
Fourth Edition
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The Basics of Effective Sign-out

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This is the fourth edition of the highly successful, pocket-sized companion “survival guide” written and edited by former Washington University residents. It is meant to complement the Washington Manual® of Medical Therapeutics and the Washington Manual® of Outpatient Internal Medicine and provide concise and practical information for those learning the basics of practicing clinical medicine. It is written assuming knowledge of basic pathophysiology and data interpretation. The target audience is primarily those beginning their internship, but this guide may be useful for medical students, residents, and anyone else on the front lines of patient care.

The fourth edition has been updated to be consistent with the most current medical practices. The pace of inpatient medicine stresses efficiency and time management, especially in our work-hour regulated environment. In keeping with the purpose of a pocket book, a deliberate attempt was made to keep the format succinct so that common workups, cross-cover calls, procedures, and other practical information would always be in a rapidly accessible format. There are also essential sections about “what not to miss” and “when to call for help” for common clinical scenarios. It is written assuming that a standard textbook of internal medicine, the Washington Manuals®, a Sanford Guide, Physicians Desk Reference, and Internet access (as well as your resident) are available nearby for reference. Furthermore, we have included a newly revised rapid-access, pocket-sized card detailing procedural skills and techniques. This card is detachable and can travel with you through the course of your residency and beyond.
We wish to thank the Washington University residents and faculty for their enthusiastic support of this project, and even more importantly, their ongoing contributions that have served to make this guide immeasurably better.

We would also like to extend our thanks to Melvin Blanchard, MD, and Vicky Fraser, MD, whose leadership and support have been vital to the continued success of this book. We appreciate the tremendous support of Thomas De Fer, MD, and Katie Sharp in their coordination of our efforts. From Wolters Kluwer/Lippincott Williams & Wilkins, we are indebted to Sonya Seigafuse, Leanne Vandetty, and Kimberly Schonberger.

E.M.K.
G.N.L.
H.F.S.
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<td>abdominal aortic aneurysm</td>
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<tr>
<td>AMA</td>
<td>against medical advice</td>
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<tr>
<td>AP</td>
<td>anteroposterior</td>
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<tr>
<td>APC</td>
<td>atrial premature contraction</td>
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<tr>
<td>ARF</td>
<td>acute renal failure</td>
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<tr>
<td>ATN</td>
<td>acute tubular necrosis</td>
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<tr>
<td>AVNRT</td>
<td>atrioventricular nodal reentrant tachycardia</td>
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<tr>
<td>AVRT</td>
<td>atrioventricular reciprocating tachycardia</td>
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<tr>
<td>BBB</td>
<td>bundle branch block</td>
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<tr>
<td>BP</td>
<td>bullous pemphigoid</td>
</tr>
<tr>
<td>CAD</td>
<td>coronary artery disease</td>
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<tr>
<td>CHF</td>
<td>congestive heart failure</td>
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<tr>
<td>COPD</td>
<td>chronic obstructive pulmonary disease</td>
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<tr>
<td>CPAP</td>
<td>continuous positive airway pressure</td>
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<tr>
<td>CXR</td>
<td>chest x-ray</td>
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<tr>
<td>D/C</td>
<td>discharge</td>
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<tr>
<td>DKA</td>
<td>diabetic ketoacidosis</td>
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<tr>
<td>ECF</td>
<td>extracellular fluid</td>
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<tr>
<td>FFP</td>
<td>fresh frozen plasma</td>
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<tr>
<td>GERD</td>
<td>gastroesophageal reflux disease</td>
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<tr>
<td>GN</td>
<td>glomerulonephritis</td>
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<tr>
<td>H&amp;P</td>
<td>history and physical examination</td>
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<tr>
<td>β-hCG</td>
<td>human chorionic gonadotropin β</td>
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<tr>
<td>Hct</td>
<td>hematocrit</td>
</tr>
<tr>
<td>HEENT</td>
<td>head, eyes, ears, nose, and throat</td>
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<tr>
<td>HTN</td>
<td>hypertension</td>
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<tr>
<td>I/O</td>
<td>input/output</td>
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<tr>
<td>JPCs</td>
<td>junctional premature contractions</td>
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<tr>
<td>JVP</td>
<td>jugular venous pressure</td>
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<tr>
<td>LBBB</td>
<td>left bundle branch block</td>
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<tr>
<td>LMWH</td>
<td>low-molecular-weight heparin</td>
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<tr>
<td>LP</td>
<td>lumbar puncture</td>
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<tr>
<td>LVH</td>
<td>left ventricular</td>
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<tr>
<td>NS</td>
<td>normal saline</td>
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<tr>
<td>NSAID</td>
<td>nonsteroidal anti-inflammatory drug</td>
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<tr>
<td>NSR</td>
<td>normal sinus rhythm</td>
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<tr>
<td>PA</td>
<td>posteroanterior</td>
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<tr>
<td>PE</td>
<td>physical examination</td>
</tr>
<tr>
<td>PICC</td>
<td>peripherally inserted</td>
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<tr>
<td>PID</td>
<td>central catheter</td>
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<tr>
<td>PUD</td>
<td>pelvic inflammatory disease</td>
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<tr>
<td>PV</td>
<td>peptic ulcer disease</td>
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<tr>
<td>PVC</td>
<td>premature ventricular contraction</td>
</tr>
<tr>
<td>RR</td>
<td>respiratory rate</td>
</tr>
<tr>
<td>RTA</td>
<td>renal tubular acidosis</td>
</tr>
<tr>
<td>SBO</td>
<td>small bowel obstruction</td>
</tr>
<tr>
<td>SBP</td>
<td>systolic blood pressure</td>
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<tr>
<td>SJS</td>
<td>Stevens-Johnson syndrome</td>
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<tr>
<td>SOB</td>
<td>shortness of breath</td>
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<tr>
<td>T</td>
<td>temperature</td>
</tr>
<tr>
<td>TDP</td>
<td>torsades de pointes</td>
</tr>
<tr>
<td>TIA</td>
<td>transient ischemic attack</td>
</tr>
<tr>
<td>TSH</td>
<td>thyroid-stimulating hormone</td>
</tr>
<tr>
<td>TSS</td>
<td>toxic shock syndrome</td>
</tr>
<tr>
<td>UTI</td>
<td>urinary tract infection</td>
</tr>
<tr>
<td>VPC</td>
<td>ventricular premature contraction</td>
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As an intern you will encounter many new challenges. These will range from very simple to most complex. Please remember that you have acquired the tools to address this adversity. And, even when you feel most overwhelmed, you are undoubtedly surrounded by a wealth of available resources that include ancillary and nursing staff, fellow interns, senior residents, and attending physicians.

Although the year ahead of you may now seem long and daunting, your tasks (other than survival) are quite achievable. The following competencies/milestones were copied from the curriculum for the inpatient general medicine rotation for the internship program at Washington University School of Medicine and Barnes-Jewish Hospital. You can see that not only are the objectives quite simple, but your rigorous work in medical school has well prepared you to master many of your goals. Throughout the year, use these objectives as a checklist to remind yourself of your accomplishments and to guide your learning in potential areas of weakness.

• Patient care:
  • Gather accurate information about patients, including performing a thorough history and physical examination.
  • Synthesize data into a prioritized problem list and differential diagnosis, and then formulate diagnostic and therapeutic plans.
  • Prioritize each day’s work.
  • Know the indications, contraindications, and risks of some invasive procedures and competently perform some invasive procedures.

• Medical knowledge:
  • Demonstrate an increasing fund of knowledge in the range of common problems encountered in inpatient internal medicine and utilize this knowledge in clinical reasoning.
  • Become familiar with the diagnostic and therapeutic approach to patients with chest pain, shortness of breath, fever, mental status changes, abdominal pain, GI bleeding,
syncope and lightheadedness, renal failure (acute and/or chronic), cirrhosis, congestive heart failure, anemia, hypertension, diabetes mellitus, pneumonia, chronic obstructive pulmonary disease, urinary tract infection, soft tissue infections, and alcohol withdrawal.

• Practice-based learning and improvement:
  • Understand your limitations of knowledge and judgment; ask for help when needed; be self-motivated to acquire knowledge.
  • Accept feedback, learn from your own errors, and develop self-improvement plans.
  • Use information technology to manage patient data and access online medical information.

• Interpersonal and communication skills:
  • Demonstrate caring and respectful behaviors with patients, families, including those who are angry and frustrated, and all members of the healthcare team.
  • Counsel and educate patients and their families.
  • Conduct supportive and respectful discussions of code status and advanced directives.
  • Facilitate the learning of students and other healthcare professionals.
  • Demonstrate the ability to convey clinical information accurately and concisely in oral presentations and in chart notes.

• Professionalism:
  • Demonstrate respect, compassion, and integrity.
  • Demonstrate a commitment to excellence and on-going professional development.
  • Demonstrate a commitment to ethical principles pertaining to provision or withholding of clinical care, confidentiality of patient information, informed consent, and other aspects of clinical care.
  • Develop an appreciation for the ethical, cultural and socioeconomic dimensions of illness, demonstrating sensitivity and responsiveness to patients’ culture, age, gender, and disabilities.

• Systems-based practice:
  • Work efficiently with others as a member of the healthcare team.
• Advocate for quality patient care and assist patients in dealing with system complexities.

• Understand and appreciate the importance of contacting the patient’s primary care provider at the time of admission or soon thereafter.

• Learn the cost-effective use of diagnostic and therapeutic technologies.
1. Don’t panic, and keep your sense of humor.

2. Take care of your patients. You are finally using your education and training.

3. Work hard, stay enthusiastic, and maintain interest.

4. Be organized and prioritize your tasks.

5. Verify everything yourself (e.g., labs, X-rays, ECGs).

6. Ask questions and ask for help! Believe it or not, you are not expected to know everything.

7. Be kind to the nurses and other ancillary staff. They can make your life much better … or much worse.

8. Choose your battles carefully. Even in the name of patient care, ugly behavior is ugly. Don’t get a reputation.

9. Sleep when you can, remember to eat, and be mindful of your own health.

10. Don’t forget your family and friends.

11. Call for consultations on your patients early in the day and have a specific question you want answered from the consultant.

12. Start thinking about discharge/disposition planning from day one.

13. Dictate discharge summaries the day the patient leaves.

PULSELESS ARREST

1. BLS algorithm: Call for help, give CPR
2. Give oxygen when available
3. Attach monitor/defibrillator when available
4. Check rhythm
5. Shockable rhythm?
6. Shock
7. Give 5 cycles of CPR
8. Check rhythm
9. Not shockable: Asystole/PEA
10. Resume CPR immediately for 5 cycles
   When IV/IO available, give vasopressor
   • Epinephrine 1 mg IV/IO
   • Repeat every 3 to 5 minutes
   or
   • May give 1 dose of vasopressin 40 U IV/IO to replace first or second dose of epinephrine
   Consider atropine 1 mg IV/IO for asystole or slow PEA rate
   Repeat every 3 to 5 minutes (up to 3 doses)
11. Check rhythm
12. Shockable rhythm?
13. Not shockable: Go to Box 4

ACLS Algorithms

During CPR

• Push hard and fast (100/min)
• Ensure full chest recoil
• Minimize interruptions in chest compressions
• One cycle of CPR: 30 compressions then 2 breaths; 5 cycles = 2 minutes
• Avoid hyperventilation
• Secure airway and confirm placement
• After an advanced airway is placed, rescuers no longer deliver "cycles" of CPR. Give continuous chest compressions without pauses for breaths. Give 8 to 10 breaths/min. Check rhythm every 2 minutes
• Rotate compressors every 2 minutes with rhythm checks
• Search for and treat possible contributing factors:
  – Hypovolemia
  – Hypoxia
  – Hydrogen ion (acidosis)
  – Hypo-/hyperkalemia
  – Hypoglycemia
  – Hypothermia
  – Toxins
  – Tamponade, cardiac
  – Tension pneumothorax
  – Thoracoabdominal (coronary or pulmonary)
  – Trauma
BRADYCARDIA
Heart rate <60 bpm and inadequate for clinical condition

1. Maintain patent airway; assist breathing as needed
2. Give oxygen
3. Monitor ECG (identify rhythm), blood pressure, oximetry
4. Establish IV access
5. Prepare for transcutaneous pacing; use without delay for high-degree block (type II second-degree block or third-degree AV block)
6. Consider atropine 0.5 mg IV while awaiting pacemaker. May repeat to a total dose of 3 mg. If ineffective, begin pacing
7. Consider epinephrine (2 to 10 µg/min) or dopamine (2 to 10 µg/kg/min) infusion while awaiting pacemaker or if pacing ineffective
8. Prepare for transvenous pacing
9. Treat contributing causes
10. Consider expert consultation

Signs or symptoms of poor perfusion caused by the bradycardia?
(e.g., acute altered mental status, ongoing chest pain, hypotension, or other signs of shock)

Figure 3-2. Bradycardia algorithm. AV, atrioventricular; bpm, beats per minute; ECG, electrocardiogram; IV, intravenous. (From the American Heart Association in collaboration with the International Liaison Committee on Resuscitation. Guidelines 2005 for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.3: management of symptomatic bradycardia and tachycardia. Circulation. 2005;112(24 suppl):IV-67-IV-77, with permission.)
If atrial fibrillation with aberrancy
- See Irregular Narrow-Complex Tachycardia (Box 11)

If pre-excited atrial fibrillation (AF + WPW)
- Expert consultation advised
- Avoid AV nodal blocking agents (e.g., adenosine, digoxin, diltiazem, verapamil)
- Consider antiarrhythmics (e.g., amiodarone 150 mg IV over 10 min)

If recurrent polymorphic VT, seek expert consultation
If torsades de pointes, give magnesium (load with 1 to 2 g over 5 to 60 min, then infusion)

If ventricular tachycardia or uncertain rhythm
- Amiodarone 150 mg IV over 10 minutes repeat as needed to maximum dose of 2.2 g/24 hours
- Prepare for elective synchronized cardioversion

If SVT with aberrancy
- Give adenosine (go to Box 7)

During Evaluation
- Secure, verify airway and vascular access when possible
- Consider expert consultation
- Prepare for cardioversion

Treat contributing factors:
- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypoglycemia
- Hypothermia
- Toxins
- Tamponade, cardiac
- Tension pneumothorax
- Thrombosis (coronary or pulmonary)
- Trauma (hypovolemia)

TACHYCARDIA With Pulses

1. Assess and support ABCs as needed
   - Give oxygen
   - Monitor ECG (identify rhythm), blood pressure, oximetry
   - Identify and treat reversible causes

2. Symptoms persist

3. Is patient stable?
   - Unstable signs include altered mental status, ongoing chest pain, hypotension, or other signs of shock
   - Note: Rate-related symptoms uncommon if heart rate <150/min

4. Perform immediate synchronized cardioversion
   - Establish IV access and give sedation if patient is conscious; do not delay cardioversion
   - Consider expert consultation
   - If pulseless arrest develops, see Pulseless Arrest Algorithm

5. Establish IV access
   - Obtain 12-lead ECG (when available) or rhythm strip
   - Is QRS narrow (<0.12 s)?

6. Narrow QRS:
   - Is rhythm regular?
     - Regular
     - Irregular

7. Does rhythm convert?
   - Note: Consider expert consultation
     - Converts
     - Does not Convert

8. If rhythm converts, probable reentry SVT (reentry supraventricular tachycardia):
   - Observe for recurrence
   - Treat recurrence with adenosine or longer-acting AV nodal blocking agents (e.g., diltiazem, β-blockers)

9. If rhythm does not convert, possible atrial flutter, ectopic atrial tachycardia, or junctional tachycardia:
   - Control rate (e.g., diltiazem, β-blockers; use β-blockers with caution in pulmonary disease or CHF)
   - Treat underlying cause
   - Consider expert consultation

10. Irregular Narrow-Complex Tachycardia
    - Probable atrial fibrillation or possible atrial flutter or MAT (multifocal atrial tachycardia)
    - Consider expert consultation
    - Control rate (e.g., diltiazem, β-blockers; use β-blockers with caution in pulmonary disease or CHF)

11. Wide QRS:
    - Is rhythm regular?
      - Regular
      - Irregular

12. Wide QRS:
    - Is rhythm regular?
      - Regular
      - Irregular

13. If ventricular tachycardia or uncertain rhythm
    - Amiodarone 150 mg IV over 10 minutes repeat as needed to maximum dose of 2.2 g/24 hours
    - Prepare for elective synchronized cardioversion

14. If atrial fibrillation with aberrancy
    - See Irregular Narrow-Complex Tachycardia (Box 11)

Figure 3-3. Advanced Cardiac Life Support Tachycardia algorithm. (From the American Heart Association in collaboration with the International Liaison Committee on Resuscitation. Guidelines 2005 for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.3: management of symptomatic bradycardia and tachycardia. Circulation. 2005;112(24 suppl):IV-67-IV-77, with permission.)
Most house officers carry a pocket-sized general manual of internal medicine in their pocket. The following are useful and practical:


Additional portable guides that you don’t want to be without:


Other things to carry with you:

- A good stethoscope
- Reflex hammer
- Guaiac developer and stool cards
- Penlight
- ECG calipers
• Ophthalmoscope/otoscope (yes, you still need this; make sure it’s charged for your call and leave it in your locker)
• ABG kit
• Alcohol wipes
• Notecards for patient information

HANDHELD COMPUTER RESOURCES

Drug References
• ePocrates: www.epocrates.com. Free (basic program).
• Tarascon Pocket Pharmacopoeia: www.tarascon.com.

Calculators
Many of these programs are embedded in other smart phone/PDA programs like ePocrates; if you want them individually, here are some.
• MedCalc, for iOS.
• MedCalc 3000, for Android.

Data Tracking

Other References
• Skyscape: www.skyscape.com.

INTERNET RESOURCES

General Sites
• American College of Physicians: www.acponline.org
• MD Consult: www.mdconsult.com.
• UpToDate: www.uptodate.com.

Cardiovascular Disease
• American College of Cardiology: www.acc.org
• American Heart Association: www.heart.org
• National Heart, Lung, and Blood Institute: www.nhlbi.nih.gov

Cerebrovascular Disease and Stroke
• The Internet Stroke Center: www.strokecenter.org
• National Institute of Neurological Disorders and Stroke:
  www.ninds.nih.gov
• National Stroke Association: www.stroke.org

Pulmonary Diseases and Allergy
• American College of Chest Physicians: www.chestnet.org
• Global Initiative for Chronic Obstructive Lung Disease:
  www.goldcopd.org
• National Heart, Lung, and Blood Institute: www.nhlbi.nih.gov

Endocrine and Diabetes
• American Diabetes Association: www.diabetes.org
• American Thyroid Association: www.thyroid.org
• National Diabetes Information Clearinghouse: diabetes.niddk.nih.gov
• National Institute of Diabetes and Digestive and Kidney Diseases:
  www.niddk.nih.gov
• The Endocrine Society: www.endo-society.org

Rheumatology
• American Arthritis Foundation: www.arthritis.org
• American College of Rheumatology: www.rheumatology.org
• National Institute of Arthritis and Musculoskeletal and Skin Diseases:
  www.niams.nih.gov
• National Osteoporosis Foundation: www.nof.org
Nephrology

- National Kidney Foundation: www.kidney.org

Oncology

- American Cancer Society: www.cancer.org
- American Society of Clinical Oncology: www.asco.org
- American Society of Hematology: www.hematology.org
- National Cancer Institute: www.cancer.gov
- National Comprehensive Cancer Network: www.nccn.org

Orthopedic Surgery

American Association of Orthopaedic Surgeons: www.orthoinfo.aaos.org

Gastroenterology

- American Association for the Study of Liver Diseases: www.aasld.org
- American College of Gastroenterology: www.acg.gi.org
- American Gastroenterological Association: www.gastro.org
- National Digestive Diseases Information Clearinghouse: digestive.niddk.nih.gov

Infectious Disease/HIV

- Centers for Disease Control and Prevention: www.cdc.gov/hiv
- Infectious Diseases Society of America: www.idsociety.org
- National Institute of Allergy and Infectious Diseases: www.niaid.nih.gov
- University of California at San Francisco HIV InSite: hivinsite.ucsf.edu
- World Health Organization: www.who.int

Geriatrics, Aging, and Osteoporosis

- Administration on Aging: www.aoa.gov
- Alzheimer’s Association: www.alz.org
• National Institute on Aging: www.nia.nih.gov
• National Osteoporosis Foundation: www.nof.org

**Complementary and Alternative Medicine**
National Center for Complementary and Alternative Medicine: nccam.nih.gov

**Online Journals**
Subscriptions are generally required. Your institution may have a site license.
• *Annals of Internal Medicine*: www.annals.org
• *British Medical Journal*: www.bmj.com
• *The Journal of the American Medical Association*: www.jama.ama-assn.org
• *The Lancet*: www.thelancet.com

**INTERNET RESOURCES FOR EVIDENCE-BASED MEDICINE**

The library at your institution is a great place to start for EBM searches. Many of them offer web-based proxy servers to their e-journal collections, as well as search engines like PubMed or Ovid.

**The Cochrane Database of Systematic Reviews**
• http://thecochranelibrary.net
• Reviews, analyzes, and synthesizes the best clinical trials by topics.
• Multidirectional links MEDLINE, EBM, and EUCLID full-text.
• Subscription required.

**PubMed**
• Maintained by the National Library of Medicine.
• Allows a user-friendly approach to medical literature with built-in search filters. Free.

**ACP Journal Club**
• www.acpjcr.org
• Evidence-based medicine reviews of journal articles.
• Subscription required.
Sleep (hours) = \frac{(\text{Discharges} + \text{Transfers})}{(\text{Admissions} + \text{Cross Cover})^2} \times \text{Number of Interns}

**A-a O\textsubscript{2} GRADIENT**

A-a gradient = PA\textsubscript{AO\textsubscript{2}} - PA\textsubscript{O\textsubscript{2}}

PA\textsubscript{AO\textsubscript{2}} = ([FiO\textsubscript{2} \times 713] - PaCO\textsubscript{2})/0.8

*(all units in mmHg)*

- Estimate for upper limit of normal in room air (in mmHg) by age = (age/4) + 4.
- Causes of increased A-a gradient: V/Q mismatch, intrapulmonary right-to-left shunt, intracardiac right-to-left shunt, impaired diffusion (room air only).

**ANION GAP (SERUM)**

AG = [Na\textsuperscript{+}] - ([Cl\textsuperscript{-}] + [HCO\textsubscript{3}])

([Na\textsuperscript{+}], [Cl\textsuperscript{-}], HCO\textsubscript{3} in mEq/L)

- Normal = 8–12 mEq/L.
- See Acid-Base section in Chapter 18 for differential diagnosis.

**ANION GAP (URINE)**

UAG = (U\textsubscript{[Na\textsuperscript{+}]} + U\textsubscript{[K\textsuperscript{+}]} - U\textsubscript{[Cl\textsuperscript{-}]} )

(U\textsubscript{[Na\textsuperscript{+}]], U\textsubscript{[K\textsuperscript{+}]], U\textsubscript{[Cl\textsuperscript{-}]} in mEq/L)

- Normal = slightly positive.
- UAG is **negative** in diarrhea-induced nongap metabolic acidosis (enhanced urinary NH\textsubscript{4} excretion).
- UAG is **positive** in distal RTA-induced nongap metabolic acidosis (impaired urinary NH\textsubscript{4} excretion).
**BODY MASS INDEX**

BMI = weight/(height)^2  
*(weight in kg, height in m)*  
- <18.5 = underweight  
- 18.5–24.9 = normal weight  
- 25–29.9 = overweight  
- >30 = obese

**CREATININE CLEARANCE/GLomerular FILTRATION RATE**

**Estimated (Cockcroft-Gault Formula)**  
CrCl = [(140 – age) × weight]/[serum Cr × 72]  
Multiply by 0.85 for women  
*(weight in kg, Cr in mg/dL)*

**Estimated (MDRD)**  
cGFR = 186.3 × (serum Cr)^−1.54 × age^−0.203 × 0.742 (if female)  
× 1.21 (if black)  
*(eGFR in mL/min per 1.73 m², Cr in mg/dL)*

**Measured (24 Hour)**  
CrCl = (U_{[Cr]} × U_{volume})/(P_{[Cr]} × 24 × 60)  
*(Cr in mg/dL, volume in mL, and time in min)*

**CORRECTED SERUM CALCIUM**

Corrected serum Ca = measured [Ca^{2+}] + [0.8 × (4.0 – measured albumin)]  
*([Ca^{2+}] in mg/dL, albumin in g/d)*

**CORRECTED SERUM SODIUM**

Corrected serum Na = measured [Na^+] + [0.016 × (measured [glucose] – 100)]  
*([Na^+] in mEq/L, [glucose] in mg/dL)*
FRACTIONAL EXCRETION OF SODIUM

\[ \text{FE}_{\text{Na}} = \left( \frac{U_{[\text{Na}^+]}}{P_{[\text{Na}^+]}} \times \frac{P_{[\text{Cr}^+]}}{U_{[\text{Cr}^+]}} \right) \times 100 \]

- \( \text{FE}_{\text{Na}} < 1\% \) in prerenal states, early ATN, contrast or heme pigment nephropathy, and acute glomerulonephritis.
- Not valid when diuretics have been given.

FRACTIONAL EXCRETION OF UREA

\[ \text{FE}_{\text{urea}} = \left( \frac{[U_{[\text{urea}]} \times P_{[\text{Cr}]}]}{[P_{[\text{urea}]}] \times U_{[\text{Cr}]}} \right) \times 100 \]

- \( \text{FE}_{\text{urea}} < 35\% \) in prerenal states.
- Not affected by diuretics.

MEAN ARTERIAL PRESSURE

\[ \text{MAP} = \left( \frac{\text{SBP} + (2 \times \text{DBP})}{3} \right) \]

OSMOLALITY (SERUM, ESTIMATED)

Calculated serum osm = \((2 \times [\text{Na}^+]) + ([\text{glucose}] / 18) + ([\text{BUN}] / 2.8)\)

- Causes of increased osmolal gap: decreased serum water, hyperproteinemia, hypertriglyceridemia, and presence of unmeasured osmoles (e.g., sorbitol, glycerol, mannitol, ethanol, isopropyl alcohol, acetone, ethyl ether, methanol, and ethylene glycol).

OSMOLAL GAP

Osmolal gap = measured \( S_{\text{osm}} \) – calculated \( S_{\text{oosm}} \)

- Good marrow response = 3.0–6.0
- Borderline response = 2.0–3.0
- Inadequate response = <2.0

RETICULOCYTE INDEX

Reticulocyte index = \( \frac{[\text{measured reticulocyte count} \times (\text{measured Hct}/45)]}{\text{maturation factor}} \)

Maturation factor = 1 + \((0.5 \times [(45 – \text{Hct})/10])\)

- Good marrow response = 3.0–6.0
- Borderline response = 2.0–3.0
- Inadequate response = <2.0
• The letters in the following refer to a standard $2 \times 2$ table presented in Table 5-1.

• **Sensitivity**: The percentage of patients with the target disease/condition who have a positive result $[A/(A + C)]$. The greater the sensitivity, the more likely the test will detect patients with the disease. High-sensitivity tests are useful clinically to **rule OUT** a disease (SnOUT) (i.e., a negative test result would virtually exclude the possibility of the disease).

• **Specificity**: The percentage of patients without the target disease/condition who have a negative test result $[D/(B + D)]$. Very specific tests are used to confirm or **rule IN** the presence of disease (SpIN).

• **Positive predictive value** (PPV): The percentage of persons with positive test results who actually have the disease/condition $[A/(A + B)]$.

• **Negative predictive value** (NPV): The percentage of persons with negative test results in which the disease/condition is absent $[D/(C + D)]$.

• **Number needed to treat** (NNT): The number of patients who need to be treated to achieve one additional favorable outcome; calculated as $1/absolute\ risk\ reduction\ (ARR)$, rounded up to the nearest whole number.

• **Number needed to harm** (NNH): The number of patients who, if they received the experimental treatment, would lead to one additional person being harmed compared with patients who receive the control treatment; calculated as $1/absolute\ risk\ increase\ (ARI)$.
<table>
<thead>
<tr>
<th></th>
<th>Disease</th>
<th>No Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Positive</strong></td>
<td>True positive (TP)</td>
<td>False positive (FP)</td>
</tr>
<tr>
<td></td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td><strong>Negative</strong></td>
<td>False negative (FN)</td>
<td>True negative (TN)</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>D</td>
</tr>
</tbody>
</table>

All with positive test = A + B
Positive predictive value (PPV) = \[
\frac{A}{A + B}
\]

All with negative test = C + D
Negative predictive value (NPV) = \[
\frac{D}{C + D}
\]

Sensitivity = \[
\frac{A}{A + C}
\]
Specificity = \[
\frac{D}{B + D}
\]
WORKING WITH ANCILLARY STAFF

• Give specific directions and use your judgment, but also give others a chance to make suggestions and solve problems. Effective use of ancillary staff can greatly increase your efficiency (see Table 6-1).

• A compliment for a job well done goes a long way (others are overworked too), and you will be remembered when you need help. When someone performs exemplary work, let his or her supervisor know.

• Criticize in private. When done, offer only nonjudgmental and constructive feedback.

• Regard ancillary staff as fellow members of the patient care team; they are often “bothering” you out of concern for the patient and not to harass you. They have valuable insight that often proves important in patient care.

• Make efforts to let team members know the plan can save you phone calls and increase sleep.

REFERRING A PATIENT

• When referring a patient to the ED or another physician, or transferring a patient, always make a courtesy call first.

• Pertinent information includes the following:
  • Who you are.
  • Patient identification information.
  • Succinct history of the problem.
  • Supporting lab data.
  • Suggestions for further evaluation.
  • Likely disposition of the patient.
  • A contact number where you or someone covering for you can be reached for questions or follow-up information.
<table>
<thead>
<tr>
<th>Resources</th>
<th>Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nursing supervisor</td>
<td>Individual or joint meetings with or without loved ones (useful for any situation below)</td>
</tr>
<tr>
<td>or manager</td>
<td></td>
</tr>
<tr>
<td>Social worker</td>
<td>Initial evaluation discharge plan. This includes nursing home, rehab, or extended care facility placement</td>
</tr>
<tr>
<td>Case coordinator</td>
<td>Return to nursing home issues; durable medical equipment; insurance disputes and concerns</td>
</tr>
<tr>
<td></td>
<td>Home health/home infusion/hospice referrals</td>
</tr>
<tr>
<td></td>
<td>Transportation issues</td>
</tr>
<tr>
<td>Risk management</td>
<td>For litigious patients/family members, if you have concerns about the case, or if you expect a poor outcome</td>
</tr>
<tr>
<td>Ombudsman or ethics</td>
<td>Helpful if there are disputes between physicians or family members</td>
</tr>
<tr>
<td>committee</td>
<td></td>
</tr>
<tr>
<td>Family members or loved</td>
<td>Proceed cautiously; if the family is in disagreement, try to remain neutral while defending the patient’s wishes</td>
</tr>
<tr>
<td>ones</td>
<td></td>
</tr>
<tr>
<td>Religious resources</td>
<td>For spiritual support and issues related to death and end of life</td>
</tr>
<tr>
<td>A new physician</td>
<td>A new physician may be the best solution if other options have failed</td>
</tr>
</tbody>
</table>
WORKING WITH DIFFICULT PATIENTS

• Be proactive and address potential concerns, expectations, or questions upfront (see Tables 6-2 and 6-3). Checking in with the patient at regular intervals builds rapport and can save you from multiple phone calls. Try to minimize waiting time and interruptions during meetings.

• Be as flexible and as accommodating as you can. Recognize that the patient may be tired of repeating his or her history or having a physical examination.

• Let the patient (and loved ones) know about the management plan at least once a day. Inform him or her of the test results and changes in the plan, and let him or her know if consultants will be coming by.

• Avoid promises that you cannot fulfill. This includes the time of discharge or the time of a diagnostic procedure.

• If more than one service is involved, designate someone to be the primary source of communication to avoid confusion.

• The patient’s “difficult” behavior may stem from lack of control over decision making and the situation or lack of insight into his or her medical condition. Past experiences, things the patient may have seen or read in the media, and fear may also play a role. Active listening, acknowledging the patient’s point of view, and reassurance can go a long way.

• Acknowledge your own frustration, seek the advice of others when necessary, and always try to do what’s best for the patient.
<table>
<thead>
<tr>
<th>Problems</th>
<th>Suggestions</th>
<th>Potential Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abuses of the system (i.e., narcotics)</td>
<td>Set boundaries; written contracts are often helpful (be specific in stating the problem and plan)</td>
<td>Notify all members of the team caring for the patient Document concisely in the chart and include on sign out See above</td>
</tr>
<tr>
<td>Manipulative patients</td>
<td>See above Coordinate care through one team member to maintain consistency</td>
<td>See above Contact security or law enforcement officials if necessary Arrange to have security nearby when you see the patient</td>
</tr>
<tr>
<td>Violent patients (see Psychiatry, Chapter 21)</td>
<td>Safety first Tell others you are seeing the patient Try to remain calm and neutral Always stand between the patient and the door Remove all potentially dangerous items that can be used against you (i.e., stethoscope, necktie)</td>
<td>See above</td>
</tr>
<tr>
<td>Patients who want to leave against medical advice (AMA)</td>
<td>First, establish decisional capacity (see Psychiatry, Chapter 21) Then, listen to the patient’s reason(s).</td>
<td>Have patient sign AMA form Carefully document discussion of risks and benefits in the chart</td>
</tr>
</tbody>
</table>

(Continued)
<table>
<thead>
<tr>
<th>Problems</th>
<th>Suggestions</th>
<th>Potential Actions</th>
</tr>
</thead>
</table>
| Homelessness or return to abusive situations | Respond in a nondefensive, non-judgmental manner  
  Calmly explain the risks and consequences of leaving  
  Explain the benefits of staying and the importance of completing the diagnostic and treatment plan  
  Be accommodating if possible  
  Enlist the help of other team/family members  
  Social work is a helpful resource | Refer patients to local shelters or to local shelters/safe havens  
  Contact proper authorities (i.e., police, Division of Aging, Child and Family Services)  
  Careful, neutral documentation in the medical record  
  Emphasize that it may be a temporary stay  
  Try to arrange for close outpatient follow-up, home health services, and other family members to check in and help |
| Refusal of nursing home placement | Social work, family members, and other team members can assist and discuss with patient  
  Consider rehabilitation as a potential place for referral | Try to arrange discharge medications and follow-up plans  
  Advise the patient to seek medical attention again if condition worsens  
  Notify the attending of record of AMA discharge |
<table>
<thead>
<tr>
<th>Problems</th>
<th>Suggestions</th>
<th>Potential Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>High likelihood that patient will abuse substances again</td>
<td>Consider chemical dependency consult</td>
<td>Ultimately, you may have to respect the patient's wishes</td>
</tr>
<tr>
<td>Concern for suicide or homicide (see Psychiatry, Chapter 21)</td>
<td>Consider psychiatry consultation, assess competency</td>
<td>Educate the patient on hazards of substance abuse</td>
</tr>
<tr>
<td>Financial difficulties</td>
<td>Social workers or case coordinators can be very helpful</td>
<td>Consider inpatient or outpatient follow-up</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Document carefully in the chart</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Place patient on suicide precautions with sitter</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Consider possible transfer to inpatient psychiatric setting</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Notify family members and loved ones</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Payment and transportation arrangements can be made</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Referral to free clinics and resources</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Assistance with applying for Medicaid, disability, etc.</td>
</tr>
<tr>
<td>Issue</td>
<td>Suggestions</td>
<td></td>
</tr>
<tr>
<td>---------------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Code status</td>
<td>Explain it in a language the patient and family members can understand</td>
<td></td>
</tr>
<tr>
<td>Intubation</td>
<td>Remain neutral, although it is appropriate to give your opinion, especially if asked</td>
<td></td>
</tr>
<tr>
<td>CPR</td>
<td>Be specific and clear (i.e., the exact interventions to be done or not to be done) in your discussion and document clearly in the medical record</td>
<td></td>
</tr>
<tr>
<td>Vasopressors</td>
<td>Communicate status with other team members</td>
<td></td>
</tr>
<tr>
<td>Cardioversion</td>
<td>Give sufficient time to consider the decision, and explain this decision can be changed.</td>
<td></td>
</tr>
<tr>
<td>Antibiotics and other</td>
<td>Be available for further discussion and questions.</td>
<td></td>
</tr>
<tr>
<td>medications</td>
<td>If patient is not able to discuss this with you, contact primary physician and family members, power of attorney</td>
<td></td>
</tr>
<tr>
<td>Nutrition (i.e., G-tubes)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phlebotomy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IV lines</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Withdrawal of support</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Comfort care</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Patient safety is a priority for all entities of patient care from physicians, to nurses, to ancillary staff. The most important advice an intern can follow concerning patient safety is to feel comfortable in your decision making, and if you don’t feel comfortable, seek help from your upper-level residents or attending physician. A little bit of caution and mindfulness can prevent unnecessary errors and potential patient harm.

