stress fracture**
Juvenile rheumatoid arthritis

**Pulmonary disease**

**Croup**
infection of upper airway – larynx, trachea, bronchi
Parainfluenza accounts for 75\%; also, influenza A/B, RSV, Mycoplasma
Barking cough ± hoarseness, stridor. “Steeple sign” on neck XR

**Epiglottitis**  
formerly due to H. flu; since vaccine, S. pyogenes, S. pneumo, S. aureus  
Cherry red epiglottis. “Thumbprint sign” on lateral neck XR  
Airway emergency – intubate/cricothyrotomy, antibiotics

**Bronchiolitis**  
infection of lower airways w/obstruction of bronchioles by mucus, edema  
Increased incidence in bottle-fed, daycare, pre-existing resp disease  
RSV accounts for >50%; also parainfluenza, adenovirus, Mycoplasma  
Starts w/mild URI Sx (sneezing, coryza), fever (38.5-39); then increasing resp distress, wheezing, tachypnea, irritability. Usually no GI Sx.  
Tx: O2, epinephrine/albuterol SVN

**Cystic fibrosis**  
Auto recessive mutation in CFTR (ΔF508 most common); impaired Cl excretion  
May present with meconium ileus in 15-20%; bronchiolitis, bronchiectasis;  
exocrine pancreatic insufficiency; nasal polyposis, sinusitis; cholecystitis; failure to thrive.  
Pulmonary disease accounts for most morbidity/mortality. Eventually become colonized w/Pseudomonas, Burkholderia spp., aspergillus. MRSA also an important pathogen  
Dx: sweat chloride, molecular testing  

**Neonatology**  
**Neonatal screening labs**  
State of Washington newborn screening tests for the following 9 disorders:  
1) PKU  
2) Congenital hypothyroidism (T4; the lowest 10% also get a TSH)  
3) MSUD  
4) CAH (17-OH progesterone)  
5) Homocystinuria  
6) Galactosemia  
7) Biotinidase deficiency  
8) Medium Chain Acyl co-A Dehydrogenase Deficiency  
9) Hemoglobinopathies

**Neonatal Cyanosis**  
Systemic: respiratory depression due to maternal opiates, magnesium sulfate, sepsis, hypoglycemia  
Heart: TOF, transposition of great arteries, total anomalous venous return, Ebstein’s anomaly, pulmonic stenosis  
Respiratory: choanal atresia, pulmonary hypoplasia, pneumonia, IRDS, meconium aspiration  
Misc: diaphragmatic hernia, intraventricular hemorrhage

**Neonatal Jaundice** (usually an unconjugated hyperbilirubinemia)  
1st 24 hours of life: erythroblastosis fetalis – Rh+ fetus, Rh- sensitized mother hemmorhage – cephalohematoma  
sepsis  
intrauterine infection – congenital toxoplasmosis, syphilis, CMV
>24 hours: physiologic jaundice – peaks 3-5th day, normalizes in 2 weeks, Tbili ≤ 12 (higher peak levels, takes longer to resolve in premies) early onset breast feeding jaundice Crigler-Najjar – auto recessive, absence of glucuronol transferase; severe jaundice, kernicterus -> seizures -> brain injury Gilbert’s syndrome – auto dominant, mild deficiency of gluc transferase; Mild, episodic jaundice precipitated by stress 

>1 week: breast milk jaundice – peaks 2-3rd week, Tbili 10-30 atresia of bile ducts cystic fibrosis Hereditary spherocytosis – auto dominant Hypothyroidism

Complications of prematurity

IRDS due to decreased levels of surfactant. ↑ risk w/diabetic mother, C section w/o labor, multiple gestation. Sx: Resp distress that worsens in 1st few hours, peaks in 48-72 hours, then gradually improves. Dx: reticulogranular pattern on CXR Tx: steroids pre-delivery to accelerate lung maturity; surfactant post delivery secondary to O2 toxicity, ventilator injury; follows IRDS. Mostly see in <1,000 g babies. Decreased alveolarization. Clinical worsening after 3rd/4th day of life, unlike IRDS.

BPD typically see after start feeding infant, onset in 1st 2 weeks of life. Can be rapidly progressive. Sx: abdominal distension, hematochezia Dx: KUB shows pneumatosis intestinalis, free air, pneumatobia Tx: d/c oral feeds, antibiotics; surgery if perforated or doesn’t respond

NEC abnormal retinal vascularization. Spontaneous regression in 90%. 10% progress – vessels proliferate, extend into vitreous humor; retinal detachment, blindness can occur

ROP
Clerkship director: Roger P. Tatum, M.D.
Web site: http://depts.washington.edu/surgstus/

REFERENCES & HELPFUL RESOURCES
1. Surgical Recall—all the basics from horses to zebras, most difficult pimping questions are found in this book
2. “A primer for third year medical students entering their surgical clerkship”, Farhood Farjah and Lorrie Langdale
4. Access to any surgical text for more in-depth reference

• A NOTE ON TERMINOLOGY: A surgical procedure is referred to as an “operation,” NOT a “surgery.” “A surgery” is a British term referring to a place where surgery is performed. Surgeons perform operations, the performance of operations is called surgery, and “a surgery” is a place. Got it? Don't call an appendectomy “a surgery,” it's an operation! It would be appropriate to say “this patient just had surgery” but NOT “this patient just had a surgery.”

WARD TIPS
• Wear comfortable shoes! Wear scrubs on non-clinic days, and nice clothes in clinic.
• Check surgery schedule for next day, read up on anatomy and basics of procedure. There is so much that they can pimp you on but 3rd years are supposed to know basic anatomy and pathophysiology. You will be unable to prepare for every case—pick one or two and know them cold then just go over the basics of the others. Surgical recall does a pretty good job of giving the bare bones facts. There will be certain operations that you will see all of the time (depending on location) like inguinal hernia repairs, lap choles, exploratory laparotomies (at HMC), so tailor your studying to your environment. If you’ve seen a case 4-5 times you’d better know the anatomy, pathophysiology, and oddball minutia cold.
• Pre-round and give yourself enough time to formulate a daily assessment and plan before rounds (review plan with intern if you get the chance), and have most of the note finished before rounds (may change plan after discussing with seniors on rounds). Follow-up on patient labs, pathology, x-ray, etc. Most importantly know everything about your patients.
• Be prepared to do dressing changes for team patients on a.m. rounds. When you enter a room where the patient has a wound that needs to be looked at (after you have cleared it with the intern) take the dressing down and have the new dressing ready to be put on. Dressing changes are your job! Have your pockets filled with dressing materials so you don’t have to fumble through the room looking for supplies while the team is trying to get through rounds.
• Dressing material to carry: Bandage shears, 2-3 ABDs, Lots of 4X4s, a drain dressing or two, and tape, +/- other material depending on the service.
• Eat when you can, sleep as much as you can, and try to study every night. Carry something to study in your coat in case of a rare moment of downtime.
• The floors all have small supply closets with crackers and other snacks—always a nice place to stock up just in case.
• Make sure your pager is loud enough to wake you up, if not consider clipping it to the chest of your scrubs when napping on call, or buying a louder pager.

• The intern will love you if you learn to help keep the Cores list (http://cores.medical.washington.edu at UW and HMC) up to date with current patient census and information. It will also help you remember the patients and stay up on what is going on with the service.

OR TIPS
• Introduce yourself to the scrub nurses, s/he will hand you the instruments you need, and keep you from doing anything really stupid. Be friendly and ask if you should write your name down for them.

• Pay attention to the operation, if you know what is going on and make an effort to help (retract etc,) you will get to do a lot more.

• Practice suturing and tying, if you do these well when given the opportunity the surgeons will notice and let you do more.

• Always try to use the restroom prior to starting a long case.

• Pull your gloves and gown prior to scrubbing, sometimes the nurses will do this for you but always check.

• Help when you can, but also stay out of the way.

• Have fun. The OR time is the best part of the surgical rotation

SAMPLE NOTES
Daily Progress Note (Post-Op check follows same format)
MS3 PN, Gen Surg B
ID: 37 yo male S/P ex lap, HD#3, POD#2, Ceftaz Day #1
S: Pain, ambulation, flatus, BM, appetite and diet, breathing and pertinent information unique to respective surgery.
O: Vitals, I&Os including breakdown of volumes. UOP should average at least 30cc/hr.
    JP/drains – note amount of fluid, color.
PE: Don’t forget to describe wounds
    Labs
    Imaging

A/P: Address all significant issues and what you want to do about them
Plan should always include:
1. Diet plan
2. Pain (controlled w/ PCA, change to po, etc.)
3. Volume status (IVF, vitals, urine)
4. Discharge plan (best guess when and what needs to happen to get there)
5. Any specific problems

Brief Operative Note
Pre-Op Dx
Post-Op Dx – same (same as above or explain difference based on operative finding)
Procedure
Surgeons, Assistants
Anesthesia – GET (general endotracheal), LMA, MAC, local, etc
EBL – ask anesthesia
Fluids – ask anesthesia
Drains – list what drains were put in
Findings – list significant findings
Specimens – list tissue sent to path
Complications – usually none... if there is a complication, ask the surgeon what you should write here or let resident write this section
Disposition – condition of patient i.e. patient taken to recovery room in stable condition

**BLOOD PRODUCTS**

Red Blood Cells: 1 unit of PRBCs raises HCT 3 points
   Indications: symptomatic anemia, large blood loss/continuing loss, low hct (< about 30) and h/o CAD

Platelets: 1 unit of platelets raises count 5,000-10,000. Given in “six packs” (six units).
   Indications: platelet count less than 20,000 (can result in spontaneous bleeding) or platelet count < 50 with bleeding or needs operation.

**SELECTED SURGERY TOPICS**

**Layers of the abdominal wall**
One of the most common pimpling questions
1. Skin and subcutaneous fat
2. Camper’s (fatty) fascia
3. Scarpa’s (membranous) fascia
4. External oblique
5. Internal oblique
6. Transversus abdominus
7. Transversalis fascia
8. Peritoneum

**Acute Abdomen** = inflamed peritoneum

Sxs/signs  Rebound tenderness, involuntary guarding, motion pain (shake the bed or tap patient’s feet)
Labs     CBC with diff, Chem 7, Ca, Mg, PO4, amylase/lipase, +/- Liver tests, type and screen, UA, urine hCG
Ddx    Think in terms of quadrants and what lives in respective quadrants
RUQ    Cholecystitis, hepatitis, PUD, perforated ulcer, pleurisy/pneumonia, pancreatitis, liver tumor, gastritis, hepatic abscess, pericarditis, cholelithiasis, hepatic abscess, nephrolithiasis, PE, MI, appendicitis (esp. in pregnancy)
LUQ    PUD, perforated ulcer, gastritis, splenic disease/rupture, abscess, dissecting aortic aneurysm, pyelonephritis, nephrolithiasis, strangulated hiatal hernia, Boerhaave’s syndrome, Mallory-Weiss tear, pneumonia, PE, MI, pleurisy
LLQ    Diverticulitis, sigmoid volvulus, perforated colon, colon cancer, UTI, SBO, IBD, PID, ectopic pregnancy, nephrolithiasis, pyelonephritis, referred hip pain, aortic aneurysm, ovarian cyst, endometriosis, gyn tumor, ovarian torsion, Fallopian torsion
RLQ    Appendicitis, mesenteric lymphadenitis, cecal diverticulitis, Meckel’s, intussusception, cecal volvulus + LLQ causes above

Other causes to consider
Gyn causes  Ovarian cyst/torsion, PID, fibroid degeneration, ectopic pregnancy, endometriosis, tumor, tubo-ovarian abscess
Thoracic  MI, pneumonia, aortic dissection, aortic aneurysm, empyema, esophageal
rupture/tear (Boerhaave’s), pneumothorax
Scrotal Testicular torsion, epididymitis, orchitis, inguinal hernia
Diffuse Uremia, porphyria, diffuse peritonitis, gastroenteritis, IBD, DKA, early appendicitis, SBO, sickle cell crisis, ischemic mesentery, lead poisoning, pancreatitis

Appendicitis
Cause: Obstruction of appendiceal lumen by lymphoid hyperplasia, fecalith, etc., (i.e., a closed loop obstruction). Continued secretion into its lumen results in increased pressure in appendix. When lumen pressure = perfusion pressure, wall becomes ischemic, and eventually ruptures spilling bacterial and fecal matter into abdominal cavity.
Complic. Perforation, peritonitis, bowel obstruction
Early sx Periumbilical pain (referred and poorly localized) that then migrates and presents as RLQ pain once peritoneal inflammation occurs, anorexia, nausea, vomiting (Pain almost always before N/V)
Signs Involuntary guarding, rebound tenderness, fever, obturator sign, psoas sign, Rovsig’s sign (pain at on opposite side when abdomen is palpated)
Labs CBC with diff, UA, pregnancy test if of childbearing age
Imaging Dx typically clinical but may get Abd XR, U/S, or CT if diagnosis is not clear on physical exam
Rx Appendectomy and 24hr Abx for non-perforated. Appendectomy and 5-7 days Abx for perforated appendix.

Cholelithiasis 10% of U.S. population, 50% are symptomatic
Risk Factors Female, obesity, multiparity, oral contraceptives, biliary stasis, chronic hemolysis, cirrhosis, TPN, IBD, hyperlipidemia (fat, forty, fertile, female)
Stones Cholesterol stones 90%, pigment stones 10%
Rx If symptomatic = Lap Cholecystectomy

Biliary Colic Obstruction, RUQ pain, nausea, vomiting usually after large meal, but no inflammation and resolves within few hours

Cholecystitis = inflammation of gallbladder due to obstruction of cystic duct
Causes Gallstones, biliary stasis (TPN, fasting)
Complic. Abscess, perforation, choledocholithiasis (stones within the common bile duct), gallstone ileus (gall stone may erode through gall bladder, enter the small bowel and lodge itself at the ileocecal valve causing a SBO)
Sx/signs RUQ pain and tenderness (longer than 1-2 hours), fever, nausea, vomiting, Murphy’s sign, right subscapular pain (referred)
Imaging U/S shows thickened gallbladder wall, gallstones, pericholecystic fluid, (all of above are non-specific signs) one of most important and specific signs is the sonographic Murphy’s sign (when positive is almost always cholecystitis)
Rx IVF, Abx, cholecystectomy (often within 72 hours of presentation)
For pain control use Demerol vs. morphine as morphine induces spasm of sphincter of Oddi

Cholangitis = bacterial infection of biliary tract (true surgical emergency)
Charcot’s triad = fever/chills, jaundice, RUQ pain
Reynold’s pentad = Charcot’s + altered mental status and shock
Rx IVF, Abx, and surgical decompression

**Hernia**

**Causes**
Increased intraabdominal pressure, obesity, pregnancy, ascites, patent processus vaginalis.

**Complic.**
Incarceration (unable to reduce), strangulation (compromised blood supply), SBO (#1 cause of SBO in children and adults with no prior Hx of surgery). Note: the smaller the defect in the fascia the more likely the herniated bowel is to strangulate.

**Types**
Most common are indirect inguinal > direct inguinal > femoral

- **Indirect inguinal**: lateral to Hesselbach’s triangle, through internal ring of inguinal canal, most common hernia in men and women.
- **Direct inguinal**: within Hesselbach’s triangle
- **Femoral**: beneath inguinal ligament down femoral canal and medial to femoral vessels. More likely to incarcerate than inguinal hernias

**Rx**
Herniorrhaphy (open or laproscopic), emergent or elective depending on complications. Often use mesh.

**Acute Pancreatitis**

**Causes**
Alcohol, gallstones, idiopathic, hypercalcemia, trauma, hyperlipidemia, ERCP (iatrogenic), cardio-pulmonary bypass, familial, drugs

**Complic.**
Pseudocyst, abscess, necrosis, ARDS, Sepsis, hypocalcemia, DIC, splenic vein thrombosis, shock and multi-organ failure

**Sxs/signs**
Epigastric pain radiating to back, nausea, vomiting, abd. tenderness, guarding, decreased bowel sounds, fever, dehydration, shock. Look for Cullen’s (periumbilical) or Turner’s (flank) signs that indicate retroperitoneal hemorrhage

**Findings**
Increased amylase, lipase, WBC, LFTs, and glucose. Decreased Hct and calcium. Pseudocyst, phlegmon, abscess, necrosis on CT. Gallstones on CT, U/S.

**Rx**
Supportive therapy: fluid resuscitation, meperidine (Demerol) for pain, NPO, NGT if needed for protracted nausea and vomiting. Surgical debridement and abx for infected necrotizing pancreatitis. CT-guided drainage and abx for pancreatic abscess.

**RANSON’S CRITERIA** (predicts mortality)

<table>
<thead>
<tr>
<th>Initially (at dx)</th>
<th>After 48 hrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt;55</td>
<td>Base Deficit &gt;4</td>
</tr>
<tr>
<td>WBC &gt;16,000</td>
<td>BUN increase &gt; 5</td>
</tr>
<tr>
<td>Glucose &gt;200</td>
<td>Serum Ca &lt;8</td>
</tr>
<tr>
<td>LDH &gt;350</td>
<td>Hct decrease &gt;10%</td>
</tr>
<tr>
<td>AST &gt;250</td>
<td>Fluid sequestration &gt;6L</td>
</tr>
</tbody>
</table>

**PROGNOSIS**

<table>
<thead>
<tr>
<th># of criteria</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-2</td>
<td>&lt;5%</td>
</tr>
<tr>
<td>3-4</td>
<td>~15%</td>
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<tr>
<td>5-6</td>
<td>~40%</td>
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<tr>
<td>7-8</td>
<td>~100%</td>
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(Am J Gastroent 77:633;1982)

**Chronic Pancreatitis** = fibrosis, calcification due to chronic inflammation

**Causes**
Alcohol, idiopathic, hypercalcemia, hyperlipidemia, familial, trauma, iatrogenic, gallstones, cystic fibrosis.

**Complic.**
Diabetes, steatorrhea, malnutrition, splenic vein thrombosis

**Sxs/signs**
Epigastric pain, weight loss, steatorrhea, diabetes

**Ddx**
PUD, pancreatic cancer, angina, AAA
Imaging
May see calcification of pancreas on AXR, CT.
May see duct dilation/stenosis on ERCP (chain of lakes)

Rx
Insulin, pancreatic enzyme replacement, pain meds, stop alcohol.
Surgery indicated for severe, refractory pain (many options including Peustow procedure, distal pancreatectomy, total pancreatectomy, others)

Pancreatic Cancer
RFs
Chronic pancreatitis, smoking, DM, FHx

Sx
Dull epigastric pain radiating to back, may be worse w/eating; weight loss, Anorexia, ± jaundice, steatorrhea if tumor occludes bile duct

PE
may be unremarkable; ± abd mass, ascites, nontender palpable gallbladder, supraclavicular nodes

Dx
Labs: elevated bili, alk phos, CA 19-9 transabdominal U/S
CT abdomen w/ and w/o contrast -> if see mass, will need surgical C/S
Endoscopic U/S with FNA
ERCP/MRCP to r/o cholangitis and chronic pancreatitis

Tx
Resect tumors without mets that don’t invade SMA/SMV, portal vein etc.
Procedure is typically Whipple; also total pancreatectomy, and others.

Small Bowel Obstruction = mechanical obstruction of intraluminal contents

Causes Adhesions = #1, hernia (# 1 in kids and in adults with no hx of abd surgery), tumor, intussusception, gallstone ileus, Meckel’s diverticulum, abscess, bowel wall hematoma, radiation enteritis, Crohn’s disease

Complic. Bowel strangulation, necrosis

Sxs/signs Abd. pain, cramping, nausea, vomiting, high-pitched bowel sounds
If strangulated bowel -> fever, severe pain, hematemeses, shock, abdominal free air, peritoneal signs, acidosis

Ddx Paralytic ileus (common in post-op pts.), electrolyte imbalance (hypokalemia is most common)

Types Complete (no colon gas), incomplete (some colon gas)

Rx NGT, IVF, Foley cath, and close observation for incomplete. Laparotomy for complete SBO.

Large bowel obstruction:
Almost always requires an operation, less common than SBO
Ddx: Colon Cancer, obstipation, volvulus.

Volvulus, cecal = twisting of cecum on itself and mesentery, usu. axial twist

Causes Idiopathic poor fixation of R colon, many have H/O abd surgery
Sxs/signs Acute abd pain, colicky RLQ pain, progresses to constant pain with vomiting, abd distention, obstipation

Dx/Imaging AXR shows dilated colon with large air-fluid level in RLQ. “Coffee bean” sign = apex aiming toward LUQ
Colonoscopy or gastrografin contrast study if AXR non-diagnostic

Rx Emergent surgery. Cecopexy if cecum is viable, R colectomy with ileostomy and mucus fistula if cecum infarcted

Volvulus, sigmoid (more common than cecal)
RiskFx  High residue diet, pregnancy, constipation, laxative abuse, think elderly persons in nursing homes and other chronically institutionalized persons

Complic.  Obstruction, necrosis, perforation of colon

Sxs/signs  Acute abd. pain, progressive distention, anorexia, cramps, nausea, vomiting, obstipation. Signs of strangulation include hemorrhagic mucosa on sigmoidoscopy, bloody fluid in rectum, peritoneal signs, fever, and hypovolemia. Signs of necrotic bowel include free air, pneumatosis

Imaging  See distended loop of sigmoid colon on AXR. "omega sign" = loop pointing towards RUQ

Dx  Sigmoidoscopy or CT with gastrografin enema

Tx  Sigmoidoscopic reduction successful in 80%. Enema study can also reduce. 40% recurrence after nonoperative reduction, so do elective sigmoid resection even if successful in reducing.

Peripheral vascular disease
If patient has PVD, likely also has other vascular disease (CAD, carotid disease, or AAA)

Risks  SMOKING is the biggest

S/Sx  Claudication, chronically cold extremity, decreased pulses, muscle atrophy

Work-up  ABIs – ratio of measured BP in ankle and arm (brachial). Normal ABI > .9. Abnormal if <.9, indicates peripheral vascular disease. ABIs < 0.4 indicate severe ischemia (resting pain) and contraindication to bypass. Angiogram needed if bypass planned.

Rx  Smoking cessation, exercise for mild disease, anti-platelet therapy. Revascularization procedures if medical therapy fails.

Fever Work-Up (6W’s)

Wind  Pneumonia, atelectasis (especially 1-2 days post-op)

Water  UTI (especially if Foley present)

Wound  5-7 days (abd abscess will wall off after 5-7 days)

Walk  DVT, PE

Wonder drugs  Drug fever especially if on prolonged Abx or new med

Whole Blood  Transfusion rxn

Fistula formation  Things that keep fistulas open (FRIENDS)

Foreign body

Radiation

(Granulomatous) Inflammation

Epithelialization

Neoplasm

Distal Obstruction

Steroids

Wound Healing  (From Essentials for Students: Plastic and Reconstructive Surgery, 1998)

Substrate/Inflammatory Phase, Days 1-4

Redness, heat, swelling, pain, loss of function. Leukocyte margination, venule dilation, lymphatic blockade, neutrophil chemotaxis, phagocytosis. Removal of clot, debris, bacteria. Lasts 1-4 days in primary intention. Healing continues until wound is closed in secondary and tertiary intention healing

Proliferative Phase, Days 4-42

Synthesis of collagen from fibroblasts, rapid gain of tensile strength
Remodeling Phase, 3 wks onward
Maturation by cross-linking of collagen, leads to flattening of scar. Dynamic, ongoing process, 9 months in adults.

Primary Intention Healing
Closure by direct approximation, flap, skin graft

Secondary Intention Healing (spontaneous healing)
Wound left open, maintained in inflammatory phase. Closure depends of contraction and epithelialization. Contraction due to force of myofibroblasts. Epithelialization occurs from margin to center, ~1mm/day.

Tertiary intention healing
Delayed wound closure, intentional interruption of healing begun as secondary intention. Performed when wound not infected and granulation tissue present.

Factors influencing wound healing
- Tissue trauma
- Malnutrition
- Hematoma
- Steroids
- Blood supply
- Chemotherapy
- Temperature
- Chronic illness
- Infection
- Technique/suturing

Skin Graft = skin separated from its bed, transplanted to another area; receives new blood supply

Spilt thickness
Epidermis + part of dermis. Donor site heals in 7-10 days
Thin graft has better take
Thick graft is more durable and has less contraction

Full Thickness
Epidermis + all of dermis. Slower vascularization.
Donor site has full thickness skin loss, which must be closed by primary intention or split thickness skin graft. Used for fingers, face.

Graft survival
1 to 48 hrs Serum imbibition, diffusion of nutrients
48hr to 4d Inosculation, capillary ingrowth
5d-> Revascularization

Factors contributing to graft loss
- Hematoma / seroma forms under graft
- Shearing forces or traumatic tissue handling
- Decreased vascularity of recipient bed
- Infection / colonization
- Traumatic tissue handling

Skin Flaps = tissue transferred from one site to another with its own vascular supply. Used to replace tissue loss due to trauma or surgery, bring in better blood supply, improve sensation, and for reconstruction

Random flap
2 types: rotation and advanced. Limited length-to-width ratio (1.5-2.1 to 1). Blood supply is from dermal and subdermal plexus.

Axial Flap (arterial flap)
Peninsular vs. island. Greater length possible, blood supply is by artery and accompanying vein.

**Musculocutaneous flap**
Consists of skin, subQ, and muscle tissue (well-vascularized). Blood supply from vessels in muscle.

<table>
<thead>
<tr>
<th>Reconstructive Ladder</th>
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<tbody>
<tr>
<td>Direct Closure</td>
</tr>
<tr>
<td>Graft</td>
</tr>
<tr>
<td>Local Flap</td>
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<tr>
<td>Distant Flap</td>
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<tr>
<td>Free Flap</td>
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**Essential Medications for Surgery Rotation** (6 Ps)

**PRNs**

- **Tylenol**: 650 PO/PR Q4-6H PRN don’t exceed 4 g/24 hours
- **Reglan**: 10 mg PO/IV QID (take 30 min prior to meals and at bedtime)
- **Zofran**: 8 mg PO/IV Q8H PRN
- **Ambien**: 5-10 mg PO QHS
- **Benadryl**: 25-50 mg PO/IV Q4-6 H PRN

**Pain**

- **Morphine**: 2.5-5 mg IV q2-3H PRN
- **Demerol**: 25-50 mg IV q3-4 H PRN
- **Dilaudid**: 1-4 mg IV q4-6H PRN
- **Percocet**: (oxycodone) 325/5 mg (or other combos) 1-2 PO Q4-6H PRN
- **Vicodin**: (hydrocodone 500/5 or other combo) 1-2 tabs PO Q4-6 H PRN

**Prophylaxis (GI and DVT)**

- **Protonix**: 40 mg PO/IV QD
- **Heparin**: 5000 U SQ q 8-12H

**Poop**

- **Docusate**: 100-200 mg PO BID

**Parasites (antibiotics)** (prophylaxis antibiotic doses are the only ones included here)

- **Ancef (Kefzol)**: 1g IV Q6H
- **Unasyn**: 1.5-3g IV Q6H
- **Zosyn**: 3.375 g IV Q6H
- **Metronidazole**: 500 mg PO QID
- **Cefoxitin**: 1-2 g IV Q6-8 H

**Pre-Op Medications** Don’t forget to restart pre-op medications as appropriate!
Clerkship directors: HMC - Eileen Bulger, M.D., UW - Jared Strote, M.D.

REFERENCES & HELPFUL RESOURCES
1. https://depts.washington.edu/emed/wordpress/ (check out procedure videos)

WARD TIPS
1. With every patient, ask yourself “Is this patient sick or not sick?” “While in the ED, does this patient need to be in a monitored bed?” and “Do they need to be admitted?” (Consider social support situation and ability to care for self outside)
2. Think ahead, get labs and imaging early, call consults early with appropriate diagnostics completed and results in front of you.
3. D/C Home if:
   ED care is completed  
   Patient has normal vital signs  
   Patient is awake, alert, and in stable condition  
   Patient is ambulatory or at baseline  
   Patient has received appropriate care, discharge instructions, follow-up

NOTES
• Strive to be legible and succinct
• Record patient Time in / time out of ED
• Address CC as documented on chart by nursing staff (this may be different than CC given to you by patient)
• Recheck vital signs if abnormal on first check
• Abbreviations

STHB said to have been HOD heroin overdose
STH said to have MVC motor vehicle collision
CBIB courteously brought in by CTLS cervical, thoracic, LOC loss of consciousness lumbar spine
AOB alcohol on breath BAL blood alcohol level
GCS Glasgow coma scale PTA prior to arrival

• Things to include in HPI (some info may be gathered from paramedic notes):
   _____ y.o. male/female referred by Dr._____ from _____hospital (or CBIB medics from _____) STH/STHB_______
   + / - LOC
   VS and GCS at scene, interventions prior to arrival (CPR, meds, fluids) complaints on arrival
document dominant hand and occupation for any UE injury

• Physical Exam (examples of items to include, as appropriate)
  Gen: AO x 3, NAD, interacts appropriately
  HEENT: NC/AT, PERRL, EOMI, TMss clear, oropharynx wnl
  CTLS: Ø tenderness, Ø stepoff, Ø contusions/abrasions/lacs
  Neck: C-collar in place, supple, Ø STS (soft tissue swelling), Ø ecchy, Ø abrasion, nl range of motion
  Lungs: CTA bilaterally, symmetric chest excursions, no crepitus
Cardiac: RRR, no murmur/gallop/rub
Abd: soft, NT/ND
Pelvis: NT, stable to AP and lateral compression
Rectal: nl prostate and tone, Ø gross blood, guaiac + or -
Ext: AT, pulses, Ø STS, Ø ecchy, Ø contusion/abrasions/lacs
Neuro: MS, CN II-XII intact, sensory intact, DTRs, gait/cerebellum, GCS (3-15)
Motor: D B T WE WF Q H DF PF
R 5 --------------------------------------------
L 5 --------------------------------------------

• Imaging/EKG: studies obtained in ED, compared to prior if available
• Labs: labs obtained in ED (often draw a “rainbow.” Order usually: red, green, blue, lavender, but in trauma get lavender and blue first—Hct and coags are priority!)
  RED = chemistries (M7, LFTs), amylase, lipase, EtOH, hCG, hep serologies, drug levels, thyroid, ANA, PSA, RPR, methemoglobin, CPK isoenzymes
  LAV = CBC, platelets, ESR
  BLUE = coags, PT, PTT, INR
  GREEN = ammonia, thiamine, chest pain panel
  PEARL = BNP

• ER Course: Describe events and time course while in ED. This is hard to remember, but very important when someone asks you why your patient was in the ED for 5 hours
  1400 Pt. Seen by med student / M.D.
  1430 Pt. In CT
  1500 CT read by radiology
  1530 D/C to home

• Assessment / Plan, Dispo, Safety
  Pt. D/C to home with instructions for wound care, meds, follow-up in ____ clinic, and instructions for when to return to ED/UCC.

SELECTED TOPICS IN EMERGENCY MEDICINE
(From Surgery 684 Student Manual, 2000, Baernstein and Sparks)

Trauma Primary survey = ABCDE
Fix deficits before moving to next step. If pt status changes, go back to A, then BCD…

Airway (If pt can talk, airway is OK)
(& C-spine) Otherwise start with chin lift/jaw thrust, then oropharyngeal/nasopharyngeal airways, then intubation, with cricothyroidotomy the last resort.
All trauma patients need c-spine immobilization.

Breathing Inspect (movement, rate, tracheal shift, JVD, accessory, chest wounds)
Auscultate (upper/lower airway (stridor, wheeze, gurgle)
Percuss (hyper-resonant or dull to percussion)
Palpate (crepitance, rib fx, flail chest)
During this stage place chest tubes, flutter valves if tension PTX, 3 sided occlusive dressings if open chest wound. May also need to intubate here if work of breathing is increased
If intubated, check for CO2 return and bilateral breath sounds!
Circulation: Assess perfusion AND bleeding
BP estimation by pulse site (pulse present = BP > than number)
  Radial = 90mmHg pressure  Femoral = 70
  Brachial = 80  Carotid = 60
Assess UOP, mental status, cap refill, skin
Important -- BP is not a reliable indicator of volume
Address massive bleeding at this time
  The treatment for hypovolemic shock is volume.  Put direct pressure on bleeding wounds; if bleeding severe and ongoing, pt may need emergent surgery.