**MOST COMMON MEDICAL ERRORS**

- **Medication errors**
  - These are the errors most commonly encountered on the ward and also the most avoidable. The most common medication errors include missed doses, wrong dosages, infusion errors, patient allergy, and orders entered on the wrong patient.
  - When discharging patients, it is important to ensure that all medications are reconciled, prescriptions are correctly filled out, and the patient has adequate instructions for use.

- **Procedure-related errors**
  - Be sure to obtain proper informed consent.
  - The most common errors encountered during procedures include incorrect patient/site, failure to review labs, line infections, incorrect documentation, and periprocedural adverse events. You should always review pertinent labs/imaging, including platelet count, and INR prior to every procedure to reduce bleeding risk.
  - A “time out” should be done before all but trivial procedures.
  - Central line–associated bloodstream infections are an unfortunate complication of procedures, but they can be minimized with sterile technique and adequate preparation.
  - Residents are always available to help/supervise any procedures that you are not comfortable with.
• Ancillary staff communication
  • An integral part of any admission is open and continued
    communication with all participants of patient care.
  • Be sure to set a comfortable climate in which other healthcare
    providers can express their opinions and concerns.

• Prophylactic measures
  • Should the patient be in some form of isolation?
  • Does the patient need DVT/PE prophylaxis?
  • Are fall precautions indicated?
  • Is the patient at risk for delirium and/or substance withdrawal?
  • Are there unnecessary lines (e.g., bladder catheters, peripheral
    IVs, peripherally inserted central lines, central lines) that are
    sources of potential infection?

ERROR DISCLOSURE

Errors are bound to occur, and expedient identification, correction, and disclosure will prevent further escalation. It is important to disclose medical error/mistakes and explain how the error occurred and what you plan to do to correct the error. Studies have shown that patients are more receptive to open admittance of errors than to attempts at concealment. Medical errors must also be relayed to not only your supervising resident but also charge nurses on the floor and your institution’s risk management department.
• The goal of risk management is to improve the quality of patient care and reduce the liability to the healthcare provider.

• Physicians are obligated to inform patients when there is an adverse event, even if the patient was not noticeably harmed.

• Disclosing adverse events should be done with the help of your attending physician and risk management (see Table 8-1). Reporting adverse events not only is important to patient care but also helps identify major system flaws.

DEFINITIONS

• Adverse event: any incident, therapeutic error, iatrogenic injury, or other undesirable occurrence that caused probable or definite harm to a patient.

• Disclosure of adverse events: an honest and empathetic discussion of clinically significant facts between providers and the patient about the occurrence of an adverse event that resulted or could have resulted in patient harm.

• Sentinel event: unexpected occurrence involving death or serious physical or psychological injury, or the risk thereof. Requires immediate investigation and response.

HOW TO RESPOND TO A POTENTIAL INCIDENT

• Arrive as soon as possible.

• Avoid assigning blame to an individual.

• Never argue in public or in the chart.

• Document the facts.

• Report injuries.

• Never state in the medical record that an incident report was completed.
### TABLE 8-1  WHEN TO CALL RISK MANAGEMENT

<table>
<thead>
<tr>
<th>Event</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint commission mandated sentinel event</td>
<td>Unanticipated death or major permanent loss of function</td>
</tr>
<tr>
<td></td>
<td>Suicide in hospital or within 72 hours of discharge</td>
</tr>
<tr>
<td></td>
<td>Unanticipated death of full-term infant</td>
</tr>
<tr>
<td></td>
<td>Abduction of any patient</td>
</tr>
<tr>
<td></td>
<td>Discharge of an infant to the wrong family</td>
</tr>
<tr>
<td></td>
<td>Rape</td>
</tr>
<tr>
<td></td>
<td>Hemolytic transfusion reaction involving major blood group incompatibilities</td>
</tr>
<tr>
<td></td>
<td>Surgical or nonsurgical invasive procedure on the wrong patient, wrong site, or wrong procedure</td>
</tr>
<tr>
<td></td>
<td>Severe neonatal hyperbilirubinemia (bilirubin $&gt;30$ mg/dL)</td>
</tr>
<tr>
<td></td>
<td>Prolonged fluoroscopy (cumulative dose $&gt;1,500$ rads) or radiotherapy to wrong body region or $&gt;25%$ above planned dose</td>
</tr>
<tr>
<td>When an error has been made</td>
<td>Informed consent</td>
</tr>
<tr>
<td>When patient makes threats of litigation</td>
<td>Confidentiality</td>
</tr>
<tr>
<td>Unexpected death or outcome</td>
<td>Disclosure</td>
</tr>
<tr>
<td>Any situation that could turn into a legal or risk issue</td>
<td></td>
</tr>
</tbody>
</table>

### HOW TO REPORT AN ERROR

- Call risk management at your institution.
- Complete an incident report.
- Discuss the situation with your resident and attending as soon as possible (see Table 8-1).
HOW TO DISCLOSE AN ADVERSE EVENT

• Speak with your resident, attending physician, and risk management first.
• Your attending should coordinate disclosure efforts and speak with the patient and family.
• If more than one service is involved, confer and collaborate on the disclosure conversation. The medical care team should deliver a consistent message.
• Give the patient and family an honest, straightforward, and prompt explanation of what occurred.
• You can apologize for the error.
• If the cause of error is uncertain, do not speculate or hypothesize. State the facts. Tell the patient that further investigation is necessary.
• Identify who will be involved with ongoing care. You may need to transfer the patient’s care if the patient–physician relationship has been compromised.
• Tell the patient and family what steps are being undertaken to prevent further error.
• Allow time for questions.
• Speak in layman’s terms.
• Be aware of body language.
• Document the discussion with the family
  • Time, date, and place
  • Names and relationships of those present
  • Documentation of discussion of event
  • Patient/family response

DOCUMENTATION

• Documentation is vitally important whenever there has been an adverse event.
• Prior to writing a note, give yourself a few minutes to recap what happened. Remember: “If it wasn’t documented, it wasn’t done.”

Do

• Record changes in patient’s condition and response to treatment.
• Add addendums when necessary.
• Write legibly.
• Use factual and objective language.
• Use only approved abbreviations.
• Chart patient nonadherence.
• Show your thought process.
• If you consider something potentially serious and rule it out, say so and why.
• Write plans for future treatment.

Don’t
• Alter documentation.
• Use imprecise terms.
• Write negative comments about the patient or family.
• Document disputes with other care providers.

INFORMED CONSENT
• This includes the type of procedure, reason or indication for the procedure, benefits and risks of the procedure with disclosure of incidence and severity of complications, and explanation of any alternative procedures.
• Must be obtained from a competent patient, legally appointed guardian, or durable power of attorney for health care.
• When a patient is unable to consent, then consent can be obtained from a spouse, parent, adult children, siblings, or grandparents.
• Two physicians can give consent in a life- or limb-threatening emergency.
• Must obtain consent of parents or legal guardians for minors.
• Minors can consent for themselves if being treated for chemical dependency, STDs, or pregnancy (excepting elective abortion in some states).
• It is also important to document informed refusal of a treatment/procedure.
Triage

BEFORE ACCEPTING ADMISSIONS

1. Obtain demographic information: name, date of birth, medical record number, current location, and attending physician.
2. Why does the patient need to be admitted? What is the admitting physician’s assessment?
3. Is the patient competent, and does he or she want to be admitted?
4. What are the patient’s chief complaints, comorbidities, relevant past medical history, and brief current history?
5. When was the last previous admission? Obtain old medical records (inpatient and outpatient). If the patient is coming in transfer, be sure to acquire all radiographs and lab results.
6. Obtain most recent vital signs, pertinent examination including mental status, key lab data, CXR, ECG, and code status. Review as many lab results and films in the ED as you can.
7. Confirm IV access.
8. Inquire about major interventions performed, medications given, and consultations pending.
9. What follow-up is necessary (i.e., lab tests that are pending, consults that need to be called, blood transfusions, antibiotics)?
10. Find out who the primary physician is and if this person has been notified.
11. Do family members need to be called?

OTHER IMPORTANT QUESTIONS TO CONSIDER

• Is this an appropriate admission for your service (i.e., Is there something you can do for the patient that no one else can do? Does a different service make more sense?)?
• Can this patient be managed as an outpatient? If yes, social services may need to be involved. In addition, arranging follow-up is crucial.
• Is the patient stable enough for the floor or for transfer from an outside hospital? Are any more treatments needed before transfer (i.e., nebulizer treatments or blood transfusions)?
• Can your staff adequately handle this patient?
• What specific interventions does this patient need that other institutions cannot provide (in the case of a hospital-to-hospital transfer)?
• Obtain collateral information from family, nursing homes, or other caregivers. Always collect and hold onto important phone contacts.
1. When called with a new admission, it is critical to review old records. However, if the patient is accessible, always see the patient first and check vital signs before digging through his or her chart. With that said, **old records are invaluable**. Most systems have old lab results, discharge summaries, and H&Ps stored as electronic versions; use these extensively, but always confirm with your own eyes and ears.

2. After assessment of the patient and examination, **admission orders should be completed as soon as possible**. This will help the nursing staff and will enable you to get appropriate lab results in a timely manner. If you need stat labs, always inform the nursing staff directly. It is also helpful to inform the nurses when your orders are complete. Telemetry orders should also be completed as soon as possible, if needed.

3. Taking a history and performing the physical examination should be well engrained by now. It is often helpful to **type or dictate the H&P right after evaluating the patient before moving on to your next admission**. If you decide to dictate, you must ensure a signed copy of the dictation makes it into the medical record chart. **A short handwritten holding note** of the current admission problems and short assessment should also be entered in the chart while awaiting the dictated H&P. Lab results can be added to the dictation as an addendum.

4. If the patient has a private primary care physician, he or she should be notified as soon as possible regarding the admission, and your plan should be communicated to the private physician. **Many private physicians or their covering partners like to be notified as soon as possible**, regardless of the time of night.

5. In summary, remember the three pearls of an admission:
   - Assess the stability of the patient immediately.
• Obtain a good H&P, even if this has already been done by another medical team.
• Write orders as soon as possible. This makes the nurses and unit clerks happy and allows you to get the lab data you need to finish your evaluation.

ADMISSION ORDERS

• Many admission diagnoses have preset clinical pathways and associated order sets (i.e., CHF, asthma), which are often helpful. Also, consider the patient’s eligibility for appropriate research studies.
• The mnemonic ADC VAANDISML may be useful:
  • Admit to ward/attending/house officers
  • Diagnosis
  • Condition
  • Vitals: e.g., routine, every shift, every 2 hours. Always include call orders (i.e., call HO for SBP >180 or <90, pulse >130 or <60, RR >30 or <10, T >38.0°C, O₂ saturation <92%)
  • Allergies and reactions
  • Activity (ad lib, bed rest with bedside commode, up to chair, etc.)
  • Nursing (strict I/O, daily weights, guaiac stools, blood sugars, Foleys, etc.)
  • Diet (NPO, prudent diabetic, low fat/low cholesterol, renal, low salt, etc.)
  • IV (IV fluids, heplock)
  • Special (wound care, consults with social work, dietitian, and PT/OT)
  • Meds: All medications should include dosage, timing, route, and indications. Don’t forget prn meds or you will be called often; if no contraindications, consider including acetaminophen, bisacodyl, docusate, and aluminum and magnesium hydroxide (Maalox).
  • Laboratory (including a.m. labs).
• Don’t forget DVT prophylaxis for every patient who is not ambulating and GI prophylaxis for critically ill patients (see below for guidelines)!
DVT PROPHYLAXIS

• Indications: patients with one or more risk factors for DVT and confined to bed; critical care patients.

• Risk factors for DVT: cardiac dysfunction (heart failure, arrhythmia, MI), malignancy, surgery, trauma (especially orthopedic), previous DVT/PE, obesity, smoking, age >40 years, inflammatory disease (e.g., inflammatory bowel disease, lupus), nephrotic syndrome, pregnancy or postpartum within 6 weeks, immobility, acquired/genetic thrombophilia, chronic lung disease, ischemic stroke, serious infections, or indwelling central venous catheter.

• Contraindications to pharmacologic prophylaxis: heparin-induced thrombocytopenia; active bleeding; preoperative within 12 hours or postoperative within 24 hours; LP or epidural within 24 hours; recent intraocular or intracranial surgery; coagulopathy.

• Recommended regimens (for medical patients):
  • Low-molecular-weight heparin (LMWH): enoxaparin 40 mg subcutaneous qday (adjust dosage for CrCl <30 mg/dL, contraindicated in ESRD) or dalteparin 5,000 units subcutaneous qday.
  • Factor Xa Inhibitor: fondaparinux 2.5 mg subcutaneous qday.
  • Unfractionated heparin (UFH): for patients <100 kg: 5,000 units subcutaneous q8h, for patients >100 kg: 7,500 units subcutaneous q8h.
  • For patients at high risk for bleeding, consider intermittent pneumatic compression or graduated compression stockings.

• For planned invasive procedures (e.g., pacemaker placement, catheterization, surgery), hold UFH 8 hours prior to procedure and LMWH 12 hours prior to procedure!

GI PROPHYLAXIS

• Gastric erosions and stress-induced ulcers can form in critically ill patients. However, not every patient needs GI prophylaxis—if patients do not have any of the risk factors listed below, prophylaxis is not necessary, even in the ICU setting! Most patients will not need GI prophylaxis.
Risk factors for stress-induced ulcers: mechanical ventilation >48 hours, coagulopathy, shock, sepsis, multi-organ system failure, hepatic failure, multiple trauma, burns over >35% of total body surface area, organ transplant recipient, head trauma, spinal cord injury, history of peptic ulcer disease or upper GI bleeding, use of anticoagulants or high-dose corticosteroids.

Recommended regimens:
- H₂ blockers: e.g., famotidine 20 mg PO/IV bid or ranitidine 50 mg PO/IV tid
- Proton pump inhibitors: e.g., omeprazole 40 mg PO qday

**ASSESSMENT/PLAN**

- This is the most important part of your note. It is useful to separate this section by problem. The assessment should include a one-line summary of the patient’s known medical problems (i.e., HTN, T2DM, CAD) and those under evaluation (i.e., fever, melena). For example, 60 y/o female with a history of hypertension, T2DM presents with new-onset chest pain. Include a short differential diagnosis of the current problem.
- The plan should be separated by problem. Cover all problems, including stable issues:
  1. Chest pain: No ECG changes, chest pain free now, will rule out MI, monitor on telemetry, continue β-blocker, nitrates, ASA, and ACE-I. NPO for stress thallium in AM assuming rules out for MI.
  2. Hypertension: Good control on current medical regimen.
  3. T2DM: Good control with A1C of 6.5%. Continue glucose checks, prudent diabetic diet. Hold PO diabetic meds while NPO. Will use insulin sliding scale while NPO.
  4. Fluids/electrolytes/nutrition (F/E/N): Monitor I/Os, urine output.
  5. Vascular access: Note patient’s sites of IV access.
  6. Prophylaxis: Indicate plans for DVT prophylaxis and GI prophylaxis if indicated (see above).
  7. Disposition: Note any anticipated discharge needs (nursing home placement, home health, home).
  8. Code status: Code status should be addressed with every patient admitted regardless of age or disease. Unexpected problems arise too often, and it is better to be prepared.
LABORATORY RESULTS AND ORDERS

It is imperative that orders and lab tests are followed up in a timely manner. You must take personal responsibility to ensure that this is completed.

PATIENT SAFETY ISSUES

Restraints

- Restraints may be needed for patients in a variety of situations. Indications for restraints include the following:
  - Protecting patients from harming themselves (e.g., self-extubation, pulling at Foley catheter, pulling at IV lines)
  - Protecting staff and/or family from patient violence
  - Facilitating medically necessary procedures
  - Preventing disoriented patients from wandering or falling
- Written orders for restraints must include the following:
  - Type of restraint and site (e.g., soft limb restraints on upper extremities, mittens)
  - Start and end times
  - Frequency of monitoring and reevaluation
- The medical reason for restraint use must be clearly documented in the chart. Patients should be reevaluated at least every 24 hours and orders renewed if necessary.
- If possible, consider the use of bedside sitters, bed alarms, veil beds, low beds, floor mats, etc., instead of physical restraints. Likewise, chemical restraints (e.g., antipsychotics and low-dose benzodiazepines) should be used only when clearly indicated. Most hospitals have written policies regarding the use of restraints—be sure your orders and documentation comply with hospital policies.

Dangerous Abbreviations for Order Writing

Each hospital may have its own list of unacceptable or dangerous abbreviations. Table 10-1 shows some of the most common ones.
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Intended Meaning</th>
<th>Misinterpretation/ Common Error</th>
<th>Correction</th>
</tr>
</thead>
<tbody>
<tr>
<td>U</td>
<td>Units</td>
<td>Mistaken for the numbers “0” or “4” or “cc”</td>
<td>Write “unit”</td>
</tr>
<tr>
<td>IU</td>
<td>International units</td>
<td>Mistaken for “IV” or the number “10”</td>
<td>Write “International Units”</td>
</tr>
<tr>
<td>Q.D. or Q.O.D</td>
<td>Daily or every other day</td>
<td>Mistaken for each other or QID</td>
<td>Write “Daily” “every other day”</td>
</tr>
<tr>
<td>Trailing zero or lack of leading zero</td>
<td>X.0 mg or .X mg</td>
<td>Decimal point is missing</td>
<td>Write X mg</td>
</tr>
<tr>
<td>MS, MSO, and MgSO</td>
<td>Morphine sulfate or magnesium sulfate</td>
<td>Confused for one another</td>
<td>Write “morphine sulfate”</td>
</tr>
<tr>
<td>μg</td>
<td>Microgram</td>
<td>Mistaken for “mg”</td>
<td>Use “mcg” or “microgram”</td>
</tr>
<tr>
<td>AU, AS, AD</td>
<td>Latin abbreviation for both ears, left ear, right ear</td>
<td>Mistaken as the Latin abbreviation “OU” (both eyes); “OS” (left eye); “OD” right eye</td>
<td>Write “both ears”; “left ear” or “right ear”</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
<td>Clarification</td>
<td></td>
</tr>
<tr>
<td>--------------</td>
<td>---------------------------</td>
<td>-------------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>cc</td>
<td>Cubic centimeter</td>
<td>Misread as “U” (Units). Use “mL”, “ml”, or write out “cubic centimeters” or “milliliters”</td>
<td></td>
</tr>
<tr>
<td>HS</td>
<td>Half strength or</td>
<td>Confused for either half strength or at bedtime. qHS mistaken for every hour.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Latin abbreviation for</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>bedtime</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TIW</td>
<td>Three times a week</td>
<td>Misinterpreted as “three times a day” or “twice a week”.</td>
<td></td>
</tr>
</tbody>
</table>

From Barnes-Jewish Hospital Department of Pharmacy. St. Louis: Washington University Medical Center; 2010.
Many programs categorize rounds into prerounds, work rounds, and attending rounds.

Prerounding

• Prerounding is primarily an intern’s responsibility.
• Usually allow 30 minutes to an hour before rounds, depending on the number of patients on your service.
• The exact responsibilities should be worked out individually with your resident.
• It is often not necessary to physically see all of your patients before work rounds. However, it is customary to see those patients with an acute problem.
• The most important tasks for prerounding are getting sign-out and catching up on the overnight events (i.e., cross-cover problems). The following example is a good general prerounding plan:

1. Get your sign-out from the night float or cross-cover team. You need to be aware of any major events that happened overnight, this will dictate how you will spend your time prerounding. Check charted vital signs on all your patients. It is also helpful to check nursing and event notes on the computer.

2. Check lab results and final results of tests (i.e., CXR, echo). Check telemetry every day on all your monitored patients.

3. See the patients. A quick check on your patients (2 to 3 minutes per patient) allows you to see how they look and if they have developed any new problems overnight. Of course, patients with more acute illness require more time.

4. For patients with private physicians, it is often helpful to discuss the plan face-to-face with them in the morning.
Daily Assessments

(i.e., try to catch them on their morning rounds). This saves you time in trying to reach them at their offices or in deciphering their progress notes.

**DAILY NOTES AND EVALUATION**

- Interns are primarily responsible for writing daily notes on each of their patients. The SOAP format is usually used for daily notes:
  - **Subjective**: Events over the past 24 hours garnered from the patient, cross-covering physician, or nursing staff.
  - **Objective**: Factual information, vitals, PE, lab results, lines, and tubes. Include microbiology results, X-rays, and other studies here. Always check final official readings of tests.
  - **Assessment/Plan**: This is the most important part of the note. It is usually categorized by problem or organ system in the order of importance. Remember to include problems such as electrolyte abnormalities, hypovolemia/hypervolemia, and nutritional status in your problem list and document code status in every note. Also include the type of IV access the patient has and the plan for DVT prophylaxis. In addition, the last category or problem should be discharge planning. Include status and goals (e.g., social work arranging placement, home oxygen).
  - Active medications are often listed in a side column. This exercise can be tedious but ensures that every medication is reviewed daily. Also include the number of days each antibiotic has been given and the expected total duration. Similar documentation can be used with other medications that require loading doses or are tapered.
  - Review the following items daily:
    - Do IV lines need to be changed?
    - Can IV meds be changed to PO?
    - Can you discontinue the Foley?
    - Do restraint orders need to be renewed?
    - Can you advance the diet and increase the activity of the patient? Is the patient moving his or her bowels? Is there any procedure or test planned that requires that patient to be NPO?
    - PT/OT and social work: Are they involved, and should they be? What is the status of discharge planning?
    - Are all meds adjusted for renal or hepatic failure?
Daily orders should be consolidated and written as early as possible. Don’t forget to order a.m. labs for the next morning (only if they are truly indicated). Every lab test and study ordered needs to be followed up. If a study needs to be done stat or ASAP, you must notify the ward clerk and nurse directly and consider talking to the radiologist directly. It is helpful to discuss a brief plan with the patient and the nursing staff. This helps them to be part of the team and also helps move things along.

SIGN-OUTS

Background
• The initiation of work-hour regulations in 2003 increased the number of physician-to-physician transfers.
• Patients cared for by a team other than their primary team are at a higher risk for adverse events. Poor sign-out processes can lead to inefficiencies, delays in care, cost increases, and harm. Negative outcomes can be mitigated by formal sign-out systems.
• Complex systems fail unless communication can be standardized. Atul Gawande, MD, demonstrates that a standardized checklist decreased the rate of major complications by 36%, deaths by 47%, and infections by nearly 50%.

Necessary Information for Effective Sign-Out
• Using a standardized format for each patient will make it easy to ensure all necessary information is included in the written sign-out.
• This checklist was adapted by Barnes-Jewish Hospital interns from a study at three major centers that compiled focus group information from residents on what is required for effective cross-cover.

Administrative Data
• Patient name, DOB, medical record or hospital number, location, admission date
• Code status
• Access
• Acuity (i.e. sick)

Background Data
• Brief HPI (1 to 2 lines).
• Admitting diagnosis/major problem, interventions tried (successful, unsuccessful).
• Other significant problems, interventions tried (successful, unsuccessful).
Daily Assessments

- Significant events of the day.
- Antibiotic dates (if applicable).
- Trends.
- Goals of care/of the hospitalization.

Cross-Cover
- Anticipatory guidance with specific if–then statements.
- Specific tests ordered that will come back during the coverage period.

Verbal Communication
- Face-to-face communication between outgoing and oncoming care providers.
- Verbally discuss each patient, tailoring conversation to acuity and complexity of the situation.
- Preferably sign-out should occur in a designated location that can allow the two, or more, physicians caring for the patients to be fully engaged in the process with minimal distraction.

DISCHARGE PLANNING

- Discharge (D/C) planning must be addressed and readdressed constantly. Proper D/C planning prevents large censuses and results in a more manageable workload for the resident and intern. D/C planning should start on admission.
- Social work should be consulted on admission if D/C needs are anticipated (assisted living, placement, transportation). Scheduled meetings with case coordinators or social workers are often helpful to reassess the situation and provide updates.

REFERENCES

• With proper planning, discharges can be smooth for you and the patient.
• In today’s health-care system many more diseases are being managed and followed in the outpatient setting. Therefore, it is critical that the patient has follow-up and has a completely reconciled medication list. In addition, the patient’s physician must be aware of any pending issues, studies, or laboratory draws scheduled prior to outpatient follow-up. Communication with all involved parties is crucial to a successful discharge process and ultimately prevents many “bounce backs.”
• Significant reimbursement disincentives will soon go into effect for those with MI, CHF, and pneumonia and who are readmitted within 30 days (for any reason!). Other diagnoses are likely to follow.

**DISCHARGE PROCESS/PEARLS**

• Obtain social work/case coordinator assistance early in the admission. Try to anticipate issues and problems early on (e.g., transportation, home oxygen, home antibiotics, or nursing/rehabilitation facility placement).

• **Make sure the patient and his or her family are aware of possible discharge dates** so they can arrange their schedules and not be caught off guard.

• **Arrange for home care services at least 24 hours before discharge** (i.e., home nursing, home laboratory draws, PT, OT).

• **Criteria for home O₂:**
  • \( \text{PaO}_2 < 55 \text{ mm Hg} \), must be measured within 48 hours of discharge.
  • \( \text{O}_2 \text{ sat} < 88\% \) on room air consistently (at rest, with exercise, or with sleep).
  • \( \text{PaO}_2 \) of 55 to 59 mm Hg or \( \text{O}_2 \text{ sat} < 89\% \) with evidence of cor pulmonale or secondary polycythemia (Hct >55%).

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12 Discharges
Discharges 45

- Discontinue Foley catheters, sitter, telemetry monitoring, and supplemental oxygen as soon as possible. Many rehabilitation facilities require that these are not present 24 hours before discharge.
- Change antibiotics and diuretics to PO the day before discharge. Avoid a.m. lab work the morning of discharge, unless absolutely necessary.
- Provide prescriptions for a 1-month supply of all medications, excluding controlled substances unless absolutely necessary.

Reconcile discharge medications with admission medications.

Make a note in the discharge summary of the patient’s current medication list as well as any medications that were discontinued or dose-adjusted during the admission. This paperwork can often be done in advance.

- Dictate the discharge summary at the time of discharge. It may seem painful at the time, but it will save you time later and prevent frustration on outpatient follow-up. It is most efficient to dictate when you are most familiar with the patient and hospital course. Take the extra 5 to 10 minutes to complete it now.
- The hospital course section of a well-organized discharge summary is generally organized by problem list.

DISCHARGE SUMMARY

Each institution has its own rules on discharge summaries. However, most should include the following items:

- Your name, the attending physician’s name, and patient name and medical record number.
- Date of admission and discharge.
- Principal and secondary diagnoses and procedures.
- Chief complaint, HPI, and allergies.
- Hospital course, including all major events, listing of major radiological and diagnostic tests and results, and all major therapeutic interventions.
- Discharge medications, diet, and activity. Again, make sure to reconcile admission and discharge medications and ensure that your dictated list is correct. Errors in this regard are a major cause of unanticipated outcomes and readmissions.
- Follow-up plans, including dates and times of outpatient appointments/studies.
- Condition on discharge.
- Copy distribution. Be sure to include any physicians outside your healthcare system who do not have access to your electronic documentation.
GENERAL POINTS

• For the first few months of residency, whenever you are called about a patient, go examine them, review the chart, and assess the situation. Communicate your impression and plan with the nursing staff and write a brief event note in the chart. Continue to do this until you feel comfortable deciding what situations can be adequately handled over the telephone with the nurses and the support staff.

• If there is any doubt, see the patient. The patient is always your number one priority.

• Once you have seen the patient, write an event note (this can be brief, depending on the situation). Things to document include the following:
  • The reason you were called to see patient (e.g., CTSP for chest pain).
  • A summary of the situation. This includes the patient’s general appearance, vitals, pertinent physical examination, pertinent laboratory, and imaging data.
  • A synopsis of your plan. This includes all medical decision making such as diagnostic tools you ordered and medications provided.
  • Outcome.

REASONS YOU MUST GO SEE A PATIENT

• Any major changes in clinical status:
  • Altered mental status or other changes in neurologic state
  • Dyspnea
  • Chest pain
  • Severe abdominal pain
  • Seizures
• Uncontrolled bleeding (hemoptysis, hematemesis, lower GI bleed, hematuria, vaginal bleeding)
• Intractable vomiting
• Severe headache
• New onset of pain
• Falls
• Any major changes in vital signs:
  • Oxygen desaturation
  • Hypotension
  • Arrhythmias (tachyarrhythmias and bradyarrhythmias)
  • Fever associated with changes in mental status, changes in other vital signs

THINGS TO CONSIDER OBTAINING BEFORE ARRIVAL AT BEDSIDE

• If the nurse’s summary of the patient sounds unstable or you are at all unsure, page your resident immediately!
  • Full set of vitals
  • Hospital chart at bedside
  • IV access
  • Oxygen (nasal cannula, face mask), respiratory therapist
  • Cardiac monitor, ECG
  • Crash/code cart
  • Chest X-ray
  • ABG kits
  • Blood cultures, if febrile
  • Basic lab results

THINGS THAT CAN WAIT

• Talking to family members; unless urgent, this can usually be handled by the primary team.
• Major adjustments in medication regimen in a stable patient (i.e., antihypertensive medicines, antibiotics).
• Consultations in nonemergent, stable situations (i.e., GI consult in patient with occult blood-positive brown stool, stable hematocrit, stable vital signs).
• Addressing code status in a stable patient. This is best addressed by the patient’s primary medical team.

APPROPRIATE TRANSFER OF PATIENTS TO THE INTENSIVE CARE UNIT

• Determine which unit is most appropriate for management of the patient. **Do not hesitate to incorporate your resident into the decision making process.**

• Speak to the resident who will be accepting the patient to inform him or her of the situation and provide sign-out.

• Have the nursing staff give report to the staff in the unit.

• Write a brief transfer note that includes the following:
  • When and why you were called to see the patient
  • One-line description of patient and his or her comorbidities
  • Your assessment of the situation
  • Your management of the situation, including diagnostic and therapeutic measures, complications, and outcome. Include code note, if appropriate
  • Your assessment and plan with brief differential diagnosis for what could be going on
  • Reason for transfer (e.g., hypotension arrhythmia, unstable vital signs, closer monitoring)
  • Vascular access (e.g., femoral line, peripheral IV)
  • Code status
  • Who has been notified and their contact information (patient’s physician, family members)
OFF-SERVICE NOTES

It is your final day on the wards, you’ve had a grueling month, and the last thing you want to do is write yet another note, let alone think about writing long, drawn-out off-service notes. However, the presence of concise off-service notes can be a lifesaver to the intern coming onto the service. The essentials include the following items:

• Date of admission
• Any new diagnoses/alterations in previous diagnoses
• Pertinent past medical history
• Hospital course (major interventions, events, procedures); this can be organized chronologically or by organ system depending on the patient. This is not a day-by-day recap of every test performed. Distill the story down to its essential details
• Current medications including day number for antibiotics
• Current pertinent PE and lab results
• Assessment and plan, including goals of care and discharge needs (skilled nursing facility placement, home IV antibiotics, etc.)

PROCEDURE NOTES

These are of vital importance as part of the documentation of the hospital course and should include the following items:

• Procedure, site of procedure. Regulatory bodies now require documentation of a pre-procedure “time-out” to confirm the patient’s name and procedure performed
• Indication(s)
• Informed consent
• Sterile prep used
• Anesthesia used
• Brief description of the procedure
• Specimens and what they were sent for. Of note, any fluids that you just spent your valuable time collecting should be hand delivered to the lab personally
• Complications
• Post-procedure disposition and pending follow-up studies (e.g., CXR post-central line placement)

DEATH/EXPIRATIONS

Interns are called on quite frequently to pronounce a death. Certain steps must be performed:
• On arrival to the bedside, you should observe for respirations, auscultate for heart sounds, palpate for a pulse, and attempt to elicit a corneal reflex. You also need to agree on an exact time of death with the nursing staff.
• Notify your attending physician, the private physician, and family immediately, even in the middle of the night. The family must be asked specifically about (1) autopsy, (2) anatomic gift donation, and (3) funeral home. Appropriate forms for an autopsy and anatomic gifts must be completed. Note: Many hospitals have specially trained personnel to handle these particular requests, so be aware that it may not be appropriate for you to approach the family regarding these issues. Notify the appropriate hospital personnel if necessary.
• Complete a death note in the progress note section of the chart. It should include the following information: “Called by nursing to see patient regarding unresponsiveness. The patient was found to be breathless, pulseless, and without heart sounds, blood pressure, and corneal reflexes. The patient was pronounced dead at 9:55 p.m. on August 29, 2012. The patient’s private physician and family were notified. The patient’s family refused both anatomic gifts and autopsy. The funeral home will be Manchester Mortuary.” The word dead must be used.
• The Certificate of Death must be completed, including your assessment of cause of death. If the patient has a private physician, the death certificate will be completed by the private physician. Also, dictate a short death summary, which should include a concise summary of the hospital course as well as the information included in the brief death note.
Yes, it could be angina or MI. So, **assess the patient ASAP.** A thorough history and physical may elucidate a variety of possible diagnoses. However, it may lead to exactly the diagnosis that you thought of initially.

Be sure to ask the nurse for vital signs. Initial verbal orders should include stat ECG, O₂ to keep saturations >92%, sublingual nitroglycerin 0.4 mg, and aspirin 325 mg to the bedside. Confirm IV access.