Disability  Glasgow Coma Scale  Coma = score < 8

Eye  4 pts if opens spontaneously  Verbal  5 if appropriate answers
  3 if opens to command  4 if confused
  2 if opens to pain  3 if inappropriate words
  1 if does not open  2 if incomprehensible
Motor  6 if obeys commands
  5 if localizes painful stimulus  If intubated, omit verbal portion
  4 if withdraws from pain
  3 if decorticate posturing
  2 if decerebrate posturing
  1 if no movement

NB: A dead patient has a GCS = 3

Exposure – remove pt’s clothing and examine (then cover to prevent hypothermia)

6 life-threatening conditions to be diagnosed during primary survey
  • Airway obstruction (Tx=intubate, or surgical airway)
  • Tension pneumothorax (Tx= fluttervalve or chest tube)
  • Open pneumothorax (sucking chest wound) (Tx= 3 sided dressing + chest tube)
  • Flail chest (more than 3 continuous ribs fractured in 2 locations). Tx= intubate
  • Cardiac tamponade (Beck’s triad = muffled heart sounds, high neck veins, and hypotension) Tx= pericardiocentesis
  • Hemothorax (decreased breath sounds dull to percussion). Tx = Chest tube

Secondary survey is a physical exam from head to toes looking for other traumatic injuries (fxs, puncture wounds, dislocations, amputations, etc)
  • Consider NGT to decompress GI tract or w/suspected OD, etc
  • Foley cath unless blood at perineum/urethral meatus or “high-riding” prostate

Imaging = Trauma Series
  1. Lateral C-spine (look at airway and for obvious c-spine fx / alignment)
  2. AP chest (to diagnose pneumothorax, hemothorax, check ET tube placement, widened mediastinum as evidence for aortic dissection, etc)
  3. AP pelvis (for fractures)
General trauma info:

1. All trauma patients get monitors (cardiac, BP, O2 sat at least), 2 or more IVs (16-18 gauge or larger), supp O2, foley after rectal exam
2. Labs: “Trauma Panel” = CBC, chem 7, Coags, Type and Cross, amylase, ABG, ETOH level, U dip and Utox, (hCG for women of child-bearing age)
3. Fluid: 3:1 rule. 3cc crystalloid for 1 cc blood. Basically bolus adults w/ 2 liters of LR, may repeat times one, if still unstable move on to blood transfusion. (Unless obviously needs blood right away). Don’t let fluid replacement delay OR.
4. Be available when trauma patients arrive; the trauma doc at Harborview will give you a task. Don’t take it personally if you are elbowed out of the way because you are taking too long to put in the foley or fem stick. It needs to be done quickly.
5. Trauma “codes” at Harborview:
   - Green= trauma doc only needs to see
   - Yellow= third year surgical resident must see and work-up patient
   - Red= “trauma code” very unstable and requires the attending or trauma fellow and chief resident to be present upon arrival to ER.

Potential spaces for life threatening internal hemorrhage
1. chest       2. pelvis       3. abdomen       4. femur       5. scalp

Shock
Five types: All result in decreased blood pressure, hypoperfusion, and eventually multiple organ system failure and death if patient not resuscitated.
1. Hypovolemic – not enough blood volume to perfuse
2. Cardiogenic – heart not pumping well enough to push adequate volume to perfuse
3. Septic – cardiac output increased, but vasculature so dilated due to inflammatory reaction that insufficient pressure to perfuse (functional hypovolemia)
4. Neurogenic – dilated vas due to spinal cord injury (loss of sympathetic tone)
5. Anaphylactic – similar to septic

Hypovolemia
Physiologic response to hypovolemia:
   Early = tachycardia, decreased urine output, narrowed pulse pressure
   Late = hypotension
   Na+/H2O retention via renin/aldosterone
   H2O retention via ADH
   Vasoconstriction via Angiotensin II and sympathetic response

Low-speed MVC (<35mph)
Likely injuries include neck strain, back strain, contusion, abrasion, concussion
History Description of crash - speed, wearing seatbelt?, airbag deployed?, LOC?, passenger or driver?, side of impact
PE Head, spine, abd, neuro and any area that hurts.
Labs 2 Hcts 30 min apart, urine dip, stool guaiac
Imaging C-spine series, Head CT for LOC, other imaging as needed
Rx Handouts on neck strain, back strain, etc.
Pain: ibuprofen 600 mg PO QID or naproxen 500 mg PO BID until F/U
F/U in UCC 4-7 days
F/U in 1-2 days with concussion or LOC
If C-spine is cleared by x-ray, but tender to palpation, 2 options:
- Have patient wear soft collar x 2 wks (including sleeping and showering), then return for Flexion-Extension films
- Do Flexion-Extension films before patient leaves ED

Lacerations

History  How and when they occurred, possible retained foreign objects?
PE      Fever, signs of infection, neurovascular integrity distal to wound, measure size and record location (draw picture)
Labs    Hct, EtOH if indicated
Imaging To look for foreign body at site, involvement of underlying bone, or head CT for patient with head laceration and intoxication
Rx      Tetanus toxoid, unless documented in past 5 years
         If >8-12 hrs old, clean but do not suture

Clean skin with Betadine, anesthetize with 1% lido w/ epi (no epi for ear, finger, nose, toes, etc.)
Shave area (do not shave eyebrows)
Irrigate with 1-2 L warm, sterile saline
Explore: joint space involved? (call ortho), hand/wrist tendons involved? (call ortho hand)

If laceration of tongue or through cheek, call OMFS
If laceration of ear with cartilage damage, call ENT
If laceration near eye, call ophtho
Suture according to table below

<table>
<thead>
<tr>
<th>Location</th>
<th>Suture</th>
<th>Suture Removal</th>
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<tbody>
<tr>
<td>Scalp</td>
<td>4-0 Dermalon</td>
<td>7 days</td>
</tr>
<tr>
<td>Face</td>
<td>5-0 Dermalon</td>
<td>5 days</td>
</tr>
<tr>
<td>Oral Mucosa</td>
<td>4-0, 3-0 Chromic gut (absorb.)</td>
<td>NA</td>
</tr>
<tr>
<td>Ext</td>
<td>3-0, 4-0 Dermalon</td>
<td>10-14 days</td>
</tr>
<tr>
<td>Over joint</td>
<td>3-0 Dermalon</td>
<td>10-14 days</td>
</tr>
<tr>
<td>SubQ</td>
<td>4-0, 3-0 Dex/Vicryl</td>
<td>NA</td>
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F/U for suture removal in UCC.
Give patient wound care instructions, antibiotic ointment, gauze and tape

IVDU Abscess

History  Fever, nausea, H/O murmur, H/O endocarditis
PE      Murmur, fluctuance, induration, cellulitis, neurovascular function distal to abscess
Labs    If T > 38.5, do CBC and blood culture x 2
         CBC if temp is nl
Imaging Consider if osteomyelitis or gas-forming organisms are of concern
Rx      Fever, large area, or deep abscess -> candidate for admission
         Hand abscesses should be seen by ortho
         Give Tetanus toxoid
         I & D Abscess:
- Consider pre-medication for pain/anxiety
- Clean area with Betadine, prep/drape in sterile fashion
- Wear mask with eye protection and sterile gloves
- Anesthetize area with lidocaine/epi
- Open abscess with scalpel. Evacuate pus. Irrigate with saline
- Pack wound with iodoform gauze, cover with dry bandage

If there is surrounding cellulitis, give antibiotics:
If MRSA, Vancomycin 1g IV q12h or Bactrim 1 tab DS PO BID x 7-14 days
If MSSA, Cephalexin 500 mg PO QID x 7 days or Dicloxacillin 500 mg PO QID

Bites
History How and when did injury occur? Animal / Human
PE Neurovascular integrity distal to wound, signs of infection, fever
Labs CBC if wound appears infected
Imaging If concern for fracture, osteomyelitis, gas-forming organisms
Rx Tetanus toxoid
Clean skin with Betadine
Irrigate with sterile saline
Do not close bite wound
Give Timentin 3.1 gm IV x 1 dose in ED
Give Augmentin 875 mg PO BID x 7 days
Ortho should see hand infections
F/U in UCC in 24-48 hrs.

Low Back Pain
History Trauma, heavy lifting, radiculopathy, bowel or bladder incontinence, “saddle anesthesia” (numbness in perineum), fever, weight loss, cancer, IVDU
PE Spinal tenderness, muscle tenderness, neuro exam of lower extremities, straight-leg raise, rectal tone, saddle anesthesia
Imaging Only if patient has concerning symptoms and signs: fever, H/O IVDU or cancer, weight loss, incontinence, decreased rectal tone or numbness
Rx For most back pain, treat conservatively
Toradol 30 mg IV x 1 dose in ED (if normal renal function)
Robaxin 1 gm IV x 1 dose in ED
D/C with ibuprofen 600 mg PO QID or naproxen 500 mg PO BID, robaxin 500-1000mg PO QID
Back physical therapy once acute episode has resolved
Ice / heat, No bed rest

Alcohol Intoxication
A few things to think about when caring for intoxicated patients:
DDx of decreased level of consciousness
Head injury, stroke, CNS infection, metabolic abnormalities, other ingestions – opiates, methanol, acetone, ethylene glycol, isopropyl alcohol, cocaine, benzodiazepines, barbiturates, carbon monoxide, inhalants (gas, glue)
Level of consciousness should improve over time: document this with repeat VS/mental status/brief neuro exams; if no improvement - suspect other Dx too
Co-morbidities that can be present in intoxicated patients
Pneumonia, upper GI bleed, subdural hematoma, hypothermia, cellulitis,
fractures, pancreatitis

**History**
- Substances used, trauma?, GI bleeding?, H/O withdrawal or seizure? If patient cannot give hx, do PE, and get history later

**PE**
- Skin, CV, Resp, Abd, Extremities (trauma, infection), Neuro. Do stool guaiac if patient cannot give history regarding GI bleed

**Labs**
- BAL
  - Do CBC, Chem 7 if other pathology is present
  - Ca, Mg, PO4 if seizures or withdrawal
  - Alcohol Screen (methanol, acetone, ethylene glycol) if warranted
  - Udup / Utox

**Rx**
- Thiamine 100 mg IV/IM at every ED visit
- 2-pt. restraints if needed
- Recheck abnormal vital signs
  - Warm blankets / dry clothing, esp. if T < 35.0C
  - Consult social work for alcohol treatment options

**Dispo**
- Assume drop of 50 mg/dL per hour in BAL, D/C when BAL <150 (Beware of withdrawal) and pt is alert, oriented, talking, and ambulating safely

In most people, EtOH level 50-100 mg/dL = disinhibition
  - 200 mg/dL = slurred speech
  - 400-500 mg/dL = coma, resp depression, vascular collapse

Tolerance develops with chronic alcohol use (you may see chronic alcoholics that will have withdrawal symptoms at a BAL of 200 mg/dL)

**Alcohol Withdrawal**
- Signs and symptoms of withdrawal include seizure, tremor, hypertension, tachycardia, insomnia, vomiting, visual hallucinations, delirium tremens

**History**
- Time of last drink, H/O withdrawal, H/O HTN, H/O head injury

**PE**
- Monitor VS (P, BP) and watch for changes

**Neuro**
- Tremor, asterixis, focal neurologic signs

**Labs**
- BAL (withdrawal unlikely if BAL > 200 mg/dL)
  - Chem 7, Mg, PO4, Ca
  - CBC

**Imaging**
- Head CT if first seizure or H/O head trauma
  - CXR if concern for aspiration, pneumonia

**Rx**
- IVF, electrolyte replacement
  - Thiamine 100-200 mg IV/IM
  - Prochlorperazine 10 mg IV/IM for nausea/vomiting
  - Diazepam 5-10 mg IV q 15 min prn

**Dispo**
- If in mild withdrawal, D/C to home, street, jail, or medical detox
- If mod-severe withdrawal, admit

**Abdominal Pain**
- History
  - N/V/D, constipation, appetite, change in stool, blood in stool, hematemesis, dysuria, hematuria, frequency, vaginal bleeding, vaginal discharge, pregnancy, LMP, trauma, drugs/EtOH/meds, cough, fever, pleuritic chest pain, chills

- PE
  - VS, skin rash, peritoneal signs, size of spleen/liver, ascites, Murphy’s sign, CVA tenderness, hernia, pelvic exam in females, rectal exam

- Labs
  - CBC, Chem 7, B-HCG, UA. Consider Hct x 2, coags, amylase, bili, LFTs,
hepatitis panel

Imaging  
CXR if suspect pneumonia  
Abd series if patient has peritoneal signs  
EKG for cardiac suspicions  
Abd US for liver, gallbladder, appendix  
Pelvic US for ectopic pregnancy, ovarian torsion/abscess  
Testicular US for torsion/abscess  
CT-KUB, non-contrast, good for kidney stones, hydronephrosis  
Abd CT with IV contrast for vascular  
Abd CT with IV and PO contrast (time-consuming). Look for abscess, diverticulitis, appendicitis, pancreatic pseudocyst

Rx  
If patient has surgical condition: IVF, CBC with plts, M7, coags, amy, bili, UA, B-HCG, EKG for older men and women, NPO, appropriate imaging, and surgical consult.  
Otherwise, Rx with appropriate meds and outpatient follow-up

Seizure  
Causes  
Epilepsy, post-traumatic, drugs, toxins, fever, electrolyte abnl, hypoxia, alcohol withdrawal  
History  
Symptoms before and after seizure, H/O seizure, drugs/EtOH/meds, H/O head trauma, trauma sustained during this event, loss of continence  
PE  
Head, neck, tongue. Look for trauma, incontinence  
Neuro (if post-ictal/Todd’s paralysis, patient may not be able to do fully)  
Repeat neuro exam  
Look for signs of alcohol withdrawal  
Labs  
IV access always, Chem 7, CBC, Ca, Mg, BAL  
Dilantin, Tegretol, phenobarb, Depakote levels  
LP if signs of meningitis, encephalitis, bleed  
Imaging  
CXR if aspiration suspected  
Head CT w/o contrast for new trauma, new focal neuro finding, no improvement in mental status, severe HA  
Head CT w/ contrast for 1st time seizure  
C-spine series for neck pain, fall, head trauma  
Rx  
Status epilepticus: Intubate, C-spine precautions, meds (Diazepam, Phenytoin)  
First-time seizure: Admit for work-up  
Epilepsy: Usually due to sub-therapeutic on meds, refill meds  
F/U with Neuro Clinic  
Withdrawal Seizure: See Alcohol Withdrawal above

Asthma  
Risk fxs  
H/O intubation, >2 admissions in past year, >3ED visits in past year, for death: H/O sudden, severe exacerbation, poverty  
History  
Triggers – URI, allergies, exposures, exercise, cold  
Meds, using meds properly?  
Symptoms – chest tightness, wheezing, cough, nocturnal cough  
PE  
Able to speak in full sentences?  
Resp: wheezing, RR, O2 sat, chest hyperinflation  
Labs / Tests  
Serial Peak Flows  
CXR if suspect PNA, CHF
ABG for severe exacerbation  
Theophylline level, if indicated  
Rx  
Assess ABC’s, give O2  
Give nebulized albuterol & ipratropium, +/- IV steroids  
Mild exac  
Several nebulized albuterol doses or MDI w/ spacer (many puffs)  
Prednisone 40 mg PO QD x 5 days  
F/U In UCC or with PMD in 2-3 days  
Severe exac  
O2, continuous nebulized albuterol, ipratropium  
Prednisolone 125 mg IV x 1 dose  
Epinephrine if pt. is deteriorating (1:1000) 0.3-0.5 mg SQ q15-20 min up to 3 doses  
Consider aminophylline 5 mg/kg IV over 45 minutes  
Admit  
Intubate for  
Apnea, mental status changes, acute respiratory acidosis pH < 7.1  
Use low tidal volumes, low resp. rate, high inspiratory flow rate  
Rx of Chronic Asthma: Education is important! Short-acting B-2 agonists, long-acting B-2 agonists, inhaled corticosteroids  

Chest Pain  
Ddx  
Acute MI, unstable angina, stable angina, PE, pericarditis, aortic dissection, mitral valve prolapse, cardiac tamponade, pneumonia, PTX, asthma, COPD, pleuritis, bronchitis, GERD, esophageal spasm, esophagitis, cholecystitis, costochondritis, rib fx, muscle strain, herpes zoster  
History  
Onset, timing, quality and character of pain, progression of pain, constant or intermittent, factors that alleviate or exacerbate, radiate?, positional?, responsive to SL NTG?, cardiac risk factors: CAD, HTN, tobacco, DM, high cholesterol, Fam Hx  
PE  
Resp: crackles?  
Chest wall: tenderness?  
CV: S3, murmur, friction rub?, neck veins, pulses  
Abd: pulsatile mass?  
Neuro  
Labs/Tests  
12-lead EKG  
Cardiac enzymes  
CBC, Chem 7, coags, Ca, ionized Ca, Mg  
Imaging  
CXR  
Rx  
For presumed coronary chest pain:  
Monitor cardiac rhythm  
O2 2-4 L via NC to keep O2 sat > 95%  
ASA 325 mg chewed  
0.4 mg SL NTG q 5 min x 3 doses (can do NTG drip 10 mcg/kg/min if chest pain persists)  
Morphine 2 mg IV prn  
Metoprolol 5 mg IV q 5 min x 3 doses (longer-acting) or esmolol 500 mcg/kg IV over 1-2 min load, then 50-250 mcg/kg/min IV drip (shorter-acting in case patient develops side-effects)  
Heparin 80 u/kg IV bolus, then 16 u/kg/hr maintenance  
Consider reperfusion therapy (thrombolytics or PTCA)  

If patient has Acute MI or unstable angina, admit and follow
If patient has stable angina and chest pain resolves quickly and is not out of ordinary for patient, then D/C home

Pericarditis  Indomethacin 50 mg PO tid or ibuprofen 800 mg PO tid
Aortic dissection  Thoracic CT w/o contrast
Stat surgery consult
Control HTN with esmolol
Add sodium nitroprusside 0.5-10 mcg/kg/min if SBP remains >130
Morphine for pain
Tension PTX  If unstable, flutter valve
If hypotensive, 1-2 L isotonic fluids
Chest tube
Muscular pain /
Costochondritis  Ibuprofen 600 mg tid-qid x 7 days

GERD  GI cocktail for immediate relief (and dx)
- Viscous lidocaine & Maalox
Smaller meals, avoid caffeine, alcohol, fatty foods
Elevate HOB
Start PPI

Overdose
History  Substances and quantity ingested, time of ingestion, prior H/O OD, drug & alcohol use
PE  Mental status, pupils, resp. rate and pattern, gag reflex
Labs  CBC, M7, B-HCG, EtOH, ASA, Tylenol, extended urine tox (calculate anion gap). Consider serum osmolality
Tests  EKG, look for prolonged QRS, prolonged QT interval
Rx  Assess ABC’s. Intubate if patient cannot protect airway
Give O2, telemetry, pulse O2, IV access, IVF if needed
Consider Thiamine 100 mg IV, glucose 25 gms IV, Naloxone 0.8 mg IV
Admit if ↓ LOC, ECG or rhythm abnl, toxic serum drug levels, or ingestion of sustained-release pills.
Evaluation by social work and/or Psych for suicide precautions

Overdose & Antidotes
Organophosphates  Atropine 1-2 mg IV then 1-4 mg IV q15 min prn
Opiates  Naloxone 0.8 mg IV load, 0.4-0.6 mg/hr IV drip
Isoniazide  Pyridoxine 1 gm per gm INH ingested over 5 min. Max 5 gms.
Acetaminophen  N-acetylcysteine 140 mg/kg per OG after lavage
Methanol  Ethanol 10 mL/kg of 10% solution load, then 1.0-2.0 mL/kg/hr
Carbon Monoxide  100%O2, hyperbaric O2
Digoxin  Digoxin antibody fragments 10-20 vials for arrhythmias
Anticholinergics  Phystostigmine 0.5-2.0 mg IV over 2 min q30 min prn

Block Absorption  Gastric Lavage, Activated charcoal
Enhance Elimination
Alkaline diuresis  For ASA, Phenobarb: 3 amps NaHCO3 to 1 L D5W, infuse at 250cc/hr
Hemodialysis  Severe ASA, lithium, methanol, ethylene glycol, mushroom
### Pain Management

**From An Introduction to Emergency Medicine by Mengert**

- **Acute Pain:** opioids are a good choice in this situation.
- **Chronic Pain (non-malignancy):** opioids not a good choice, choose NSAIDS, APAP (but screen for renal impairment, h/o ulcer or GIB, allergies, and liver disease).

### NSAIDS

<table>
<thead>
<tr>
<th>Medication</th>
<th>Usual Dosing</th>
<th>Max Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASA</td>
<td>325-975 mg PO q4-6hr</td>
<td>4000mg/24hr</td>
</tr>
<tr>
<td>Ketorolac (Toradol)</td>
<td>30-60 mg IM, then 30 mg IM q6hr or 10 mg PO q6hr</td>
<td>120mg/24hr IM 40mg/24hr PO</td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>300 mg PO QID up to 800 mg PO tid</td>
<td>2400 mg/24hr</td>
</tr>
<tr>
<td>Naproxen</td>
<td>250-500 mg PO BID</td>
<td>1250 mg/24hr</td>
</tr>
<tr>
<td>Naproxen sodium</td>
<td>550 mg PO, then 275 PO q6-8hr up to 550 mg PO bid</td>
<td>1375 mg/24hr</td>
</tr>
</tbody>
</table>

### OPIOIDS

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Codeine</td>
<td>30-60 mg PO q4-6hr</td>
</tr>
<tr>
<td>Meperidine (Demerol)</td>
<td>50-150 mg PO q3-4hr</td>
</tr>
<tr>
<td></td>
<td>25-50 mg IV q15-60 min prn</td>
</tr>
<tr>
<td></td>
<td>50-125 mg IM q3-4hr</td>
</tr>
<tr>
<td>Hydrocodone (Vicodin)</td>
<td>5-10 mg PO q4-6hr</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>5 mg PO q4-6hr</td>
</tr>
<tr>
<td>Hydromorphone (Dilaudid)</td>
<td>2 mg PO q4-6hr</td>
</tr>
<tr>
<td></td>
<td>1-2 mg IV q4-6hr</td>
</tr>
<tr>
<td></td>
<td>1-2 mg IM/SC q4-6 hr prn</td>
</tr>
<tr>
<td>Morphine</td>
<td>2-5 mg IV q10min prn</td>
</tr>
<tr>
<td></td>
<td>5-10 mg IM/SC q4hr prn</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>1-2 µg/kg IV, then 1µg/kg IV q5-10min prn</td>
</tr>
<tr>
<td></td>
<td>100 µg IM q1-2hr</td>
</tr>
</tbody>
</table>
NEUROLOGY

Clerkship director: Eric Kraus, M.D., ekraus@u.washington.edu, 206-598-0216
Web site: http://courses.washington.edu/neural/

NEUROLOGICAL EXAMINATION

A. Mental and communication status
   1. Education level
   2. Level of consciousness
      Alert Delirium Obtunded Stupor Coma
   3. Mood and psychomotor activity
   4. Orientation (time, place, person, body parts, left-right, awareness of illness)
   5. Calculation, spelling
   6. Speech function (fluency, comprehension, repetition, naming, reading, writing)
   7. Memory (immediate, short term, long term)
   8. Ability to follow complex commands
   9. Mini-mental status examination (MMSE)

B. Cranial nerve functions
   1. Olfactory (aromatic smell)
   2. Optic
      a. Acuity (Snellen card) Here are a few examples:
         acuity (near, corrected) 20/20 OU [normal vision in both eyes]
         acuity (near, uncorrected) 20/100 OD, 20/50 -2 OS [left eye, 2/6 missed on 20/50 line]
      b. Fundi (vessels, disc border, cup/disc ratio), visual fields
   3. Oculomotor, Trochlear, Abducens
      a. Pupillary reaction (light, accommodation, afferent pupillary defect), extraocular movements, nystagmus
   4. Trigeminal
      a. Muscles of mastication
      b. Sensation of face (test all 3 divisions) and cornea
      c. Sensation of mucous membranes and noxious smell
      d. Jaw jerk
   5. Facial
      a. Muscles of facial expression, palpebral fissures
      b. Taste anterior 2/3
   6. Acoustic
      a. Cochlear (finger rub, tuning fork)
      b. Vestibular (nystagmus, past pointing)
   7. Glossopharyngeal, Vagus
      a. Palate rise to phonation (say “ah”) and gag
      b. Voice and articulation
      c. Taste posterior 1/3
   8. Spinal accessory
      a. Sternocleidomastoid
      b. Upper trapezius
   9. Hypoglossal
      a. Tongue movement
b. Bulk
C. Motor function
1. Strength
   a. Direct testing
      Grades:
      | Grades | Description                                      |
      |--------|--------------------------------------------------|
      | 0      | No muscle contraction                            |
      | 1      | Trace visual or palpable movement                |
      | 2      | Movement with gravity eliminated                 |
      | 3      | Movement against gravity but not resistance      |
      | 4      | Movement against resistance but can be overcome  |
      | 5      | Normal                                           |
   b. Functional testing
      i. Walking on toes and heels
      ii. Deep knee bend
      iii. Hopping on one foot
      iv. Arm drift
2. Tone
   a. Spasticity
   b. Rigidity (lead-pipe, cogwheel)
   c. Hypotonic or flaccid
3. Bulk
D. Reflexes
1. Deep tendon Grades:
<table>
<thead>
<tr>
<th>Grades</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No response</td>
</tr>
<tr>
<td>Tr</td>
<td>Reinforcement required</td>
</tr>
<tr>
<td>1</td>
<td>Diminished</td>
</tr>
<tr>
<td>2</td>
<td>Normal, average</td>
</tr>
<tr>
<td>3</td>
<td>Brisker than normal</td>
</tr>
<tr>
<td>4</td>
<td>Clonus</td>
</tr>
</tbody>
</table>
   Use “+ or -” to indicate smaller differences
2. Abdominal
3. Babinski – use up or down arrow to indicate
4. Hoffman
5. Frontal lobe (glabellar, snout, palpomental)
6. Other (cremasteric, bulbocavernosis)
E. Sensory function (use sensory maps and draw pictures as needed)
1. Primary (thalamic) sensation
   a. Light touch
   b. Pain
   c. Temperature
   d. Vibration
   e. Proprioception
2. Discriminative (cortical) sensation
   a. Stereognosis
   b. Graphesthesia
   c. Two-point discrimination
   d. Point localization
   e. Extinction with double simultaneous stimulation (DSS)
3. Romberg - evaluation of balance with eyes closed and feet together reflects proprioceptive and touch function in the legs and feet
F. Cerebellar function, station, and gait
   1. Balance on one foot with eyes open
   2. Walking
      a. Wide or narrow base
      b. Normal or reduced arm swing
      c. Tandem gait (heel-to-toe)
      d. Ataxia
   3. Rapid alternating movements (RAM)
   4. Finger-nose-finger (FNF) and heel-knee-shin (HKS) tests

G. Abnormal movements
   1. Tremor (note predominant component)
      a. Rest (Parkinsonian)
      b. Postural
      c. Kinetic (action)
   2. Involuntary movements (dystonia, chorea, tic)
   3. Bradykinesia

H. Meningeal and mechanical signs
   1. Neck stiffness
   2. Brudzinski’s sign
   3. Kernig’s sign
   4. Straight leg raise
   5. Pressure tenderness of bone, muscle, and nerves

I. Vascular status
   1. Auscultation of head and neck
   2. Auscultation of heart
   3. Palpate extremity vessels

MINI-MENTAL STATUS EXAMINATION (MMSE)
(See Psychiatry section for complete MMSE.)

SELECTED TOPICS IN NEUROLOGY

Stroke Syndromes
   • Anterior cerebral artery – contralateral leg weakness, grasp reflex, gegenhalten, abulia, gait disorder, perseveration, urinary incontinence.
   • Middle cerebral artery
      o Superior division: contralateral arm/face greater than leg weakness, Broca’s aphasia (left MCA stroke).
      o Inferior division: mild or transient motor/sensory deficit, Wernicke’s aphasia (left MCA stroke), neglect, sometimes visual field cut.
   • Posterior cerebral artery – contralateral visual field cut; sometimes memory loss, color anomia, alexia without agraphia, hemisensory loss, mild hemiparesis.
   • Lacunar – hypertensive lipohyalinosis; four types: (1) pure motor hemiparesis with face, arm, leg equally affected; (2) pure hemisensory loss; (3) dysarthria-clumsy hand syndrome; (4) ataxic hemiparesis with ipsilateral incoordination out of proportion to degree of weakness.

Spinal Cord Syndromes
   • Central Cord: mid- to lower cervical spine injury leading to bilateral arm greater than leg motor paresis, dysesthesias, areflexia, patchy sensory involvement.
• Brown-Sequard: hemisection of the spinal cord causing ipsilateral motor paralysis and spasticity; ipsilateral loss of tactile/vibratory sensation and proprioception; contralateral pain/temp loss; and ipsilateral loss of all sensation at the level of the lesion.

• Anterior Cord: injury to the anterior portion of the spinal cord, usually secondary to anterior spinal artery stroke; characterized by complete paralysis and hypalgesia (pain/temp) at the level of injury with preservation of posterior column sensory modalities (fine touch, vibration, proprioception) at and below lesion.

• Conus Medullaris: injury at T12/L1 leading to both upper and lower motor neuron deficits (muscle atrophy, weakness, spasticity, neurogenic bladder) with variable sensory loss.

• Cauda Equina: injury to the nerve roots inferior to the conus medullaris causing weak or flaccid lower extremities with partially preserved sensation; absent knee/ankle jerks; asymmetric saddle anesthesia; and loss of bowel/bladder/sexual function.

Mnemonics:

• Altered Mental Status: TIPS AEIOU
  - Trauma, Temperature
  - Infection
  - Psychiatric
  - Space Occupying, Stroke, Subarachnoid Hemorrhage, Shock
  - Alcohol, other drugs, Ammonia
  - Endocrine, Electrolytes
  - Insulin
  - Oxygen (lack of), Opiates
  - Uremia

• Peripheral polyneuropathy: DANG THERAPIST
  - Diabetes
  - Alcohol
  - Nutritional (vit B12, B1, B6, E)
  - Guillain-Barre
  - Toxic (lead, arsenic, other metals, meds)
  - HEReditary (Charcot-Marie-Tooth)
  - Recurrent (chronic inflammatory demyelinating
    Polyradiculoneuropathy - CIDP)
  - Amyloid
  - Porphyria
  - Infectious (leprosy, HIV, Lyme, diphtheria, mononucleosis)
  - Systemic (uremia, hypothyroidism, lupus, Sjogren’s, Wegener’s)
  - Tumors (paraneoplastic, CIDP assoc with myeloma)
# MOTOR CORD LEVELS

<table>
<thead>
<tr>
<th>Level</th>
<th>Motor Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>C4</td>
<td>Spontaneous breathing</td>
</tr>
<tr>
<td>C5</td>
<td>Shoulder shrug / deltoid</td>
</tr>
<tr>
<td>C6</td>
<td>Biceps reflex / elbow flexion / wrist extension</td>
</tr>
<tr>
<td>C7</td>
<td>Triceps reflex / elbow extension / wrist flexion</td>
</tr>
<tr>
<td>C8/T1</td>
<td>Finger abduction / opposition of thumb</td>
</tr>
<tr>
<td>T12</td>
<td>Cremasteric reflex</td>
</tr>
<tr>
<td>L1/L2</td>
<td>Hip flexion</td>
</tr>
<tr>
<td>L2/L3/L4</td>
<td>Hip adduction / quadriceps</td>
</tr>
<tr>
<td>L4</td>
<td>Patellar reflex</td>
</tr>
<tr>
<td>L5</td>
<td>Great toe dorsiflexion</td>
</tr>
<tr>
<td>S1/S2</td>
<td>Ankle jerk reflex / foot plantar flexion</td>
</tr>
<tr>
<td>S2-S4</td>
<td>Anal wink / rectal tone</td>
</tr>
</tbody>
</table>

Do you like my dermatomes?
WARD TIPS

1. There are three courses that fulfill this requirement: rehabilitation, geriatrics, and palliative care

2. This section of the manual addresses the rehab track. Please review your general medicine to help with geriatrics. The palliative care track will give you a chance to work on your interpersonal skills and develop sensitivity to the issues patients face at the end of life; this is obviously highly individualized, and does not lend itself to a list of factoids that could be included in this manual.