**Major Causes of Chest Pain**
- Heart/vascular: angina, MI, acute pericarditis, aortic dissection
- Lungs: pneumonia, PE, pneumothorax
- GI: esophageal spasm, GERD, PUD, pancreatitis
- Other: costochondritis, herpes zoster, rib fracture, anxiety

**Things You Don’t Want to Miss (Call Your Resident)**
- MI
- Aortic dissection
- PE
- Pneumothorax

**Key History**
- Quickly review chart.
- Take a focused chest pain history including quality, duration, radiation, changes with respiration, diaphoresis, and N/V.
- Review ECG. If cardiac etiology is suspected, give NTG SL if SBP >90. Also, make the patient chew the aspirin, if not already given during the day.
Focused Examination

- General: Does the patient appear distressed or ill?
- Vitals: Hypotension is an ominous sign. Tachycardia may be from a PE or from pain. Bradycardia may be from AV block with inferior MI. Take BP in both arms to evaluate for aortic dissection. Fever may raise suspicion for PE.
- Chest: Check for chest wall tenderness and any skin lesions. Listen for murmur, rubs, or gallops. Assess JVP.
- Lungs: Listen for crackles, absent breath sounds on one side, friction rub.
- Abdomen: Examine for distension, tenderness, and bowel sounds.
- Extremities: Edema or evidence of deep venous thrombosis. Examine pulses bilaterally in both upper and lower extremities to assess for aortic dissection.

Laboratory Data

- Obtain an ECG if you haven’t already. Review telemetry, if available.
- Check ABG if respiratory distress or low saturations are present; serial troponins (q12h x 2), portable CXR.
- Consider CT angiography or V/Q scan if PE is suspected. Consider contrast CT or TEE if aortic dissection is suspected.

Management

- Cardiac: If evidence of acute MI on ECG (ST elevation of 1 mm or more in two contiguous leads or new LBBB) and history, call a stat cardiology consult for consideration of reperfusion therapy (thrombolytics or angioplasty). Ensure the patient is on a monitor, has IV access, has oxygen 2 L by NC, and has received an aspirin. Consider administering β-blockers, nitrates, morphine, heparin (LMWH or UFH). If the chest pain persists, consider loading with clopidogrel, 300 mg PO, and starting a glycoprotein IIb/IIIa inhibitor (e.g., eptifibatide or tirofiban). Metoprolol, 5 mg IV, may be initiated and repeated every 5 minutes for a total dose of 15 mg.
- Angina: NTG SL, 0.4 mg 3 times every 5 minutes, assuming SBP >90. Consider β-blockers, IV or transdermal NTG, heparin, and antiplatelet agents. Place on telemetry. If the patient is still having chest pain after NTG SL 3 times, consider administration of morphine and initiating NTG drip to titrate until chest pain free.
• Aortic dissection: Arrange for immediate transfer to CCU/MICU. Start nitroprusside or labetalol for BP control. Stat vascular/thoracic surgery consultation.

• Pulmonary: If PE, ensure adequate oxygenation and administer LMWH or UFH.

• Pneumothorax: Tension pneumothorax requires immediate needle decompression in the second intercostal space in the mid-clavicular line, followed by chest tube. Other pneumothoraces involving >20% of the lung require a surgery consult for chest tube placement.

• GI: Antacids such as aluminum hydroxide, 30 mL PO prn q4-6h (avoid in patients with renal failure), famotidine, 20 mg PO bid, or omeprazole, 20 mg PO qday. Elevate the head of the bed, especially after meals.

**Refractory Chest Pain**

• Reevaluate the patient for causes of chest pain—has your initial impression changed?

• Repeat ECG, vital signs, physical exam.

• For ongoing cardiac ischemia, particularly with elevated troponins and/or ST segment depression, start a NTG drip, consider further antiplatelet agent therapy such as clopidogrel or glycoprotein IIb/IIIa inhibitor, and consider an urgent cardiology consult.

**ABDOMINAL PAIN**

What are the patient’s vital signs? How severe is the pain? Is this a new problem? If vital signs are stable, inform the nurse you will be there shortly and to call you immediately if things worsen.

**Major Causes of Abdominal Pain**

Figure 15-1 lists some causes of abdominal pain by location. Generalized abdominal pain may be due to various causes such as appendicitis (at its inception); intestinal infection, inflammation, ischemia, and obstruction; peritonitis of any cause; diabetic ketoacidosis; uremia; sickle cell crisis; acute intermittent porphyria; ruptured aneurysm; and acute adrenocortical insufficiency.

**Things You Don’t Want to Miss (Call Your Resident)**

• AAA rupture

• Bowel rupture, perforation, or ischemia
Figure 15-1. Major causes of abdominal pain.
• Ascending cholangitis
• Acute appendicitis
• Retroperitoneal hematoma

**Key History**

• Check BP, pulse, respirations, $O_2$ saturations, and temperature.
• Quickly look at the patient and review the chart.
• Take a focused history, including quality, duration, radiation, changes with respiration, location, N/V (bilious vs. nonbilious), last bowel movement, and any hematemesis, melena, or hematochezia.
• For women of childbearing age, ask about their last menstrual period.
• Mesenteric ischemia often has pain out of proportion to examination. Consider this, especially with a history of atrial fibrillation and vascular disease and in elderly patients.

**Focused Examination**

• General: Is the patient distressed or ill-appearing?
• Vitals: Repeat now, especially BP.
• HEENT: Check for icterus.
• Chest: Check for any skin lesions. Listen for murmur, rubs, or gallops. Assess JVP.
• Lungs: Listen for crackles, absent breath sounds on one side, friction rub.
• Rectal: Must be performed. Assess for hemorrhoids and anal fissures. Guaiac for occult blood.
• Pelvic: If indicated by history.

**Laboratory Data**

• Consider CBC, electrolytes, ABG, lactate, LFTs, amylase, lipase, $\beta$-hCG, and UA.
• Films to consider include flat and upright abdominal films, CXR, and ECG. Abdominal CT, ultrasound, or both may be required.

**Management**

• The initial goal is to determine if the patient has an acute abdomen and needs surgical evaluation and treatment.
• An acute abdomen includes such signs as rebound tenderness or guarding and conditions such as ruptured viscus, abscess, or hemorrhage. See Acute Abdomen section in the General Surgery consult section (Chapter 21).
• Other conditions can be managed using a more detailed approach, after the acute abdomen has been ruled out.
• Keep the patient NPO. Ensure large-bore IV access and run maintenance fluids.

ACUTE ALTERED MENTAL STATUS

• What are the patient’s vital signs? Are the changes acute or subacute? Is the patient confused or is there a change in the level of consciousness? Any recent fall or trauma? Is the patient a diabetic? Does the patient have a cardiac history? Could this be an effect of a prescribed medicine? Could there be an infection? Could this be withdrawal from alcohol or another substance?
• Initial verbal orders to consider: Think of TONG (Thiamine, Oxygen, Naloxone, and Glucose). Have the nurse obtain blood sugar and oxygen saturations.
• Acute mental status changes associated with fever or decreased consciousness require that you see the patient immediately.

Major Causes of Acute Altered Mental Status
Think DELIRIUMS:
• D = Drug effect or withdrawal (e.g., EtOH; narcs, benzos, anticholinergics; especially in the elderly, even in low doses)
• E = Emotional (e.g., anxiety, pain)
• L = Low Po₂ (e.g., MI, PE, anemia) or high Pco₂ (e.g., COPD)
• I = Infection
• R = Retention of urine or feces
• I = Ictal states
• U = Undernutrition/underhydration
• M = Metabolic (electrolytes, glucose, thyroid, liver, kidney)
• S = Subdural, acute CNS processes (e.g., head trauma, hematoma, hydrocephalus, CVA/TIA)

Things You Don’t Want to Miss (Call Your Resident)
• Sepsis or meningitis
• Intracranial mass or increased intracranial pressure
Top Ten Workups

• Alcohol withdrawal with or without delirium tremens
• Acute CVA
• Neuroleptic malignant syndrome

Key History
• Check BP, pulse, respirations, $O_2$ saturations, temperature, and blood sugar.
• Quickly look at the patient and review the chart.
• Confirm no falls or trauma.
• Review orders for new meds or narcotics.
• If possible, take a focused history, including onset and level of responsiveness. Confirm this history with the family or staff.

Focused Examination
• General: Does the patient appear ill or distressed? Is the patient protecting his/her airway?
• HEENT: Look for signs of trauma; pupil size, symmetry, and response to light; papilledema and nuchal rigidity.
• Chest: Check for any skin lesions. Listen for murmur, rubs, or gallops.
• Lungs: Listen for crackles and equal breath sounds.
• Abdomen: Look for ascites, jaundice, and other signs of liver disease.
• Neurologic: Evaluate for weakness, asterixis, rigidity, or asymmetry. Perform a mental status examination.

Laboratory Data
• Consider CBC, electrolytes, LFTs, ABG, TSH, ammonia level, UA, cultures, ECG, and CXR. Other studies that may be required are lumbar puncture, CT, and EEG.
• The performance of a head CT before lumbar puncture is controversial but is generally not required for nonelderly, immunocompetent patients who present without focal neurologic abnormalities, seizures, or diminished level of consciousness.

Management
• Management is based on findings on examination and laboratory data. If meningitis is suspected, lumbar puncture should be performed as detailed above. In addition, empiric antibiotics should be started stat (see Chapter 21, Neurology consult section, for antibiotic choices).
Alcohol withdrawal needs to be treated urgently with benzodiazepines, usually with chlordiazepoxide 50 to 100 mg PO q6-8h or lorazepam 0.5 to 1 mg PO/IV/IM q6-8h (prn or scheduled). Thiamine, 100 mg IV/IM, should also be administered, especially before any glucose.

**ACUTE RENAL FAILURE**

- What are the patient’s vital signs? How much urine has been produced in the last 24 hours? In the last 8 hours? Does the patient have a Foley catheter inserted? What are the patient’s recent electrolytes, especially potassium, BUN, creatinine, and bicarbonate?
- If the patient does have a Foley catheter inserted, ask the nurse to flush the catheter with 30 mL NS. If the patient does not have a Foley catheter, ask the nurse to place one now. Tell the nurse you will see the patient shortly.

**Major Causes of Oliguria**

Oliguria is generally defined as <500 mL of urine per 24 hours. Major causes of oliguria can be broken down as follows:

- **Prerenal**: volume depletion, congestive heart failure, vascular occlusion
- **Renal**: glomerular, tubular/interstitial (acute tubular necrosis caused by drugs or toxins), and vascular
- **Postrenal**: obstruction (BPH), clogged Foley catheter, stones

**Things You Don’t Want to Miss (Call Your Resident)**

- Hyperkalemia
- Severe acidosis
- Acute, marked uremia
- Life-threatening volume overload

**Key History**

- Check BP, pulse, respirations, $O_2$ saturations, and temperature.
- Quickly look at the patient and review the chart.
- Take a focused history.
- Determine volume status. Review ins and outs over the past few days. Any new medications (e.g., ACE inhibitors, diuretics, NSAIDs, IV contrast dye)?
Focused Examination

- General: Does the patient appear sick?
- Vitals: Check orthostatics and weight over the past few days.
- Cardiovascular: Look for JVD, friction rub, and skin turgor.
- Abdomen: Evaluate for ascites or enlarged bladder.
- Genitourinary: Check for enlarged prostate.

Laboratory Data

- UA: Look for cells, casts, protein.
- Check serum electrolytes and urine electrolytes; calculate FE_{Na} (and/or FE_{urea} if the patient is on diuretics); consider urine eosinophils, ABG, and ECG.
- Renal ultrasound should be ordered within 24 hours to rule out hydronephrosis and evaluate the renal system.

Management

- The minimum acceptable urine output is 30 mL/h. If flushing the Foley catheter did not help, ask the nurse to change the Foley catheter.
- Initial management should be directed at treating life-threatening electrolyte disorders and correcting volume contraction and hypotension. Obtain diagnostic urinary studies before administering diuretics. Don’t forget to adjust drug doses based on glomerular filtration rate.
- Calculate the fractional excretion of sodium:

\[ \text{FE}_{Na} = \left( \frac{U_{[Na^+]}}{P_{[Cr]}} \right) \times \left( \frac{P_{[Na^+]}}{U_{[Cr]}} \right) \times 100 \]

This equation is most useful with oliguric renal failure but may be helpful in nonoliguric renal failure.

- FE_{Na} > 1% to 2% with oliguria is almost always ATN but can be prerenal with diuretics.
- FE_{Na} < 1% with oliguria is generally prerenal: volume depletion, severe CHF, nephrotic syndrome, NSAID or dye toxicity, sepsis, cyclosporine toxicity, acute glomerulonephritis, and hepatorenal syndrome.
- Calculate FE_{urea} in nonoliguric renal failure or if diuretics have been given. FE_{urea} < 35% is consistent with prerenal state.
- If hyperkalemia is suspected, order an ECG and stat serum potassium.
A stat renal consult is required if the patient needs urgent dialysis. Indications for urgent dialysis include AEIOU:

- **A** = Acidemia (pH < 7.2)
- **E** = Electrolyte disorder (e.g., hyperkalemia when unable to manage medically, see Chapter 17, Fluid and Electrolytes)
- **I** = Intoxication (e.g., alcohol, salicylates, theophylline, lithium)
- **O** = Overload (e.g., pulmonary edema when unable to manage medically)
- **U** = Uremia (encephalopathy, pericarditis)

**Prerenal causes** can be initially managed with a small volume challenge, such as 500 mL NS bolus depending on the cardiovascular status of the patient. This can be followed by NS at a set rate. Specific criteria should be given to the nursing staff (i.e., call HO if urine output is < 30 mL/h). Alternatively, if congestive heart failure is suspected, the patient may need diuresis. Escalating doses of furosemide can be used, and urine output and daily weights can be assessed. With a fluid challenge, the creatinine level often trends down by the next morning if the cause is prerenal.

**Postrenal causes** can be potentially managed by placing a Foley catheter. If immediate flow is obtained, urethral obstruction is likely. If a Foley cannot be placed due to obstruction, consider a urology consultation.

To prevent **contrast-induced ARF**, euvolemia is essential. Use ½ NS or NS at 1 mg/kg/h for 6 to 12 hours before and 6 to 12 hours after the procedure. Due to conflicting study results, the use of acetylcysteine remains controversial in most circumstances.

### HEADACHE

- What are the patient’s vital signs? How severe is the headache? Has there been a change in consciousness? Are there any new focal CNS symptoms? Has the patient had similar headaches in the past; if so, what precipitates or relieves them?

- **If the headache is severe and acute or associated with N/V, changes in vision, other focal CNS findings, fever, or decreased consciousness, the patient should be seen immediately.** Otherwise, inform the nurse you will see the patient shortly.
Major Causes and Types of Headache

- Tension
- Vascular (migraine, subarachnoid hemorrhage)
- Cluster
- Drugs
- Temporal arteritis
- Infectious (meningitis, sinusitis)
- Trauma
- CVA
- Severe hypertension
- Mass lesions

Things You Don’t Want to Miss (Call Your Resident)

- Meningitis
- Subarachnoid hemorrhage or subdural hematoma
- Mass lesion associated with herniation

Key History

- Check BP, pulse, respirations, \( O_2 \) saturations, and temperature.
- Quickly look at the patient and review the chart.
- A detailed, well-focused history is the best method for evaluating a headache. Most are tension or migraine type, but more serious conditions need to be ruled out.

Focused Examination

- General: Does the patient appear ill or distressed?
- HEENT: Look for signs of trauma, pupil size, symmetry, response to light, papilledema, nuchal rigidity, temporal artery tenderness, and sinus tenderness.
- Neurologic: Thorough examination is mandatory, including mental status.

Laboratory Data

- Consider CBC and ESR if temporal arteritis suspected.
- **Head CT** should be considered for:
  - A chronic headache pattern that has changed or a new severe headache occurs.
  - A new headache in a patient older than 50 years.
  - Focal findings on neurologic examination.
• **If meningitis is suspected, an LP should be performed.** The performance of a head CT before lumbar puncture is controversial but is generally not required for nonelderly, immunocompetent patients who present without focal neurologic abnormalities, seizures, or diminished level of consciousness.

**Management**

• **The initial goal is to exclude the serious life-threatening conditions** mentioned previously. After such conditions have been excluded, management can focus on symptomatic relief.

• For suspected bacterial meningitis, start antibiotics immediately. See Chapter 21, Neurology consult section, for antibiotic choices.

• For suspected subdural hematoma or subarachnoid hemorrhage, obtain CT scan. If positive, a neurosurgery consultation should be obtained.

• Tension headaches and mild migraines can be treated with acetaminophen 650 to 1,000 mg PO q6h prn or ibuprofen 200 to 600 mg PO q6-8h; consider sumatriptan 25 mg PO for moderate to severe migraine headaches; can repeat 25 to 100 mg q2h for maximum of 200 to 300 mg/d.

• Severe migraines may require an opiate. Sumatriptan and ergotamine are usually most effective in the prodromal stage. These agents are contraindicated in patients with angina, uncontrolled hypertension, hemiplegia, or basilar artery migraine.

**HYPOTENSION AND HYPERTENSION**

**HYPOTENSION**

• What are the patient’s vital signs? Is the patient conscious, confused, or disoriented? What has the patient’s blood pressure been? What was the reason for admission?

• **Hypotension requires that you see the patient immediately.**

• If impending or established shock is suspected, ensure IV access (at least 20G IV) and consider having the patient placed in the Trendelenburg position (i.e., head of bed down). However, use of the head-down position has been significantly challenged as not helpful and potentially harmful.

**Major Causes of Hypotension**

• Cardiogenic (rate or pump problem)

• Hypovolemic
• Septic shock
• Anaphylaxis

Things You Don’t Want to Miss (Call Your Resident)
• You will likely want to let your resident know about any patient with significant hypotension.
• Shock is inadequate tissue and organ perfusion. This is best assessed by looking at end organs: brain (mental status), heart (chest pain), kidneys (urine output), and skin (cool, clammy).
• Shock is a clinical diagnosis defined as an SBP <90, with evidence of inadequate tissue perfusion.

Key History
• Check BP (both arms), pulse, respirations, O₂ saturations, and temperature.
• Quickly look at the patient and review chart.

Focused Examination
• General: How distressed or sick does the patient look?
• Vitals: Repeat now and often. Elevated temperature and hypotension suggest sepsis.
• Cardiovascular: Heart rate, JVP, skin temperature, color, and warmth. Capillary refill.
• Lungs: Listen for crackles, breath sounds on both sides.
• GI: Any evidence of blood loss?
• Neurologic: Mentation, symmetric movements.

Laboratory Data
Consider troponins, ECG, ABG, CBC, electrolytes, and CXR.

Management
• Examine the ECG and take the pulse yourself. Check BP in both arms. A compensatory sinus tachycardia is an expected appropriate response to hypotension. However, check the ECG to ensure that the patient does not have atrial fibrillation, SVT, or ventricular tachycardia, which may cause hypotension because of decreased diastolic filling. Bradycardia may be seen in autonomic dysfunction or heart block.
• Most causes of shock require fluids to normalize the intravascular volume. Use normal saline or lactated Ringer’s. The exception is cardiogenic shock, which may require preload and afterload reduction, inotropic and/or vasopressor support, and transfer to an ICU.
• **Hypovolemic, anaphylactic, and septic shock require fluids.** Use boluses of 500 mL to 1 L. If no response, repeat bolus or leave fluids open.

• **Anaphylactic shock requires epinephrine**, 0.3 mg IV immediately and repeated every 10 to 15 minutes as required. Epinephrine is the most important component of management. Hydrocortisone, 200 mg IV, and diphenhydramine, 25 mg IV, should also be administered. Diphenhydramine is adjunctive and not first-line treatment. H2 antihistamines probably add little. Glucocorticoids do nothing for the emergent symptoms and take hours to become effective; they are given to prevent prolonged or recurrent anaphylactic reactions.

• **In septic shock, IV fluids and antibiotics can resolve the shock.** However, continuing hypotension requires ICU admission for vasopressors.

• Cardiogenic shock can be the result of an acute MI or worsening CHF. However, other causes of hypotension and elevated JVP include acute cardiac tamponade, PE, and tension pneumothorax. These always need to be considered.

**HYPERTENSION**

• What are the patient’s vital signs? What has the patient’s blood pressure been? What is the reason for admission? What BP medications has the patient been taking? Does the patient have signs of hypertensive emergency (end-organ damage)?

• The rate of rise of the BP and the setting in which the high BP is occurring are more important than the level of BP itself. Elevated blood pressure alone, in the absence of symptoms or new or progressive end-organ damage, rarely requires emergent therapy.

• **Hypertensive emergencies require that you see the patient immediately.** Prior to your arrival, make sure the patient has an IV and order an ECG.

**Hypertensive Emergencies**

• Encephalopathy
• Intracranial hemorrhage
• Unstable angina or MI
• Acute left ventricular failure with pulmonary edema
• Aortic dissection
• Eclampsia
• Renal insufficiency (new or worsened)
Hypertensive Urgencies
• Blood pressure >180/110
• Optic disc edema
• Severe perioperative hypertension

Things You Don’t Want to Miss (Call Your Resident)
Hypertensive emergencies: hypertension with acute end-organ system damage.

Key History
• Check BP, pulse, respirations, O₂ saturations, and temperature.
• Quickly look at the patient and review the chart. Get an ECG.

Focused Examination
• General: Is the patient distressed or ill-appearing?
• Vitals: Repeat BP yourself in both arms.
• HEENT: Check fundi for papilledema, retinal hemorrhages, or other hypertensive changes.
• Lungs: Listen for crackles, breath sounds on both sides.
• Neurologic: Mentation, confusion, delirium, focal neurologic deficits.

Laboratory Data
Consider troponins, ECG, ABG, CBC, electrolytes, UA, and CXR.

Management
• Treat the patient, not the BP reading. Acute lowering of BP in asymptomatic patients with long-standing hypertension can be dangerous.
• Permissive hypertension is usually advised by neurologists for patients with an acute ischemic stroke, unless the BP is severely elevated (>220/120) or conditions such as ACS, decompensated CHF, dissections, encephalopathy, ARF, and eclampsia coexist.
• Hypertensive emergencies require an ICU setting. The goal is to reduce the MAP by no more than 25% in the first 2 hours. IV hydralazine, nitroprusside, labetalol, esmolol, enalaprilat, and fenoldopam are often used. While arranging transfer to the ICU, certain wards allow medications to be started. Consider IV nitroglycerin for hypertension associated with MI or pulmonary edema. Nitroprusside and labetalol are useful in aortic dissection. Nitroprusside is also used for patients with
encephalopathy but often requires intra-arterial blood pressure monitoring.

- Hypertensive urgencies can usually be managed with oral medications with the goal of reducing BP over 24 to 48 hours. Examples include captopril 25 to 50 mg PO, clonidine 0.1 to 0.2 mg PO, or labetalol 200 to 400 mg PO. These can be repeated or titrated every 2 to 4 hours. Close follow-up is essential. PO/SL short-acting nifedipine should not be used in most medical patients.

COMMON ARRHYTHMIAS

- Obtain the vital signs, including temperature. Any chest pain or shortness of breath? What is the patient’s mental status?
- Review the telemetry and order a stat ECG. **Patients with chest pain, shortness of breath, altered mental status, or hypotension need to be seen immediately.**

**Major Causes of Rapid Heart Rate and Slow Heart Rates**

- **Rapid rates:**
  - Regular: Sinus tachycardia, SVT, ventricular tachycardia, atrial flutter
  - Irregular: Atrial fibrillation with rapid ventricular rate (RVR), multifocal atrial tachycardia

- **Slow rates:**
  - Drugs (β-blockers, CCB, digoxin)
  - Sick sinus syndrome
  - MI (especially inferior)
  - AV block

**Things You Don’t Want to Miss (Call Your Resident)**

- Ventricular tachycardia
- Unstable SVT
- Hypotension
- Angina or MI

**Key History**

- Check BP, pulse, respirations, O₂ saturations, and temperature.
- Quickly look at the patient and review the chart, while waiting for a 12-lead ECG.
Focused Examination

- General: Does the patient look sick or distressed?
- Vitals: Repeat now.
- Cardiovascular: Heart rate, jugular venous pulse, skin temperature and color, capillary refill.
- Lungs: Listen for crackles and breath sounds on both sides.
- Neurologic: Evaluate for confusion or change in level of consciousness.

Laboratory Data

In addition to ECG, consider troponins, ABG, CBC, electrolytes, and CXR.

Management

- **Always complete the ABCs first and ensure O₂ and IV access.** Place the patient on monitor or telemetry; consider transfer to a monitored bed on a cardiology floor.

- **If the patient is hypotensive and has atrial fibrillation with RVR, SVT, or ventricular tachycardia, emergency cardioversion may be required.**

- First, call your resident. If the patient is unstable with serious signs or symptoms, a ventricular rate greater than 150, or both, you should prepare for immediate cardioversion. The patient may require sedation. Serious signs and symptoms per ACLS protocol include chest pain, shortness of breath, decreased level of consciousness, hypotension and shock, congestive heart failure, and acute MI. Refer to the proper ACLS algorithm at this point (see Chapter 3).

- **Atrial fibrillation with RVR but without evidence of hemodynamic compromise** can be rate controlled with diltiazem, metoprolol, esmolol, or digoxin. Amiodarone can also be considered, though there is a risk of pharmacologic cardioversion. Amiodarone is generally considered the best choice if heart failure and/or an accessory pathway is present.
  - **Diltiazem**, 0.25 mg/kg IVP over 2 minutes; if no response, repeat 0.35 mg/kg IVP over 2 minutes; follow with an IV infusion at 5 to 15 mg/h. Diltiazem is the agent of choice in most patients. Verapamil can also be used, 2.5 to 10 mg IVP, may repeat 5 to 10 mg IVP after 15 to 30 minutes for a maximum of 20 mg.
  - **Metoprolol**, 2.5 to 5 mg IVP over 2 minutes every 5 minutes to a total of 15 mg followed by oral dosing. Preferred agent if ischemia is suspected or present.
• **Esmolol**, 0.5 mg/kg over 1 minute loading dose, followed by 50 μg/kg/min, maximum 300 μg/kg/min.

• **Digoxin**, 0.25 to 0.5 mg IVP; then 0.125 to 0.25 mg IVP every 4 to 6 hours to a total dose of 0.75 to 1.35 mg; followed by oral dosing. Digoxin’s effect will take longer than other agents and is much less commonly used for acute rate control. It may, however, be used for this purpose for those with CHF.

• **Amiodarone**, 150 mg IV over 10 minutes, followed by infusion of 1 mg/min for 6 hours, then 0.5 mg/min for 18 hours, is not FDA-approved for the treatment of atrial fibrillation, but studies have shown it to be effective. Its onset of action is slower than calcium channel blockers and β-blockers. Also, be cautious if atrial fibrillation has been present >48 hours as amiodarone can cause conversion to sinus rhythm and put the patient at risk for cardioembolic stroke.

• **SVT without evidence of hemodynamic compromise** can sometimes be broken with Valsalva maneuver, carotid sinus massage (one side at a time and always listen for bruits first), or both. If still in SVT, try **adenosine**, 6 mg rapid IV push, followed by 12 mg rapid IV push if necessary. Always remember to flush with at least 20 mL of NS after each IV push. If the complex width is narrow with stable BP, verapamil, 2.5 to 5 mg IV, or diltiazem, 10 mg IV, can be used. Adenosine should be given with significant caution if WPW is suspected. If wide complex, manage as stable VT.

• For ventricular tachycardia, if pulseless or without BP, manage as ventricular fibrillation. If ventricular tachycardia with serious signs or symptoms, consider immediate synchronized cardioversion. If stable, follow the ACLS protocol (see Chapter 3).

**FEVER**

• What are the patient’s vital signs? What was the reason for admission? Is this a new finding? Any associated symptoms (e.g., cough, headache, change in mental status, and N/V)? Any antipyretics or current antibiotics? Any recent surgeries or procedures?

• Order CXR, blood and urine cultures.

• **Patients with symptoms concerning for meningitis or hypoten-**

sion need to be seen immediately.
**Major Causes of Fever**

- Infections: Best to think of by site—lung, urine, IV sites, blood, CNS, abdomen, and pelvis. Confirm immune status. **Immunocompromised patients may warrant much more aggressive evaluation and empiric therapy** (e.g., post-chemo-therapy neutropenic fever).
- Drug-induced fever: Antibiotics and many other drugs have been implicated.
- Postoperative atelectasis (though often invoked, there is minimal evidence to support this contention).
- Neoplasms.
- Rheumatologic diseases.
- Deep venous thrombosis/pulmonary embolism.
- Fever of unknown origin.

**Things You Don’t Want To Miss (Call Your Resident)**

- Meningitis
- Septic shock, particularly in neutropenic patients
- Endocarditis

**Key History**

- Check BP, pulse, respirations, $O_2$ saturations, and temperature.
- Quickly look at the patient and review the chart.

**Focused Examination**

- General: Does the patient appear ill? Check all catheter sites (IV, central line, Foley, G-tube, etc.).
- Vitals: Repeat now. Tachycardia is an expected finding with fever. Recheck blood pressure.
- Cardiovascular: Heart rate, jugular venous pulse, skin temperature, and color. Any new murmurs? Capillary refill.
- Lungs: Listen for crackles and breath sounds on both sides.
- Abdomen: Assess for localized tenderness and bowel sounds.
- Extremities: Check calves for signs of deep venous thrombosis and joints for effusions.
- Neurologic: Mentation, photophobia, neck stiffness, Brudzinski's or Kernig's signs.
Laboratory Data

- Consider CBC, blood cultures (two sets at different sites; if a central line is present, be sure to get one peripheral set as well), CMP, UA and culture, sputum culture and Gram’s stain, CXR.
- LP if meningitis is suspected.
- Fluid collections (e.g., pleural effusion, ascites) may need to be tapped.
- Consider *Clostridium difficile* toxin stool testing.

Management

- Make sure the patient is hemodynamically stable. Review medications and obtain cultures. Give antipyretics (acetaminophen 650 mg PO/PR or ibuprofen 400 mg PO q6-8h prn). Ensure IV access and consider maintenance fluids for insensible losses.
- Consider antibiotics carefully. **If the patient is hemodynamically stable, immunocompetent, not toxic appearing, with no clear source of infection, it may be prudent to withhold antibiotics and await culture results.**
- Patients with fever and hypotension require broad-spectrum antibiotics and IV fluids or pressors to manage the hypotension. Septic shock is an emergency (see above).
- Patients with fever and meningitis symptoms require antibiotics immediately. Do not wait for the LP to be completed. Start the antibiotics, then begin the LP.
- Consider changing or removing Foley catheters and any indwelling IV sites.

Febrile Neutropenia

- Patients with fever and neutropenia (<1000 cells/mm³) require a careful physical examination, with particular attention paid to mucosal surfaces, lungs, skin, and vascular access sites.
- Blood cultures for bacteria and fungi should be drawn; also consider urine culture, sputum culture, LP, and CXR if clinically indicated.
- Broad-spectrum antibiotics should be started.
- Choices for initial therapy include fourth-generation cephalosporin, carbapenem or an antipseudomonal penicillin, with or without aminoglycoside.
- If a catheter-related infection is suspected or the patient is known to be colonized with penicillin-resistant pneumococcus or methicillin-resistant *Staphylococcus aureus*, consider adding vancomycin to the above regimen.
What follows is adapted from the Barnes Jewish Hospital Stem Cell Transplant Unit Febrile Neutropenia Pathway. **The recommendations are based on antibiotic resistance patterns specific to Barnes Jewish Hospital. Consult your hospital’s antibiogram to tailor antimicrobial therapy to local resistance patterns.**

- **Definition of neutropenic fever:** Temperature >38.3°C, or ≥38.0°C for at least 1 hour, with ANC ≤500 or anticipate ANC to fall <500.
- **Workup:** Obtain blood cultures × 2, physical exam, CXR, UA, and culture.

**Initial treatment:**

- **Cefepime** 1 g IV q8h. *If PCN allergy: ciprofloxacin 400 mg IV q12h or aztreonam 2 g IV q8h.*
- **Vancomycin** 1 g IV q12h if any of the following are present: Severe mucositis, clinical evidence of catheter-related infection, known colonization with resistant *Streptococcus* or *Staphylococcus*, sudden temperature spike > 40°C, hypotension, or sepsis.
- **Consider metronidazole** 500 mg IV q8h, if suspected oropharyngeal or intra-abdominal source.
- **Consider addition of gentamicin** 5 mg/kg IV q24h × 72 hours, if clinically unstable.
- **Tailor antibiotics based on culture results.**

**Treatment of persistent fevers:** new fever after afebrile ≥48 hours or persistently febrile ≥72 hours and cultures negative.

- **If clinically unstable:** change GNR coverage to meropenem 500 mg IV q6h or ciprofloxacin 400 mg IV q12h ± aminoglycoside.
- **If clinically stable:** continue current regimen and tailor based on culture results.
- **If persistently febrile >5 days and cultures negative:**
  - If the patient is NOT on anti-mold prophylaxis and NO identified clinical sites suspicious for fungal infection: use echinocandin.
  - If the patient is NOT on anti-mold prophylaxis and clinical site suspicious for fungal infection (excluding sinusitis, see below): use voriconazole, weight-based dosing for IV and PO administration.
  - If the patient is on anti-mold prophylaxis and NO identified clinical sites suspicious for fungal infection:
• Clinically stable: no change in antifungals, monitor closely.

• Clinically unstable: **amphotericin B lipid complex (ABLC)** 5 mg/kg IV qday. If already receiving amphotericin product, obtain an ID consult for antifungal selection.

• If the patient is on anti-mold prophylaxis and clinical site suspicious for fungal infection: Use ABLC 5 mg/kg IV qday.

• If the patient has suspected fungal sinusitis: ABLC 5 mg/kg IV qday.

**Duration of antibiotics:**

• Discontinue vancomycin after 72 hours if cultures are negative for coagulase-negative staphylococci, oxacillin-resistant *Staphylococcus aureus*, cephalosporin-resistant streptococci, or *Corynebacterium jeikeium*.

• Discontinue double GNR coverage (e.g., aminoglycoside) after 72 hours if cultures are negative for GNR and the patient is clinically stable.

• Culture negative for 3 to 5 days:
  - Afebrile and ANC ≥500, discontinue after 48 hours.
  - Afebrile and ANC <500, continue antibiotics until ANC ≥500 for 48 hours.
  - Febrile and ANC ≥500, reassess after 4 to 5 days.
  - Febrile and ANC <500, continue antibiotics until neutropenia resolves.

• Culture positive:
  - Remove line if *Pseudomonas* spp., *Stenotrophomonas maltophilia*, *Acinetobacter* spp., vancomycin-resistant enterococcus, *S. aureus*, *C. jeikeium*, and *Candida* spp.
  - All other organisms and tunnel catheter infections, consider removing line.
  - Continue antibiotics until ANC ≥500 for 7 days or for 14 days total, whichever is longer.

• UTI: Continue antibiotics until ANC is ≥500.

• Pneumonia:
  - Bacterial: Until ANC ≥500 × 7 days or for 14 days total, whichever is longer.
  - *Aspergillus* spp. (suspected or proven): Voriconazole (weight-based dosing) and consider ID consult.
SHORTNESS OF BREATH

• What are the patient’s vital signs, including temperature? When was the onset of SOB and what was the reason for admission? Does the patient have reactive airway disease or COPD? Is the patient getting oxygen?

• Order oxygen and an ABG kit to the bedside. Patients with SOB need to be seen immediately.