USEFUL ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>AAROM</td>
<td>Active Assistive Range of Motion</td>
</tr>
<tr>
<td>ADL</td>
<td>Activities of Daily Living</td>
</tr>
<tr>
<td>AFO</td>
<td>Ankle Foot Orthosis</td>
</tr>
<tr>
<td>AKA</td>
<td>Above Knee Amputation</td>
</tr>
<tr>
<td>Amb</td>
<td>Ambulation</td>
</tr>
<tr>
<td>AROM</td>
<td>Active Range of Motion</td>
</tr>
<tr>
<td>BKA</td>
<td>Below Knee Amputation</td>
</tr>
<tr>
<td>CGA</td>
<td>Contact Guard Assist</td>
</tr>
<tr>
<td>CPM</td>
<td>Continuous Passive Motion</td>
</tr>
<tr>
<td>DF</td>
<td>Dorsiflexion</td>
</tr>
<tr>
<td>EOB</td>
<td>Edge of Bed</td>
</tr>
<tr>
<td>E Stim</td>
<td>Electrical Stimulation</td>
</tr>
<tr>
<td>FIM score</td>
<td>Functional Independence Level</td>
</tr>
<tr>
<td>FWW</td>
<td>Forward Wheel Walker</td>
</tr>
<tr>
<td>HEP</td>
<td>Home Exercise Program</td>
</tr>
<tr>
<td>I</td>
<td>Independent</td>
</tr>
<tr>
<td>IADLS</td>
<td>Instrumental Activities of Daily Living</td>
</tr>
<tr>
<td>MMT</td>
<td>Manual Muscle Test</td>
</tr>
<tr>
<td>NWB</td>
<td>Non-Weight Bearing</td>
</tr>
<tr>
<td>PF</td>
<td>Plantar Flexion</td>
</tr>
<tr>
<td>PROM</td>
<td>Passive Range of Motion</td>
</tr>
<tr>
<td>SBA</td>
<td>Stand By Assist</td>
</tr>
<tr>
<td>SLP</td>
<td>Speech Language Pathology</td>
</tr>
<tr>
<td>TLSO</td>
<td>Thoracic Lumbar Sacral Orthosis</td>
</tr>
<tr>
<td>WFL</td>
<td>Within Functional Limit</td>
</tr>
</tbody>
</table>

SAMPLE PROGRESS NOTE

24 hour course – events overnight from nursing/therapists
Subjective – what the patient is concerned about
Objective – vitals and exam
Laboratory
Imaging
Assessment/Plan by problem:
  Medical issues
Spinal stability/orthoses – plan for repeat films? How long should C-collar/TLSO be worn?
Impaired mobility – PT progress update
Impaired self care – OT progress update
Impaired cognition – SLP progress update (neuropsychological testing?)
Impaired swallowing – same
Adjustment to disability – Rehab Psychology progress update
Disposition

Physical Therapy
Includes ROM, stretching, strengthening, coordination/balance and endurance exercises, mobility training (bed mobility, transfers, gait with assistive devices, wheelchair mobility), and home safety evaluation.

Occupational Therapy
Includes ROM (especially hands), stretching, strengthening (pinch and grip measurements to follow handstrength progress), splinting needs, functional tasks (ADLs – eating, continence, personal hygiene, toilet/shower transfers, dressing; IADLs - money management, kitchen evaluation, etc).

Speech Language Pathology
Encompasses three domains:
  o Cognitive – screening exam, memory strategies.
  o Swallow – bedside swallow evals, Barium swallow studies, diet recommendations and aspiration precautions.
  o Communication – language.

American Spinal Injury Association (ASIA) Classification of Spinal Cord Injury Severity
A: complete injury; no sensory/motor in sacral segments S4-S5
B: incomplete; sensory but no motor below neurologic level of lesion
C: incomplete; motor function preserved below level, but more than half of muscles have
   Strength grade less than 3
D: incomplete; motor function preserved below level, more than half of muscles have
   Strength grade 3 or more
E: normal
REFERENCES & HELPFUL RESOURCES
1. Habif, Skin Disease: Diagnosis and Treatment; 2001
3. The Electronic Textbook of Dermatology (www.telemedicine.org/stamfor1.htm)

BASIC HISTORY FOR SKIN LESIONS
How long has the lesion been present?
Has the lesion changed or spread?
How does the lesion bother you?
Previous treatments and results
Associated systemic symptoms (esp. rheumatic sx, myalgias, arthralgias, etc)
Relationship to heat, cold, occupation, sun exposure, medications, hobbies
Family history (esp. of melanoma, atopy, psoriasis)

PHYSICAL EXAM OF SKIN
Examine all skin areas, mucous membranes, nails, and scalp
Describe type of lesions (see descriptive terms below); shape, color, texture
Describe margins (well-demarcated, sharp, irregular, ill-defined)
Describe number & arrangement of lesions
Describe distribution or location
Measure and record size (mm or cm)

TERMINOLOGY
Primary Lesions
Macule
Circumscribed, flat discoloration of skin <5mm diameter
Patch
Flat discoloration >5mm in diameter
Papule
Elevated, solid lesion up to 5mm diameter
Plaque
Elevated, flat-topped lesion >5mm diameter
Nodule
Elevated, solid lesion >5mm diameter
Wheal
Firm, edematous, erythematous plaque due to fluid in dermis
Vesicle
Circumscribed, elevated, fluid-filled lesion <5mm diameter
Bulla
Fluid-filled lesions >5mm in diameter
Pustule
Circumscribed, elevated lesion containing purulent exudate

Secondary Lesions
Scales
White, dry flakes caused by shedding of stratum corneum
Crusts
Formed by dried serous and serosanguineous exudate
Erosion
Circumscribed depression caused by focal loss of epidermis
Ulcer
Focal loss of epidermis and dermis, heals with scarring
Excoriation
Erosion caused by scratching (usu. linear)
Fissure
Linear loss of epidermis with sharply-defined vertical walls
Atrophy
Depression in skin resulting from thinning of epidermis or dermis
Lichenification
Thickened epidermis with accentuated skin lines (scratching)
Scar
Abnormal formation of connective tissue; dermal damage
Lesion Arrangement
Annular  Arranged in circle or ring
Arcuate  Arranged in incomplete rings, arc-like pattern
Linear  Arranged in straight lines
Reticulate  Arranged in net-like pattern
Morbilliform  Like measles with discrete or confluent lesions

SPF = Minimal erythema dose with a sunscreen
Minimal erythema dose without a sunscreen
A sunscreen with SPF 10 will allow a person who normally burns with 10 minutes of sun exposure to have up to 100 minutes of sun exposure without burning.

USEFUL TECHNIQUES
KOH exam: use to look for fungal infection. Remove scales from surface of lesions with #15 scalpel blade and place on slide. Add KOH (15-20%), place cover slip, and heat gently. Examine under microscope for hyphae and spores. Keep looking, sometimes it takes awhile.

Scraping for scabies: Scrape suspected lesion with #15 scalpel and place on slide. Add drop of mineral oil; place cover slip. Look for feces, ova, and mites.

Liquid Nitrogen Cryotherapy: Apply to lesions and 1-2mm of surrounding skin. Preferred technique is rapid freezing and slow thawing. Thaw time of 15-20 seconds. Use for warts, actinic keratoses, seborrheic keratoses.

SELECTED TOPICS IN DERMATOLOGY
Malignant and Pre-malignant lesions
Basal Cell Carcinoma
Description  Most common skin malignancy. Occurs on sun-exposed skin – face, scalp, ears, neck. Metastases are rare.
Types  
Nodular: most common, pearl white, dome-shaped papule with overlying telangiectasias, rolled translucent border. Friable tissue, can bleed and ulcerate.
Pigmented: nodular type with melanin pigment
Superficial: least aggressive form, seen on trunk and ext
Micronodular: resembles nodular with microscopic tumor cells extending beyond the clinical margins
Morpheaform (sclerosing): least common, looks like scar tissue (waxy, pale)
Rx  Shave biopsy for diagnosis, sometimes punch biopsy
Electrodesiccation and curettage for smaller lesions
Excision, Mohs surgery

Squamous Cell Carcinoma
Description  2nd most common skin malignancy. Occurs on sun-exposed areas – head, neck, hands. Other risk factors include radiation exposure, chronic inflammation/infection, burn/scar tissue (Marjolin’s ulcer), HPV infection Lesion has poorly defined margin with adherent yellowish cutaneous horn
Ddx  AK, keratoacanthoma, Bowen’s disease
Rx  Excisional biopsy and F/U as needed
If SCC, do wide local excision, can do Moh’s surgery
**Actinic Keratosis**

**Description** Poorly defined lesions with fine white scale. Feels like a rough spot, occurs on sun-exposed skin – face, scalp, ears, neck, upper ext. Actinic chelitis refers to actinic lesions at the lips. Can progress to SCC

**Rx**
Liquid nitrogen cryotherapy
Topical 5-FU, F/U in 6-12 months

**Bowen’s disease**

**Description** SCC-in-situ. Raised, red plaque with dry scale seen in sun-exposed areas, penis. Less common than AK, full-thickness lesion.

**Ddx** Psoriasis, eczema, SK, superficial BCC

**Rx**
Biopsy for diagnosis
Excision with confirmed margins. Can also do electrodesiccation and curettage, cryosurgery, topical 5-FU. Do regular follow-up

**Melanoma**

**Description** 3rd most common skin malignancy, high mortality rate. Risk factors include atypical nevi, family hx, fair skin, h/o sunburn, congenital nevi. Look for ABCDEs (Asymmetry, Border irregularity, Color irregularity, Diameter ≥6 mm, and Evolution or changes in lesion). Types include superficial spreading, nodular, lentigo maligna, acral lentiginous, and amelanotic.

**Types** Superficial spreading, nodular, lentigo maligna, acral lentiginous, amelanotic

**Ddx** Benign nevi, atypical nevi, SK, solar lentigo, pigmented BCC, angiookeratoma, hemangioma

**Rx**
Do complete excisional biopsy for diagnosis (no shave bx)
Palpate regional nodes
If melanoma -> wide local excision with appropriate margins. Sentinel node mapping, biopsy, and dissection when appropriate

**Common Benign Skin Tumors**

**Seborrheic Keratosis**

**Description** Well-circumscribed, tan to black, rough surface, appears “stuck on” skin, waxy, and may have retained keratin cysts. Located on trunk, extremities, face, and scalp. Increasing incidence with age.

**Ddx** Melanoma. (melanoma usually has more color variation and smoother surface)

**Rx**
For cosmesis, to R/O melanoma or removal of irritated skin. Use liquid nitrogen cryotherapy, curettage, or shave excision.

**Acrochordon (skin tag)**

**Description** Hyperplastic epidermis, pedunculated, flesh-colored to brown, increasing incidence with age. Seen at neck, axilla, inguinal regions.

**Ddx** Wart, nevus

**Rx**
For cosmesis or irritation. Use electrocautery or scissor excision at base

**Dermatofibroma**

**Description** Firm, elevated papules/plaques or nodules made of dermal tissue. Can be brown, purple, yellow, red or pink. Usually on anterior surface of legs.

**Dx**
Fitzpatrick’s sign – dimpling or retraction of lesion beneath skin with lateral compression.
Rx For cosmesis or histologic diagnosis. Use excisional biopsy or cryo.

Keratoacanthoma
Description Papular lesions that enlarge rapidly, can have central umbilication. Lesion involutes after 4-6 mo, leaving hypopigmented scar. Located on sun-exposed skin – face, upper ext, lower ext.
Ddx SCC, wart, molluscum contagiosum
Rx Usually excisional or shave biopsy, because keratoacanthoma can be difficult to differentiate from SCC. Can also do electrodesiccation.

Epidermoid Cyst (sebaceous cyst)
Description Firm, SubQ, keratin-filled cyst seen on face, posterior neck, and trunk. Cysts can rupture, leading to acute inflammation
Rx Remove to prevent rupture and scarring. Incise cyst wall and remove contents of lesion.

Sebaceous Hyperplasia
Description Soft, yellow, dome-shaped papules, can have central umbilication. Seen in middle-aged and elderly persons on forehead, cheeks, nose.
Ddx BCC
Rx Laser therapy, electrodesiccation, topical bichloracetic acid, cryosurgery

Common Derm Problems
Acne
Description Pustular/papular eruptions most common on face, chest, and back.
Rx Topical treatments include salicylic acid, benzoyl peroxide, tretinoin, adapalene. Topical antibiotics include erythromycin and clindamycin – use for infected pores. Oral antibiotics (erythromycin, tetracycline) can also be used. Oral isotretinoin (Accutane) is used for severe cystic acne. Use caution when prescribing for women.

Rosacea
Description See in patients >30 yo. Papular/pustular eruptions combined with telangiectasia, flushing at forehead, nose, cheeks and surrounding eyes. Chronic inflammation of nose can lead to rhinophyma.
Rx Avoid hot, spicy foods and sunlight. Topical metronidazole or sulfacetamine. Oral tetracycline, erythromycin, or minocycline for pustules.

Psoriasis
Description Scaly plaques at elbows, knees, scalp. Can have nail and joint involvement. Psoriatic arthritis occurs in 5-8% of people with psoriasis
Rx Topicals: calcipotriol, steroids, anthralin, tazarotene. Phototherapy includes UVB (UVA sometimes helps), PUVA, and narrow-band UVB. Systemic therapies include methotrexate, cyclosporine, acitretin, biologics.

Seborrheic Dermatitis
Description Inflammatory dermatitis with yellow, greasy, adherent scale at scalp, eyebrows, nasolabial folds, external ear
Rx Ketoconazole cream, antidandruff shampoo (coal tar, selenium, zinc pyrithione),
topical steroids. Also consider ketoconazole shampoo. Can give short course of oral ketoconazole or fluconazole. UVB phototherapy and oral corticosteroids are also effective.

**Warts**

**Description** Benign epithelial proliferations caused by HPV infection. Transmitted by skin-to-skin contact. Flesh-colored papules with black dots (thrombosed capillaries) in center. Often on fingers, plantar surface, knees, elbows.

**Rx** Topical salicylic acid, liquid nitrogen, cryotherapy. No therapy proven to be more effective than others. Can regress spontaneously.

**Herpes Zoster**

**Description** Reactivation of varicella virus leads to preeruptive pain, hyperesthesia, followed by vesicles in dermatomal pattern.

**Rx** Most effective when started within 48 hrs. Acyclovir 800 mg PO 5x per day for 7-10 days. Valacyclovir 1000 mg TID or famciclovir 500 mg TID.

**Candidiasis**

**Description** Pustules and red glistening skin with cigarette-paper scale. Seen at mucous membranes, intertriginous areas

**Dx** KOH exam

**Rx** Topical ketoconazole, miconazole, clotrimazole apply BID x 10 days.

**Fungus**

**Description** Cutaneous infection – red, slightly elevated, scaly border. Nail infection – see distal subungual onychomycosis

**Dx** KOH exam, can do fungal culture for nail infection

**Rx** Topical antifungals for cutaneous infection – butenafine, terbinafine, clotrimazole, miconazole. Topical therapy is not effective for nail infections. Systemic therapy is 50-80% effective. Terbinafine or Intraconazole 200mg/day x 6wks for fingernails and x12 wks for toenails. Ciclopriox (Penlac) topical solution can be used x 1 yr.