Major Causes of Shortness of Breath

• Pulmonary: Asthma, COPD, pulmonary embolism, pneumonia
• Cardiovascular: CHF, MI/ischemia, cardiac tamponade
• Others: Pneumothorax, obstruction (e.g., mucous plug), anxiety

Things You Don’t Want to Miss (Call Your Resident)

• Inadequate tissue oxygenation (i.e., hypoxia)
• Tension pneumothorax
• Airway obstruction

Key History

• Check BP, pulse, respirations, \( O_2 \) saturations, and temperature.
• Quickly look at the patient and review the chart. Get an ECG, ABG, and CXR if the patient looks sick.

Focused Examination

• General: Does the patient appear ill or distressed?
• Vitals: Repeat now. Check for pulsus paradoxus.
• Cardiovascular: Heart rate, JVP, skin temperature and color, capillary refill.
• Lungs: Listen for crackles and breath sounds on both sides, evidence of consolidation or effusion.
• Neurologic: Mentation

Laboratory Data

• Consider ABG, ECG, troponins, CBC, D-dimer, V/Q scan, and CXR.
• If you have any doubt at all, get an ABG—if you think about it you should do it. Beware of relying on pulse oximetry alone.
Management

- Order empiric oxygen to keep saturations >92%. Be cautious if the patient has COPD and is a retainer of CO₂—in that case, keep O₂ saturations around 88% to 90% and check ABG. Remember that the O₂ saturation tells you nothing about pH or PCO₂.
- For asthma or COPD, administer albuterol and ipratropium by nebulizer, q2-4h until stable. Consider IV corticosteroids, methylprednisolone, 60 mg IV q6h, and antibiotics if needed.
- For COPD, is the patient volume overloaded? Raise the head of the patient’s bed. Administer furosemide, 20 to 40 mg IV, and albuterol nebulizer. Consider morphine or nitroglycerin. Assess for adequate diuresis.
- For suspected cardiac tamponade, order a stat cardiac echo and cardiology consult.
- For pulmonary embolism, often the patient is tachycardic and tachypneic and has a sudden onset of SOB. The classic, though not usually present, ECG findings are S1, Q3, and T3 (S waves in lead I, Q waves in lead III, inverted T waves in lead III). If suspicion is high, consider starting heparin or LMWH. Ensure that the patient has no history of bleeding disorders, PUD, recent CVA, or surgery. Obtain CT pulmonary angiography or a V/Q scan.
- Acute respiratory failure is generally defined by ABG of PO₂ <60 or PCO₂ >50 with a pH <7.3 while on room air. Ensure that the patient hasn’t received narcotics recently. If so, consider naloxone, 0.2 mg IV. Acute respiratory acidosis with a pH <7.2 usually requires mechanical ventilation.

GASTROINTESTINAL BLEEDING

- What are the patient’s vital signs? When was the onset of bleeding and what is the reason for admission? Is the bleeding upper (coffee ground emesis, melena) or lower (hematochezia)? How much blood has been lost?
- Confirm that the patient has IV access (at least 18G) and recent CBC and INR. Type and cross-match blood. If the patient is tachycardic or hypotensive, see the patient immediately.

Major Causes of Gastrointestinal Bleeding

- Upper: Esophageal varices, Mallory-Weiss tear, peptic ulcer, esophagitis, neoplasm, aortoenteric fistula (history of AAA repair).
• Lower: Diverticulosis, angiodysplasia, neoplasm, IBD, infectious colitis, anorectal disease (hemorrhoids, fissures).

**Things You Don’t Want to Miss (Call Your Resident)**
GI bleeding leading to hypovolemic shock

**Key History**
• Check BP, pulse, respirations, O₂ saturations, and temperature. Orthostatic BP.
• Quickly look at the patient and review the chart.

**Focused Examination**
• General: How distressed or sick does the patient look?
• Vitals: Repeat now.
• HEENT: Check for conjunctival pallor or scleral icterus.
• Cardiovascular: Heart rate, JVP, skin temperature and color, capillary refill.
• Abdomen: Check for tenderness and bowel sounds, look for ascites.
• Rectal: Must be performed. Guaiac stool.
• Neurologic: Evaluate level of consciousness and ability to protect airway.

**Laboratory Data**
• Consider CBC, coags, and CMP.
• The initial CBC may be deceptive in acute GI bleeding.

**Management**
• Insert two large-bore IVs (16G to 18G), type and cross pRBCs. It can take up to 8 hours for CBC to equilibrate, so initial Hct may falsely appear normal or unchanged. In the absence of renal disease, high BUN suggests GI bleeding. Check coags and platelets to exclude bleeding disorders. Is the patient receiving anticoagulants? If so, stop the anticoagulant and consider reversal with FFP or vitamin K.
• Consider whether special blood products are required based on comorbidities (e.g., irradiated or washed RBCs). Also consider whether the patient needs premedication with acetaminophen/diphenhydramine based on prior transfusions.
• Bolster the intravascular volume by giving IV fluids (normal saline), especially while awaiting blood products. Keep the patient NPO.
• For upper GI bleeding, insert a nasogastric tube and perform lavage to assess if active bleeding is present. Suppress acid with PO (or IV) proton pump inhibitor therapy. GI consult for endoscopy. If bleeding has stopped and the patient is hemodynamically stable, elective endoscopy can be performed within the next 24 hours. Otherwise, urgent endoscopy may be required for diagnosis and treatment.

• For active variceal bleeding in patients with cirrhosis, start IV octreotide (somatostatin), 50 μg bolus, then 50 μg/h, start prophylactic IV ceftriaxone 1 g/day, correct coagulation deficits, replace pRBCs as needed. Call a GI consult as urgent endoscopy may be required.

• For lower GI bleeding, correct fluid status. If hemodynamically stable, obtain GI consult for colonoscopy. If unstable, an urgent tagged RBC scan should be scheduled. Also, consider arteriography.

• Surgery consult/indications include the following:
  • Aortoenteric fistula
  • Uncontrollable or recurrent bleeding
  • Bleeding episode requiring transfusion of more than 6 units pRBCs
  • Visible naked vessel seen in peptic ulcer by endoscopy
GENERAL PRINCIPLES

• *Pain* is defined as “an unpleasant sensory or emotional experience associated with actual or potential tissue damage.”

• Many complex biopsychosocial factors influence the outward manifestations of pain, both acute and chronic. As a result, pain assessment is subjective; there is no such thing as an objective “pain-o-meter.” We must rely on patient description.

• In the hospital, pain rating scales (e.g., “1 to 10 pain scale” or the “Wong-Baker FACES Pain Rating Scale”) are frequently used to standardize these descriptions. Pain rating scales have poor inter-rater reliability but good intra-rater reliability, so they may be helpful for tracking changes in a patient’s pain.

• The pain management strategy should be appropriate to the degree of pain (Table 16-1) and should be put into place concurrently with attempts to diagnose and treat the source of the pain.

• Pain is often an undertreated symptom. In the vast majority of circumstances, acute pain can be treated without compromising diagnostic evaluation or the patient’s clinical condition.

• Complete absence of pain is often an unrealistic goal.

• Patients should be frequently reassessed during analgesic treatment.

• Scheduled analgesics are often more effective than prn administration.

• For chronic pain, long-acting pain medications can improve adherence and reduce some side effects and can be coupled with immediate-release pain analgesics for breakthrough pain at doses of 5% to 15% of the total daily dose.

• Avoid IM injections. Subcutaneous opioids are equally efficacious and less painful than IM injections. Uptake can be erratic and unpredictable with both.

• Before prescribing pain medications, always consider comorbidities, allergies, drug interactions, and potential side effects.
<table>
<thead>
<tr>
<th>Agent</th>
<th>Oral</th>
<th>Parenteral</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1. Mild pain: nonopioid (and/or adjuvant)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acetaminophen</td>
<td>650 mg q4-6h prn or 1,000 mg q6h prn (max: 4 g/d, 2 g/d if liver disease)</td>
<td>1,000 mg IV q6h prn</td>
</tr>
<tr>
<td>Aspirin</td>
<td>325–650 mg q4-6h pm</td>
<td>—</td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>200–800 mg q6-8h pm</td>
<td>—</td>
</tr>
<tr>
<td>Ketorolac</td>
<td>10 mg q4-6h prn (max: 40 mg/d)</td>
<td>30–60 mg IM or 15–30 mg IV × 1; 15–30 IM/IV q6h prn; combined PO/IM/IV not to exceed 5 days</td>
</tr>
<tr>
<td>Gabapentin (for neuropathic pain)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Starting dose 300 mg qhs, titrating to a max of 3,600 mg/d divided q8h</td>
<td>—</td>
</tr>
<tr>
<td>Pregabalin</td>
<td>100–600 mg/d, divided into 2–3 doses</td>
<td>—</td>
</tr>
<tr>
<td><strong>Step 2. Moderate pain: opioid formulated for mild/moderate pain (and/or nonopioid, and/or adjuvant)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tramadol</td>
<td>25–100 mg q4-6h (max: 400 mg/d)</td>
<td>—</td>
</tr>
<tr>
<td>Hydrocodone/acetaminophen 5/325 mg (e.g., Vicodin, Norco, Lorcet)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1–2 tablets q4-6h prn</td>
<td>—</td>
</tr>
</tbody>
</table>
**Step 3. Severe pain: opioid formulated for moderate/severe pain (and/or nonopioid, and/or adjuvant)**

<table>
<thead>
<tr>
<th>Medicine</th>
<th>Dose/Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxycodone/acetaminophen 5/325 mg (e.g., Percocet, Tylox, Roxicet)</td>
<td>1–2 tablets q4-6h prn</td>
</tr>
<tr>
<td>Morphine</td>
<td>Immediate-release tablets, 10–30 mg q2-4h pm&lt;sup&gt;cd&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Suppository, 10–20 mg q4h prn</td>
</tr>
<tr>
<td>Morphine, extended release (e.g., MS Contin, Oramorph SR)</td>
<td>15 mg q8-12h</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>2–4 mg q3-4h prn</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.5–4 mg SC/IM/IV q3-6h prn&lt;sup&gt;f&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Transdermal, 12.5–100 μg/h&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>50–100 μg IV q1-2h&lt;sup&gt;g&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>a</sup>Most patients will require 900 to 1,500 mg/d to achieve pain control.
<sup>b</sup>There are multiple dosage formulations.
<sup>c</sup>Peak serum concentration of most short-acting oral morphine preparations occurs after 1 hour; doses can be given as frequently as q2h without stacking/overlapping of doses.
<sup>d</sup>Initial dosing of short-acting oral morphine for opioid-naïve patients is 5 to 10 mg q4h prn.
<sup>e</sup>Not appropriate for the initial management of acute pain.
<sup>f</sup>Initial dose of hydromorphone for opioid-naïve patients is 0.2 to 0.6 mg IV q2-3h prn.
<sup>g</sup>Should only be used by those certified in conscious sedation and with appropriate monitoring/emergency equipment.
• Select alternative agents to meperidine, propoxyphene (no longer FDA-approved in the United States), and codeine to limit potential side effects and drug interactions. At least 10% of the US population lacks the appropriate enzyme to convert codeine to the active compound morphine.

• One should always be aware of the “hidden” acetaminophen in combination products and not exceed the 4 g/d limit (2 g/d in those with liver disease).

• Consider obtaining a pain management consult for other options in persistent, severe, uncontrolled pain.

• Trust your instincts. If you think a patient is displaying aberrant drug-related behaviors, set boundaries and stick with them. Questions to ask:
  • Does the patient only ask for pain medications when you are in the room?
  • Do others observe different behaviors when you leave the room?
  • Is the patient “splitting” the staff, playing one against the other?
  • Is the patient talking or resting comfortably? This may be quite misleading in patients with chronic pain and should not be assumed to be a sign of deception in such patients.
  • Is the patient allergic to every pain medication except the one he or she is requesting?
  • Is the patient unwilling to accept any adjunctive nonopioid treatment?
  • Is the patient very sleepy or lethargic and still asking for more?

EQUIPOTENT ANALGESIC DOSES OF OPIOIDS

Equipotent analgesic doses are approximate, and clinical conversions should be done carefully.

1. Calculate the total opioid dose used in the previous 24 hours.
2. Convert the total dose to an oral morphine equivalent using Table 16-2.
3. Convert from oral morphine equivalent to the new opioid.
4. Give 50% of the calculated daily dose to account for incomplete cross-tolerance between opioids. Conversion to or from methadone is not as straightforward (see Table 16-2).
<table>
<thead>
<tr>
<th>Drug</th>
<th>SQ/IV Dose (mg)</th>
<th>PO Dose (mg)</th>
<th>Duration (hours)</th>
<th>Half-Life (hours)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Short half-life opioids</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Morphine</td>
<td>10</td>
<td>30</td>
<td>4</td>
<td>2–3.5</td>
</tr>
<tr>
<td>Hydrocodone</td>
<td>—</td>
<td>30</td>
<td>4</td>
<td>3–4</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>—</td>
<td>20</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>—</td>
<td>20</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>1.5</td>
<td>7.5</td>
<td>4</td>
<td>2–3</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.1</td>
<td>—</td>
<td>1–2</td>
<td>1.5–6</td>
</tr>
<tr>
<td><strong>Long-acting opioids</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Methadone</td>
<td>—</td>
<td>1.7–6.7</td>
<td>4–48[^b]</td>
<td>15–60[^c]</td>
</tr>
</tbody>
</table>

When converting to methadone, use a starting dose of 10% of the 24h IV morphine equivalent dose (24h dose of IV morphine) \( \times 0.10 \) = total daily methadone; divide by 2 for q12h, 3 for q8h dosing. Expect steady state in 5–7 days, dose adjust weekly.

[^a]: Duration of analgesic effect is for single-dose administration. To achieve steady state, it takes 5 half-lives; exert caution when starting long half-life opioids.
[^b]: Duration of action of single-dose methadone is 4 to 8 h and after 3 to 5 days of repeated doses 22 to 48 h.
[^c]: There is highly significant variability in the pharmacokinetic parameters of methadone.
5. Schedule the dosing frequency based on the analgesic half-life (e.g., for morphine: q4h, MS Contin: q8-12h; oxycodone: q4h, OxyContin: q12h).

6. Divide the calculated 24-hour dosage by the number of doses to be given daily.

7. Add prn doses of the new opioid (short-acting form) at 5% to 15% of the total daily dose for breakthrough pain.
BASAL REQUIREMENTS

Water

• Basal water requirement may be calculated as follows:
  • For the first 10 kg of body weight, 4 mL/kg/h PLUS
  • For the second 10 kg of body weight, 2 mL/kg/h PLUS
  • For remaining weight above 20 kg, 1 mL/kg/h

• Fever, increased respiratory rate, and sweating can all increase insensible water losses. Insensible losses increase by 100 to 150 mL/d for each degree of body temperature above 37°C.

Electrolytes

• Sodium: 50 to 150 mmol/d (as NaCl). Most of this is excreted in the urine.
• Chloride: 50 to 150 mmol/d (as NaCl).
• Potassium: 20 to 60 mmol/d (as KCl), assuming renal function is normal. Most of this is excreted in the urine.

Carbohydrates

• Dextrose: 100 to 150 g/d.
• IV dextrose administration minimizes protein catabolism and prevents ketoacidosis.

MAINTENANCE INTRAVENOUS FLUIDS

• Basal requirements of water, electrolytes, and carbohydrates can be conveniently administered as 0.45% NaCl in 5% dextrose plus 20 mmol/L KCl.
• Fluid losses can be divided as urinary losses and all other losses.
  • Urinary losses for the average adult are 0.5 to 1 mL/kg/h (e.g., 70 kg person produces approximately 40 to 60 mL/h or 1,200 mL/d).
• Other losses (i.e., water lost in sweat, stool, hydration, insensible losses) total approximately 800 mL/d.

• For average-sized adults, 2 to 3 L (90 to 125 mL/h) of this IV solution per day is sufficient (i.e., D5⅓NS + 20 mmol KCl/L @ 100 mL/h).

• Patients with hypovolemia require more aggressive fluid resuscitation, generally with 0.9% NaCl. Patients with renal failure or CHF may require less.

• GI and renal losses may significantly increase the loss of water, Na⁺, and K⁺. Serum electrolytes should be followed closely in these situations.

ELECTROLYTE ABNORMALITIES

Hyponatremia

Etiology

• **Hypotonic hyponatremia** is caused by primary water gain or Na⁺ loss. Na⁺ loss may be the result of renal or extrarenal causes.

• **Hypertonic hyponatremia** is caused by an increase in extracellular solute concentration (e.g., hyperglycemia or IV mannitol administration).

• **Isotonic hyponatremia** (pseudohyponatremia) occurs as a result of a decrease in the aqueous phase of plasma (e.g., hyperproteinemia, hyperlipidemia). The concentration of Na⁺ per liter of plasma water is normal.

Evaluation

• A careful H&P should be done, paying close attention to fluid status and the neurologic examination.

• Plasma osmolality, urine osmolality, and urine [Na⁺] should be measured.

• Refer Figure 17-1.

Treatment

• Mild asymptomatic hyponatremia generally requires no treatment.

• For isovolemic and hypervolemic hypotonic hyponatremia, consider fluid restriction.

• For hypovolemic hypotonic hyponatremia, consider saline therapy. **Too rapid correction may result in osmotic demyelination or central pontine myelinolysis.**
Plasma osmolality

High (>300 mOsm/kg)
- Hypertonic Hyponatremia
  - Hyperglycemia
  - Mannitol
  - Hyperproteinemia
  - Hyperlipidemia

Normal (275–300 mOsm/kg)
- Isotonic Hyponatremia
  - “Pseudohyponatremia”

Low (<275 mOsm/kg)
- Hypotonic Hyponatremia

Maximal volume of maximally dilute urine? ($U_{osm}<100$ mOsm/kg)

No
- Assess ECF volume

Increased
- Hypervolemic Hypotonic Hyponatremia
  - CHF
  - Cirrhosis
  - Nephrotic syndrome
  - Renal failure
  - Fluid restriction
  - Consider normal saline + loop diuretics

Normal
- Isovolemic Hypotonic Hyponatremia
  - SIADH
  - Drugs
  - Physical and emotional stress
  - Glucocorticoid deficiency
  - Hypothyroidism
  - Renal failure

<10 mmol/L
- Extrarenal sodium loss
  - GI (vomiting, tube drainage, diarrhea)
  - Skin (sweating, extensive burns)
  - 3rd spacing (peritonitis, pancreatitis)
  - Treatment: Fluid restriction (1.5 L)

>20 mmol/L
- Renal sodium loss
  - Diuretics
  - Sodium wasting nephropathy
  - Hypoaldosteronism
  - Post-obstructive diuresis
  - Nonoliguric ATN
  - Treatment: Isotonic saline

Decreased
- Hypovolemic Hypotonic Hyponatremia
  - Urine sodium concentration

Yes
- Primary polydipsia

Figure 17-1. Evaluation of hyponatremia.
Chronic asymptomatic hyponatremia: Correct slowly, about 5 to 8 mEq/L over 24 hours.

Severe symptomatic hyponatremia (CNS symptoms): Usually treated with hypertonic saline; however, this must be done with great care and very close monitoring. Aim to correct 1 to 2 mmol/L for 3 to 4 hours; once patient is stable, taper off such that the rise in Na⁺ does not exceed 10 to 12 mmol/L over 24 hours.

The quantity of Na⁺ required to increase the plasma Na⁺ by a given amount can be estimated as follows:

\[
\text{Na⁺ deficit (mmol)} = \text{desired change in } [\text{Na⁺}] \times \text{TBW}
\]
\[
\text{TBW} = 0.6 \times \text{body weight (kg)}
\]

For example, if the desired change in [Na⁺] is 8 mmol in a 70 kg patient, then 336 mmol of Na⁺ would be required \((42 \times 8 = 336)\). This would be 0.65 L hypertonic (3%) saline (336 mmol ÷ 513 mmol/L) or 2.2 L isotonic (0.9%) saline (336 mmol ÷ 154 mmol/L).

Hypernatremia

**Etiology**

- Hypernatremia is caused by Na⁺ gain or water deficit (the latter is much more common).
- **Water deficit** caused by decreased intake may be seen in patients with limited access to water (e.g., mental status alteration, intubated patients) or impaired thirst.
- **Water loss** may be the result of renal or extrarenal causes.
- Rarely, hypernatremia may result from excess Na⁺ intake (e.g., hypertonic saline or NaHCO₃).

**Evaluation**

- A careful H&P should be done, paying close attention to fluid status and the neurologic examination.
- Plasma osmolality, urine osmolality, and urine [Na⁺] should be measured.
- Solute excretion rate = urine osmolality × urine volume.
- Refer Figure 17-2.

**Treatment**

- Underlying conditions should be treated (e.g., hyperglycemia, diarrhea).
Flowchart and Text:

- **Assess ECF volume**
  - **Increased**
    - Administration of hypertonic saline or NaHCO₃
    - Urine osmole excretion rate >750 mOsm/d?
      - Yes
        - Diuretic
        - Osmotic diuresis (hyperglycemia, mannitol, high-protein diet)
      - No
        - Renal response to DDAVP?
          - Increased
            - Central diabetes insipidus
          - Unchanged
            - Nephrogenic diabetes insipidus
  - **Not increased**
    - Minimum volume (≤500 mL/d) of maximally concentrated urine (>800 mOsm/kg)?
      - Yes
        - Insensible water loss (fever, exercise, heat exposure, burns)
        - GI water loss (osmotic diarrhea, viral gastroenteritis)
        - Remote renal water loss
      - No

Figure 17-2. Evaluation of hypernatremia.

- ECF volume should be restored in hypovolemic patients with isotonic saline.
  
  \[
  \text{Free water deficit (L) = (plasma [Na⁺] – 140)/140 \times TBW (L)}
  \]

  \[
  \text{TBW = 0.6 \times body weight (kg)}
  \]

- As with hyponatremia, **correcting hypernatremia too rapidly is potentially dangerous.** The rate of correction of the plasma [Na⁺] should not exceed 0.5 mmol/L/h and the [Na⁺] should decrease by
no more than 12 mmol/L over the first 24 hours. With chronic asymptomatic hypernatremia, lower [Na⁺] more slowly, about 5–8 mmol/L/d.

- Don’t forget to take into account ongoing losses. The safest route is PO or NG tube administration of water. Alternatively, ½NS (0.45%), ¼NS (0.225%), or D5W can be given IV. Reassess volume status and [Na⁺] every 8 to 12 hours.
- Central diabetes insipidus is treated with intranasal dDAVP.
- Nephrogenic diabetes insipidus may be reversible by treating the underlying disorder or eliminating the offending drug (e.g., lithium).

**Hypokalemia**

- Defined as a [K⁺] < 3.5 mmol/L, the clinical features vary greatly. Myalgias and weakness are common complaints.
- Severe hypokalemia can result in an increased risk of arrhythmias. The [K⁺] level of cardiac patients is generally maintained above 4 mmol/L.

**Etiology**

- **Decreased intake**: This is infrequently the sole cause but can exacerbate other causes of hypokalemia.
- **Transcellular shifts**: Metabolic alkalosis, insulin, stress-induced catecholamine release, β-adrenergic agonists, and anabolic states all cause K⁺ to shift into cells.
- **Nonrenal and renal loss**: Renal loss of K⁺ may be caused by increased distal K⁺ secretion (mineralocorticoid excess, Liddle’s syndrome) or increased distal tubular flow rate (loop and thiazide diuretics, Bartter and Gitelman syndromes). GI causes include vomiting and diarrhea. Coexistent hypomagnesemia should be ruled out.

**Evaluation**

- When the etiology of hypokalemia is not immediately apparent, renal K⁺ excretion and acid–base status can help identify the cause.
- The transtubular potassium gradient is calculated as follows:

  \[
  \text{TTKG} = \frac{U_{[K^+]}}{P_{[K^+]}} / \left( \frac{U_{\text{osm}}}{P_{\text{osm}}} \right)
  \]

  - TTKG <2 suggests a nonrenal source of K⁺ loss.
  - TTKG >4 suggests inappropriate renal K⁺ secretion.
- Acid–base status: Hypokalemia is generally associated with metabolic alkalosis. The finding of metabolic acidosis implies lower GI losses, distal RTA, or DKA.
Treatment

- $K^+$ may be repleted either orally or IV. It is difficult to provide an algorithmic approach to replacing $K^+$ as the degree of total depletion does not correlate well with plasma levels.
- It is generally safer and more cost-effective to replace $K^+$ via the oral route. Caution should be used in replacing $K^+$ in patients with renal insufficiency. A reasonable estimate in patients with normal renal function is that every 10 mmol of KCl will increase the serum level by 0.05 to 0.1 mmol/L.
- Severe hypokalemia or patients who cannot take anything PO should be treated with IV KCl. The rate of infusion should not exceed 10 mmol/h for a peripheral line. Rates higher than this should be administered via a central line and rates >20 mmol/h require observation unit/ICU monitoring. Rates >40 mmol/h should only occur in the ICU for patients with life-threatening hypokalemia.
- If hypomagnesemia is present, this should be supplemented as well.

Hyperkalemia

- Defined as a $[K^+] > 5$ mmol/L, the most serious effect is cardiac toxicity. Toxicity also depends on the acuity of the hyperkalemia. Gradually developing, chronic, modest hyperkalemia in the range of 5.0 to 5.6 mmol/L is generally tolerated and does not necessarily require aggressive treatment.
- If a suspicious result is received, consider repeating it stat; $[K^+]$ can be obtained on an ABG.
- An ECG must be obtained. Refer to ECG interpretation section in Chapter 19. Look for peaked T waves, prolonged PR interval, and QRS duration. Consider continuous cardiac monitoring.

Etiology

- Pseudohyperkalemia is caused by $K^+$ movement out of cells associated with venipuncture. This may be seen with repeated fist clenching, prolonged tourniquet time, hemolysis, leukocytosis, or thrombocytosis.
- Increased $K^+$ intake is an unusual cause of hyperkalemia but may be seen in the setting of excess $K^+$ replacement, renal insufficiency, or both.
- Transcellular shifts: Acidosis, insulin deficiency, drugs (e.g., succinylcholine, β-blockers), hypertonicity (e.g., hyperglycemia), hemolysis, tumor lysis, rhabdomyolysis, and hyperkalemic periodic paralysis all cause potassium to shift out of cells.
• Decreased renal K⁺ excretion is a common cause of chronic hyperkalemia. Major causes of decreased renal potassium excretion include
  • Acute or chronic kidney disease
  • Decreased effective circulating volume
  • Hypoaldosteronism
    ▪ Primary adrenal insufficiency
    ▪ Hyporeninemic hypoaldosteronism (type 4 RTA)
  • Drugs (e.g., NSAIDs, ACE inhibitors, angiotensin receptor blockers, cyclosporine, heparin, spironolactone, triamterene, amiloride, trimethoprim, pentamidine)

Evaluation
• Rule out pseudohyperkalemia by repeating the serum electrolytes. Consider drawing the sample without the use of a tourniquet or fist clenching.
• Obtain a stat ECG and an ABG (if acidosis is a concern).
• Assess the patient’s urine output and renal function.
• Examine the patient, paying particular attention to ECF volume status.
• Review the patient’s medication list.
• Assessment of TTKG and plasma renin and aldosterone levels may be useful when etiology is not immediately apparent.
  • TTKG > 10 suggests that renal tubular mechanisms for K⁺ secretion are intact. In this case, hyperkalemia could be due to high K⁺ intake and/or decreased effective circulating volume causing diminished distal solute delivery and hence decreased K⁺ secretion.
  • TTKG < 7 implies impaired K⁺ secretion caused by hypoaldosteronism, aldosterone resistance, or hyporeninemic hypoaldosteronism.

Treatment
• Stop all exogenous K⁺ and potentially offending drugs.
• Not all hyperkalemia requires immediate aggressive treatment. This is particularly true of CKD/ESRD patients who often have mild hyperkalemia (5.0 to 5.6 mmol/L). Unless there are clinical signs of significant hyperkalemia, the acute management below is usually unnecessary.
• Severe hyperkalemia or hyperkalemia with ECG changes requires emergent treatment. Do not do this by yourself. Call your resident immediately.
• **Acute treatment**
  
  - **Calcium gluconate** 10%, 10 mL IV over 2 to 3 minutes decreases cardiac membrane excitability. The effect occurs in minutes but lasts only 30 to 60 minutes. It can be repeated after 5 to 10 minutes if the ECG does not change. Use with extreme caution in patients receiving digoxin.
  
  - **Insulin**, 5 to 10 units IV, causes an intracellular shift of $K^+$ in 10 to 30 minutes. The effect lasts for several hours. Glucose, 100 g IV (2 amp D50), should also be administered to prevent hypoglycemia and the patient’s blood sugar should be checked in 1 to 2 hours.
  
  - **NaHCO$_3$** 1 ampule (i.e., 50 mmol HCO$_3^-$ in 50 mL) IV can also be used to cause an intracellular shift of $K^+$, and the effect can last several hours. This treatment should probably be reserved for patients with severe hyperkalemia and metabolic acidosis. Patients with end-stage renal disease seldom respond and may not tolerate the Na$^+$ load.
  
  - **$\beta_2$-adrenergic agonists** can be used to cause an intracellular shift of $K^+$.
  
  - **Diuretics** (e.g., furosemide, 40 to 120 mg IV) enhance $K^+$ excretion provided renal function is adequate.
  
  - **Cation exchange resins** (sodium polystyrene sulfonate; Kayexalate) enhance $K^+$ excretion from the GI tract, but single doses are only mildly effective. Though previously very common, the FDA now recommends that Kayexalate NOT be given in sorbitol solution due to the risk of intestinal necrosis. Kayexalate reconstituted in water may be given PO (15 to 30 g) or as a retention enema (50 g in 150 mL of tap water). It may be beneficial to coadminister an alternative laxative, such as lactulose or MiraLAX, when giving PO Kayexalate. Doses may be repeated q4-6h.
  
  - **Dialysis** may be necessary for severe hyperkalemia when other measures are ineffective and for patients with renal failure.
  
• **Chronic treatment** is aimed at the underlying condition. Dietary $K^+$ should be restricted. Metabolic acidosis should be corrected. Drugs causing hyperkalemia should be avoided. Administration of exogenous mineralocorticoid may be effective for select patients.
GENERAL PRINCIPLES

• Changes in acid–base balance occur as a result of changes in $[\text{H}^+]$ and $[\text{HCO}_3^-]$.

• Acidemia (pH $<7.37$) results from either decreased $[\text{HCO}_3^-]$ or increased $\text{PCO}_2$.

• Alkalemia (pH $>7.43$) results from either increased $[\text{HCO}_3^-]$ or decreased $\text{PCO}_2$.

• An ABG, electrolyte panel, and a serum $[\text{HCO}_3^-]$ are required to assess acid/base status.

• Stepwise approach to an ABG:
  1. Examine the pH. Is the patient acidic or alkaline?
  2. Establish the primary disturbance.
      a. Examine the $[\text{HCO}_3^-]$. In primary metabolic disorders, it moves in the same direction as the pH.
      b. Examine the $\text{PCO}_2$. In primary respiratory disorders, it moves in the opposite direction as the pH.
      c. A combined disorder is present when 1) pH is normal but $\text{PCO}_2$ and $[\text{HCO}_3^-]$ are both abnormal or 2) changes in both $\text{PCO}_2$ and $[\text{HCO}_3^-]$ can cause the change in pH.
  3. Is there adequate respiratory or metabolic compensation? If there is not adequate compensation, there may be a combined disorder present (Table 18-1).
  4. If a metabolic acidosis is present:
      a. Calculate the anion gap: $\text{AG} = [\text{Na}^+] - ([\text{Cl}^-] + [\text{HCO}_3^-])$.
      b. If no gap is present, calculate the urine anion gap: $\text{UAG} = U_{[\text{Na}^+]} + U_{[\text{K}^+]} - U_{[\text{Cl}^-]}$. A negative UAG suggests GI $\text{HCO}_3^-$ losses, whereas a positive UAG suggests an RTA.
  5. If there is an anion gap, assess the delta gap:
      a. $\text{AG}_{\text{correct}} = \text{AG} + ((4 - \{\text{albumin}\}) \times 2.5)$
      b. $\Delta \text{AG} = \text{AG}_{\text{correct}} - 10$
<table>
<thead>
<tr>
<th>Disorder</th>
<th>Abnormality</th>
<th>Primary Changes</th>
<th>Compensatory Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>[HCO₃⁻] loss or [H⁺] gain</td>
<td>↓[HCO₃⁻]</td>
<td>↓PCO₂ by 1.0 – 1.3 mm Hg for every 1.0 mmol/L ↓[HCO₃⁻]</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>[H⁺] loss or [HCO₃⁻] gain</td>
<td>↑[HCO₃⁻]</td>
<td>↑PCO₂ 0.6 – 0.7 mm Hg for every 1 mmol/L ↑[HCO₃⁻]</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>Alveolar hypoventilation</td>
<td>↑PCO₂</td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td></td>
<td></td>
<td>↑[HCO₃⁻] 1.0 mmol/L for every 10 mm Hg ↑PCO₂</td>
</tr>
<tr>
<td>Chronic</td>
<td></td>
<td></td>
<td>↑[HCO₃⁻] 3.0–3.5 mmol/L for every 10 mm Hg ↑PCO₂</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>Alveolar hyperventilation</td>
<td>↓PCO₂</td>
<td>↓[HCO₃⁻] 2.0 mmol/L for every 10 mm Hg ↓PCO₂</td>
</tr>
<tr>
<td>Acute</td>
<td></td>
<td></td>
<td>↓[HCO₃⁻] 4.0 – 5.0 mmol/L for every 10 mm Hg ↓PCO₂</td>
</tr>
<tr>
<td>Chronic</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
c. $\Delta [\text{HCO}_3^-] = 24 - [\text{HCO}_3^-]$

d. $\Delta \text{AG} = \Delta [\text{HCO}_3^-]$ indicates simple AG metabolic acidosis.

e. $\Delta \text{AG} > \Delta [\text{HCO}_3^-]$ indicates AG metabolic acidosis and metabolic alkalosis.

f. $\Delta \text{AG} < \Delta [\text{HCO}_3^-]$ indicates AG metabolic acidosis and nongap metabolic acidosis.

### METABOLIC ACIDOSIS

#### Etiology and Diagnosis

See Table 18-2.

#### Treatment

- Treatment of the underlying condition should be the primary focus.
- Severe acidosis (pH < 7.20) may require treatment with parenteral NaHCO$_3$. Rapid infusion should be considered only for very severe acidosis.

### TABLE 18-2 Causes of Metabolic Acidosis

<table>
<thead>
<tr>
<th>Increased Anion Gap</th>
<th>Normal Anion Gap</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methanol</td>
<td>GI [HCO$_3^-$] loss (diarrhea, urinary diversion, small bowel, biliary, pancreatic, cholestyramine, or ingestion of Ca or Mg chloride)</td>
</tr>
<tr>
<td>Uremia</td>
<td>Ingestion of exogenous acids</td>
</tr>
<tr>
<td>Diabetic ketoacidosis; alcoholic ketoacidosis</td>
<td>Proximal (type 2) renal tubular acidosis</td>
</tr>
<tr>
<td>Paraldehyde</td>
<td>Classic distal (type 1) renal tubular acidosis</td>
</tr>
<tr>
<td>Lactic acidosis</td>
<td>Hyperkalemic (type 4) renal tubular acidosis</td>
</tr>
<tr>
<td>Ethylene glycol</td>
<td>Early renal insufficiency</td>
</tr>
<tr>
<td>Salicylates</td>
<td>Expansion acidosis (rapid saline administration)</td>
</tr>
<tr>
<td></td>
<td>Drug-induced hyperkalemia (K$^+$-sparing diuretics, trimethoprim, pentamidine, ACE inhibitors, nonsteroidal anti-inflammatory drugs, cyclosporine), carbonic anhydrase inhibitors</td>
</tr>
</tbody>
</table>
• Overaggressive correction should be avoided to prevent overshoot alkalosis.
• Adverse effects of parenteral NaHCO$_3$ include pulmonary edema, hypernatremia, hypokalemia, and hypocalcemia. Monitor electrolytes frequently.

**METABOLIC ALKALOSIS**

**Etiology**
• Metabolic alkalosis may be caused by HCO$_3^-$ gain, H$^+$ loss, or volume contraction.
• Vomiting and diuretic use are the two most common causes.
• See Table 18-3.

**Treatment**
• Treatment of the underlying condition should be the primary focus.
• Correct hypokalemia and hypomagnesemia.

<table>
<thead>
<tr>
<th>Cl$^-$ Responsive (Urine Cl$^-$ &lt;10 mmol/L)</th>
<th>Cl$^-$ Unresponsive (Cl$^-$ &gt;10 mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gastrointestinal</strong></td>
<td></td>
</tr>
<tr>
<td>Vomiting, NG suction</td>
<td>K$^+$ or Mg$^{2+}$ depletion</td>
</tr>
<tr>
<td>Villous adenoma</td>
<td>Bartter’s syndrome</td>
</tr>
<tr>
<td>Congenital chloridorrhea</td>
<td>Hypercalcemia</td>
</tr>
<tr>
<td>Cystic fibrosis</td>
<td></td>
</tr>
<tr>
<td><strong>Renal</strong></td>
<td><strong>Hypertensive</strong></td>
</tr>
<tr>
<td>Diuretics</td>
<td></td>
</tr>
<tr>
<td>Posthypercapnic state</td>
<td>Primary aldosteronism</td>
</tr>
<tr>
<td>Nonreabsorbable anions</td>
<td>Hyperreninemic</td>
</tr>
<tr>
<td>(penicillin)</td>
<td>hyperaldosteronism</td>
</tr>
<tr>
<td>Exogenous alkali (NaHCO$_3$, massive</td>
<td>Exogenous mineralocorticoid</td>
</tr>
<tr>
<td>transfusion, antacids, acetate, citrate)</td>
<td></td>
</tr>
<tr>
<td>Contraction alkalosis</td>
<td></td>
</tr>
</tbody>
</table>

**Adrenal enzyme defects**

Primary aldosteronism
Hyperreninemic hyperaldosteronism
Exogenous mineralocorticoid
Pseudohyperaldosteronism (licorice, carbenoxolone, tobacco chewing, Liddle’s syndrome)
• Chloride-responsive metabolic alkaloses should be treated with isotonic NS.
• Chloride-unresponsive metabolic alkaloses do not improve with saline administration.
  • K⁺-sparing diuretics (e.g., amiloride, spironolactone) are effective for mineralocorticoid excess.
  • In patients with normal renal function, alkalosis from excessive alkali administration will resolve quickly once the HCO₃⁻ load is withdrawn.
  • Acetazolamide may be useful if alkalosis persists despite the above interventions or if saline administration is limited by volume overload.

**RESPIRATORY ACIDOSIS**

**Etiology**
- ↑PCO₂ is almost always the result of alveolar hypoventilation.
- In **acute respiratory acidosis**, the pH ↓0.08 for every 10 mm Hg ↑PCO₂ above 40 mm Hg.
- In **chronic respiratory acidosis**, the pH ↓0.03 for every 10 mm Hg ↑PCO₂ above 40 mm Hg.
- Renal compensation takes several days to develop fully.
- See Table 18-4.

**Treatment**
- Treatment is directed at the underlying condition.
- Potentially contributing drugs should be stopped or counteracted (e.g., naloxone, flumazenil).

<table>
<thead>
<tr>
<th>Table 18-4</th>
<th>Causes of Respiratory Acidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central respiratory depression (drugs, sleep apnea, obesity, CNS disease)</td>
<td></td>
</tr>
<tr>
<td>Airway obstruction (foreign body, laryngospasm, severe bronchospasm)</td>
<td></td>
</tr>
<tr>
<td>Neuromuscular abnormalities (polio, kyphoscoliosis, myasthenia, muscular dystrophy)</td>
<td></td>
</tr>
<tr>
<td>Parenchymal lung disease (COPD, pneumothorax, pneumonia, pulmonary edema, interstitial lung disease)</td>
<td></td>
</tr>
</tbody>
</table>
Acid–Base Disorders

• Ventilatory assistance may be required (CPAP, BiPAP, or mechanical ventilation).
• Avoid NaHCO$_3$ administration as this can worsen hypercapnia (HCO$_3^-$ combines with H$^+$ in the tissues to form CO$_2$ + H$_2$O).

**TABLE 18-5** Causes of Respiratory Alkalosis

| Central stimulation (anxiety, pain, hyperventilation syndrome, head trauma, CVA, tumors, fever/infection, salicylates, thyroxine, progesterone) |
| Hypoxemia (any causes) |
| Airway irritation |
| Decreased lung compliance (CHF, fibrosis) |
| Pulmonary embolism |
| Hepatic insufficiency/failure |
| Pregnancy |
| Hyperthyroidism |
| Overzealous mechanical ventilation |

**RESPIRATORY ALKALOSIS**

**Etiology**

• It is important to remember that tachypnea/hyperventilation does not necessarily imply a simple respiratory alkalosis. If you have any uncertainty, obtain an ABG.
• See Table 18-5.

**Treatment**

• Treatment is directed at the underlying condition.
• Psychogenic hyperventilation may be treated by rebreathing from a paper bag.
ECG

TACHYCARDIA

• Tachyarrhythmias are broadly categorized as wide-complex tachycardia (WCT) and narrow-complex tachycardia (NCT).

• In the hemodynamically unstable patient with a tachyarrhythmia, immediately ask for help, initiate ACLS if warranted, and place defibrillator pads to prepare for electrical cardioversion.

• In the otherwise stable patient, some time and thought can lead to a satisfying diagnosis!

Narrow-Complex Tachycardias

• NCTs are almost always supraventricular in origin. When dealing with NCTs it is useful to first assess whether the rhythm is regular or irregular (Figure 19-1).

• If the tachyarrhythmia is regular, a standard 12-lead ECG should be examined for p-waves. The rhythm can then be further categorized by assessing whether the p-wave is closer to the R-wave that precedes it (short R-P tachycardia) or the R-wave that follows it (long R-P tachycardia).

• In the case of long R-P tachycardia consider the diagnoses of 1) sinus tachycardia, 2) atrial tachycardia, or 3) atrial flutter.

• In the case of short R-P tachycardia, consider the diagnosis of 1) AVnRT or 2) AVRT.

• If the tachyarrhythmia is irregular, the diagnosis is almost always 1) atrial fibrillation, atrial flutter (with variable block) or 2) multifocal atrial tachycardia (MAT).

Sinus Tachycardia

• Remember, sinus tachycardia is almost always secondary to another process.
• In patients with underlying causes for sinus tachycardia such as hypovolemia, pulmonary emboli, or MI, an elevated heart rate may be the patient’s only means for maintaining cardiac output (heart rate \times stroke volume = cardiac output), and thus treatment of sinus tachycardia in these patients with negative chronotropic agents (β-blockers, calcium channel blockers) is ill-advised.

• A thorough workup for underlying causes including those mentioned above as well as infection, anemia, anxiety, exertion, thyroid disease, certain drugs or other substances, autonomic neuropathy (especially of diabetes), and inflammation should be pursued before sinus tachycardia is treated with medications that slow the heart rate.

• In the patient without heart failure, intravenous fluids aid in the management of most conditions that cause sinus tachycardia and are a useful reflex starting point in management.

• If telemetric data are available at the onset of the arrhythmia, an examination of a graph of heart rate over time will typically show gradual increase to the fast rate with sinus tachycardia, whereas other NCTs will show abrupt change in rate, signifying the change in rhythm from sinus.

**Atrial Fibrillation**

• Atrial fibrillation is the result of numerous disorganized foci of depolarization within the atrium and is represented on the standard 12-lead ECG with an irregularly irregular R-R interval and no discernible p-waves (Figure 19-2).

![Atrial fibrillation](image)

**Figure 19-2.** Atrial fibrillation.
New onset atrial fibrillation and atrial flutter usually have an underlying cause that must be investigated.

A convenient mnemonic for common causes of atrial flutter or fibrillation is PIRATES: Pulmonary disease, Ischemia, Rheumatic or valvular heart disease, Anemia, Thyrotoxicosis, Ethanol/Electrolytes, Sepsis.

Because the atrioventricular (AV) node can usually not conduct at a rate greater than 180 bpm, if the ventricular response is at a higher rate, strongly suspect the presence of a coexisting pre-excitation syndrome.

Likewise, if the patient in atrial fibrillation on no negative chronotropic agents has a ventricular rate less than 120, strongly suspect node dysfunction. This is important when considering cardioversion.

In the hemodynamically stable patient, as you search for the underlying cause of atrial fibrillation, consideration should be given to a rate-control versus rhythm-control strategy, depending on the reversibility of the atrial fibrillation.

Rate control is typically achieved with β-blockers and calcium channel blockers, or digoxin in patients with heart failure and lower blood pressures (see below).

Anticoagulation should also be considered, depending on the patient’s risk of stroke, regardless of whether sinus rhythm can be restored.

For new atrial fibrillation of unknown time period, or period greater than 48 hours, care must be taken to avoid cardioversion with pharmacologic or electrical means due to the risk of thromboembolism.

Atrial Flutter

Atrial flutter is the result of large reentrant foci of ectopy within the atria. There are several types of atrial flutter, but the rhythm should be suspected when the atrial rate is around 300 bpm (Figure 19-3).

Figure 19-3. Atrial flutter.
• Workup and management are identical to those for atrial fibrillation; consideration must be given to rate control, rhythm control, and anticoagulation.

• Because the AV node cannot usually conduct impulses at a rate greater than 180 bpm, the ventricular rate corresponding to atrial flutter is usually slower and frequently around 150 bpm.

• One frequent misdiagnosis occurs when the ECG is read as “sinus tachycardia with first-degree heart block” with a ventricular rate of around 150 bpm. If the p-p interval is identical to the r-r interval, strongly suspect atrial flutter with 2:1 block, with half the flutter waves buried within the QRS interval.

Management of Atrial Flutter and Fibrillation

• Rate control can typically be achieved with calcium channel blockers such as diltiazem, β-blockers such as metoprolol, or digoxin.

• Diltiazem can be given as an IV push, with a typical initial dose 10 to 15 mg, followed by a continuous infusion, with heart rate and blood pressure parameters communicated to the nursing staff for them to titrate the dose (i.e., please titrate to HR < 100 and SBP > 100).

• Metoprolol is usually given in increments of 5 mg IV, with up to three consecutive doses at 5-minute intervals, followed by a PO regimen, such as 12.5 mg PO q6h.

• Digoxin, while also a negative chronotrope, is not an antihypertensive, making it useful for slowing the heart rate in patients with lower blood pressures. Taking caution in patients with renal failure and electrolyte abnormalities, digoxin can be given in increments of 0.25 mg IV q4h. Keep in mind that digoxin will take longer to exert its effect than diltiazem or metoprolol. Ask for help prior to using other agents such as amiodarone, as these confer a risk of cardioversion.

Multifocal Atrial Tachycardia

• MAT is an irregularly irregular rhythm that is the result of multiple distinct foci of pacemaker activity within the atria.

• MAT is identified by the presence of three or more distinct p-waves that reliably give rise to QRS complexes.

• Each p-wave should have a unique morphology and PR interval, owing to their distinct locations in relation to the surface electrocardiographic leads and the AV node.

• Management does not differ from atrial fibrillation, although the etiology of this rhythm is usually pulmonary in nature, and anticoagulation need not be considered as patients are not at increased risk for thromboembolism.
AVnRT and AVRT

- AVnRT (AV-nodal reentrant tachycardia) and AVRT (AV reentry tachycardia) are distinct, pathophysiologically different arrhythmias.
- AVnRT arises from conduction tissue of differential speed within the AV node.
- AVRT arises when conduction occurs, often through a muscular bridge, between the atria and ventricles independent of the AV node.
- AVnRT typically occurs at a rate of around 180 bpm.
- AVRT can run much faster, owing to the fact that it does not depend on the AV node to sustain its rates.
- Strongly suspect one of these rhythms when a tachycardia is regular at rates greater than 170 bpm.
- As always, in the hemodynamically unstable patient, call for help, initiate ACLS if necessary, and prepare for electrical cardioversion!
- **In the hemodynamically stable patient, attempt to make a diagnosis by “breaking the rhythm.”**
  - This can be done by first asking the nurse to set up a 12-lead ECG and run a rhythm strip.
  - Initially, ask the patient to perform *vagal maneuvers* (i.e. Valsalva).
  - If this does not work, listen for carotid bruits and if there are none, attempt carotid massage.
  - Never attempt bilateral simultaneous *carotid massage*, for risk of cerebral hypoperfusion!
  - If neither of these produces a satisfactory result, ask for help and have a nurse bring vials of adenosine to the bedside.
  - **Adenosine** will block conduction through the AV node, “breaking” AVRT or AVnRT.
  - To administer adenosine, have a nurse place a y-stopcock onto a standard IV. Prepare the patient by telling them they may have an unpleasant sensation upon administration.
  - Always have the defibrillator close by in the event cardioversion or pacing is required.
  - Have the nurse push 6 mg followed immediately by a saline flush, as adenosine will be rapidly metabolized in the arm prior to reaching the heart without a flush.
  - If this does not produce a satisfactory result, try the same maneuver with 12 mg of adenosine.
  - If the adenosine has successfully exerted an effect, but the rhythm is not in fact AVRT or AVnRT, QRS complexes will be suppressed,
and only atrial activity will appear on the rhythm strip. This might aid in the diagnosis of the rhythm, if suppression of the QRS complexes shows flutter waves or a nonsinus p-wave (as in atrial tachycardia).

**Atrial Tachycardia**

- A sinus p-wave should be positive in leads II, III, and aVF and negative or biphasic in lead V1, with a PR interval of 120 to 20 ms.
- If an NCT arises where the morphology of the p-wave is unlike that of a sinus p-wave, the rhythm is atrial tachycardia.
- It is useful to distinguish atrial tachycardia from sinus tachycardia, as the search for underlying causes will differ.
- Management of atrial tachycardia is geared toward slowing conduction through the AV node, usually with β-blockers or calcium channel blockers.

**Wide-Complex Tachycardias**

- At the beginning of your internship, you should always ask for help before attempting to manage patients with WCT!
- In the unstable patient with WCT, time to defibrillation is the strongest predictor of good outcomes and survival. As always with an unstable patient, call for help, initiate ACLS protocol, and place defibrillator pads to prepare for electrical cardioversion if necessary.
- In the stable patient, a WCT can be the result of a supraventricular rhythm (including sinus tachycardia) with bundle branch block, a supraventricular rhythm with aberrant conduction, or ventricular tachycardia (VT).

**Diagnosis of WCT**

- Key features on history, labs, and the ECG can help make the diagnosis.
- **In a patient with structural heart disease, history of MI, or significant abnormalities of magnesium or potassium levels, a WCT is almost always VT, given that the patient does not have underlying bundle branch block** (Figure 19-4).
- The presence of capture or fusion beats on ECG clinches a diagnosis of VT as well.
- There have been numerous proposed algorithms to aid in the diagnosis of WCT as supraventricular tachycardia versus VT, the most well-known of which is the Brugada Criteria, found elsewhere.
Remember, in a patient with chest pain and a new WCT, acute MI is a very likely diagnosis, and you should immediately instruct the nurse to administer aspirin and call for help!

**Management of WCT**

- Management of stable WCT depends on the diagnosis.
- In the case of VT, options include electrical cardioversion or attempted pharmacologic cardioversion.
- In the case of SVT, management is per the NCT section above.
- In either case, the patient is at high risk for something bad happening and you should call for help before initiating treatment!

**Nonsustained Ventricular Tachycardia**

- Often as an intern, you will be called for “beats of NSVT” or “run of NSVT.”
- NSVT, or nonsustained ventricular tachycardia, is defined as fewer than 30 consecutive wide-complex beats that are ventricular in origin.
- These usually occur in patients with heart failure or toxin ingestion. Given that these patients are usually hemodynamically stable, nothing need be done on an emergent basis.
- As always, if the patient demonstrates hemodynamic instability or altered mental status during the episodes, ask for help, as the patient likely requires treatment.
- Large studies have been done to evaluate the outcomes for patients who are treated with antiarrhythmic agents to suppress ectopic beats. In these studies, those patients whose ectopic beats were effectively suppressed had higher mortality, whereas those simply treated with β-blockade had improved survival.
- Therefore, in most patients with NSVT, titrating up a β-blocker as tolerated is currently the most effective management.
- NSVT alone is rarely an indication for placement of an intracardiac cardioverter-defibrillator (ICD).
Ventricular Fibrillation and Torsades de Pointes

• Close this book and call a Code Blue! These rhythms should prompt immediate initiation of ACLS and rapid defibrillation.
• In the case of torsades, IV magnesium can aid restoration of normal sinus rhythm.

BRADYARRHYTHMIAS AND DISORDERS OF CONDUCTION

Sinus Bradycardia

• Sinus bradycardia is defined as sinus rhythm with a rate of less than 60 bpm.
• Sinus bradycardia is most often caused by increased vagal tone, antiarrhythmic agents, ischemia, and conduction system degeneration (with long-standing hypertension, diabetes, or increased age).
• Sinus bradycardia is common in sleeping patients.
• Sinus bradycardia should be treated only if symptoms of hypotension exist. That is to say, if you are called for sinus bradycardia, and the patient is asymptomatic with an acceptable blood pressure, this patient requires no treatment.
• Look for offending agents that can cause bradycardia, such as β-blockers, calcium channel blockers, digoxin, or other medications. Discontinue these immediately.
• Acute treatment can consist of the anticholinergic agent atropine and/or placement of defibrillator pads with transcutaneous pacing.
• Atropine can be given in increments of 0.5 mg IV until the heart rate is satisfactory.
• Atropine has a half-life of 2 hours, so after resolution with atropine, care must be taken to ensure the bradycardia does not return.
• Infusions such as dopamine or isoproterenol can be considered as pharmacologic means to restore and maintain the heart rate. Call for help prior to initiating these treatments.
• Transcutaneous pacing should be employed if necessary, but is exceedingly uncomfortable for the patient. Transvenous pacing should be considered in the symptomatically bradycardic patient, with the help of an upper level resident or cardiology fellow.
First-Degree AV Block
- The PR interval is greater than 200 ms, but all p-waves result in a QRS complex (Figure 19-5).
- First-degree block can be caused by increased vagal tone, electrolyte abnormalities, conduction system degeneration, and drugs (calcium channel blockers, β-blockers, and other antiarrhythmic agents are common causes). Ischemia can in specific instances prolong the PR interval.
- First-degree block rarely requires treatment. If severe, consideration can be given to discontinuation of offending drugs.

Mobitz Type I and Mobitz Type II Second-Degree AV Block
In second-degree block, not all p-waves result in a QRS complex.

Mobitz Type I Block (Wenckebach)
- Mobitz I is identified when the **PR interval progressively increases** between PQRS complexes, until a p-wave is not conducted to the ventricle and does not result in a QRS complex (Figure 19-6).
- Mobitz I usually occurs as a result of slowed conduction within the AV node.
- The QRS complexes should not change in morphology.
- Etiologies are the same for first-degree block.
- Mobitz type I block is usually asymptomatic.
- Given that it is asymptomatic, and **does not usually progress to worsening heart block, this type of block requires no treatment**.
- If symptomatic bradycardia ensues, treatment is the same as that for sinus bradycardia.

**Figure 19-5.** First-degree AV block.

**Figure 19-6.** Mobitz type I (Wenckebach), second-degree AV block.
Mobitz Type II Block

- Mobitz type II block is diagnosed when the PR interval is fixed, but one or more p-waves do not result in a QRS complex (Figure 19-7).
- Mobitz II usually occurs as a result of slowed conduction just below the AV node.
- Blocked conduction may occur in a fixed ratio (two, three, or four conducted p-waves for one nonconducted p-wave).
- Causes are similar to Mobitz type I block.
- Because Mobitz type II block is at high risk for degenerating into complete AV block, these patients must be closely monitored.
- Treatment usually involves placement of an external pacemaker, regardless of symptoms.

Third-Degree (Complete) AV Block

- No p-waves are conducted; complete AV dissociation occurs (Figure 19-8).
- QRS complexes that arise do so as the result of intrinsic pacemakers within or below the level of the AV node.
- “Escape” rhythms usually occur in the acute setting that obviate the development of asystole. If a narrow QRS occurs, the rhythm originates from the AV node’s intrinsic pacemaker and is referred to as “junctional.” If a wide QRS occurs, the rhythm originates from somewhere within the ventricles and is termed “idioventricular.”

Figure 19-7. Mobitz type II, second-degree AV block.

Figure 19-8. Complete heart block.
Third-degree block carries a high risk of degeneration to asystole. Therefore, regardless of symptoms, an immediate plan for transcutaneous and/or transvenous pacer, with eventual permanent pacemaker, should be implemented.

Escape Rhythms

- Stimulation of the His-Purkinje system and the ensuing QRS complex is driven by the fastest active intrinsic pacemaker within the heart. Usually, this is the sinus node.
- In the event of sinus node failure, the AV node’s pacemaker may resume pacemaking responsibilities for the heart, resulting in a junctional rhythm. Junctional rhythm can be recognized when a narrow-complex QRS (or a QRS similar to the one caused by sinus rhythm) arises without any detectable atrial activity (p-waves). The rate is usually between 40 and 60 bpm.
- In the event of failure of both the junctional and sinus pacemakers, pacemakers within the ventricle may resume pacemaking responsibilities. If this occurs, the rhythm is termed idioventricular rhythm. Because the pacemakers are located within the ventricle, conduction does not necessarily occur through the His-Purkinje system, and the QRS complex is wide. The rate is typically around 40 bpm. Thus, idioventricular rhythm can be identified by a wide, regular, and slow rhythm that occurs in the absence of atrial activity.
- Treatment is the same as for patients with third-degree heart block. Your resident or fellow should be called, and plans for increasing the heart rate using drugs such as dopamine or dobutamine or temporary pacing should be considered.

MYOCARDIAL ISCHEMIA

- Remember to keep the clinical picture in mind when interpreting ECGs. In the symptomatic patient, very subtle ECG changes could represent significant ischemia. Likewise, in the asymptomatic patient, consider whether you would have ordered an ECG in the first place. As always, if you are unsure, ask for help!
- When confronted with a patient with suspected ischemia, it is always useful to obtain an old ECG for comparison! If the old ECG is normal, interpretation of the new ECG is less complicated. Often, the patient will have baseline abnormalities on the old ECG, and changes from that baseline could represent a pathologic process.
ST Segment and T-waves

- Myocardial ischemia is characterized by symmetric T-wave inversion, flat or downsloping ST depression, or both.
- ST elevation MI (STEMI), representing severe ischemia, will present with ECG changes as shown in Figure 19-9, depending on when in the pathological process the ECG is taken.
- The distribution of leads in which ECG changes representing ischemia are observed can often help localize the region of myocardium that is ischemic (Table 19-1).
- If changes are observed in leads I and aVL, ischemia likely involves the high lateral wall; the culprit vessel is typically a proximal diagonal artery or the circumflex artery.
- If changes are observed in leads II, III, and aVF, ischemia likely involves the inferior wall; the culprit lesion is likely in the distal circumflex artery or mid- to distal right coronary artery.
- Changes in leads V1–V2 represent the septal region of the heart; consider a left main or proximal LAD lesion.

Figure 19-9. ST and T-wave changes in STEMI.
TABLE 19-1  ECG LOCALIZATION OF MYOCARDIAL INFARCTION

<table>
<thead>
<tr>
<th>Area of MI</th>
<th>ECG Abnormality</th>
<th>Artery Involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septal</td>
<td>ST elevation and Q-waves in V1–V2</td>
<td>Proximal LAD, septal perforators</td>
</tr>
<tr>
<td>Anteroseptal</td>
<td>ST elevation and Q-waves in V1–V4</td>
<td>Left anterior descending</td>
</tr>
<tr>
<td>Anterior</td>
<td>ST elevation and Q-waves in V3–V4</td>
<td>Left anterior descending</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>ST elevation and Q-waves in I, aVL, V3–V6</td>
<td>Mid-LAD or circumflex</td>
</tr>
<tr>
<td>Extensive anterior</td>
<td>ST elevation and Q-waves in I, aVL, V1–V6</td>
<td>Proximal LAD</td>
</tr>
<tr>
<td>Lateral</td>
<td>ST elevation and Q-waves in I, aVL, V6</td>
<td>Circumflex</td>
</tr>
<tr>
<td>High lateral</td>
<td>ST elevation and Q-waves in I, aVL</td>
<td>Circumflex</td>
</tr>
<tr>
<td>Inferior</td>
<td>ST elevation and Q-waves in II, III, aVF</td>
<td>Right coronary artery</td>
</tr>
<tr>
<td>Posterior</td>
<td>Tall R and ST depression V1–V2</td>
<td>RCA or circumflex</td>
</tr>
<tr>
<td>Right ventricular</td>
<td>ST elevation V4R</td>
<td>Proximal RCA</td>
</tr>
</tbody>
</table>

- Changes in V3–V5 represent a continuum of myocardium extending from the anterior LV to lateral LV; consider mid- to distal LAD and proximal to mid-circumflex lesions.
- In a symptomatic patient, a new LBBB could represent severe ischemia, and you should immediately call for help!
- Keep in mind that in patients with known LBBB, there should be some anteroseptal ST segment elevation and lateral ST segment depression. Likewise, in RBBB, there should be some anteroseptal ST depression. Absence of these changes could represent ischemia in the symptomatic patient with known bundle branch block and warrant further workup.
- Similarly, patients with LVH or on digoxin (Figure 19-10) will often have lateral ST segment depression, and interpretation of those changes as ischemic or nonischemic must be made taking
into account all clinical factors (e.g., symptoms) and laboratory values (e.g., cardiac enzymes).

**R-Waves**

- The utility of precordial R-waves in interpreting ECGs is often underappreciated.
- Examining leads V1–V6 sequentially, the R-wave should be larger than the S-wave by lead V3, and larger in each subsequent lead than the previous. If this pattern is not present, the patient is said to have poor R-wave progression.
- The differential for poor R-wave progression includes obesity, incorrect lead placement, and pulmonary disease; however, in the setting of a patient who is suspected of having myocardial ischemia, poor R-wave progression could represent the equivalent of a q-wave. The interpretation of an ECG with poor R-wave progression should read “cannot rule out anterior infarct, age indeterminate.” As always, interpret the ECG in the setting of all clinical and laboratory information at hand.
- The true posterior region of the myocardium is poorly represented on the standard 12-lead ECG. The presence of an R-wave in lead V1 with ST depression should alert the clinician to a possible STEMI of that region of the heart. Call for help if you note this ECG finding in the symptomatic patient!

**Q-Waves**

- Pathologic q-waves are longer than 40 ms in duration and larger than one third the QRS amplitude or one fourth the R-wave amplitude.
- Q-waves usually represent a region of the myocardium that has suffered an infarct in the past.
- However, as always in the symptomatic patient, your threshold for recognizing a change as acute should be higher, and close attention should be paid to newly found q-waves.

**Management**

- Management of acute myocardial ischemia should usually involve your resident. Aspirin 162 mg PO is always a good starting point.
As internists, there is no medicine we give that affects mortality as significantly as aspirin in the setting of acute MI!

Conditions Mimicking STEMI

- **ST elevations in the symptomatic patient should be treated as STEMI until proven otherwise. Still, it is useful to know that certain other conditions will also cause ST elevations on the ECG.**

- **Pericarditis will cause diffuse ST elevations** (Figure 19-11). Certain clues, however, can point away from the diagnosis of STEMI. Clinically, the patient may have the classic pericarditis presentation, with chest pain and shortness of breath that is relieved with leaning forward or with NSAIDs. The patient may also have features on history that predispose to pericarditis. On ECG, the ST elevations will often not follow a single arterial distribution. Classically, the ECG will also show depression of the PR segment.

- **Takotsubo cardiomyopathy can also cause diffuse ST elevations.** Takotsubo cardiomyopathy is an increasingly recognized condition that occurs during periods of great emotional or physiological stress; patients present with symptoms identical to that of acute MI. In addition to ST elevations, segmental myocardial wall motion abnormalities also occur. Typically, though Takotsubo cardiomyopathy occurs in the absence of pericardial coronary artery occlusion, cardiac catheterization is needed to make this diagnosis of exclusion.

- **A late sequel of a STEMI is the formation of ventricular aneurysm,** which can also present with ST elevations. Clues to this diagnosis include the presence of ST elevations in a patient with recent STEMI but without new symptoms. The diagnosis is clinched with ventricular imaging such as echocardiography.

- **Severe hyperkalemia,** usually with levels greater than 5.5 mg/dL, can present with **sharply peaked T-waves,** imitating the changes found during the hyperacute phase of a STEMI (Figure 19-12).

![Figure 19-11. Pericarditis.](image)
As hyperkalemia worsens, the QRS will widen. Strongly suspect hyperkalemia in the patient with known renal disease or on high doses of spironolactone, ACE inhibitors, or angiotensin receptor blockers. ECG changes should trigger immediate treatment of hyperkalemia, initially with 2 g IV calcium gluconate, followed by measures to drive potassium out of the intravascular space and eliminate it from the body.

**RADIOGRAPHY**

**CHEST X-RAY**

The chest X-ray is by far the most common radiograph you will order and need to interpret. When reading a chest X-ray, like any radiograph, the most important consideration is to be systematic. Be sure to check the name and date on the image and compare with old images whenever possible.

**Technique**

- Is the exposure correct? Underexposure can cause you to see things that aren’t there, while overexposure can cause pathology to disappear. You should be able to faintly see the intervertebral disc spaces through the cardiac silhouette.
- Is the patient properly positioned? The spinous processes and trachea should be midline. The clavicular heads should be equidistant from the spinous processes. Rotated films distort the appearance of the cardiac silhouette and hila.
- Is the frontal image posterior-anterior (PA) or anterior-posterior (AP)? AP images are often obtained in emergent situations or when the patient cannot stand. A two-view (PA and lateral) examination is optimal if the patient can tolerate it. A normal cardiac silhouette will appear larger on an AP exam due to its proximity to the X-ray source.
- Was the image taken at full inspiration? Small lung volumes can produce vascular crowding and apparent mediastinal widening and atelectasis.
Lines and tubes

- If the patient is intubated, check the position of the endotracheal tube (should be a minimum of 2 cm above the carina with 3 to 5 cm optimal).

- Central venous catheters should follow expected venous courses and should generally terminate in the superior vena cava, near the level of the carina along the right aspect of the mediastinum. The end of the catheter should travel along the long axis of the superior vena cava (vertically).

- Nasogastric and enteric feeding tubes may be partially visualized. Ensure they do not coil in the esophagus or extend outward into the lung due to endobronchial placement.

Airway

- The trachea should be midline and not deviated.

- The trachea will deviate away from the side of a pneumothorax if there is tension physiology. In cases of volume loss such as lobar collapse, it will deviate toward the affected side.

Bones

Systematically look at the sternum, ribs, clavicles, spine, and shoulders for fractures, osteolytic or osteoblastic lesions, and arthritic changes.

Diaphragm

- The sides of the diaphragm should be equal and slightly rounded. The right side may be slightly higher. Elevation of one side may suggest paralysis, loss of lung volume on that side, diaphragmatic eventration, or diaphragmatic tear (in the setting of trauma).

- Look for blunting of the costophrenic angles suggesting small pleural effusions, best seen on the lateral view.

- Flat hemidiaphragms are indicative of hyperexpansion, often seen in emphysema.

- Check for free air under the diaphragm on an upright radiograph, which can be seen with bowel perforation. If you think you’ve detected free intraperitoneal air, let your resident know right away.

Soft Tissues

Examine the soft tissues for symmetry, subcutaneous air, edema, and breast tissue.
Heart and Mediastinum

- Maximal heart width greater than half of the chest width suggests cardiomegaly or pericardial effusion.
- The aortic knob should be distinct.
- Mediastinal widening may indicate thoracic aortic dissection or aneurysm, lymphadenopathy, or mass. In obese patients, it may be related to mediastinal fat deposition.
- Mediastinal and tracheal deviation can be seen with a tension pneumothorax. As above, the trachea will deviate away from the side of the pneumothorax if tension physiology is present.
- Use lateral images to confirm findings on frontal images and look for retrocardiac pathology, such as lower lobe pneumonias or hiatal hernias.

Hilar Structures

- The left hilum is usually 2 to 3 cm higher than the right. They are generally of equal size.
- Enlarged hila suggest lymphadenopathy or pulmonary artery enlargement. Use the lateral image to help differentiate.

Lung Markings

- Look for normal lung markings all the way out to the chest wall to rule out pneumothorax. If lung markings are not seen to the periphery, look for a thin white visceral pleural line. **Be sure not to miss this! If you think you’ve detected a pneumothorax, let your resident know right away.**
- Normal lung markings taper as they travel out to the periphery and are smaller in the upper lungs. Lung markings in the upper lung fields that are as large as or larger than those in the lower lung (“cephalization”) suggest pulmonary edema.
- Kerley’s B lines (small linear densities perpendicular to the pleural surface often best seen in the lung bases) are seen in congestive heart failure.
- Hyperlucent lungs with increased retrosternal clear space on the lateral image are seen in emphysema.
- Examine the lungs for areas of consolidation and nodules.
- Obscuration of all or part of the heart border (silhouette sign) implies that a lesion is contiguous with or abuts the heart border and likely lies within the right middle lobe or lingula.
- A small pleural effusion is suggested by blunting of the costophrenic angle, best seen on the lateral image. Larger effusions obscure the
shadow of the diaphragm and produce an upward-curving shadow along the chest wall. A straight horizontal air-fluid level indicates a concurrent pneumothorax (“hydropneumothorax”).

- Lateral decubitus films can be done to ensure that the effusion is free flowing and large enough to attempt thoracentesis (usually >1 cm on lateral film). The side of the effusion should be down.

### PLAIN ABDOMINAL FILMS

Ordered quite frequently, plain abdominal films (“KUB” or “obstructive series”) are usually a first-line screening study and subsequent studies may be needed to clarify or identify pathology. An obstructive series consists of a frontal image and left lateral decubitus or upright image and should be ordered when evaluating for perforation or small bowel obstruction. Again, a systematic approach is key.

### Bones

- Examine the bones first or else you’ll forget.
- Begin with the spine, then ribs, pelvis, and upper femurs. Look for arthritis, fractures, and osteolytic or osteoblastic lesions.