**Scabies**

**Description** Patient with present with intense itching, usually worse at night. Look for linear or curvilinear burrows, slightly elevated, at the web spaces of fingers/toes, wrists, inguinal region, or abdomen. Infection is contagious and spreads among household members.

**Dx** Apply mineral oil to burrow. Scrape with #15 scalpel blade and put on slide. Apply cover slip and look for mites, feces

**Rx** Permethrin, apply from neck down. Repeat in 1 wk
Wash bedding and clothing in hot water, can use anti-itch lotion
Can use Ivermectin 200 µg/kg PO x 1 dose
Cutaneous manifestations of systemic disease

Acanthosis Nigricans: Thickened, hyperpigmented skin seen at posterior neck, axilla, groin. Marker of insulin resistance. Associated with obesity and diabetes mellitus.

Neurofibromatosis: Café au Lait macules, dermal and SubQ neurofibromas. Neurofibromatosis is an autosomal dominant disorder affecting ectodermal tissues.


Pyoderma gangrenosum: Rapidly enlarging exudative ulceration of skin. Associated with IBD, rheumatoid arthritis, Chronic hepatitis, myelodysplasia, polycythemia vera, AML, CML, myeloma, Waldenstrom’s macroglobulinemia, myelofibrosis

Malignancies (paraneoplastic syndromes)

- Carcinoid syndrome – carcinoid tumors in small bowel, lung
- Dermatomyositis – assoc with breast, lung, ovary, GI malignancies
- Sudden eruption of multiple SKs – assoc with adenocarcinoma
- Pemphigus – assoc with hematologic malignancy and CLL
- Pruritis – assoc with many malignancies, esp. Hodgkin’s disease

Differential diagnoses by type of lesion

<table>
<thead>
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<th>Papulosquamous diseases</th>
<th>Purpuric lesions</th>
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<td>Psoriasis</td>
<td>Bruise</td>
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<tr>
<td>Lichen Planus</td>
<td>Vasculitis</td>
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<tr>
<td>Pityriasis rosea</td>
<td>Meningococcemia</td>
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<tr>
<td>Secondary syphilis</td>
<td></td>
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<tr>
<td>Lichen simplex chronicus</td>
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</tbody>
</table>

| Atopic eczema                                                                           | Pustular lesions           |
| Seborrheic dermatitis                                                                  | Bacterial folliculitis     |
| Stasis dermatitis                                                                      | Acne                      |
| Nummular dermatitis                                                                    | Furunculosis              |
| Tinea                                                                                  | Candidiasis               |
| Candidiasis                                                                            | Herpes simplex            |
| Xerosis                                                                               | Herpes zoster             |
| Ichthyosis                                                                             |                           |
| Contact dermatitis                                                                     |                           |
| Dyshidrotic eczema                                                                     |                           |
| Neurodermatitis                                                                        |                           |
| Mycosis fungoides                                                                      |                           |

| Vesiculobullous diseases                                                               |                            |
| Dermatitis herpetiformis                                                               | Bulous impetigo            |
| Pemphigus                                                                              | Herpes simplex             |
| Pemphigoid                                                                            | Herpes zoster              |
| Acute contact dermatitis                                                               | Erythema multiforme        |

6
<table>
<thead>
<tr>
<th>Differential diagnoses by region</th>
<th>Back</th>
<th>Buttocks</th>
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<tbody>
<tr>
<td>Chest</td>
<td>Acne</td>
<td>Acne</td>
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<tr>
<td>Acne</td>
<td>Acne</td>
<td>Cutan. T-cell lymphoma</td>
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<tr>
<td>Actinic keratosis</td>
<td>Becker’s nevus</td>
<td>Furunculosis</td>
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<tr>
<td>Darier’s disease</td>
<td>Cutan. T-cell lymphoma</td>
<td>Herpes Simplex</td>
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<td>Eruptive syringoma</td>
<td>Dermatographism</td>
<td>Hidradenitis suppurativa</td>
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<td>Keloid</td>
<td>Keloids</td>
<td>Psoriasis</td>
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<tr>
<td>Seborrheic dermatitis</td>
<td>Melanoma</td>
<td>Tinea</td>
</tr>
<tr>
<td>Tinea Versicolor</td>
<td>Tinea Versicolor</td>
<td>Anus</td>
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<tr>
<td>Grover’s disease</td>
<td>Seborrheic keratosis</td>
<td>Hidradenitis suppurativa</td>
</tr>
<tr>
<td>Elbow/Knee</td>
<td>Areolae</td>
<td>Psoriasis</td>
</tr>
<tr>
<td>Dermatitis herpetiformis</td>
<td>Eczema</td>
<td>Vitiligo</td>
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<td>Lichen simplex chronicus</td>
<td>Paget’s disease</td>
<td>Warts</td>
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<tr>
<td>Psoriasis</td>
<td>Seborrheic keratosis</td>
<td>Strep cellulitis</td>
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<td>Axilla</td>
<td>Actinic keratosis</td>
<td>Groin</td>
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<td>Acanthosis nigracans</td>
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<td>Squamous cell carcinoma</td>
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<td>Candidiasis</td>
<td>Eczema</td>
<td>Erythrasma</td>
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<tr>
<td>Contact dermatitis</td>
<td>Epidermal cyst</td>
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<td>Erythrasma</td>
<td>Keloid</td>
<td>Intertrigo</td>
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<td>Furunculosis</td>
<td>Lupus erythematosus</td>
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<td>Hailey-Hailey disease</td>
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<td>Molluscum contagiosum</td>
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<td>Hidradenitis suppurativa</td>
<td>Ramsey-Hunt syndrome</td>
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<td>Impetigo</td>
<td>Seborrheic dermatitis</td>
<td>Seborrheic keratosis</td>
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<tr>
<td>Lice</td>
<td>Venous lake</td>
<td>Striae</td>
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<tr>
<td>Trichomycosis axillaries</td>
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<td>Tinea</td>
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<tr>
<td>Sole of Foot</td>
<td>Palm</td>
<td>Dorsum of Hand</td>
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<tr>
<td>Cutaneous larva migrans</td>
<td>Callus/corn</td>
<td>Actinic keratosis</td>
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<tr>
<td>Dyshidrotic eczema</td>
<td>Contact dermatitis</td>
<td>Atopic dermatitis</td>
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<td>Erythema multiforme</td>
<td>Cowden’s disease</td>
<td>Contact dermatitis</td>
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<tr>
<td>Hand, foot, mouth dz</td>
<td>Dyshidrotic eczema</td>
<td>Cowden’s disease</td>
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<td>Hyperkeratosis</td>
<td>Erythema multiforme</td>
<td>Erythema multiforme</td>
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<tr>
<td>Melanoma</td>
<td>Hand, foot, mouth dz</td>
<td>Granuloma annulare</td>
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<tr>
<td>Nevi</td>
<td>Keratolysis exfoliativa</td>
<td>Keratoacanthoma</td>
</tr>
<tr>
<td>Pitted keratolysis</td>
<td>Psoriasis</td>
<td>Lentigo</td>
</tr>
<tr>
<td>Psoriasis, pustular</td>
<td>Pyogenic granuloma</td>
<td>Paronychia</td>
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<tr>
<td>Scabies</td>
<td>Scabies</td>
<td>Psoriasis</td>
</tr>
<tr>
<td>Syphilis, secondary</td>
<td>Syphilis, secondary</td>
<td>Scabies</td>
</tr>
<tr>
<td>Tinea</td>
<td>Tinea</td>
<td>Tinea</td>
</tr>
<tr>
<td>Wart</td>
<td>Vesicular ID reaction</td>
<td>Vesicular ID reaction</td>
</tr>
</tbody>
</table>
GENERAL POINTS FOR ALL STUDIES
1. Check patient name and date
2. Type of study (position, contrast)
3. Old films for comparison?

HOW TO READ A CXR
1. PA or AP, supine or upright
2. Pt rotated? Check for vertebral and clavicle symmetry.
3. Lung volumes
4. Tube & line placement
   - ETT 3-5 cm above carina
   - NGT in stomach
   - FT in stomach/duodenum
   - Central line in SVC/R atrium
   - Swan in PA
5. Pneumothorax: check apices on upright film, deep sulcus sign at bases
6. Pleural effusion, pleural thickening
7. Mediastinum: normal contour, wide
8. Heart: normal size, cardiomegaly
9. Lung parenchyma: masses, opacites, look for silhouette sign
10. Soft tissues: foreign bodies, SQ air, breast shadows
11. Bones: fractures, osteopenia, abnormalities

Silhouette Sign = obscuring of normal borders on radiograph caused by intrathoracic lesion.
Obscured R heart border = R middle lobe
Obscured L heart border = Lingula
Obscured diaphragm = Lower lobe

ABDOMINAL PLAIN FILMS
Indications: appendicitis, perforation, ischemia, abscess, obstruction, urinary calculi, severe pain
Order supine & upright or supine & left lateral decubitus
Evaluation:
1. Soft tissues (fat lines)
   - Mucosal swelling, luminal distention
2. Gas patterns: intraluminal, extraluminal
   - Intrapitoneal gas
   - Portal venous gas
   - Pneumobilia
3. Calcifications: enterolith, gallstones, kidney stones
4. Fluid collections: intraluminal, extraluminal

CT BASICS
Acute blood is bright on non-contrast study
Epidural hematoma = lens shaped collection (biconvex), confined by suture lines. Usually hyperdense fluid collection between skull and dura.
Subdural hematoma = Crescentic shaped fluid collection, can cross suture lines. Acute is usually hyperdense, subacute and chronic will be isodense or hypodense. Due to tearing of bridging veins.
SAH = hyperdense fluid in CSF

**MRI BASICS**

<table>
<thead>
<tr>
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<th>T1</th>
<th>T2</th>
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<tbody>
<tr>
<td>Water</td>
<td>Bright</td>
<td>Dark</td>
</tr>
<tr>
<td>Fat</td>
<td>Bright</td>
<td>Less bright</td>
</tr>
<tr>
<td>Calcium</td>
<td>Dark</td>
<td>Dark</td>
</tr>
<tr>
<td>Gas / Air</td>
<td>Dark</td>
<td>Dark</td>
</tr>
<tr>
<td>Flowing blood</td>
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<td>Dark</td>
</tr>
<tr>
<td>Gadolinium</td>
<td>Bright</td>
<td>Bright</td>
</tr>
<tr>
<td>Bone</td>
<td>Dark</td>
<td>Dark</td>
</tr>
</tbody>
</table>

**ULTRASOUND BASICS**

Hyperechoic = white
Hypoechoic = black
Bone, air, fat = white
Fluid = black

**Risks for Adverse Reactions to Contrast Material**

- Renal insufficiency
- Previous reaction to contrast material
- Asthma
- Concomitant use of nephrotoxic agents: aminoglycosides, NSAIDS
- Advanced Age
- Multiple medical problems
- Food or medication allergies

**Types of Contrast Material**

**High Osmolality**

- Ionic: Hypaque, Conray

**Low Osmolality**

- Ionic: Hexabrix
- Nonionic: Omniscan, ProHance, Visipaque, Isovue, Ultravist, Optiray

High Osm. Contrast agents are more likely to cause adverse reactions. Low osm. contrast agents are more expensive.

**How to prevent Renal Insufficiency in patients given Contrast**

1. D/C nephrotoxic meds if possible before exam
2. Use smallest amount of contrast possible
3. Allow several days between contrast studies
4. Hydrate!
If patient can take orals, then 500cc pre-study and 2-3L post-study over 24 hours. For IV, give 100mL/hr for several hours before study, then continue for 24 hours.
MAMMOGRAPHY
Screening Mammography
Bilateral cranio-caudal and medio-lateral-oblique views
85-90% Sensitivity
5-10% call back rate

Diagnostic Mammography
Spot magnification views targeted at diagnostic problem

CXR FINDINGS
Pleural Effusion
300-500cc fluid needed to blunt lateral costophrenic angles on PA CXR
150cc fluid needed to blunt posterior costophrenic angle on lateral CXR

Diagnosis
Get bilateral decubitus films or ultrasound

Causes
Bilateral
CHF, volume overload, collagen vascular disease, drug reaction, pulmonary embolus, viral pleuritis, bilateral pneumonia
Unilateral
TB, cancer, pancreatitis, aortic dissection/transection, pneumonia, central obstructing mass, constrictive pericarditis

Pneumothorax
Differentiate from skin fold!!
See PTX apically on upright film. Look for “deep sulcus” sign on supine film.

Diagnosis
Get upright CXR, maybe inspiratory-expiratory views

Pleural Calcifications
Causes
Asbestos plaques, old empyema, old hemothorax
Diagnosis
Get oblique films or CT

Average Radiation Exposure from Common Radiology Studies

<table>
<thead>
<tr>
<th>Film or Study</th>
<th>MilliRoentgens / film</th>
</tr>
</thead>
<tbody>
<tr>
<td>PA CXR</td>
<td>30</td>
</tr>
<tr>
<td>Lateral CXR</td>
<td>70</td>
</tr>
<tr>
<td>KUB</td>
<td>650</td>
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<tr>
<td>Body CT</td>
<td>2500</td>
</tr>
<tr>
<td>Head CT</td>
<td>5000</td>
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<tr>
<td>Mammo</td>
<td>600 per view</td>
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<tr>
<td>AP pelvis</td>
<td>520</td>
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<tr>
<td>Wrist PA, Obl</td>
<td>10</td>
</tr>
<tr>
<td>Elbow AP, lat, ext rot</td>
<td>50</td>
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<tr>
<td>AP shoulder</td>
<td>180</td>
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<tr>
<td>Lateral cervical spine</td>
<td>150</td>
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<tr>
<td>Fluoroscopy</td>
<td>800-1000 per min</td>
</tr>
</tbody>
</table>

ORDERING RADIOLOGIC TESTS
Always include a brief patient ID and the sx that have led to ordering the exam. Don't simply write “rule out…” History will help the radiologist interpret the exam, and s/he may even suggest changing the study to something more appropriate.

<table>
<thead>
<tr>
<th>Patient Condition</th>
<th>Test to Order</th>
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<tr>
<td>Skull Fracture</td>
<td>CT scan</td>
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<tr>
<td>Head Trauma</td>
<td>Head CT w/o contrast</td>
</tr>
<tr>
<td>Intracranial hemorrhage</td>
<td>Head CT w/o contrast</td>
</tr>
<tr>
<td>Acute Stroke</td>
<td>Head CT w/o contrast</td>
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<tr>
<td>CNS tumor</td>
<td>CT or MRI w/ contrast</td>
</tr>
<tr>
<td>Multiple Sclerosis</td>
<td>MRI w/ contrast</td>
</tr>
<tr>
<td>Abdominal Trauma</td>
<td>CT or DPL</td>
</tr>
<tr>
<td>AAA</td>
<td>CT w/ contrast</td>
</tr>
<tr>
<td>Abdominal Abscess</td>
<td>CT w/ contrast</td>
</tr>
<tr>
<td>Cholelithiasis, cholecystitis</td>
<td>Ultrasound</td>
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<tr>
<td>Choledocolithiasis</td>
<td>Ultrasound, ERCP</td>
</tr>
<tr>
<td>Bowel Obstruction</td>
<td>AXR, supine and upright</td>
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<tr>
<td>Appendicitis</td>
<td>AXR, ultrasound, or CT</td>
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<tr>
<td>Nephrolithiasis</td>
<td>AXR, CT KUB</td>
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<tr>
<td>Diverticulitis (non-acute)</td>
<td>Barium Enema, CT</td>
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<tr>
<td>Pregnancy or Gyn pathology</td>
<td>Ultrasound</td>
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<td>Upper GI Bleed</td>
<td>UGI series or endoscopy</td>
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<tr>
<td>Lower GI Bleed</td>
<td>Barium Enema or colonoscopy</td>
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<td>Pyloric Stenosis</td>
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<td>Meckel’s Diverticulum</td>
<td>Nuclear Med Scan (Meckel’s scan)</td>
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<td>Bone Metastases</td>
<td>Bone Scan</td>
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<td>Pneumonia</td>
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<td>Hemoptysis</td>
<td>CXR</td>
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<td>PE</td>
<td>V/Q Scan, CT w/ contrast, PAgram</td>
</tr>
<tr>
<td>Aortic aneurysm or dissection</td>
<td>CT w/ contrast</td>
</tr>
<tr>
<td>Aortic tear</td>
<td>Angiogram</td>
</tr>
<tr>
<td>Carotid Stenosis</td>
<td>Ultrasound with Duplex</td>
</tr>
<tr>
<td>52yo M with worst HA of his life</td>
<td>CT w/o contrast</td>
</tr>
<tr>
<td>31yo F w/ HIV, presents with confusion, left arm weakness</td>
<td>CT w/ contrast (R/O lymphoma, toxo, PML)</td>
</tr>
<tr>
<td>41yo M w/ UGI bleed, falling hematocrit and peritoneal signs</td>
<td>Upright CXR, Upright AXR, supine AXR (R/O perforation)</td>
</tr>
<tr>
<td>78yo F w/ abd pain, distention, vomiting x 3d</td>
<td>Supine and Upright AXR (Obstruction)</td>
</tr>
<tr>
<td>28yo F IVDU, 2 day H/O bilateral LE weakness. L-spine radiographs are normal.</td>
<td>MRI of spine (osteomyelitis, abscess)</td>
</tr>
<tr>
<td>67yo M smoker with 4 episodes of pneumonia in past 5 months</td>
<td>CT w/ contrast (malignancy)</td>
</tr>
<tr>
<td>67yo M with hypercalcemia, mental status changes, and abnormal SPEP</td>
<td>Skeletal survey (multiple myeloma)</td>
</tr>
</tbody>
</table>
As students are now required to obtain additional credits in surgery, an attempt has been made here to VERY BRIEFLY present a few high-yield facts pertinent to several subspecialty rotations. If you would like further information, please contact the program coordinator for the rotation you are interested in:

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**OPHTHALMOLOGY**

**References & Helpful Resources**
- Basic Ophthalmology for Medical Students and Primary Care Residents (Cynthia A. Bradford, M.D. Executive Editor, 8th edition, 2004)

**Eye Exam**
1. Visual acuity
2. Pupillary reactions
3. Extraocular movements (remember LR-6 SO-4, the rest CN 3)
4. Direct ophthalmoscopy – check red reflex, view fundus, in particular vessels, fovea/macula and optic disk
5. Slit lamp exam
6. Optional
   - Tonometry
   - Anterior chamber depth assessment
   - Confrontation field testing
   - Color vision testing
   - Fluorescein staining
   - Eversion of the upper lid

**Acute Visual Loss**

**Differential Diagnosis:**
1. Media opacities
2. Angle closure glaucoma (EMERGENCY!! Watch for painful red eye!)
3. Hyphema (blood in the anterior chamber following trauma)
4. Vitreous hemorrhage
5. Retinal detachment (watch for the symptom of flashing lights, inc. floaters, loss of peripheral vision; distinguish from ocular migraine)
6. Central retinal artery occlusion (EMERGENCY!! Watch for painless sudden loss of vision with cherry red spot on fundoscopic exam!)
7. Optic neuritis (may occur in multiple sclerosis)
8. Head trauma
9. Ischemic optic neuropathy
10. Giant cell arteritis (watch for ↑ESR, headaches, jaw claudication)
Gradual Visual Loss
Differential Diagnosis:
1. Cataract
2. Chronic papilledema
3. Open angle glaucoma (glaucomatous cupping is optic cup to disc ratio over 0.5, and intraocular pressure over 21)
4. Macular degeneration
5. Diabetic/hypertensive retinopathy
6. Sarcoidosis
7. Sickle cell anemia
8. CMV retinitis (occurs in AIDS and immunocompromised patients)
9. Syphilitic chorioretinitis

Red Eye
Differential Diagnosis:
1. Acute angle closure glaucoma
2. Iritis or iridocyclitis
3. Herpes simplex keratitis
4. Conjunctivitis
5. Episcleritis/scleritis
6. Adnexal disease
7. Sunconjunctival hemorrhage (not an emergency)
8. Pterygium
9. Keratoconjunctivitis sicca (dry eyes)
10. Abrasions/foreign bodies

Myopia: refractive error leading to loss of far vision
Hyperopia: refractive error leading to loss of near vision
Astigmatism: refractive error caused by an irregular curvature of the cornea leading to inability to focus
Presbyopia: loss of vision that occurs with aging
Amblyopia: disruption of normal development of vision due to strabismic v. refractory form of deprivation or occlusion.

Strabismus:
Eso- inward   Hypo – downward   Phoria - latent
Exo – outward  Hyper – upward   Tropia – manifest
For example: exotropia of the left eye means an obvious (to a student) deviation of the left eye outward

ORTHOPEDICS
References & Helpful Resources (nothing is required, but these may help)
• Netter’s Orthopaedic Anatomy—helpful book that fits in a coat pocket, but not necessary. Anatomy is what you really need to know and this allows you brush up briefly before a case.
• Physical Examination of the Spine and Extremities—this is best for those going into ortho, but can improve your physical exam if you can get your hands on it.
• Surgical Exposures in Orthopaedics, The Anatomic Approach—You will most often be pimped on anatomy as surgery progresses. If you have access to this book, it will give you a first look at the approaches which may be helpful

**General Tips**
• You can’t know everything. It is most important to work hard and be interested
• You have to work hard, so don’t plan on doing anything else that month
• The test at the end is hard. Try to get through the packet of reading they gave you, but the subject is too big to know everything.

**Ward Tips**
• Be helpful and interested. This is the most important thing for being successful in your ortho rotation. Those who do the best are the ones who are always available, always attentive and willing to do whatever without excessive brownnosing.
• Rounds are fast. Watch the residents then copy them
• Carry the box of dressing changes
• See the Surgery ward tips for additional suggestions

**Sample Notes**
The daily note will vary based on the rotation, but is going to be much more focused. Use the notes of the junior resident as a guide.

ID: 37 y/o male s/p ORIF of R acetabular fx  
S: ask about pain, mention ROM, weight bearing status, dressing or splinting  
O: afvss (afebrile, vital signs stable)  
  wound cdi (clean dry and intact), dressing intact  
  any new imaging  
A/P: include ABX, pain control, ROM or weight bearing status, and DC plans

**Physical Exam**
This is focused on the musculoskeletal system. The area of focus will vary based on your service and you can learn the specific tests as appropriate. Review the shoulder or knee exam as appropriate from your ICM course, Bates, or if you have access to it Physical Examination of the Spine and Extremities. It would be better to go over the exam with a willing resident, learn, be interested, and do it right from then on, than to do a poor exam for half the rotation. Do not forget to be aware of the neuro exam.

**Selected Topics in Orthopedics** (The emphasis will depend on the service)  
**Shoulder service:** know the differential for shoulder pain – rotator cuff tears, impingement, dislocation, fractures, arthritis, etc. You be familiar with a few operations such as shoulder replacement (full and hemi), Rotator cuff repair (open), and ream and run.  
**Trauma service:** just try to make it through without falling over.  
**Sports medicine:** know all of your musculoskeletal anatomy.  
In some clinics, you may need to be familiar with the differential for knee pain – meniscal tears, ACL rupture, arthritis, etc. or hip pain – bursitis, fracture, arthritis, etc.
Ward Tips
1. In Seattle, you will be able to choose where you would like to do this rotation (depending on how many students there are)—HMC, UWMC, CHRMC or VA.
2. If you are interested in oto as a career, do a 4-week rotation. The UW is the best place to get a letter of recommendation, but has long hours due to free flap procedures.

Selected Topics in Otolaryngology
Otitis Media (OM)

- Etiology: eustachian tube dysfunction (esp kids w/horizontal tubes) -> negative pressure develops in middle ear -> transudation of serous fluid -> inoculation with bacteria from nasopharynx
  - ↑ risk: cleft palate, GERD, adenoidal hypertrophy, bottle-feeding, passive tobacco
  - often follow URI – viruses increase bacterial colonization, suppress immune response
  - in adults – consider nasopharyngeal CA
- Organisms
  - Acute: S. pneumo, H. flu, M. catarrhalis
  - Chronic: Pseudomonas, Staph, E. coli, anaerobes
- Diagnosis
  - Sx: otalgia/ear tugging, otorrhea, fever ± vertigo, tinnitus, facial paralysis
  - PE: pneumatic otoscopy is gold std – decreased TM mobility
- Treatment
  - Antibiotics: 1st line – amoxicillin 90 mg/kg/d; also bactrim, augmtenin, cefuroxime. Ototopic 1st line for chronic suppurative OM.
  - Transcan for immunocompromised, treatment failures
  - Pressure equalization tubes for recurrent OM (3 x in 6 mo), chronic OM w/effusion
  - Consider adenoidectomy if need > 1 set of tubes

- Complications of untreated OM
  - Hearing loss
  - Labyrinthitis
  - Perilymphatic fistula
  - TM perforation
  - Tympanosclerosis
  - Cholesteatoma
  - Mastoiditis
  - Epidural abscess
  - Meningitis

Acute bacterial rhinosinusitis

- mucosal edema -> obstruction of sinus ostia -> impaired mucociliary clearance -> bacterial overgrowth
- maxillary, anterior ethmoid most often involved
- S. pneumo, H. flu, M. catarrhalis most often involved in acute disease
- Dx when URI doesn’t resolve in 10 days & accompanied by:
  - Nasal congestion & drainage, post nasal drip
  - Unilateral facial pain/pressure
  - Fever, cough, fatigue
  - Maxillary dental pain
• Hyposmia/anosmia
• Ear fullness/pressure
- Tx: augmentin, fluoroquinolone or TMP-SMX 10-14 days + nasal steroids. 50% spontaneously resolve. Increasing bacterial resistance.

Peritonsillar abscess = pus between pharyngeal tonsil and its capsule
- most common ages 20-40
- predisposition with chronic tonsillitis, acute tonsillitis, s/p antibiotic failure
- Pathophysiology
  • majority: tonsillitis -> peritonsillar cellulitis -> PTA
  • direct spread from parotitis, trauma, odontogenic origin
- Most are mixed aerobic/anaerobic w/S. pyogenes, Fusobacterium spp most common
- Sx: “hot potato voice,” localized sore throat, trismus
- PE: bulging soft palate w/deviated uvula
- Dx: needle aspiration, lateral neck XR, CT w/contrast
- Tx: aspiration/I&D, clinda/cefuroxime or unasyn

Retropharyngeal abscess
- Pus in space posterior to buccopharyngeal fascia, anterior to alar & prevertebral fascia; bound laterally by carotid sheaths, extends from base of skull to bifurcation of trachea
- Most common in young kids -> ruptured suppurative retropharyngeal lymph node
- In adults, due to contiguous spread/trauma (intubation, endoscopy)
- Organisms, Sx similar to PTA + neck stiffness, drooling, stridor
- PE: bulging of posterior pharyngeal wall
- Dx: lateral neck CXR, CT w/contrast. DO NOT do needle aspiration
- Tx: airway protection, surgical drainage
- Complications: mediastinitis, jugular venous thrombosis, aspiration pneumonitis + many more

Epistaxis
- Anterior = bleeding from Kiesselbach’s plexus; 80% of cases
- Posterior = bleeding from Woodruff’s plexus (sphenopalantine a.); 20% of cases, mostly >50 yo
- Severe recurrent epistaxis in adolescent male -> think juvenile nasopharyngeal angiofibromatosis
- Treatments (try these more or less in order): compression, Afrin & lido spray, silver nitrate cautery, anterior/posterior nasal packs, embolization, arterial ligation

Tidbits
Stridor = noise during inspiration and/or expiration due to upper airway obstruction. Most common cause in
  Early childhood -> laryngomalacia
R/o septal hematoma to prevent perforation, saddle-nose deformity
Suspect T-bone fracture w/ hearing loss, dizziness, facial weakness, hemotympanum, Battle’s sign
Samter’s triad: ASA sensitivity, nasal polyposis and bronchospasm
Laryngeal cancer is most common HN cancer; most commonly arises in glottis; majority are SCCAs. Prior HN cancer increases risk of esophageal cancer 8X

TRANSPLANT SURGERY

References & Helpful Resources
Organ Transplantation (2nd edition) by Frank P. Stuart, et al.

A few words about the UWMC Transplant Service and some commonalities:
• You will receive a Transplant Services Medication Protocol Handbook – generally gives you all the rules you need to know. You’ll get the book above, too.
• Pre-rounds start at 7 AM, followed by ICU rounds at 8 AM, then liver rounds at 9 or so AM, then kidney and pancreas rounds at 1 PM (different attendings apply)
• When the values for medication levels become available, usually later in the morning/early afternoon, the team writes orders for medications
• Request to follow a few patients, otherwise it will be first come, first serve
• There is no call for students (theoretically) but it’s still 24/7 until you’ve seen a few of the liver and kidney transplants. Same applies to weekends.
• Post-surgery, liver transplant recipients go to Transplant ICU where you will follow but NOT manage them, while kidney recipients go to the floor
• Keep in mind that the hours are terrible for everyone but you. When there is an organ, they have to transplant.
• Post-surgery, every recipient gets Bactrim for PCP prophylaxis and ganciclovir for 3 months for CMV prophylaxis
• Liver donor/recipients are matched only by ABO Rh+/-, while the kidney donor/recipients are matched also by the HLA class as rejection is more of a problem in the latter
• Early fever in most post-transplant pts is usually attributable to anti-T-cell antibody administration. When there has been no such administration, do a fever of unknown origin work-up. Don’t forget to include a post-operative abscess, CMV infection, and rejection in your differential diagnosis
• Alcohol dependence/abuse are no longer absolute contraindications to liver transplantation. Theoretically, the patient must demonstrate abstinence for 6 months, but they’re becoming less strict about it

Kidney Transplant Complications:
1. Delayed Graft Function d/t ATN
2. Nephrotoxicity of calcineurin inhibitors (cyclosporine and tacrolimus)
3. Hemolytic Uremic Syndrome
4. CMV infection
5. Pyelonephritis
6. BK virus associated nephropathy (at 6 months or later)
7. Recurrent renal disease
8. Rejection
   • Hyperacute: circulating preformed cytotoxic anti-donor antibodies directed to ABO blood group antigens are present – happens after revascularization – no salvage to allograft.
   • Accelerated Acute: rapidly progressive, within the first week, with infiltrates of lymphocytes, macrophages, and plasma cells. Treat
with anti-T-cell antibodies and pulse corticosteroids. Long-term function usually compromised, though partial salvation is possible.

- **Acute Tubular Interstitial Cellular Rejection:** T cell mediated, occurs within the first year post-transplant, with injuries to tubules. May be reversed with steroids, possibly with addition of anti-T-cell antibody. Repeated episodes place the recipient at risk for chronic rejection.

- **Chronic Rejection:** slow and progressive deterioration in renal function with histological changes involving tubules, interstitium, and renal capillaries. Conventional treatment doesn’t appear to alter the course.

**Liver Transplant Complications**
1. Hypertension due to cyclosporine (also leads to hyperkalemia) and steroids; treat with nifedipine, labetalol, beta blockers, and diuretics
2. Renal insufficiency due to cyclosporine or ATN
3. Hepatic Artery Thrombosis (may lead to intrahepatic abscess)
4. Biliary Leak (may lead to abdominal abscess and peritoneal infection)
5. Diabetes Mellitus (due to prednisone, cyclosporine, tacrolimus, weight gain, hepatitis C)
6. Hyperlipidemia – treat with pravastatine 20 mg qd
7. Metabolic bone disease d/t steroids
8. Biliary strictures
9. Skin cancer

**Liver Transplant Patient Selection**
- The pt must meet minimal listing criteria before placed in the waiting list (Child-Tucotte-Pugh score of at least 7 for most cases of cirrhosis)
- After, the pt is awarded a priority based on the current UNOS organ allocation scheme (MELD). MELD score=0.957*Log e (creatinine mg/dl)+0.378*Log e (bilirubin mg/dl) + 1.120 * Log e (INR) + 0.643 (you’ll need a special calculator for this!)
- Consequently to above, you will manage VERY ILL patients who’re awaiting liver transplantation. Keep an eye out for variceal hemorrhage, hepatic encephalopathy, ascites, and spontaneous bacterial peritonitis. Fever or delirium are very bad signs and must be diagnosed aggressively.
PROCEDURES

REFERENCES & HELPFUL RESOURCES
Pfenninger, Procedures for Primary Care Physicians, 1st ed; 1994.