### Lines and Tubes

- Nasogastric/orogastric tubes should terminate in the left upper quadrant, with the proximal-most side port past the gastroesophageal junction.
- Enteric feeding tubes should course past the stomach into the right abdomen and then cross back over the midline to the left (following the course of the duodenum) to terminate in the jejunum.

### Soft Tissues

- Systematically study the soft tissues looking for evidence of masses or calcifications. Calcifications can be seen over the gallbladder, renal shadows, ureteral courses; in the right lower quadrant (appendicolith); or overlying the uterus (fibroids). Phleboliths (vascular calcifications) are commonly seen in the pelvis and usually have a lucent center.
- Be sure to carefully look for free air under the diaphragm (upright film) or next to the liver edge (left lateral decubitus film). Free air is indicative of bowel perforation. If you see this, let your resident know immediately!
Gastrointestinal Structures

• Look for the gastric bubble. A large air-distended stomach suggests some form of obstruction or dysfunction.

• Observe the bowel gas pattern. A small amount of air is generally seen in the colon, while the small bowel is generally devoid of air. Fecal material is often visible in the colon although large amounts may be seen in constipation.

• The colon may become distended (colon diameter >6 cm or cecum >9 cm) in colonic obstruction or ileus. Unless the distension is severe, the haustral markings are maintained. Large bowel markings are differentiated from small bowel markings by wider spacing and incomplete crossing of the lumen. When the ileocecal valve is incompetent, large bowel obstruction may also cause gaseous distension of the small bowel.

• Distension of the small bowel (>3 cm in diameter) may be seen in mechanical obstruction or ileus. Small bowel striations are much more numerous, completely cross the lumen, and may become effaced with dilatation.

• With mechanical obstruction, there is distension proximal to the obstruction and clearing of air distally. The appearance of ileus is much less distinct. There is discontinuous air in the small and usually large bowel. The degree of distension is also less remarkable.

• Air-fluid levels unfortunately do not always distinguish mechanical obstruction from ileus, as they may be seen in both conditions.

PREPARATION FOR PROCEDURES

General Points

• Perform plain radiographs prior to contrast studies. Perform iodinated contrast studies prior to barium studies. Barium can cause metallic streak artifact on CT, which obscures findings to the point that it may preclude the exam until the barium is cleared from the bowel.

• Consult your radiology department if you have questions about what study to order or to confirm preparation for procedures. Some preparations are institution specific (see Table 19-2).

• Studies requiring no preparation include chest and abdominal radiographs, C-spine series, transthoracic echo, as well as those listed below.
<table>
<thead>
<tr>
<th>Procedure</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CT</strong></td>
<td>Usually none if noncontrast. If intravenous contrast is to be administered, patient should be NPO for 4–6 h</td>
</tr>
<tr>
<td>Chest/extremity/head</td>
<td>No IV contrast needed if looking for renal stone or retroperitoneal bleed. Other types of studies usually require IV contrast if possible and may require oral depending on the patient and the indication. Discuss special protocol needs (liver, pancreas, renal protocol) and need for oral contrast with the radiologist</td>
</tr>
<tr>
<td>Abdominal/pelvic</td>
<td>Usually none; sometimes oral contrast material will be given</td>
</tr>
<tr>
<td><strong>MRI</strong></td>
<td>NPO starting 6 h prior to procedure</td>
</tr>
<tr>
<td><strong>Ultrasound</strong></td>
<td>4 glasses of water 1 h prior; no voiding 1 h prior to exam</td>
</tr>
<tr>
<td>Abdominal</td>
<td>NPO 6 h prior to procedure</td>
</tr>
<tr>
<td>Pelvic</td>
<td>NPO if concern for aspiration</td>
</tr>
<tr>
<td><strong>Gastrointestinal studies</strong></td>
<td>NPO starting midnight the day of procedure</td>
</tr>
</tbody>
</table>

- Barium swallow (used to evaluate pharynx and esophagus typically for dysphagia workup)
- Modified barium swallow (used to evaluate for possible aspiration during feeding)
- Upper GI (used to evaluate the esophagus, stomach, and proximal small intestine; typically used to look for ulcers)
<table>
<thead>
<tr>
<th>Procedure</th>
<th>Instructions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small bowel follow-through</td>
<td>NPO starting midnight the day of the procedure</td>
</tr>
<tr>
<td>Barium enema</td>
<td>NPO after midnight; bowel prep varies by indication. Consult the radiology department</td>
</tr>
<tr>
<td>HIDA scan</td>
<td>NPO starting midnight the day of procedure</td>
</tr>
<tr>
<td>PET scan</td>
<td>NPO starting midnight the day of procedure. In diabetic patients, glucose must be under reasonable control (&lt;200 mg/dL)</td>
</tr>
<tr>
<td><strong>Genitourinary studies</strong></td>
<td>No dietary restriction</td>
</tr>
<tr>
<td>Cystogram</td>
<td>Full bladder</td>
</tr>
<tr>
<td><strong>Interventional studies</strong></td>
<td>If the patient is having a procedure that will require sedation</td>
</tr>
<tr>
<td>EGD/ERCP</td>
<td>(almost all procedures other than tube or line change) the patient</td>
</tr>
<tr>
<td>Colonoscopy</td>
<td>should be NPO 4–6 h before the exam</td>
</tr>
<tr>
<td><strong>Endoscopic studies</strong></td>
<td>NPO starting 6 h prior to procedure</td>
</tr>
<tr>
<td>Flexible sigmoidoscopy</td>
<td>Clear liquids 1 d prior to procedure; 1 gallon of GoLYTELY 1 cup per 15 min until done on night prior to procedure (consider NG tube if not able to complete); NPO after midnight</td>
</tr>
<tr>
<td></td>
<td>Clear liquids starting with dinner the night before; NPO after midnight</td>
</tr>
<tr>
<td></td>
<td>Mag citrate at 8 PM, 1 glass of water every 2 h until 10 PM, then 3 Dulcolax tablets at 10 PM, and 1 Dulcolax suppository at 6 AM</td>
</tr>
</tbody>
</table>
Studies requiring the patient to be NPO include CT scans with contrast, abdominal ultrasound, gastrointestinal studies, HIDA scan, PET scan, and those listed below.

• Remember to restart the diet postprocedure or if procedure is canceled.

**Contrast Reactions**

• Everyone feels a sense of warmth or flushing and many patients experience nausea and/or vomiting during contrast administration—these are not allergies.

• For known contrast sensitivity (e.g., hives, rash), consider prednisone, 50 mg PO q6h × 4 doses prior to the exam. The last dose should be administered 1 hour prior to the administration of contrast. Diphenhydramine, 50 mg PO, can also be added, 1 hour prior to contrast. Specific protocols vary by institution.

• Although only nonionic contrast is used for CT (check your institution for confirmation), if your patient has had a major event with previous contrast administration (e.g., shock or airway compromise), discuss this with the radiologist prior to ordering a test. Patients who have had life-threatening reactions in the past should not receive intravenous contrast again, even with premedication. Allergic reactions generally do not occur with PO contrast.

• The contrast used in MR examinations is a gadolinium preparation, not iodinated contrast. There is cross-reactivity in some patients, so those with severe contrast reaction to nonionic intravenous CT contrast should receive premedication for gadolinium contrast as well. See above for premedication regimen. Those with only mild sensitivity generally do not require premedication.

• There is a relationship between gadolinium administration and nephrogenic systemic sclerosis in the setting of impaired renal function. Patients with creatinine clearance <30 and acute renal failure and/or those on dialysis are at higher risk. Consult with your radiologist about preparatory regimens, reduction in the gadolinium load, or performing the study without contrast.

**Contrast Nephropathy**

• Contrast nephropathy is a common complication of any procedure involving iodinated IV contrast (e.g., radiological studies, angiograms).

• Risk factors include the presence of renal insufficiency, diabetes, reduced intravascular volume, and large amounts of infused contrast.
Strategies for prevention include:

- **IV hydration:** 1 mg/kg/h of 0.45 or 0.9 NS for 6 to 12 hours before and 6 to 12 hours after the procedure. A sodium bicarbonate solution, 3 ampules of NaHCO₃ (150 mEq total) in 1 L D5W, can also be used. This much NaHCO₃ should not be put in saline due to the high sodium load.
- **N-acetylcysteine** 600 mg bid × 2 doses before the procedure and 2 doses after the procedure may also be added but is of uncertain benefit in most patients.
- Contrast should be given cautiously or not at all to anyone with a Cr >2.0 and those with acute renal failure.

**Gastrointestinal Radiology**

- GI studies can be uncomfortable and require patient cooperation and mobility. If your patient is paralyzed, demented, angry, or delirious, the study will likely be suboptimal or may not be able to be performed.
- General rules for the “barium versus Hypaque” dilemma (call the radiologist if you have specific questions):
  - Barium is bad in pleural or peritoneal spaces so avoid this if perforation, obstruction, or a fistula is suspected. Do not use if the patient is likely to need a laparotomy or CT soon.
  - Hypaque is bad in lungs, so avoid in cases of possible aspiration.

**Cardiac Studies**

In general:
- No smoking 2 hours prior to test; remove nicotine patches the morning of the test.
- Small sips of water with medication are fine; however, depending on the type of exam certain cardiac medications (i.e., β-blockers) may need to be held. Consult with the radiology department.
- For specific recommendations, refer to Table 19-3.

**MRI**

- Absolute contraindications are pacemakers, ICDs, cochlear implants, any metal in the globe, and ferromagnetic intracranial aneurysm clips.
- Relative contraindications are recent operations (less than a few weeks) and recent vascular stenting. Prosthetic hip joints or metal implants are not generally contraindicated, but their artifact
may obscure any adjacent lesions. If you have questions, consult MRIsafety.com or call the radiologist.

- These examinations can be relatively long. The patient must be able to lie still and cooperate—consider mild sedation (e.g., lorazepam) if necessary. The tube is quite small, and patients may get claustrophobic.

### Ultrasound

- Abdominal ultrasounds can be used to evaluate the gallbladder, liver, and kidneys. The pancreas is typically not well visualized so consider CT instead.
- Ultrasound can also locate pockets of fluid to guide paracentesis or thoracentesis.
- It is important the patient be made NPO as bowel gas can obstruct the image.

<table>
<thead>
<tr>
<th>Test</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary angiogram</td>
<td>NPO after midnight</td>
</tr>
<tr>
<td>Stress echo, dobutamine stress echo, or exercise stress test</td>
<td>NPO after midnight</td>
</tr>
<tr>
<td>Nuclear stress test (walking)</td>
<td>Hold AM doses of calcium channel blockers and β-blockers</td>
</tr>
<tr>
<td>Adenosine or dipyridamole (Persantine) nuclear stress test (nonwalking)</td>
<td>NPO after midnight</td>
</tr>
<tr>
<td></td>
<td>Hold AM doses of calcium channel blockers and β-blockers</td>
</tr>
<tr>
<td></td>
<td>For diabetics on insulin only, half the normal insulin dose and eat a light meal 3 h prior to procedure (no fats or dairy products)</td>
</tr>
<tr>
<td></td>
<td>Avoid any xanthine-containing products (e.g., chocolate, caffeine) and theophylline or Persantine for 24 h prior to procedure</td>
</tr>
</tbody>
</table>
ANTIBIOTICS

General Principles
Three main issues must be addressed when selecting antibiotics:

• Expected pathogen and patient demographics (i.e., nosocomial, immunocompromised)
• Patient allergies and possible drug-drug interactions
• Renal and hepatic function

Vancomycin

• Used in severe β-lactam-resistant gram-positive bacterial infections and to treat gram-positive bacterial infections in β-lactam allergic patients.

• Therapeutic trough concentrations (draw 30 min prior to next scheduled dose):
  • Uncomplicated skin and soft tissue infections: 10 to 20 μg/mL.
  • All other infections: 15 to 20 μg/mL.

• It can be difficult to initially achieve vancomycin concentrations in this narrow therapeutic range due to significant variability between patients. These therapeutic ranges are narrower than previous recommendations, due to both increased MIC breakpoints for MRSA and increasing reports of vancomycin nephrotoxicity.

• For all patients, the empiric dose of vancomycin should be 15 mg/kg, up to a maximal single dose of 2.25 g and a total daily empirical dose of 4.5 g. Dose should be rounded to the nearest 250 mg.

• Initial dosing frequency and monitoring are determined by patient factors (Table 20-1). Vancomycin should be avoided if possible in those with a CrCl <30 mL/min and not on dialysis.

• Presuming the current dose is ≤1,500 mg and the patient has stable renal function of ≥30 mL/min, subsequent dosing can be adjusted as shown in Table 20-2. Consider obtaining an ID or pharmacy consultation for patients on doses >1,500 mg.
### TABLE 20-1  EMPIRIC VANCOMYCIN DOSING

<table>
<thead>
<tr>
<th>CrCl (mL/min)</th>
<th>Dosing*</th>
<th>Monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;90</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age ≤ 35 years</td>
<td>15 mg/kg q8h</td>
<td>Draw trough level immediately prior to the fourth dose</td>
</tr>
<tr>
<td>Age &gt;35 years</td>
<td>15 mg/kg q12h</td>
<td>Draw trough level immediately prior to the fourth dose</td>
</tr>
<tr>
<td>50–90</td>
<td>15 mg/kg q12h</td>
<td>Draw trough level immediately prior to the fourth dose</td>
</tr>
<tr>
<td>30–49</td>
<td>15 mg/kg q24h</td>
<td>Draw trough level immediately prior to the third dose</td>
</tr>
<tr>
<td>&lt;30</td>
<td>15 mg/kg × 1</td>
<td>Random level 24h later</td>
</tr>
<tr>
<td>Continuous venous-venous hemodiafiltration</td>
<td>15 mg/kg q24h</td>
<td>Draw trough level immediately prior to the third dose</td>
</tr>
<tr>
<td>Sustained low-efficiency daily dialysis (SLEDD)</td>
<td>15 mg/kg initially and then after each SLEDD</td>
<td>Draw trough level immediately prior to the third dose</td>
</tr>
<tr>
<td>Peritoneal dialysis</td>
<td>15 mg/kg × 1</td>
<td>Random level 24h later</td>
</tr>
<tr>
<td>Intermittent hemodialysis (HD)</td>
<td>10–15 mg/kg to a max of 1.5 g after each HD</td>
<td>Draw trough level immediately prior to third HD session</td>
</tr>
</tbody>
</table>

*Round dose to nearest 250 mg; maximum single dose 2.25 g; maximum daily dose 4.5 g.

Adapted from Casabar E, Portell J, eds. Tool Book: Drug Dosing and Usage Guidelines. 10th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital, Washington University Medical Center; 2012.

### Aminoglycosides

- Aminoglycosides are primarily used for serious aerobic gram-negative infections; they are also used synergistically with β-lactam antibiotics in the treatment of select gram-positive infections. Adverse effects include ototoxicity, nephrotoxicity, and neuromuscular paralysis.
<table>
<thead>
<tr>
<th>Vanc Trough (μg/mL)</th>
<th>Currently q8h Dosing</th>
<th>Currently q12h Dosing</th>
<th>Currently 24 h Dosing</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤5</td>
<td>Consider alternative therapy</td>
<td>↑ dose by 250–500 mg and continue q8h</td>
<td>↑ dose by 250–500 mg and continue q12h</td>
</tr>
<tr>
<td>5.1–10</td>
<td>↑ dose by 250 mg and continue q8h (or no change if uncomplicated skin infection)</td>
<td>↑ dose by 250 mg and continue q12h (or no change if uncomplicated skin infection)</td>
<td>↑ dose by 250 mg and continue q24h (or no change if uncomplicated skin infection)</td>
</tr>
<tr>
<td>10.1–15</td>
<td>No change</td>
<td>No change</td>
<td>No change</td>
</tr>
<tr>
<td>15.1–20</td>
<td>↓ dose by 250 mg and continue q8h (or continue same dose q12h)</td>
<td>↓ dose by 250 mg and continue q12h (or continue same dose q24h)</td>
<td>↓ dose by 250 mg and continue q24h</td>
</tr>
<tr>
<td>20.1–25</td>
<td>Hold vancomycin</td>
<td>Hold vancomycin</td>
<td>Hold vancomycin</td>
</tr>
<tr>
<td>&gt;25</td>
<td>Hold vancomycin</td>
<td>Hold vancomycin</td>
<td>Hold vancomycin</td>
</tr>
</tbody>
</table>

Adapted from Casabar E, Portell J, eds. Tool Book: Drug Dosing and Usage Guidelines. 10th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital, Washington University Medical Center; 2012.
Serum Cr should be monitored frequently during therapy.

Baseline audiometry is recommended for patients whose treatment is planned for >2 weeks.

**Extended interval (i.e., infrequent) dosing regimens are generally preferred**, but there are contraindications (Table 20-3).

Calculate the correct **dosing weight** (DW):

- **Calculate ideal body weight** (IBW):
  - IBW (male) = 50 kg + 2.3 (height in inches − 60)
  - IBW (female) = 45.5 kg + 3.4 (height in inches − 60)

- **Underweight**: if IBW > ABW (actual/current body weight) then DW = ABW

- **Normal weight**: if IBW < ABW < 1.2 (IBW) then DW = IBW

- **Obese**: if ABW > 1.2 (IBW) then DW = IBW + 0.4 (ABW − IBW)

**Traditional Aminoglycoside Dosing**

- Calculate a loading dose as per Table 20-4.
- Calculate the maintenance dose as per Table 20-5.
- Initial trough and peak levels are drawn immediately prior to and 1 hour after the start of the third dose. Goal levels when the CrCl is >30 mL/min are presented in Table 20-6. Goal level for CrCl <30 mL/min and those on renal replacement therapy are shown in Table 20-7.

**Extended Interval Dosing**

- Confirm that there are no contraindications to extended interval dosing (EID) (Table 20-3).
- Calculate loading dose based on DW (Table 20-8).
- For non-cystic fibrosis patients, the dosing interval is adjusted using the nomograms in Figure 20-1.

**ANTICOAGULATION**

- Before initiating anticoagulant therapy, ensure that the patient has no history of active peptic ulcers, recent stroke or bleeding, or recent surgery.
- All patients should have a digital rectal examination to document stool occult blood status.
<table>
<thead>
<tr>
<th>Indications</th>
<th>Traditional Dosing</th>
<th>Extended Interval Dosing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical dose assuming normal renal function</td>
<td>1–2 mg/kg q12h (based on indication)</td>
<td>5 mg/kg q24h</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8 mg/kg q24h for CF patients</td>
</tr>
<tr>
<td>Bone and joint infections</td>
<td>Endocarditis</td>
<td>CNS infections</td>
</tr>
<tr>
<td>Gram-positive infections</td>
<td>Mycobacterial infections</td>
<td>Cystic fibrosis</td>
</tr>
<tr>
<td>Septic shock</td>
<td>Skin/soft tissue infections</td>
<td>Febrile neutropenia</td>
</tr>
<tr>
<td>Urinary tract infections</td>
<td></td>
<td>Gram-negative rod bacteremia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intra-abdominal infections</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Open fracture infections</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pneumonia</td>
</tr>
<tr>
<td>Contraindications</td>
<td>Peritoneal dialysis (relative)</td>
<td>&gt;20% BSA burns</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anasarca</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cr &gt; 1.5 mg/dL without febrile neutropenia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cr &gt; 1.9 mg/mL with febrile neutropenia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CrCl &lt; 30 mL/min</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hearing loss</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Liver dysfunction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pregnancy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Septic shock</td>
</tr>
<tr>
<td>HD/CVVHDF/SLEDD</td>
<td>Indicated</td>
<td>Contraindicated</td>
</tr>
<tr>
<td>PD</td>
<td>Renal consult</td>
<td></td>
</tr>
</tbody>
</table>

CF, cystic fibrosis; HD, hemodialysis; CVVHDF, continuous venous–venous hemodiafiltration; SLEDD, sustained low-efficiency daily dialysis; PD, peritoneal dialysis.

Adapted from Casabar E, Portell J, eds. Tool Book: Drug Dosing and Usage Guidelines. 10th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital, Washington University Medical Center; 2012.
TABLE 20-4 AMINOGLYCOSIDE LOADING DOSE (TRADITIONAL DOSING)

<table>
<thead>
<tr>
<th>Indication</th>
<th>Gentamicin&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Tobramycin&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Amikacin&lt;sup&gt;c&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endocarditis</td>
<td>1 mg/kg</td>
<td>3.5 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Gram-positive infections</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urinary tract infections</td>
<td>1.5 mg/kg</td>
<td>6.5 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Bone and joint infections</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mycobacterial infections</td>
<td>1.5 mg/kg</td>
<td>6.5 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Skin and soft tissue infections</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Septic shock</td>
<td>2 mg/kg</td>
<td>8 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Other infections (when extended dosing is contraindicated)</td>
<td>2 mg/kg</td>
<td>8 mg/kg</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Round gentamicin to nearest 10 mg.  
<sup>b</sup>Round tobramycin to nearest 10 mg.  
<sup>c</sup>Round amikacin to nearest 50 mg.

Adapted from Casabar E, Portell J, eds. Tool Book: Drug Dosing and Usage Guidelines. 10th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital, Washington University Medical Center; 2012.

Unfractionated Heparin Weight-Based Dosing

- **Typical initial bolus** (round to the nearest 100 units):
  - Acute MI: 60 units/kg, maximum 4,000 units
  - Acute DVT/PE: 80 units/kg, maximum 6,000 units
  - Non-DVT/PE or acute MI: 60 units/kg, maximum of 6,000 units
  - High risk of bleeding: Consider smaller bolus

- **Typical initial IV infusion rate**:
  - Acute MI: 12 units/kg/h, maximum 1,000 units/h
  - Acute DVT/PE: 18 units/kg/h. If BMI >40, consider 14 units/kg/h
  - Non-DVT/PE or acute MI: 14 units/kg/h
  - High risk of bleeding: Consider 12 units/kg/h

- Check aPTT 6 hours after initial bolus and 6 hours after each rate change. After two consecutive aPTTs are therapeutic (60 to 94 s), the aPTT should be monitored each morning.

- CBC should be monitored every 72 hours while on IV heparin.

- Dosage adjustments based on aPTT are shown in Table 20-9.
Low-Molecular-Weight Heparin Dosing

- **Enoxaparin**: 1 mg/kg subcutaneously (SC) q12h (unstable angina, NSTEMI, or venous thromboembolism). For DVT/PE, 1.5 mg/kg q24h is an alternative. If CrCl < 30 mL/min use 1 mg/kg SC q24h.

- **Dalteparin**: 120 units/kg SC q12h (unstable angina or NSTEMI) or 200 units/kg SC q12h (venous thromboembolism).

- **Fondaparinux** (synthetic selective factor Xa inhibitor): 7.5 mg SC q24h (DVT/PE); use 5 mg SC q24h if wt <50 kg, 10 mg SC q24h if wt >100 kg.

- For all ACS patients, round doses down to nearest 10 mg. For all others, round to the nearest 10 mg.

- Individual hospitals are likely to have established protocols for LMWH use in ACS. The enoxaparin ACS protocol at Barnes-Jewish Hospital is presented in Table 20-10.
### TABLE 20-6 GOAL AMINOGLYCOSIDE LEVELS FOR CrCl > 30

<table>
<thead>
<tr>
<th>Goal Amino糖苷类水平 (μg/mL)</th>
<th>Gentamicin/ Tobramycin</th>
<th>Amikacin (μg/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trough</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gram-positive infections</td>
<td>3–5</td>
<td>10–15</td>
</tr>
<tr>
<td>Endocarditis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urinary tract infections</td>
<td>6–8</td>
<td>20–25</td>
</tr>
<tr>
<td>Bone and joint infections</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin and soft tissue infections</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mycobacterial infections</td>
<td>8–10</td>
<td>25–30</td>
</tr>
<tr>
<td>Septic shock</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other infections (when extended dosing is contraindicated)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Adapted from Casabar E, Portell J, eds. Tool Book: Drug Dosing and Usage Guidelines. 10th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital, Washington University Medical Center; 2012.

### TABLE 20-7 GOAL AMINOGLYCOSIDE LEVELS FOR CrCl < 30

<table>
<thead>
<tr>
<th>CrCl &lt; 30 mL/min</th>
<th>Level Timing</th>
<th>Drug</th>
<th>Goal (μg/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVVHDF SLEDD</td>
<td>Random level 24 h after dose</td>
<td>Gentamicin</td>
<td>&lt;1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tobramycin</td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td></td>
<td>Amikacin</td>
<td>&lt;4</td>
</tr>
<tr>
<td>HD</td>
<td>Random level just prior to next HD</td>
<td>Gentamicin</td>
<td>&lt;1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tobramycin</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Amikacin</td>
<td>&lt;4</td>
</tr>
</tbody>
</table>

CVVHDF, continuous venous–venous hemodiafiltration; SLEDD, sustained low-efficiency daily dialysis; PD, peritoneal dialysis; HD, hemodialysis.

Adapted from Casabar E, Portell J, eds. Tool Book: Drug Dosing and Usage Guidelines. 10th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital, Washington University Medical Center; 2012.
<table>
<thead>
<tr>
<th>AMINOGLYCOSIDE LOADING DOSE (EXTENDED INTERVAL DOSING)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gentamicin/ Tobramycin</strong></td>
</tr>
<tr>
<td>5 mg/kg × 1</td>
</tr>
<tr>
<td>Adjust maintenance dose based on nomograms</td>
</tr>
</tbody>
</table>

Adapted from Casabar E, Portell J, eds. Tool Book: Drug Dosing and Usage Guideline. 10th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital, Washington University Medical Center, 2012.
• Dosages need to be adjusted in patients with renal failure; **unfractionated heparin is recommended for patients with a CrCl < 10 or on hemodialysis. Antifactor Xa can be checked in patients with CrCl 10 to 30 mL/min. Risk of bleeding is increased in patients with anti-Xa levels above 0.8 units/mL.**

• Dosing in patient with a BMI >40 is uncertain. Monitoring anti-Xa levels is recommended in these patients.

• There is currently no validated nomogram for adjusting LMWH dosing based on anti-Xa levels in adults.
TABLE 20-9  UNFRACTIONATED HEPARIN WEIGHT-BASED DOSING NOMOGRAM

<table>
<thead>
<tr>
<th>aPTTa</th>
<th>Bolus</th>
<th>Rate (units/kg/h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;40</td>
<td>3,000 units(^b)</td>
<td>↑ 3</td>
</tr>
<tr>
<td>40–50</td>
<td>2,000 units(^c)</td>
<td>↑ 2</td>
</tr>
<tr>
<td>51–59</td>
<td>None</td>
<td>↑ 1</td>
</tr>
<tr>
<td>60–94</td>
<td>None</td>
<td>No change</td>
</tr>
<tr>
<td>95–104</td>
<td>None</td>
<td>↓ 1</td>
</tr>
<tr>
<td>105–114</td>
<td>Hold for 30 min</td>
<td>↓ 2</td>
</tr>
<tr>
<td>&gt;114</td>
<td>Hold for 1 h</td>
<td>↓ 3</td>
</tr>
</tbody>
</table>

\(^a\)aPTT can vary depending on lab; check local scale.
\(^b\)For acute DVT/PE, a higher bolus of 80 units/kg recommended.
\(^c\)For acute DVT/PE, a higher bolus of 40 units/kg may be used.

---

**Warfarin Dosing**

- Warfarin dosing must be individualized!
- The onset of action of warfarin is 24 to 72 hours, but full effect is not achieved until 5 to 7 days.
- Numerous common drugs *increase the effect of warfarin* including amiodarone, azole antifungals, fluoroquinolones, macrolides, metronidazole, TMP/SMX, NSAIDs, acetaminophen, and PPIs.
- Drugs that *decrease the effect of warfarin* include carbamazepine, phenobarbital, phenytoin, rifampin, and ritonavir.
- Initial and subsequent dosing of warfarin may be guided by WarfarinDosing.org (last accessed August 14, 2012), which has been widely used and scrutinized.
- **Atrial fibrillation/atrial flutter, goal INR 2 to 3:**
  - For cardioversion, if rhythm has been present >48 hours, anticoagulate for 3 weeks prior to procedure (or perform TEE prior to cardioversion) and 4 weeks afterward.
  - For patients with chronic or paroxysmal atrial fibrillation (rate or rhythm control):
    - Calculate CHADS\(^2\) score: 1 point each for CHF, hypertension, age > 75 years, DM; 2 points for prior ischemic stroke or TIA.
<table>
<thead>
<tr>
<th>Indication</th>
<th>Estimated CrCl (mL/min)</th>
<th>Dosing (Round Down to Nearest 10 mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSTEMI</td>
<td></td>
<td>1 mg/kg SC q12h</td>
</tr>
<tr>
<td></td>
<td>&gt;30</td>
<td>1 mg/kg SC q24h</td>
</tr>
<tr>
<td></td>
<td>10–30</td>
<td>1 mg/kg SC q12h</td>
</tr>
<tr>
<td>STEMI</td>
<td>&gt;30</td>
<td>Age &lt;75 30 mg IV bolus x 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>With 1 mg/kg SC q12h x 2 doses (max 100 mg/dose for the first two doses)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Then 1 mg/kg SC q12h</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age ≥75 NO IV bolus 0.75 mg/kg q12h</td>
</tr>
<tr>
<td></td>
<td></td>
<td>× 2 doses (max 75 mg/dose for the first two doses)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Then 0.75 mg/kg q12h</td>
</tr>
<tr>
<td></td>
<td>10–30</td>
<td>Age &lt;75 30 mg IV bolus x 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>With 1 mg/kg SC × 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Then 1 mg/kg SC q24h</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age ≥75 NO IV bolus 1 mg/kg q24h</td>
</tr>
</tbody>
</table>

Adapted from Casabar E, Portell J, eds. Tool Book: Drug Dosing and Usage Guideline. 10th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital, Washington University Medical Center; 2012.
If CHADS2 = 0, nothing, or ASA 75 to 325 mg daily
If CHADS2 = 1, warfarin, dabigatran 150 mg BID (75 mg BID if CrCl 15 to 30), or rivaroxaban 20 mg qday (15 mg qday if CrCl 15 to 50) preferred; ASA acceptable if elevated bleeding risk
If CHADS2 ≥2, warfarin dabigatran 150 mg BID (75 mg BID if CrCl 15 to 30) or rivaroxaban 20 mg qday (15 mg qday if CrCl 15 to 50) is recommended.

- **DVT/PE, goal INR 2 to 3:**
  - Initial anticoagulation with UFH, LMWH, or fondaparinux; warfarin can be started the same day. Overlap warfarin with one of above for at least 4 to 5 days and until INR ≥ 2 for 2 consecutive days.
  - First episode due to reversible risk factor: 3 months of treatment.
  - First episode, unprovoked: at least 6 to 12 months of treatment.
  - >1 episode: lifelong treatment.
  - VTE with cancer: anticoagulation until cancer resolution or development of contraindication. VTE recurrence rates are lower with LMWH than with standard warfarin therapy.

- **Tissue or St. Jude’s valve in aortic position, goal INR 2 to 3:**
  - Any tissue valve: 3 months anticoagulation, then ASA 325 mg lifelong.
  - St. Jude’s in aortic position: lifelong anticoagulation.

- **Mechanical valve (except St. Jude’s in aortic position), goal INR 2.5 to 3.5:**
  - Lifelong anticoagulation.
  - Consider adding ASA for caged ball or caged disc valves, CAD, history of stroke, or peripheral embolism.

**Treatment of High INR**

- Recommendations for the treatment of supratherapeutic warfarin anticoagulation are presented in Table 20-11.

- **Treatment must be individualized!**

- Vitamin K can be given in equivalent dosages PO or IVPB. Subcutaneous administration is not recommended due to unpredictable response.

- Oral administration is preferred for minor bleeding. IV administration should be reserved for major bleeding.
<table>
<thead>
<tr>
<th>INR &lt; 4.5</th>
<th>If <strong>NO risk factors</strong> for bleeding, lower or hold next dose and monitor frequently; when INR approaches desired range, resume dosing at lower dose. If <strong>rapid reversal</strong> required for surgery, hold warfarin and give vit K PO 2.5 mg; expect INR to reduce within 24 h. If INR still elevated may give another 2.5 mg vit K.</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.5 &gt; INR &lt; 9</td>
<td>If <strong>NO risk factors</strong> for bleeding, skip next 1–2 doses and monitor frequently; when INR in desired range, resume dosing at lower dose. If <strong>risk factors</strong> for bleeding, skip next dose and give vit K PO 2.5 mg; when INR in desired range, resume dosing at lower dose. If <strong>rapid reversal</strong> required for surgery, hold warfarin and give vit K PO 5 mg; expect INR to reduce within 24 h. If INR still elevated may give another 2.5 mg vit K.</td>
</tr>
<tr>
<td>INR &gt; 9</td>
<td>Hold warfarin, give vit K PO 2.5–5 mg and monitor frequently; expect INR to reduce within 24–48 h. Additional vit K may be given if necessary. When INR in desired range, resume dosing at lower dose.</td>
</tr>
<tr>
<td>Any INR with serious or life-threatening bleeding</td>
<td>Hold warfarin. Give fresh frozen plasma (2–4 units), prothrombin complex concentrate (25–50 units/kg), or recombinant factor VIIa. Give vit K 5–10 mg slow IVPB; IV vit K can be repeated if necessary q12h.</td>
</tr>
</tbody>
</table>

*Adapted from Casabar E, Portell J, eds. Tool Book: Drug Dosing and Usage Guideline. 10th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital, Washington University Medical Center; 2012.*
• If vitamin K is given IVPB, administer slowly to minimize risk of anaphylactoid reaction.

• The onset of action of oral vitamin K is 6 to 12 hours and 1 to 2 hours for IV. The peak effect is in 24 to 48 hours and 12 to 14 hours, respectively.

• Vitamin K is not recommended in the following circumstances:
  • INR < 4.5 with no active bleeding with and no surgery or procedure planned within 24 hours
  • INR ≥ 4.5 and <9 with no risk factors for bleeding or falling with and no surgery or procedure planned within 24 hours
10 COMMANDMENTS OF CONSULTATION

Guiding principles for effective medicine consults were first suggested by Lee Goldman and colleagues in a paper published in the *Archives of Internal Medicine* in September 1983—they remain highly relevant today. The so-called 10 commandments for effective consultations are presented.

1. **Determine the question being asked.**
   As a guiding principle, and when taking the initial phone call from the requesting service, always determine the specific medicine-related question he or she wants answered. This will be helpful, especially in situations in which the patient has an extensive and complicated previous medical history. Thus, a typical consultation note should begin by stating a specific problem, such as “Called to see this 78-year-old woman with type 2 diabetes and hypertension for perioperative glucose control ….”

2. **Establish urgency.**
   Always determine if the consult is emergent, urgent, or elective.

3. **Look for yourself.**
   Seldom do the answers to the consultation question lie in the chart; more often than not, independent data gathering is required, including reviewing prior admissions. Often the patient requires further testing. In many cases, the data review combined with a complete history and physical exam from an internal medicine perspective will establish the diagnosis.

4. **Be as brief as appropriate.**
   It is not necessary to repeat in detail the data already in the primary team’s note; obviously as much new data independently gathered should be recorded.

5. **Be specific.**
   Try to be goal oriented and keep the discussion and differential diagnosis concise. When recommending drugs, always include dose, frequency, and route.
6. **Provide contingency plans.** Try to anticipate potential problems (e.g., if using escalating doses of a $\beta$-blocker for rapid atrial fibrillation, make sure that regular BP checks are instituted). Staff (nursing/ancillary) on other floors are not used to treating medicine patients.

7. **Honor thy turf.** Remember to answer the questions you were asked; it is not appropriate to engage the patient in a detailed discussion of whether surgery is indicated or likely to succeed. In situations in which you are asked by the patient about the surgical procedure, defer to the primary team rather than speculating on the technical aspects of the surgery.

8. **Teach, with tact.** Share your insights and expertise without condescension.

9. **Talk is cheap, and effective.** Communicate your recommendations directly to the requesting physician. There is no substitute for direct personal contact. Next month the shoe may be on the other foot, and the same resident may be coming to evaluate a surgical abdomen on one of your medicine admissions.

10. **Follow up.** Suggestions are more likely to be translated into orders when the consultant continues to follow up.

**PREOPERATIVE CARDIOVASCULAR RISK ASSESSMENT**

- Preoperative evaluation and intraoperative management are aimed at eliminating or treating risk factors to reduce the risk of cardiac events (MI, unstable angina, CHF, arrhythmia, and death).
- Risk assessment guidelines have been published by the American College of Cardiology/American Heart Association Task Force (http://circ.ahajournals.org/content/116/17/e418.full.pdf, last accessed June 22, 2012). History, physical examination, and ECG are important components of a thorough clinical assessment and help determine the extent of diagnostic testing required.
- Patients who have mild CAD on cardiac cath or successful revascularization and have no new clinical symptoms probably have a similar risk for events as patients without CAD.
- The American College of Cardiology/American Heart Association algorithm for preoperative cardiac risk assessment is detailed in Figure 21-1.

1 Unstable coronary syndromes, decompensated CHF, significant arrhythmias, and severe valvular disease.
2 Walking at 4 mph on level ground, climbing stairs, climbing hills, riding a bicycle at 8 mph, golfing, bowling, throwing a baseball/football, carrying 25 lb (groceries from the store to the car), scrubbing the floor, raking leaves, mowing the lawn. Stable CAD, compensated or prior CHF, DM, CKD, and cerebrovascular disease.
3 Consider perioperative beta blockade. LOE, level of evidence; MET, metabolic equivalent.
• **β-blockers**: While multiple smaller studies support the use of perioperative β-blockade, the POISE trial, while confirming a decrease in cardiac events with aggressive β-blockade, showed an increase in overall mortality and stroke (Lancet 2008;371:1839). The ACC/AHA recommendations were released before the POISE trial was published. Currently, it seems reasonable to treat high-risk patients (multiple clinical risk factors or with CAD), especially those undergoing vascular surgery (http://circ.ahajournals.org/content/120/21/e169.full.pdf, last accessed June 21, 2012). β-blockers should be started as early as possible prior to surgery, with careful titration to a target resting heart rate of 50 to 60 bpm. Patients already taking β-blockers should be continued on their medication.

• ACE inhibitors should be considered for patients with systolic heart failure.

• Patients taking calcium channel blockers should continue this medication throughout the perioperative period.

• Continuation of preoperative antihypertensive treatment throughout the perioperative period is critical, particularly if the patient is on clonidine (risk of rebound hypertension). Consider switching the patient to IV or transdermal formulations of medications if the patient will be NPO for an extended period of time.

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**DERMATOLOGY**

**Toxic Epidermal Necrolysis**

• **Emergency: call a dermatology consult immediately!** Toxic epidermal necrolysis (TEN) is the most severe variant of a disease spectrum that consists of bullous erythema multiforme (EM) and Stevens-Johnson syndrome (SJS).

• Pertinent information: Drug history. **Drugs are nearly always the cause**; TEN typically occurs within the first 8 weeks of therapy. The most common offenders are sulfonamides, anticonvulsants, penicillin, NSAIDs, antiretroviral medications, and allopurinol. Fewer than 5% of patients report no history of medication use.

• Typical symptoms: Prodrome of high fever, cough, sore throat, burning eyes and malaise 1 to 3 days before the onset of skin involvement.

• Physical exam: High fever, painful erythema of skin, blisters and/or erosions, mucosal erosions, and conjunctival erythema. Involvement of two or more mucosal surfaces is necessary for diagnosis. The skin eruption is usually painful, a key to making the diagnosis.
Workup: CBC, CMP, CXR (in the setting of respiratory distress), skin biopsy.

Diagnosis: physical exam findings and skin biopsy.

Degree of skin involvement: SJS <10%, SJS-TEN overlap 10% to 30%, TEN >30%.

Treatment:

- **Stop suspect drug(s) as well as all other nonessential medications.**
- Transfer to burn unit/ICU.
- IV fluids, NG tube.
- Sterile protocol, antiseptic solution, and nonadherent dressings.
- Antibiotics only if high suspicion for sepsis.
- Consider **systemic steroids and/or IVIG**. Their use is decided on a case-by-case basis.
- Ophthalmology consult.
- OB/GYN consult for vaginal involvement; urology consult for urethral involvement.

Clinical pearls: Hepatitis occurs in 10%. Most patients have anemia and lymphopenia. Neutropenia is associated with poor prognosis. Hypothermia is more indicative of sepsis than fever. Patients with HIV, systemic lupus, or a bone marrow transplant have a higher risk of developing TEN.

Prognosis: TEN has a mortality rate of 30% to 40% and is most often secondary to sepsis, renal failure due to hypovolemic shock, or ARDS.

**Toxic Shock Syndrome**

- **Emergency: call an ID consult immediately!**
- Pertinent information: Age of patient, immunocompromised status, menstrual history, recent surgeries, diabetes, chronic cardiac or pulmonary disease.
- History: Painful erythematous skin eruption, sudden onset of high fever, nausea/vomiting, diarrhea, sore throat, confusion, headache, myalgias, and hypotension.
- Physical exam: Fever >102°F; erythematous rash initially appearing on trunk, spreading to arms and legs and involving palms and soles, progressing to diffuse erythema and edema. Involves the oral mucosa. In 10 to 12 days there is desquamation of the top layer of the epidermis (not full thickness as in TEN).
• Workup: Blood cultures, CBC (leukocytosis), CMP (hyponatremia, hypokalemia, hypocalcemia, elevated LFTs), ECG (arrhythmias), CXR. To consider: serologic testing for Rocky Mountain spotted fever, leptospirosis, measles, hepatitis B surface antigen, antinuclear antibody, VDRL, monospot antibody, rapid strep test, LP (should be normal).

• Diagnosis: Clinical (fever, typical rash, hypotension, multi-organ involvement).

• Treatment:
  - **Remove any infected foreign bodies.**
  - **Empiric IV antibiotics effective against Streptococcus and Staphylococcus:** clindamycin 900 mg IV plus IV vancomycin; tailor therapy when culture results available.
  - Aggressive IV fluids.
  - **Consider IVIG.**
  - Monitor for hypotension/shock.
  - Oxygen therapy.

• Clinical pearls: Staphylococcal scalded skin syndrome has a similar clinical picture; however, it occurs in immunocompromised adults. *Staphylococcus aureus* is the most common cause of TSS; however, exotoxin-producing streptococci can cause a similar clinical picture associated with higher mortality. Clindamycin suppresses synthesis of the TSS toxin (TSST-1), while β-lactamase–resistant antibiotics may increase synthesis of TSST-1 and therefore should be included in the antibiotic regimen.

**Necrotizing Fasciitis**

• **Emergency: call a surgeon now!**

• Pertinent information: Recent surgical or traumatic wound.

• Typical signs and symptoms: Involved area becomes erythematous, indurated, and severely painful. Within hours it can become dusky blue to black, indicating necrosis. Crepitus may be present due to subcutaneous gas formation.

• Workup and diagnosis: Blood cultures, CK, plain radiography for soft tissue gas, and non-contrast CT if clinical diagnosis not obvious or to delineate depth of infection.

• Treatment:
  - **Wide surgical debridement** and tissue culture.
  - Initial **empiric IV antibiotics** effective against streptococci, anaerobes, MRSA, and gram-negative bacilli. Include clindamycin in regimen.
Clinical pearls: Time is essential, call for surgical help immediately! Mortality rate is as high as 25%.

**Pemphigus Vulgaris (PV) and Bullous Pemphigoid (BP)**

- Urgent dermatology consult.
- Pertinent info: Flaccid or tense bullae, percentage of body surface area involved, mucosal involvement.
- Typical signs and symptoms: Flaccid bullae and erosions with mucosal involvement (PV) and pruritus with tense bullae in an elderly person (BP).
- Workup and diagnosis: Skin biopsy with direct immunofluorescence (BP and PV) and serum for indirect immunofluorescence (PV).
- Treatment:
  - PV: Steroids, mycophenolate mofetil, azathioprine, rituximab.
  - BP: Steroids (topical or systemic), tetracycline, dapsone, nicotinamide, and mycophenolate mofetil.
- Clinical pearls: PV is associated with a higher mortality than BP. Thus, PV requires aggressive treatment. Most patients do not require hospitalization unless infected or having difficulty maintaining fluid balance. Etiologies of blistering diseases include infectious, autoimmune, allergic hypersensitivity, metabolic, paraneoplastic, and genetic.

**Vasculitis**

- Urgent consult.
- Pertinent info: Drug history, known connective tissue disease, malignancy, or infection.
- Typical skin findings: **palpable purpura** (dark reddish-purple lesions that do not blanch and may blister or ulcerate) on the lower extremities or dependent areas. Eruption may be itchy, painful, or asymptomatic.
- Workup: CBC, CMP, urinalysis, skin biopsy, CXR, throat culture for strep, ESR, hepatitis panel, cryoglobulins, ANA, RF, antiphospholipid antibody, ANCA, SPEP.
- Diagnosis: Skin biopsy; concern for systemic involvement if fever, arthralgias, abdominal pain, pulmonary symptoms, hematuria, or proteinuria.
Approach to Consultation

• Treatment:
  • **Discontinue potential causative drug** (e.g., ASA, sulfonamides, penicillin, barbiturates, amphetamines, PTU).
  • **Treat underlying disorder** (e.g., infection, malignancy, or connective tissue disease).
  • Antihistamines, NSAIDs, colchicine, dapsone, antimalariais.
  • If systemic involvement: Prednisone, azathioprine, cyclophosphamide, IVIG, plasmapheresis.

• Clinical pearls: Up to 50% of cases are idiopathic. Thrombocytopenia is associated with nonpalpable purpura. DIC and warfarin necrosis cause extensive purpura. *Neisseria* sepsis and Rocky Mountain spotted fever also cause petechiae and purpura. Acral hemorrhagic papules, pustules, or vesicles may result from thrombi or septic emboli.

**Hypersensitivity Syndrome (Severe Drug Reaction)**

• Urgent consult.
• Pertinent information: Drug history (sulfonamides, anticonvulsants, allopurinol).
• Physical exam: blanchable erythematous macules, exfoliative dermatitis, or EM (target-like) lesions; edema of face or extremities, fever, lymphadenopathy, hepatosplenomegaly.
• Workup: CBC (with eosinophil count), liver function panel, urinalysis, and CXR.
• Treatment:
  • **Stop offending drug.**
  • Oral/topical steroids and antihistamines for symptoms.
• Clinical pearls: The syndrome, also known as **DRESS** (“drug reaction with eosinophilia and systemic symptoms”), develops within 8 weeks of starting the causative drug. Up to 50% develop fulminant hepatic necrosis if the drug is not stopped early in the course. Some anticonvulsants cross-react in 70% to 80% of patients (phenytoin, carbamazepine, and phenobarbital). Valproic acid does not cross-react. Patients should warn first-degree relatives that they, too, may be at high risk for a reaction due to a hereditary component.

**Erythroderma (Generalized Exfoliative Dermatitis)**

• Urgent consult.
• Pertinent info: Prior skin disorder, drug history, duration of erythroderma.
• Physical exam: Diffuse erythema of skin leading to exfoliative dermatitis, pruritus, keratoderma, shivering/chills, alopecia.
• Most common causes: Idiopathic, drug reaction, lymphoma and leukemia (including cutaneous T-cell lymphoma), atopic dermatitis (eczema), psoriasis, contact dermatitis, seborrheic dermatitis.
• Labs: CBC, BMP, albumin, calcium, SPEP, peripheral blood smear for Sézary cells.
• Diagnosis: Clinical features, skin biopsy.
• Treatment:
  • Treat underlying skin condition if known (e.g., psoriasis, eczema).
  • Discontinue any suspect drugs.
  • Search for and treat underlying malignancy.
  • Topical steroid ointment (mid-potency, e.g., triamcinolone 0.1% ointment, consider wearing a sauna suit), emollients, systemic antihistamines.
  • Monitor closely for electrolyte and fluid imbalances, high-output cardiac failure, renal failure, sepsis, and hypothermia.
• Clinical pearls: 20% of cases are idiopathic. The course and prognosis depend on the underlying etiology. A diligent search for the underlying cause is often required. Most patients do not require hospitalization unless infected.

**NEUROLOGY**

**Acute Focal Neurologic Deficit (Stroke)**

**Emergent.** If the time of onset is known to be **within 4.5 hours, call neurology immediately, as the patient may be a candidate for thrombolytic therapy.**

• Pertinent information: There are two broad categories of stroke:
  • **Ischemic** (including TIA): sudden-onset focal neurologic deficit attributable to a vascular territory. TIA is a focal deficit that completely resolves within 24 hours. Most clear within 5 to 10 minutes. Ischemic stroke accounts for more than 80% of strokes.
  • **Hemorrhagic:** Includes intracerebral hemorrhage and subarachnoid hemorrhage.
• Symptoms and signs: Stroke causes negative neurologic symptoms, such as weakness, loss of sensation, visual loss, and aphasia. Positive symptoms, such as pain, paresthesias, jerking
Headache can occur and is more common with hemorrhagic stroke than ischemic stroke.

**History:**

- What was the time of onset? This is the time the patient was last seen or known to be normal, not when he or she was found with the deficits. If the patient awakens with deficits, consider the time he or she went to sleep as the time of onset.
- How did the symptoms begin (suddenly or gradually)? How are they progressing (stable, worsening, or improving)?
- Are there associated symptoms (severe headache, nausea, or vomiting may suggest hemorrhagic stroke)?
- Are there any known contraindications for thrombolytics (see below)?
- What is the patient’s past neurologic, medical, and surgical history?
- What are the patient’s medications (especially anticoagulants)?

**Physical exam:** Check vital signs (especially blood pressure and oxygen saturation). Look for evidence of recent head injury. Oral trauma suggests unwitnessed seizure. Do a cardiopulmonary exam to evaluate for arrhythmia and concurrent cardiac ischemia and to monitor for signs of aspiration. Perform a focused neurologic exam.

**Workup:** Immediate evaluation consists of a stat noncontrast head CT to evaluate for hemorrhage, finger stick glucose, stat CBC, and stat coagulation panel. Also check ECG, CXR, urine drug screen, troponin, and chemistry panel.

**Treatment of ischemic stroke:**

- If the time of onset is well established to be within 3 hours, and there are no contraindications for thrombolysis, then **IV TPA** is the only approved specific treatment for ischemic stroke. Strong contraindications for TPA include blood pressure >185/110; blood sugar >400 or <50; seizure at onset; stroke in the last 3 months; history of intracranial hemorrhage; anticoagulant therapy and INR >1.7 or elevated PTT; platelets <100; recent major surgery within 14 days; head injury within 3 months; GI or GU bleeding within 21 days; arterial puncture at a noncompressible site within 10 days; rapid improvement of symptoms. If the time of onset is between 3 and 4.5 hours, there are additional contraindications. These include age >80; anticoagulant use (even with
normal PT/INR); history of a prior disabling stroke AND diabetes.

- Blood pressure management: Generally stroke patients are allowed to be hypertensive to improve perfusion to the ischemic penumbra. If the mean arterial pressure exceeds 130 mm Hg, consider a dose of IV labetalol (if HR >80) or hydralazine (if HR <80). If they have received TPA, there are tighter blood pressure goals to reduce the risk of hemorrhagic conversion. Treatment of the blood pressure is dependent on the risk of end-organ damage (i.e., cardiac ischemia, acute renal failure).

- Airway management: Intubation may be required if the stroke has compromised level of consciousness to the degree that the patient is unable to maintain an adequate airway. This is seen most often in brain stem strokes and strokes affecting a large territory.

- Reimaging is required if the patient develops worsening of the neurologic examination. Head CT is sufficient to evaluate for likely causes, such as hemorrhage, midline shift, herniation, and hydrocephalus.

Seizure

- Level of urgency: urgent or emergent. If the patient experiences multiple seizures, or has not returned to baseline, then an emergent neurology consult is indicated. If the patient has returned to baseline after a single convulsion, neurologic evaluation should be obtained on an urgent basis.

- Description:
  - Status epilepticus is defined as multiple seizures without regaining full consciousness between the episodes, or a single seizure that lasts longer than 5 minutes.
  - Symptoms and signs of focal brain disease: Patients may describe an aura prior to the episode or have postictal focal weakness.

- Pertinent history:
  - Has the patient returned to baseline?
  - Has he or she had seizures before?
  - What did the seizure look like (description of onset, movements, head turning, eye position, loss of continence, oral trauma, ability to respond to questions)?
  - Are there any known metabolic derangements (e.g., hypoglycemia or hyperglycemia)? Any known drug or toxic exposure?
• Any signs of infection?
• Any history of head trauma?
• Physical exam: Evaluate for signs of head injury, oral trauma, or meningismus. Determine level of consciousness and assess for deficits of cranial nerves or motor function.
• Workup: Seizures are usually a symptom of an underlying problem. Consider a structural lesion (neuroimaging), metabolic derangements (check finger stick glucose, chemistry panel, magnesium, calcium), or infection (check urinalysis and CXR).
• Treatment: Most important, remain calm!
  • **Single seizure:** If the seizure is ongoing, note the time. Check for level of consciousness, ability to follow commands. Look for forced eye deviation or nystagmus. Protect the patient’s airway, but do not insert objects into the mouth if he or she is convulsing. Turn the patient on his or her side and pad the bedrails. Provide oxygen by face mask, check vital signs, obtain finger stick blood sugar. Anticonvulsants are not needed acutely for a single seizure.
  • **Status epilepticus:** If the seizure has been continuous for 5 minutes, or if there have been multiple seizures without full return to baseline, then the patient is in status. *This should be considered a neurologic “code.”* Protect the airway, have an intubation kit available, obtain ABG, cardiac monitor, and pulse oximetry (many of these monitors will be difficult to interpret if there are ongoing convulsions). Ensure multiple working IVs are in place. Do a stat finger stick glucose and send CBC, chemistry, magnesium, urine and serum toxicology, alcohol level, urinalysis, and drug levels for any anticonvulsants the patient should be taking. Give **thiamine 100 mg IV** followed immediately by **50 mL of 50% glucose**. Do not wait for the results of the Accu-Chek as hyperglycemia is transient and far less damaging than prolonged hypoglycemia.
    - **Lorazepam** in 2 mg boluses, up to 0.1 mg/kg (4 to 8 mg) at a rate of 1 mg/min, should be administered IV if possible. PR diazepam (0.5 mg/kg to a maximum of 20 mg) or IM midazolam (0.2 mg/kg) can be given if there is no IV access. This should be repeated if there continues to be seizure activity.
    - An intravenous anticonvulsant should rapidly follow the benzodiazepine, usually **phenytoin** (15 to 20 mg/kg). Blood pressure will need to be monitored, as this may cause hypotension. The maximal infusion rate is 50 mg/min.
Alternatively, fosphenytoin can be used. It can be infused at rates up to 150 mg/min and has less risk of hypotension and arrhythmia, but is a pro-drug that must be converted prior to becoming an active anticonvulsant.

- If the patient continues to have refractory seizures, efforts should be made to transfer him or her to an ICU, with EEG monitoring. Aggressive anticonvulsant therapy requiring intubation and mechanical ventilation may be necessary.
- Watch for rhabdomyolysis and hyperthermia as complications of prolonged convulsions.

Meningitis

- **Level of urgency: emergent.**
- Pertinent information: Bacterial meningitis usually presents rapidly with progressive encephalopathy, headaches, and fever and is a neurologic emergency. Viral meningitis/encephalitis generally follows a more benign clinical course; however, it can also leave patients with severe neurologic deficits if not recognized and treated early. In HIV and other immunocompromised patients, fungal infections, such as Cryptococcus, should also be considered.
- History: Patients with bacterial meningitis frequently note generalized myalgias, sore throat, and/or fatigue prior to the major clinical deterioration that brings them to medical attention. Fever, headache, and alteration in level of consciousness are common presenting features. Seizures or photophobia may also be present. An acute confusional state in a febrile patient should always raise the suspicion for bacterial meningitis. Viral meningitis usually presents with symptoms of altered consciousness, fever, personality changes, headache, and flu-like symptoms. Remember to ask the patient about any recent travel or sick contacts.
- Physical exam: Evaluation of mental status is essential. Meningeal signs should be checked: Brudzinski’s sign (neck flexion resulting in spontaneous flexion of the knees) and Kernig’s sign (passive extension of the knees with hips flexed resulting in eye opening and verbal response). Papilledema suggests increased intracranial pressure, which may result from brain edema or sagittal sinus thrombosis. Rash may suggest a specific infectious etiology, typically *Neisseria meningitidis*.
- Workup: Neuroimaging should be obtained to rule out a mass as the cause of the altered mental status. Start with a noncontrast head CT. Blood cultures and **urgent lumbar puncture** are required. See Table 21-1 for CSF interpretation.
<table>
<thead>
<tr>
<th>Condition</th>
<th>Color</th>
<th>Pressure (mm H₂O)</th>
<th>Cells (#/mL)</th>
<th>Protein (mg/dL)</th>
<th>Glucose (mg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Clear</td>
<td>70–180</td>
<td>0–5 mononuclear</td>
<td>15–45</td>
<td>45–80 (two-thirds of serum glucose)</td>
</tr>
<tr>
<td>Bacterial meningitis</td>
<td>Opalescent</td>
<td>↑(may be normal)</td>
<td>&gt;5 to many thousands PMNs</td>
<td>50–1,500</td>
<td>0–45</td>
</tr>
<tr>
<td>Viral infection</td>
<td>Clear or opalescent</td>
<td>Normal (may be slightly ↑)</td>
<td>&gt;5–2,000, mostly lymphs</td>
<td>20–200</td>
<td>Normal (may be slightly ↓)</td>
</tr>
<tr>
<td>Tuberculous meningitis</td>
<td>Clear or opalescent</td>
<td>↑(may be normal)</td>
<td>&gt;5–500 lymphs</td>
<td>45–500</td>
<td>10–45</td>
</tr>
<tr>
<td>Fungal meningitis</td>
<td>Clear or opalescent</td>
<td>Normal or ↑</td>
<td>&gt;5–800 lymphs</td>
<td>Normal or ↑</td>
<td>Normal or ↓</td>
</tr>
<tr>
<td>Carcinomatous meningitis</td>
<td>Clear or opalescent</td>
<td>Normal or ↑</td>
<td>&gt;5–1,000 lymphs</td>
<td>Up to 500</td>
<td>Normal or ↓</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>Bloody or xanthochromic</td>
<td>↑(may be normal)</td>
<td>Many RBCs; ratio of WBC:RBC same as blood</td>
<td>Up to 2,000</td>
<td>Normal</td>
</tr>
</tbody>
</table>
• Treatment: Antibiotics should not be withheld if the head CT and lumbar puncture cannot be obtained rapidly. Obtain blood cultures prior to starting antibiotics, as a significant percentage of bacterial meningitis patients will have bacteremia. Antibiotic coverage should include ceftriaxone 2 g IV q12h and vancomycin dosed for weight and creatinine clearance (see Chapter 20). Ampicillin 2 g IV q4h can be considered for patients at risk for Listeria. If viral meningitis is suspected, start acyclovir 10 mg/kg (ideal body weight) q8h empirically while the HSV PCR is pending. The PCR will remain positive for several days after initiating treatment, so acyclovir should not be delayed. See Table 21-2 for additional treatment recommendations.

OBSTETRICS AND GYNECOLOGY

The OB/GYN History and Physical Examination

• Perform a basic GYN H&P prior to calling a consult!

• Gynecologic history:
  • Menstrual history:
    ▪ Last menstrual period (LMP)?
    ▪ Age at menarche?
    ▪ Age at menopause? Any postmenopausal bleeding?
    ▪ If a menstrual problem seems central to the chief complaint, consider asking about menstrual length, inter-menstrual duration, amount of menstrual bleeding (number of pads/day, degree of soilage of pads), and presence of dysmenorrhea.
  • Pap history: Date of last pap? Any history of abnormal paps?
  • Contraceptive history: Currently using anything to prevent pregnancy?
  • Hormone use: Any history of exogenous hormone use (e.g., in postmenopausal women)?
  • Sexual history: Currently sexually active? History of any STIs?
  • Gynecologic procedure history: Date and type of any gynecologic procedures/surgeries, for example, bilateral tubal ligation (BTL), diagnostic laparoscopy for endometriosis, unilateral or bilateral salpingo-oophorectomy (USO/BSO), loop electrosurgical excision procedure (LEEP)?
<table>
<thead>
<tr>
<th>Age or Clinical Setting</th>
<th>Likely Organism</th>
<th>Empiric Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immunocompetent 18–50 years</td>
<td><em>Streptococcus pneumoniae</em>, <em>Neisseria meningitidis</em></td>
<td>Ceftriaxone + vancomycin</td>
</tr>
<tr>
<td>&gt;50 years</td>
<td><em>S. pneumoniae</em>, <em>Listeria monocytogenes</em>, gram-negative bacilli, <em>Mycobacterium tuberculosis</em></td>
<td>Ceftriaxone + ampicillin + vancomycin</td>
</tr>
<tr>
<td>Head trauma, CSF shunt, neurosurgery</td>
<td>Staphylococci, gram-negative bacilli, <em>S. pneumoniae</em></td>
<td>Cefepime + vancomycin</td>
</tr>
<tr>
<td>Immunocompromised</td>
<td><em>L. monocytogenes</em>, gram-negative bacilli, also <em>S. pneumoniae</em> and <em>Haemophilus influenzae</em></td>
<td>Cefepime + vancomycin + ampicillin</td>
</tr>
<tr>
<td>HIV</td>
<td><em>Cryptococcus neoformans</em></td>
<td>Amphotericin B + flucytosine</td>
</tr>
</tbody>
</table>
• Obstetric history:
  • Ever been pregnant? How many times? Any complications? Any miscarriages?
  • If the patient is currently pregnant, ask:
    ■ Name of prenatal care provider/clinic? EDC (estimated date of confinement, or due date)?
    ■ Any complications during this pregnancy?
    ■ If the pregnant patient is admitted to medical services, please give the OB/GYN consultation service a courtesy call.
    ■ Any symptoms of vaginal bleeding, leakage of fluid, or decreased fetal movement? Or abdominal trauma? If yes, call OB urgently.

• Physical exam: The physical exam may help you delineate between gynecologic versus nongynecologic causes of pelvic symptoms. Components may include the abdominal, vaginal speculum, and bimanual pelvic exam; and/or rectovaginal exam; and/or wet prep. What you are able to include will depend upon your level of experience and comfort and access to materials.

  • Abdominal exam: Gaseous distension may indicate distended bowels or pneumoperitoneum. A fluid wave may indicate ascites or hemoperitoneum. Solid masses may indicate a benign process, such as uterine leiomyomas, or a malignant process (ovarian or other cancer). Uterine fundal tenderness may indicate pelvic inflammatory disease or postpartum endometritis. Adnexal tenderness may be associated with ectopic pregnancy, other adnexal mass/cyst, or ovarian torsion. Suprapubic tenderness may be indicative of cystitis. Call the consult team immediately if either rebound or guarding is present, as this could constitute a surgical emergency!

  • Speculum exam: Note any friability, bleeding, or discharge. If you suspect yeast, bacterial vaginosis, trichomoniasis, or PID, swab the posterior fornix (where the discharge pools) and perform a wet prep/KOH prep if possible. If you suspect gonococcal or chlamydial infection, send a urine or endocervical swab test for GC/Ch. Notify the OB/GYN consult team immediately if you see heavy, active bleeding!

  • Bimanual exam: Cervical motion tenderness. Note any fundal tenderness or uterine masses. Note any adnexal masses or tenderness. The ovaries are often pea-size or not felt at all. If you suspect pain from a posterior mass, you can perform a rectovaginal exam, in which you can feel masses on the posterior surface of the uterus with the rectal finger.
• Examining the discharge: The GC/Ch test will take about 2 days. However, information can be gathered from point-of-care examination of any vaginal discharge you collected. On the wet prep, you might see white blood cells (associated with PID and tubo-ovarian abscesses), clue cells (associated with bacterial vaginosis), red blood cells (may be present with inflammation or active bleeding), or motile trichomonads. On the KOH prep, look for pseudohyphae (associated with candidiasis) or a positive whiff test (the presence of a fishy odor associated with bacterial vaginosis). A pH test can also be helpful—pH < 4.5 may indicate normal vaginal flora or yeast, whereas a pH > 4.5 is associated with bacterial vaginosis, trichomoniasis, GC, blood, or menopause.

• Imaging: Review any abdominopelvic MRI, CT, and ultrasounds that the patient already has. Bear in mind that a CT scan cannot always distinguish the characteristics of adnexal masses well; an ultrasound is better. If further delineation of the pelvic organs is indicated, the OB/GYN consult team should be able to help guide you with which study to order and how it should be performed (sometimes OB/GYN will perform a pelvic ultrasound; at other institutions, it is performed by radiology).

Vaginal Discharge

Vaginitis

• The most common entities are bacterial vaginosis, vaginal candidiasis, and trichomoniasis.

• Diagnosis: The key symptoms, signs, and wet prep/KOH prep findings for the most common causes of infectious vaginitis are summarized in Table 21-3.

• Treatment: Listed here are the most common treatments for vaginitis. For additional treatment options, see www.cdc.gov.
  
  • Vaginal candidiasis (most commonly caused by Candida albicans):
    - **Fluconazole** 150 mg PO in a single dose.
    - **Clotrimazole** 1% cream 5 g intravaginally for 7 to 14 days (use if pregnant).
  
  • Bacterial vaginosis:
    - **Metronidazole** 500 mg PO bid for 7 days.
    - Metronidazole gel, 0.75%, 5 g intravaginally, once daily for 5 days.
<table>
<thead>
<tr>
<th>Condition</th>
<th>Signs and Symptoms</th>
<th>Exam Findings</th>
<th>pH</th>
<th>Wet Mount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial vaginosis</td>
<td>Increased discharge (white, thin)</td>
<td>Thin, whitish-gray homogeneous discharge, sometimes frothy</td>
<td>&gt;4.5</td>
<td>Clue cells (&gt;20%) Shift in flora Amine odor after adding KOH</td>
</tr>
<tr>
<td></td>
<td>Increased odor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Candidiasis</td>
<td>Increased discharge (white, thick)</td>
<td>Thick, curd-like discharge</td>
<td>&lt;4.5</td>
<td>Hyphae or spores</td>
</tr>
<tr>
<td></td>
<td>Pruritus</td>
<td>Vaginal erythema</td>
<td></td>
<td>Note: can be mixed infection with BV, Trichomonas vaginalis, or both and have higher pH</td>
</tr>
<tr>
<td></td>
<td>Dysuria</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Burning</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trichomoniasis</td>
<td>Increased discharge (yellow, frothy)</td>
<td>Yellow, frothy discharge with or without vaginal or cervical erythema</td>
<td>&gt;4.5</td>
<td>Motile trichomonads Increased WBCs</td>
</tr>
<tr>
<td></td>
<td>Increased odor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pruritus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dysuria</td>
<td></td>
<td></td>
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</tbody>
</table>

• Trichomoniasis (because this is an STI, encourage the patient to have her partner treated):
  - **Metronidazole** 2 g PO in a single dose (preferred).
  - Metronidazole 500 mg PO bid for 7 days (alternative).

**Cervicitis**

- Pertinent information: Most commonly caused by *Neisseria gonorrhoeae*, *Chlamydia trachomatis*. *Trichomonas vaginalis* is also associated with cervicitis.
- Diagnosis: Suspect if yellow or green cervical discharge. The cervix may be inflamed (e.g., the “strawberry cervix” of *T. vaginalis*). Perform a urine or endocervical GC/Ch probe for definitive diagnosis.
- Treatment: Adapted from the most recent CDC guidelines at www.cdc.gov. If either *gonorrhoeae* or *Chlamydia* is diagnosed, empirically treat for the other. Encourage the patient to have her partner treated.
  - *N. gonorrhoeae*:
    - **Ceftriaxone** 125 mg IM in a single dose (preferred).
    - Cefixime 400 mg PO in a single dose (second line, use if above unavailable).
    - **Fluoroquinolones are no longer recommended due to high resistance rates.**
  - *C. trachomatis*:
    - **Azithromycin** 1 g PO in a single dose (for easy dosing or if pregnant).
    - Doxycycline 100 mg PO bid for 7 days.

**Pelvic Inflammatory Disease**

- The presence of cervical motion tenderness, fever, leukocytosis, or worsening pelvic pain should raise suspicion for pelvic inflammatory disease (which can be caused by any one of a number of organisms).
- PID often requires inpatient treatment, but can sometimes be treated on an outpatient basis if specific criteria are met. Either form of treatment requires close follow-up with an OB/GYN.
- If inadequately treated, PID can lead to infertility, chronic pelvic pain, sepsis, and even death.
- If PID is suspected, an OB/GYN consult should be obtained. For treatment guidelines, see www.cdc.gov.
Ectopic Pregnancy

- **Pertinent information:** Notify the OB/GYN team immediately if you suspect ectopic pregnancy. A ruptured ectopic pregnancy is a surgical emergency.
- **History:**
  - Last menstrual period.
  - Symptoms: Missed menstrual period, abdominal pain, and/or vaginal spotting.
  - Possible risk factors for ectopic pregnancy: Prior ectopic pregnancy, history of tubal ligation or other tubal surgery, history of prior gynecologic infection or PID.
- **Physical exam:**
  - Vital signs, include orthostatics.
  - General: Note degree of pallor, weakness, responsiveness, pain.
  - Cardiovascular: Note presence of tachycardia, quality of peripheral pulses.
  - Abdomen: may palpate presence of a mass, rebound or guarding (signs of peritonitis), abdominal distension.
  - Pelvic exam: may or may not see bleeding via cervical os, cervical dilation, slightly enlarged uterus, palpable adnexal mass.
- **Labs:** CBC, T&S, qualitative urine hCG. If positive urine hCG, obtain a **quantitative** serum β-hCG.
- **Imaging:** Abdominal and pelvic ultrasound (transvaginal) to determine location of pregnancy, gestational age, and fetal heart motion.
- **Treatment:**
  - IV access and fluid resuscitation (have cross-matched blood available).
  - Removal of the ectopic pregnancy: Medical (methotrexate) versus surgical management will be based on characteristics of the ectopic, stability of the patient, and patient preference. The OB/GYN consult team will determine the mode of treatment.

Vaginal Bleeding

- **Pertinent information:** Severity of bleeding will dictate the extent of your workup prior to calling a consult. See Figure 21-2 for the most common pathologic etiologies of vaginal bleeding. Vaginitis
and cervicitis also cause vaginal spotting and can occur in any of these age groups.