12-lead ECG
Indications: Chest pain, pre-op, baseline for patients with cardiovascular risk factors
Technique:
1. Place patient in supine position
2. Clean skin with alcohol wipes (optional)
3. Place limb leads (not over bones—increases artifact)
   RA, LA: usually placed at upper chest near shoulders or on arm
   RL, LL: usually placed at lower abdomen or at ankles
   Anterior chest leads
   V1 4th intercostals space at R sternal border
   V2 4th intercostals space at L sternal border
   V3 Halfway between V2 and V4
   V4 5th intercostals space at L midclavicular line
   V5 L anterior axillary line directly lateral to V4
   V6 L midaxillary line directly lateral to V5
4. Record and print tracing
5. Remove leads
6. For 3-lead monitoring: White on right upper chest, black on left upper chest, red on left lower chest. (white on right, smoke over fire)

Ankle-Brachial Indices (ABI)
Indications: Presence of peripheral arterial disease
Technique:
1. Have patient in supine position so arms, legs and heart are at same level.
2. Use blood pressure cuff and Doppler to measure systolic BP in both arms and record.
3. Use Doppler to identify location of dorsalis pedis and/or posterior tibialis pulses, mark location bilaterally.
4. Wrap BP cuff around lower leg. Using Doppler to listen to signal, inflate cuff until signal disappears, then slowly deflate until pulse signal returns. Record pressure at which pulse is heard (systolic) by Doppler at DP and PT in both ankles.
5. To calculate the AAI divide the highest SBP from each ankle (either DP or PT) by the highest SBP reading from the upper extremities.
6. AAI = ankle / arm systolic pressure
   - > 1.3 = suggests noncompressible, calcified vessels
   - 0.91-1.3 = normal
   - 0.41-0.9 = mild to mod peripheral arterial dz (range for claudication)
   - <0.4 = Severe peripheral arterial dz. (range for critical leg ischemia and rest pain)
**Lumbar Puncture**

**Indications**  
Suspected CNS infection, SAH, Guillain-Barre syndrome, MS, SLE  
Measure intracranial pressure (pseudotumor cerebri)

**Contraindications**  
Increased intracranial pressure (except to dx pseudotumor cerebri), supratentorial mass lesion, thrombocytopenia, bleeding dyscrasia

**Complications**  
Post-LP headache, brain herniation if mass lesion present or increased intracranial pressure, bloody tap if venous plexus punctured.

**Technique**

1. Obtain informed consent
2. Position patient with back near edge of bed in lateral recumbent position. Have patient flex hips and draw knees up to chest to increase curvature of spine.
3. Palpate iliac crests and identify L3 and L4 interspaces.
4. Open tray, wear sterile gloves, and set up tubes in order, 1-4.
5. Prep and drape skin in sterile fashion
6. Infiltrate skin with 1% lidocaine
7. Use 20-22 gauge spinal needle. Insert at interspace with needle angled slightly toward umbilicus (cephalad). Keep level of needle in line with horizontal plane.
8. A course resistance can be felt as the needle passes through the paraspinous ligaments and a “pop” may be felt when needle passes through the dura.
9. Withdraw stylus fully to check for fluid.
10. Once fluid is obtained, place stopcock and manometer on hub of needle to obtain opening pressure.
11. Fill tubes in order, 2-3cc per tube
12. Once fluid has been collected, replace stylus and withdraw needle.
13. Cover site with sterile dressing and have patient remain lying down in supine position for 2 hours.
14. Observe tubes for occult blood. Decreasing amounts of blood in tubes 1-4 suggests a bloody tap, while increasing or steady amounts suggests an CNS bleed.
15. Send fluid for analysis:
   - Tube #1: glucose, protein, protein electrophoresis
   - Tube #2: Gram stain, culture, bacteria, fungal, TB, viral
   - Tube #3: cell count, differential
   - Tube #4: VDRL, India ink, cytology

**Paracentesis**

**Indications**  
Determine cause of ascites (diagnostic) or to relieve symptoms of ascites (therapeutic)

**Contraindications**  
Coagulopathy, thrombocytopenia, pregnancy

**Complications**  
Infection, perforated viscous, hemorrhage, abdominal wall hematoma, renal failure, hypotension

**Technique**

1. Obtain informed consent
2. Consider supine and upright AXR before procedure
3. Have patient urinate or use catheter to empty bladder
4. Have patient in supine or semi-recumbent position
5. Examine abdomen looking for shifting dullness & percuss level of dullness.  
   Usual site if insertion is 2-3 cm below umbilicus at midline. If there is scarring or a hernia at the midline, then pick a point one-third the distance between umbilicus and ASIS (left side)
6. Use sterile technique; prep and drape site, wear mask
7. Inject local anesthetic (lido with epi) in skin and SubQ tissues, all the way to the peritoneum.
8. Using a Z technique, insert 18-22 gauge needle attached to 20-30cc syringe, aspirating continuously.
9. When fluid comes back, fill syringe to send for studies. Then remove needle from catheter and attach tubing. Insert into Vacutainer to drain excess fluid.
10. After fluid is removed, pull catheter out and apply pressure to site.
11. Send fluid for cell count, differential, albumin, total protein, Gram stain, culture. Can also do amylase, cytology, AFB stain if clinically indicated.
12. If you need to determine if the fluid is transudate vs. exudate a serum LDH and albumin should be sent at the same time.

Thoracentesis

Indications Determine cause of pleural effusion, or relieve symptoms of large pleural effusion

Contraindic. Coagulopathy, major respiratory impairment on contralateral side, very small effusions, herpes zoster on chest wall

Complications Pneumothorax, hemothorax, infection, hypotension, reexpansion pulmonary edema, hepatic or splenic puncture

Technique
1. Obtain informed consent
2. Have patient sit up and lean forward slightly.
3. Percuss chest wall to find fluid level. If effusion is small, use ultrasound to mark location and quantify amount of fluid.
4. Usual needle insertion site for fluid removal is posterolateral back, 1-2 interspaces below fluid level. Do not use site below 8th intercostal space on back. Mark location.
5. Use sterile technique, wear mask, and prep and drape site.
6. Use 25 gauge needle to infiltrate skin and subQ with lidocaine.
7. Switch to 22 gauge, 1 1/2 inch needle and infiltrate anesthetic to the superior border of rib and into intercostals muscles. Aspirate continuously. Avoid the inferior portion of the rib as this is where the neurovascular bundle is located.
8. Use 18 gauge needle attached to 50 cc syringe for thoracentesis and insert in same tract as anesthesia needle, aspirate continuously.
9. When fluid is obtained, fill syringe, then fill containers.
10. After removing fluid, withdraw needle while patient exhales or valsalvas to decrease chance of pneumothorax.
11. Apply sterile dressing to site.
12. Send fluid for cell count, differential, LDH, total protein, Gram stain, culture, pH.
14. If patient develops hypotension, desaturation, or respiratory distress during procedure, stop, obtain CXR, or do immediate needle decompression for pneumothorax.
15. If you need to determine if the fluid is transudate vs. exudate a serum LDH and albumin should be sent at the same time.

**Chest Tube Placement**

**Indications**
Pneumothorax, hemothorax, empyema, recurrent pleural effusion

**Contraindic.**
Bleeding dyscrasia, anticoagulation, empyema caused by AFB

**Technique**
1. Obtain informed consent
2. Check coags / platelets
3. Consider sedating patient (painful)
4. Use 18-20 French tube for pneumothorax, 32-36 French tube for fluid or hemothorax
5. Assemble suction/drainage equipment and connect to suction
6. Position patient in supine position, elevate head of bed 30-60 degrees. Usual insertion site is at anterior axillary line at 4th or 5th intercostals space. Mark site.
7. Prep and drape in sterile fashion. Wear gown and mask.
8. Anesthesia at pleural insertion site: anesthetize skin over rib using 2 gauge needle, 10 cc syringe, 1% lidocaine. Anesthesia at incision site (rib below rib of pleural insertion). Using 22 gauge needle and 1% lidocaine, infiltrate subQ, muscle, periosteum, and parietal pleura.
9. Make 2-4 cm incision through skin and tissues over rib. Extend incision with blunt dissection using Kelly clamp, working towards superior aspect of rib above tunneling the course of the chest tube before entering the chest cavity.
10. Push Kelly clamp through parietal pleura. Inside pleural cavity, open clamp, then withdraw. Air or fluid should rush out.
11. Check to see that pleural space has been entered with finger.
13. Place tube in pleural space. Direct tube superior, medial, posterior for fluid drainage. Direct tube superior and anterior for pneumothorax. All ventilation holes need to be in pleural space.
15. Use 1-0 or 2-0 silk or nylon to suture chest tube in place.
16. Cover site with 4x4 gauze (with Y cuts to fit around tube)
17. Tape gauze and tube in place
18. Obtain CXR to confirm placement
19. Remove chest tube when there is less than 150cc of fluid in 24hrs and no air leak.

**Arterial Blood Draw**

**Indications**
O2 and CO2 monitoring during CPR, confirm need for home O2 therapy, diagnose O2/CO2 exchange problem, acid/base imbalance

**Contraindic.**
Unable to palpate arterial pulse, bleeding dyscrasia

**Complications**
Hematoma, thrombosis, nerve damage, getting venous blood

**Technique**
1. Do Allen test before radial artery puncture to confirm ulnar collateral flow.
2. Select ABG kit or load syringe with small amount of heparin, then empty (just need to coat the syringe)
3. Palpate artery with 2 fingers and immobilize along its course with 2-3 fingers.
4. Prep skin with betadine/alcohol or chlorhexadine.
5. Consider small amount of local anesthetic in conscious patients (but this will distort anatomy and make pulse harder to feel)
6. Hold barrel of syringe like a pencil with bevel up.
   Radial: extend wrist, support arm and wrist with towel or firm surface. Insert needle where radial pulse is prominent at 45-60 degree angle
   Brachial: extend and supinate arm. Insert needle at median aspect of antecubital fossa, just above elbow crease at 45-60 degree angle
   Femoral: insert needle 2-3cm below inguinal ligament at 60-90 degree angle
7. Blood should enter and fill syringe. You may need to aspirate with a large syringe or a hypotensive patient.
8. Remove needle quickly and apply pressure to site for 5-10 minutes to prevent hematoma.
9. Place syringe on ice and transport to lab

**Central Venous Catheterization**

*Indications* Fluid administration, CVP monitoring, dialysis, long-term IV Rx
*Contraindic.* Coagulopathy, thrombosis, distorted anatomy or landmarks, anticoagulation

*Complications* Pneumothorax, air embolism, cath/guidewire embolism, arrhythmias, central vein perforation, central vein thrombosis

**Technique**
1. Obtain informed consent
2. Get supplies, gown, mask, gloves
3. Prep & drape area in sterile fashion (subclavian, IJ, femoral)
4. Infiltrate skin and subQ with lidocaine
5. Use finder needle to locate vein
6. After venous blood is aspirated, insert large-bore needle at same site
7. After venous blood is aspirated with large-bore needle, grasp hub of needle and brace hand against patient (non-dominant hand).
8. Lower needle to angle parallel to vein, aspirate to confirm flow.
9. Remove syringe and insert guidewire
10. Remove needle over guidewire, hold wire in place with gauze (NEVER let go of the wire).
11. Make 3-4mm incision with scalpel at site.
12. Pass dilator 3-4cm over guidewire to dilate tissue down to vessel (not through).
13. Pass catheter over guidewire
14. Advance catheter and remove guidewire
15. Aspirate blood and flush ports.
16. Suture in place
17. Post-procedure stat CXR to confirm placement and R/O PTX.

**Subclavian Vein** (lower infection risk, higher risk of PTX, most comfortable for patient)
1. Have patient in Trendelenburg, place towel roll between scapulae.
2. Insert needle below clavicle, 2cm inferior to the junction between lateral 1/3 and medial 2/3 of clavicle. Aspirate continuously.
3. Keep needle parallel to floor and aim first for clavicle. Once you hit bone, aim down to just below clavicle.
4. Once under clavicle, advance needle 4-5 cm

**Internal Jugular Vein** (straight pass to SVC, PTX is less common)
1. Turn patients head 45-60 degrees to contralateral side. Find the triangle formed by 2 heads of sternocleidomastoid and clavicle. Insert needle at apex of triangle, lateral to carotid artery.
2. Insert needle at 70 degree angle to skin, aim for nipple. Aspirate continuously.
3. Do not insert needle more than 1.5 inches

**Femoral Vein** (fast and easy, no risk of PTX, risk of retroperitoneal bleed, higher risk of infection, patient needs to be immobile)
1. Palpate femoral artery below inguinal ligament. Vein is medial to artery.
2. Insert needle approx. 1cm medial to artery and 2cm below inguinal ligament. Insert at 45 degree angle to skin, bevel up.

**Skin Biopsy**
**Preparation:** Forms for consent, pathology, microbiology
- Anesthetic, syringe and needles
- Alcohol and betadine wipes
- Punch or scalpel
- Suture or Drysol
- Forceps, needle driver, scissor
- Specimen containers
- Antibiotic ointment and dressing

**Site selection:** Choose fresh lesions for vesiculobullous eruptions. Otherwise, use mature or active lesions. Choose site that will give minimal scarring and best wound healing.

**Anesthesia:** 1% lidocaine with epi is used for most derm procedures.
- No epi for fingers, nose, penis, toes
- Use 30 gauge needle to inject, put needle in quickly and inject slowly
- Can add bicarb to lidocaine to reduce pain (1 part bicarb to 10 parts lido)

**Danger Zone 1** = temporal branch of facial nerve
**Danger Zone 2** = marginal mandibular branch of facial nerve
**Danger Zone 3** = posterior triangle, accessory nerve

**Punch Biopsy**
**Indication** Full-thickness removal of small skin lesions. (Do not use on eyelids, lips, penis).
**Uses** Used for dermal and subdermal processes

**Technique**
1. Obtain informed consent
2. Select punch size to just cover size of lesion
3. Clean area of skin with antiseptic solution
4. Inject local anesthetic
5. Position punch perpendicular to skin and lesion, push into skin while rotating instrument. Usually you will feel a “give” when SubQ tissue is reached.
6. Remove punch and cut base of specimen with scissors, place in specimen cup.
7. Apply pressure for hemostasis and if desired, insert 1-2 sutures for closure (simple interrupted, vertical mattress)
8. Send specimen for pathology, culture, IF as needed
Fusiform Excisional Biopsy

Indications
Use for squamous cell carcinoma, basal cell carcinoma, suspected melanoma

Technique
1. Obtain informed consent
2. Draw an outline of the incision lines on skin with marking pen.
3. Incision lines should be parallel to skin tension lines (wrinkle lines). Length of fusiform excision should be 3x the width of lesion. Corner angles should be 30 degrees. (Can be 45 degrees with very thin skin)
4. Clean, prep and drape lesion in sterile fashion.
5. Inject local anesthetic
6. Make incision according to outline, keeping scalpel blade perpendicular to skin surface. Depth of wound should be uniform throughout.
7. If there is tension when skin margins are brought together, undermine the edges under skin in the SubQ tissue using sharp or blunt method.
8. Use electrocautery to achieve hemostasis if necessary
9. For small lesions without tension, do single-layer closure.
10. For larger lesions or with tension, close in 2 layers. Use absorbable SubQ suture for deep sutures (Dexon, absorbable Vicryl). Use interrupted or running suture for dermal closure.

<table>
<thead>
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<th>Suture Removal</th>
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<td>5-6 days</td>
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<tr>
<td>Neck</td>
<td>5-7 days</td>
</tr>
<tr>
<td>Scalp</td>
<td>5-7 days</td>
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<tr>
<td>Trunk</td>
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