- History: Note date of last normal menstrual period; the frequency, quantity, and duration of the bleeding; whether there is a history of bleeding disorders; and whether the patient is on hormones, contraception, or anticoagulants. Inquire whether she has ever had a history of any gynecologic problems in the past and whether she has a family history of any gynecologic problems, including gynecologic cancer. **Suspect malignancy in any postmenopausal patient with vaginal bleeding.**

- Physical exam: An abdominal exam, speculum exam, and pelvic exam should be performed in order to determine the source and extent of bleeding. Rule out urologic and GI sources of bleeding: spread the labia to inspect the urethra, send a macro/micro urinalysis (straight cath specimen), and perform a digital rectal exam/guaiac. Vaginal bleeding may be coming from the uterus via the cervical os, the cervix itself, or the vaginal walls.

- Labs: CBC, T&S, PT, PTT, INR, hCG, TSH, prolactin.

- Imaging: Abdominopelvic imaging (usually ultrasound) to characterize pregnancy status and anatomic causes of bleeding (fibroids, polyps, ovarian masses, endometrial stripe thickness).
Pathologic specimens:
• OB/GYN will perform pap and endometrial sampling if indicated.
• Postmenopausal women and women with chronic anovulation, obesity, or age greater than 35 should all have endometrial sampling. These women are at high risk for hyperplasia and malignancy.

Treatment:
• IV access, fluid resuscitation, transfusion as needed.
• Strict I/Os, pad counts.
• Consult OB/GYN, as treatment will depend upon etiology.

Pelvic Mass
• Pertinent information: Call an OB/GYN consult immediately if you suspect ovarian torsion! Ovarian torsion is a surgical emergency, and prompt surgical treatment could enable a young woman to keep her ovary. Ovarian torsion is clinically diagnosed. It may present with constant or intermittent severe abdominal pain, a tender pelvic mass, and low-grade temperatures. Pelvic ultrasound may demonstrate one enlarged ovary.
• History: Obtain a thorough OB/GYN history.
• Differential diagnosis: Pelvic masses may be gynecologic in origin or may arise from the urinary tract or bowel. Age is an important determinant of the likelihood of malignancy. Table 21-4 outlines the most common causes of pelvic masses for each age group. Additionally, an infectious cause (e.g., tubo-ovarian abscess) can occur in association with PID.
• Physical exam: A gynecologic exam should be performed. Ascites or pleural effusion heightens the suspicion for ovarian cancer.
• Labs:
  • CBC and hCG (in reproductive age women).
  • Tumor markers: Obtain in consultation with OB/GYN.
• Imaging: The OB/GYN consult team will help guide imaging. Transvaginal pelvic ultrasound will help to delineate the origin and internal features of the mass. If malignancy is suspected or confirmed, further imaging (CT, MRI) may be indicated to determine the extent of disease. A barium enema or colonoscopy will help to exclude a gastrointestinal etiology.
• Treatment: Varies greatly, depending on etiology.
<table>
<thead>
<tr>
<th>Adolescent</th>
<th>Reproductive</th>
<th>Perimenopausal</th>
<th>Postmenopausal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Functional cyst</td>
<td>Functional cyst</td>
<td>Fibroids</td>
<td>Ovarian tumor (malignant or benign)</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>Pregnancy</td>
<td>Epithelial ovarian tumor</td>
<td>Functional cyst</td>
</tr>
<tr>
<td>Dermoid/other germ cell tumors</td>
<td>Uterine fibroids</td>
<td></td>
<td>Bowel, malignant tumor or inflammatory</td>
</tr>
<tr>
<td>Obstructing vaginal or uterine abnormalities</td>
<td>Epithelial ovarian tumor</td>
<td></td>
<td>Metastasis</td>
</tr>
<tr>
<td>Epithelial ovarian tumor</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The Ophthalmologic History and Examination

- **Basic ophthalmic history:**
  - Chief complaint: Course/extent of vision loss, unilateral/bilateral symptoms, central/peripheral visual symptoms, constant/intermittent symptoms, presence of pain.
  - Prior ophthalmic history: Known ophthalmic conditions, prior eye surgery or trauma, contact lens/eye medication use, and relevant past medical/family history.

- **Basic ophthalmic exam:**
  - **Visual acuity (extremely important):** acuity for each eye (have the patient wear his/her glasses). If no vision chart is available, have the patient attempt to read your name badge, the newspaper, or signs around the room.
  - Pupillary exam: Are the pupils/iris visible through the cornea? Are they round and equal in size? Do they react to light?
  - Motility exam: Determine whether the eyes are aligned in primary gaze; test ocular motility and determine if nystagmus is present.
  - Visual field exam: Test each individual eye (start with the eye with better vision); determine whether the patient can count fingers at the edges of central vision.
  - External exam: Are the eyes proptotic? Is eyelid position normal? Is the eye red?
  - Fundus exam. Determine whether there is a red reflex. Look for optic nerve swelling, retinal hemorrhage, or retinal detachment. It is often helpful to communicate the level of confidence of your fundus exam to the consultant.

**Injury**

*Trauma with Possible Ruptured Globe*

- **Ocular emergency: call a consult immediately!**
- Pertinent information: Age, PMH including ocular history, allergies, current medications including ocular medications, specific ocular complaints, history of events preceding trauma, specific chemicals or items involved, any history of possible foreign body (e.g., hammering on metal).
- Typical symptoms: Pain, decreased vision, history of trauma.
• Physical exam findings suggestive of a ruptured globe: 360° subconjunctival hemorrhage, full-thickness corneal or scleral laceration, peaked or irregular pupil, exposed intraocular contents, hyphema (bleeding in the anterior chamber).

• Treatment:
  • Place a shield over the involved eye; do not press on eye or touch ocular contents.
  • NPO (determine last meal).
  • IV antibiotics: Cefazolin or vancomycin 1 g IV immediately; also give fluoroquinolone (e.g., ciprofloxacin or moxifloxacin PO or IV).
  • Administer tetanus toxoid as needed.
  • Antiemetic as needed to prevent Valsalva.
  • Orbital CT scan (axial and coronal).
  • Surgical repair as soon as possible.
  • Clinical pearl: In trauma with eyelid laceration, do not try to repair the laceration. Allow ophthalmologist to assess extent.

Acute Chemical Splash

• Ocular emergency: Begin irrigation, and then call a consult immediately!

• Pertinent information: Age, PMH including ocular history, allergies, current medications including ocular medications, specific ocular complaints, history of events, specific chemicals and amounts involved, time/duration of contact, irrigation at scene.

• Symptoms: Possible pain, blurry vision, foreign body sensation, tearing, photophobia.

• Physical exam:
  • Mild to moderate splash: Sloughing of entire epithelium, hyperemia, mild chemosis (edema of bulbar conjunctiva), eyelid edema, first- or second-degree periocular skin burns.
  • Moderate to severe: Pronounced chemosis, perilimbal (at the junction of cornea and sclera) blanching, corneal edema or opacification, anterior chamber reaction/no view of the anterior chamber or iris, increased intraocular pressure, second- or third-degree burns.

• Workup: Slit-lamp exam, evert eyelids; check pH, intraocular pressure.

• Immediate treatment (prior to contacting consult service):
  • Copious irrigation with 1 L of any available irrigant (e.g., NS, 1/2 NS, or LR), and keep irrigating until you talk with the consultant.
• May give 1 to 2 drops of topical anesthetic.
• Do not use acid or alkali to neutralize splash.
• Check pH 5 minutes after irrigation; continue irrigation until pH is neutral (i.e., 7).

• Treatment after irrigation (ideally instituted by ophthalmologist):
  • Debride necrotic tissue.
  • Topical antibiotic ointment: bacitracin/polymyxin, erythromycin, or ciprofloxacin ophthalmic qid.
  • Additional treatments including cycloplegia, the use of topical steroids, and intraocular pressure control should be instituted by an ophthalmologist with plans for frequent follow-up.

Corneal Ulcer

• This is an emergency; call a consult immediately!

• Pertinent information: Age, past ocular history (previous ulcers or herpetic infections?), history leading up to event (recent trauma or contact lens use).

• Symptoms: pain, photophobia, decreased vision, with or without discharge.

• Physical exam: Focal corneal opacity and overlying epithelial defect (abrasion), anterior chamber reaction, with or without hypopyon (white blood cells layering out in anterior chamber), eyelid edema, lagophthalmos (incomplete eyelid closure).

• Workup: Routine cultures taken of corneal scrapings (done by ophthalmologist), includes gram stain, KOH prep, fungal culture.

• Treatment:
  • Moxifloxacin drops q1-4h and cycloplegia with scopolamine 0.25% tid.
  • If ulcer is severe, patient will be admitted and given topical fortified antibiotic drops.
  • Daily ophthalmology follow-up.

• Clinical pearls: Contact lens wearers are at much higher risk. Bacteria, fungi, HSV, and *Acanthamoeba* are all possible causes. Inpatients who cannot close their eyes fully are at increased risk for exposure keratopathy and corneal ulcers. Frequent lubrication, use of a moisture chamber, or tarsorrhaphy (performed by an ophthalmologist) can help avoid further corneal damage.
Approach to Consultation

Corneal Abrasion or Foreign Body

- This is usually not an emergency, but consult if in doubt.
- Pertinent information: Age, past ocular history, history of events leading up to event, occupational history (i.e., grinding, drilling, trauma), contact lens use, type of foreign body.
- Symptoms: Acute pain, tearing, photophobia, foreign body sensation.
- Workup:
  - Blue light or slit-lamp exam with fluorescein to detect epithelial defect (seen as green fluorescent spot).
  - Look for foreign body or rust ring.
  - Measure and record dimension of defect.
  - Evert eyelids to look for hidden foreign bodies.
  - Look for associated corneal ulcers (white opacity seen before fluorescein instilled).
- Treatment:
  - Remove foreign body (preferably by an ophthalmologist).
  - Non-contact lens wearers: Treat with bacitracin/polymyxin or erythromycin ointment q2-4h or polymyxin B/trimethoprim drops tid.
  - Contact lens wearers: Moxifloxacin drops qid or tobramycin or ciprofloxacin ointment q2-4h. No contact lens use until cleared by an ophthalmologist.
  - Cycloplegia with cyclopentolate 1% tid if any significant photophobia.
  - Follow-up next day with an ophthalmologist and follow-up as needed thereafter.
- Clinical pearls: If there is any chance of penetrating injury, call a consult immediately.

Painless Acute Vision Loss

Central Retinal Artery Occlusion

- Ocular emergency: call a consult stat! Like a CVA, “time is vision!”
- Pertinent information: Age, PMH/PSH/ocular history, allergies, current meds including ocular meds, specific ocular complaints, history of preceding event, vision loss.
- Symptoms: Painless, unilateral, acute loss of vision, prior history of amaurosis fugax (painless, temporary, uniocular vision loss).
- Physical exam: Whitening of the retina with a “cherry red” spot in the center of the macula, afferent pupillary defect
(Marcus Gunn pupil), narrowed arterioles, occasionally arteriolar emboli/plaque visible.

- Workup: ESR in patients over 50 years of age, fasting blood sugar, CBC, PT/PTT; in younger patients, also check ANA, RF, FTA-ABS, SPEP, sickle cell test, and antiphospholipid antibodies. Check blood pressure. Patient may need carotid Doppler and cardiac echo.

- Treatment:
  - Call an ophthalmologist! Permanent visual loss likely after 90 to 120 minutes.
  - Ocular massage: Have patient close eyes; apply pressure to the globe for 5 to 15 seconds, then release. Repeat several times.

- Clinical pearls:
  - Ask about symptoms of giant cell arteritis and check ESR in all patients over 50 years of age.
  - Etiologies include embolus (carotid or cardiac), thrombosis, giant cell arteritis, collagen vascular disease, hypercoagulable states, and rare causes (i.e., migraine, Behçet’s disease, syphilis).

**Retinal Detachment**

- Ocular urgency: call a consult.
- Pertinent information: Age, PMH/PSH/ocular history, allergies, current meds, specific complaints, history of preceding trauma or surgery.
- Symptoms: Painless unilateral decreased vision with associated flashes and floaters, curtain or veil across vision, relative visual field defect.
- Physical exam: Typically a “white and quiet” appearing eye—externally looks normal, usually no afferent pupillary defect unless a large retinal detachment. Fundus exam reveals a white, billowing, or wrinkled retina.
- Workup: Slit-lamp exam and dilated fundus exam.
- Treatment:
  - **No acute intervention.**
  - Low-level activity.
  - Needs a retina specialist evaluation.
- Clinical pearls: Risk factors include recent eye surgery, ocular trauma, high myopia, and a retinal detachment in contralateral eye. A peripheral retinal tear or hole may present with only flashes or floaters and no change in vision. These still require urgent ophthalmologic intervention.
Painful Acute Vision Loss

**Acute Angle-Closure Glaucoma**

- **Ocular emergency: call a consult immediately!**
- Pertinent information: Age, PMH/PSH/ocular history, allergies, current meds including ocular meds, specific ocular complaints, family history, recent surgery, recent laser surgery, cardiovascular/pulmonary status, electrolyte/renal status.
- Symptoms: Severe pain, blurry vision, colored halos around lights, frontal headache, nausea/vomiting.
- Physical exam: Conjunctival injection; fixed, mid-dilated pupil (usually in one eye); shallow anterior chamber; acutely elevated intraocular pressure (40s or above).
- Workup: Slit-lamp exam, measure intraocular pressure.
- Treatment:
  - In the acute setting manage the patient’s pain and nausea as needed.
  - Medical treatment involves ophthalmic drops and systemic medications to decrease intraocular pressure and medications to manage associated inflammation—consult ophthalmology first.
  - Definitive (laser) treatment by ophthalmologist.
- Clinical pearls: Be aware of patient’s cardiovascular and pulmonary status; evaluate electrolyte/renal status before starting systemic medications for ocular pressure control. Etiology can be from an anatomic pupillary block, neovascular, anterior displacement of lens-iris diaphragm (i.e., choroidal effusion, tumor), malignant glaucoma, medications (i.e., mydriatics, anticholinergics).

Papilledema/Optic Nerve Swelling

- **Emergency: Call a consult immediately!**
- Pertinent information: Age, PMH/PSH/ocular history, allergies, current meds including ocular meds, specific ocular complaints.
- Symptoms: Transient vision loss (“gray-outs”), headache, nausea/vomiting, diplopia, visual field defects, pain with eye movement.
- Physical exam: Unilateral/bilateral swollen, hyperemic discs; blurring of disc margins; obscuration of vessels; can have normal or abnormal papillary responses and color vision (can have sixth nerve palsy from increased ICP).
- Workup: Check BP; careful ocular exam (pupils, color vision, exam of fundus); urgent orbital and head CT. Consider LP (after head CT), CBC, TSH, ESR. Consider a neurology consult.
Treatment: **Treat underlying cause.**

Clinical pearls: Etiologies you may see on a medical service are vast and include pseudotumor cerebri, subdural/subarachnoid hemorrhage, AVM or sagittal sinus thrombosis, intracranial tumors, brain abscess, meningitis/encephalitis, hydrocephalus, malignant HTN, uveitis, infiltrative disease (i.e., sarcoid, TB, syphilis), ischemic optic neuropathy (i.e., giant cell arteritis), central retinal vein occlusion, papillitis (i.e., multiple sclerosis/optic neuritis, diabetic eye disease). It is often helpful to communicate the level of confidence of your fundus exam to the consultant.

**Infection**

*Endophthalmitis*

- **Ocular emergency:** Call a consult immediately!
- Pertinent information: Age, PMH/PSH/ocular history, allergies, current meds including ocular meds, history of ocular surgery or trauma.
- Symptoms: Unilateral painful eye, decreased vision.
- Physical exam: Moderate injection, hypopyon (pus behind the cornea), poor red reflex or view to the back of the eye.
- Treatment:
  - Emergent ophthalmologic evaluation.
  - Admission.
  - **May need culture and injected antibiotics.**
- Clinical pearls: A diagnosis of endophthalmitis must be considered first in any patient with recent ocular surgery!

*Herpes Zoster Virus*

- This is not an ocular emergency but urgent referral to an ophthalmologist is strongly recommended.
- Pertinent information: Age, PMH, ocular history, immunocompromised/AIDS.
- History: Skin rash and discomfort, blurred vision, eye pain, red eye.
- Physical exam: Skin vesicles in dermatomal distribution respecting the midline (Hutchinson sign, if rash involves the tip of the nose, high chance of eye being involved), conjunctivitis, uveitis, scleritis, cranial nerve palsy.
- Treatment:
  - If younger than 40 years, evaluate for immunosuppression.
• **Oral antiviral** (e.g., acyclovir 800 mg PO 5 times a day) for 7 to 10 days.
• Erythromycin ointment to skin lesions bid.
• Artificial tears or erythromycin ophthalmic ointment to eye as needed.
• Follow-up with ophthalmologist.

**The Red Eye**

**Conjunctivitis**

- This is not an emergency (Figure 21-3).
- Pertinent information: Age, possible contacts (1 to 3 weeks prior to onset of symptoms), past ocular history, allergies or previous history of allergic conjunctivitis; make sure there is no pain involved for allergic and viral conjunctivitis.

- **Symptoms:**
  - Viral: Unilateral red eye, possible associated URI symptoms, mild itching, morning crusting or discharge.
  - Allergic: Bilateral itching, mild redness, watery discharge.
  - Bacterial: Acute purulent discharge, eyelid edema, decreased vision.

- **Physical exam:**
  - Viral: Conjunctival injection, crusting, follicles.

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**Figure 21-3. Algorithm for red eye.**
• Allergic: Lid swelling, chemosis, papillae.
• Bacterial: Purulent discharge.

• Workup for bacterial: Gram stain and culture for *Staphylococcus*, *Streptococcus*, *H. flu*, *N. gonorrhoeae*.

• Treatment:
  • Viral:
    ■ No specific medical treatment necessary, self-limited illness.
    ■ Symptomatic treatment with cool compresses or artificial tears.
    ■ Naphazoline may be prescribed tid × 7 days.
    ■ Wash hands, towels, pillowcases. Use separate towels. No contact lens use until asymptomatic for at least 7 days.
    ■ Avoid close contact (especially rubbing eyes and touching others) without washing hands first.
    ■ Protect yourself. Wash your hands frequently around patients with suspected viral conjunctivitis and avoid touching your eyes.
  • Allergic:
    ■ Eliminate inciting agent.
    ■ Cool compresses and artificial tears.
    ■ Olopatadine or nedocromil antihistamine drops bid.
  • Bacterial:
    ■ Topical trimethoprim/polymyxin B or bacitracin ointment qid for 5 to 7 days.
    ■ For *H. flu*: Amoxicillin/clavulanate (20 to 40 mg/kg/d in three divided doses).
    ■ For GC: Ceftriaxone 1 g IM and empiric treatment for *Chlamydia* with azithromycin 1 g PO × 1. Treat sexual partners.

ORTHOPEDIC SURGERY

Prior to Calling the Consult
• Examination: A thorough examination of the patient should be made prior to calling an orthopedic consult. This should include neurovascular exam (pulses, sensation, motor function) of the involved extremity. All wounds should also be examined.
• Radiography: Prior to calling a consult, obtain and evaluate X-rays of the affected joint/bone. Usually two views, an AP and a lateral...
view, of the affected area are sufficient. Special cases are listed below:

- **Shoulder/proximal humerus**: AP, true AP, axillary, scapular lateral (or Y) views. An axillary view is especially important. This can be hard for the radiology techs to perform when the patient has a painful shoulder, but this view is essential to determine that the shoulder is not dislocated.

- **Hip fractures** (femoral neck/intertrochanteric fractures): AP hip, cross-table lateral (not a frog leg lateral), AP ortho pelvis.

- **Pelvis/acetabular fractures**: AP ortho pelvis, Judet (oblique) views, inlet/outlet views.

- **Ankle**: AP, lateral, mortise views.

- **Foot**: AP, lateral, medial oblique views; and a Harris heel (axial) view if a calcaneus fracture is suspected.

- Consultation with the orthopedic service is preferred, prior to obtaining a CT, MRI, or bone scan. Plain X-rays should always be obtained prior to ordering more advanced imaging.

- For fractures, acute dislocations, suspected compartment syndrome, joint sepsis, cauda equina syndrome, or diabetic infections; make patients NPO and discontinue all anticoagulants.

- If not already done, order basic pre-op labs: CBC, BMP, PT/INR, PTT, and type and screen. If infection is suspected, include an ESR and CRP. If the patient is febrile, order blood cultures.

- Certain orthopedic issues are better managed in an outpatient setting. Examples include chronic pain in the spine or extremity, chronic rotator cuff tears, ankle sprains, patients requesting joint injections. For these types of problems, consider having the patient follow-up with an orthopedist once they are discharged from the hospital, rather than calling an inpatient orthopedic consult.

- Rule out medical or general surgical causes of orthopedic symptoms, such as cholecystitis causing right shoulder pain, myocardial ischemia causing left shoulder pain, and inguinal hernia causing hip pain.

- If an orthopedist has operated on the patient within the past several months, even for an unrelated problem, consider a courtesy call to alert the orthopedist that the patient has been admitted. Any unexpected hospital admissions may affect the patient’s post-op rehab course.

**Fractures**

- **Open fractures and fractures with associated neurovascular compromise** are emergencies: Order X-rays and basic pre-op labs, make patient NPO, discontinue any anticoagulants, and call a...
consult immediately! Any open wound in the same limb segment as a fracture means that the fracture is open unless proven otherwise by the orthopedic resident.

• History: Mechanism of injury and pre-injury level of activity (e.g., does the patient walk, do they use a walker, are they wheelchair bound, what kind of work do they do?).

• Physical exam: Complete distal neurologic and vascular exam. Examine joints proximal and distal to the injury. Carefully examine all extremities to rule out other injuries. Take down splints/dressings to perform exams, unless the fracture has been reduced by another physician or outside hospital prior to your exam. Err on the side of taking down the dressing, as open fractures have been missed by referring EDs.

• Describing fractures: Attempt to delineate the following prior to calling consultation:
  • Fracture pattern (transverse, oblique, spiral).
  • Displacement: How far are the fragments away from each other? Which direction, anterior, posterior, medial, lateral?
  • Angulation: By how many degrees do the fragments relate to each other?
  • Shortening: How much do the fragments overlap?
  • Comminution: Is it a “clean” break or are there multiple small fragments about the fracture site?
  • Open versus closed: Any break in the skin in the vicinity of a fracture must be considered an open fracture until proven otherwise with a careful examination of the wound.

• Initial management:
  • Closed fractures are treated on an individual basis based on particular bone involvement and amount of displacement.
  • Open fractures require emergent operative debridement and fixation.
  • Keep patients NPO until they are evaluated by the ortho team.
  • Open fractures require a tetanus booster and antibiotics, usually cefazolin with or without gentamicin depending on the size and contamination of the wound. If there is fecal or barnyard contamination of the wound, consider anaerobic coverage (metronidazole or clindamycin) as well.

Septic Joint

• This is an emergency: call an orthopedic consult immediately after complete examination, X-rays and labs obtained. Make the patient NPO and discontinue any anticoagulants.
• Pertinent history: Warmth, painful range of motion, tenderness, fever, inability to ambulate/use extremity.

• Pertinent exam: Neurovascular exam (pulses, sensation, motor), effusion/fluctuance, erythema, warmth. Considerable pain with passive range of motion.

• Workup: Plain radiographs, CBC, BMP, ESR, CRP, and blood cultures (if febrile). The diagnosis is confirmed with joint aspiration. Aspiration may be done by the primary physician or an orthopedic consultant. **Do not aspirate a joint through cellulitis** if at all possible. Synovial fluid should be sent for stat Gram stain, cell count, crystals, and cultures (aerobic and anaerobic). **Antibiotics should not be administered until a joint aspirate is obtained.**

• Diagnosis:
  
  • Septic arthritis is diagnosed with a synovial fluid leukocyte count generally >50,000/mm³, a positive Gram stain, or a positive culture result.
  
  • An inflammatory/autoimmune arthropathy typically has a synovial fluid leukocyte count of 10,000 to 50,000/mm³ with positive crystals (for gout or CPPD disease) and negative Gram stain and culture results.

• Treatment:
  
  • **Operative drainage:** *Neisseria* spp. are exceptions to this rule, as they are highly responsive to antibiotic therapy; operative debridement is not necessary. A privately taken history may be of great importance in these cases.
  
  • Appropriate intravenous antibiotics as determined by cultures. The course of antibiotics is typically 6 weeks. Consider an infectious diseases consult and long-term venous access (e.g., PICC line or tunneled central venous catheter) if intravenous antibiotics are needed.
  
  • Clinical pearls: *S. aureus* is the most common organism in septic arthritis. *N. gonorrhoeae* is also prevalent in sexually active adolescents and adults, whereas *Salmonella* is more common in patients with sickle cell disease.

**Compartment Syndrome**

• **Compartment syndrome is an emergency, call a consult immediately** after complete examination and X-rays. Make the patient NPO and discontinue any anticoagulants.

• Definition: Compartment syndromes are caused by elevated hydrostatic pressure within a fixed osteofascial space, leading to
tissue ischemia as compartment pressure exceeds capillary pressure (i.e., the pressure in the compartment prevents blood flow out of and into the affected area). Elevated hydrostatic pressure commonly occurs from bleeding or swelling from within the compartment or from persistently elevated externally applied pressure.

• When to consider this: The most specific signs and symptoms of compartment syndrome are pain out of proportion to injury, pain with passive stretch of the muscles in the involved compartment, and hard tense compartments. Paresthesias, pallor, pulselessness, and paralysis may or may not be present (all are more indicative of arterial insufficiency). All external circumferential dressings should be removed before examining a patient for compartment syndrome.

• History: A typical history may include the following:
  • Trauma (fracture or muscle contusion)
  • Ischemia (vascular injury, extended compression)
  • Venous obstruction
  • Massive inflammation from snake or insect bites
  • Bleeding into the compartment (consider in anticoagulated patients)
  • Infiltration of fluid into a compartment (paint gun injuries, IV infiltration)
  • Tight circumferential dressings

• Physical exam: Directly palpate the concerning area to determine “tightness” of compartments. Compare with the contralateral side. Passively range the muscles that traverse the compartment (i.e., in the forearm, passively flex and extend the fingers). Check for pulses, sensation, and motor function. Continue to monitor the patient with serial exams.

• When clinical signs are equivocal, or when the patient is obtunded or not cooperative with the exam, compartment pressures may be measured by an orthopedic consultant. Compartment pressures $>30$ mm Hg (or a diastolic blood pressure to compartment pressure difference $<30$ mm Hg in hypotensive patients) are diagnostic of compartment syndrome.

• Treatment: Make patient NPO immediately upon suspicion of diagnosis. Obtain plain X-rays and any indicated pre-op labs. If compartment syndrome is confirmed, the orthopedics team will proceed with emergent fasciotomy.

• Clinical pearls: Remember that rhabdomyolysis can occur with compartment syndrome from muscle necrosis. Administer IV fluids, follow urine output, creatinine, and CPK.
Acute Cauda Equina Syndrome

- **This is an emergency, call a consult immediately** after examination and X-rays. Again, make the patient NPO and discontinue any anticoagulants.
- Definition: Cauda equina syndrome is caused by a lesion in the spinal canal located in the lumbar spine, between the conus medullaris and the lumbosacral nerve roots, resulting in urinary retention, bowel incontinence, saddle anesthesia, severe lower extremity neurologic deficit, and anal sphincter laxity.
- History: Suspect in a patient with low back pain and the previously mentioned signs and symptoms.
- Physical exam: A complete lower extremity neurologic exam should be performed including lower extremity strength, sensation, and reflexes. A rectal exam must be performed to assess both rectal tone and perianal sensation. Remember that the cauda equina functions as the peripheral nervous system. Therefore, in a complete cauda equina injury, all peripheral nerves to the bowel, bladder, perianal area, and lower extremities will be lost, resulting in absent bulbocavernosus, anal wink, and lower extremity reflexes. Nerve root tension signs such as pain on straight leg raise are likely to be present as well. On occasion, pain may radiate down the leg that is not being flexed (crossover pain) in addition to radiating down the leg being flexed.
- Workup: Stat AP and lateral views of the lumbar spine and a stat MRI of the lumbar spine. If the patient has had a prior discectomy, obtain MRI with gadolinium contrast. If the patient has had previous spinal surgery with instrumentation, obtain a CT myelogram.
- Treatment: Keep patient NPO, obtain necessary preoperative blood work, and discontinue anticoagulants. If cauda equine syndrome is confirmed, the orthopedic spine team will proceed with emergent operative decompression.

Diabetic Foot Ulcer/Infections

- Pertinent information: The acuity of these infections is dictated by the patient’s systemic symptoms. If the patient is febrile and/or hemodynamically unstable, call a consult immediately.
- Definition: Diabetic ulcerations occur after patients lose the protective sensation in their feet. Patients may present with an advanced infection due to lack of pain.
- History: Recent glycemic control (have insulin requirements been increasing in order to maintain the same level of plasma glucose?)
prior amputations or debridements, history of optic or renal dysfunction, or cardiac disease.

• Physical examination: Obtain a thorough neurovascular exam including pulses, sensation, and motor function. Examine all wounds taking note of depth, purulence, necrotic tissue, exposed bone, and proximal extension. If peripheral pulses are absent or diminished, perform an ankle arm index (AAI) and consider vascular surgery consult (see “Ischemic Ulcer of the Lower Extremity” in the General Surgery section).

• Workup: CBC, BMP, coagulation panel, ESR, and CRP. Obtain AP, lateral, and oblique views of the ankle and foot. Do not obtain an MRI prior to an orthopedic consult.

• Treatment: Surgical management will vary based on the wound and can range from bedside debridement to limb amputation. Antimicrobial therapy should be based upon tissue or bone culture results when possible, but broad-spectrum agents (e.g., piperacillin/tazobactam or meropenem with or without vancomycin) should be started promptly for signs of hemodynamic compromise. Consider an infectious diseases consultation, as well as placement of a long-term venous access device if an extended course of intravenous antibiotics is anticipated.

Oncology Patients

• The cancers that most commonly metastasize to the bone are breast, prostate, lung, thyroid, and renal cell carcinoma. Less common cancers are multiple myeloma, leukemia, lymphoma, and melanoma.

• Any oncology patient with pain in an extremity or in the spine may have a bony metastasis and an impending fracture. The appropriate X-rays should be obtained, and an orthopedic consult should be called if a lesion is visualized. Impending fractures are important to recognize and treat early, because once the bone is fractured, fixation can become much more difficult.

• Oncology patients with spinal metastases resulting in neurological compromise may do better with surgical decompression than with radiation alone. An orthopedic consult should be called for these patients.

Management of Postoperative Orthopedic Patients

• Orthopedic patients are frequently admitted to a general medicine service postoperatively for significant medical comorbidities or unexpected medical complications.
Approach to Consultation

• Orthopedic procedures can result in significant blood loss, so labs should be carefully followed in the 48 hours following any surgery. Special attention should be given to the H/H, coags, and urine output. It is not unusual for the patient’s hematocrit to decrease over the 48 hours following a large orthopedic procedure as the patient may ooze into the surgical bed for several days. No heparin drips or therapeutic anticoagulation should be started without discussing it with the orthopedic team.

• For all orthopedic patients, and especially patients with hip or pelvic pathology, there is a high risk of DVT. Unless they are at high risk for significant bleeding, patients should have TED hose and SCDs as well as prophylactic doses of heparin or LMWH. Patients at very high risk may be treated with warfarin postoperatively.

• Aggressive physical therapy is necessary for postoperative recovery. All patients should have a physical therapy consult and orders to be out of bed tid unless otherwise specified by the orthopedic consultant. The orthopedic consultant will indicate the weight-bearing status of the affected extremity.

• Orthopedic procedures can result in significant pain. Adequate pain control is important to allow postoperative mobilization and physical therapy.

OTOLARYNGOLOGY

Airway Emergencies

• Call a consult or the airway pager immediately for assistance!

• Pertinent history: Note onset, duration, progression, and severity of stridor (the degree of stridor may not necessarily indicate the severity of obstruction). Does it occur with inspiration, expiration, or both? Are there voice changes? History of prior intubation, neck trauma, head and neck cancer/surgery, irradiation to the neck, or tracheostomy?

• Pertinent physical exam: Cardinal sign of upper airway obstruction is stridor secondary to turbulent airflow. Inspiratory stridor usually indicates partial obstruction above the vocal cords (i.e., trauma/fractures, foreign bodies, hematomas, edema). Expiratory stridor usually indicates obstruction at or below the vocal cords. Biphasic stridor suggests partial obstruction at the level of the vocal cords. Other signs of respiratory distress may include restlessness, suprastrernal/subcostal retractions, and hoarseness (which usually denotes laryngeal pathology). A muffled or “hot potato” voice suggests supraglottic involvement, such as obstruction due
to an abscess or angioedema. Coughing or choking may be due to vocal cord paralysis, aspiration, reflux, or an anatomic abnormality (laryngeal cleft or TE fistula).

- **Workup:** In a true airway emergency, workup is deferred until a stable airway is established. In a less emergent setting, diagnostics include arterial blood gas, CBC, CXR, soft tissue airway films (may demonstrate supraglottic edema or subcutaneous emphysema), CT scan of the neck, and C-spine films in cases of trauma.

- **Treatment:**
  - Cool humidified air helps to thin secretions and prevent crust formation. Use a face mask or face tent rather than nasal cannula if possible.
  - Oxygen per nasal cannula, face mask, face tent, or non-rebreather may be beneficial regardless of measured oxygen saturations.
  - **Systemic corticosteroids** may be used if edema is suspected. Dexamethasone 10 mg and methylprednisolone 125 mg are most commonly used as acute treatment, with methylprednisolone having a somewhat faster onset.
  - **Nebulized racemic epinephrine** works quickly, acting as a topical vasoconstrictor; however, it is short acting, and there may be a rebound effect once it dissipates. In addition, it can cause acute elevations in blood pressure which can be problematic in some patients. If there is a lack of improvement with epinephrine, one must be concerned about a fixed structural obstruction.
  - Heliox refers to an 80%:20% helium–oxygen mixture. It relies on decreased density of helium to transport oxygen past the obstructive site. Usually used as a temporizing measure.

- **Clinical pearls:**
  - Nasopharyngeal airway (nasal trumpet) is beneficial for patients with oropharyngeal obstruction but normal respiratory drive. It provides support to the airway at the soft palate and base of the tongue.
  - Likewise, an oropharyngeal airway may treat ventilatory obstruction due to a relaxed tongue. It is not well tolerated in fully conscious patients.
  - Transoral intubation is the standard for airway control. Contraindications include C-spine fractures and some types of laryngeal or tracheal trauma. The laryngeal mask airway (LMA) is another option for emergent airway control,
especially as a temporizing measure. Use of this device is becoming more widespread and has the advantage of being placed without direct laryngoscopy. Endotracheal tubes can also be passed through some types of LMAs.

• Consultation is advised when a difficult intubation is anticipated (and a fiberoptic intubation would be preferred), for airway distress refractory to medical interventions, or when exam of the upper airway is indicated (i.e., rule out vocal cord paralysis, neoplasm, foreign body).

• While pulse oximetry should be monitored in a patient with airway concerns, note that saturations may stay above 95% until complete airway obstruction or respiratory exhaustion occurs. The pulmonary reserve for many patients is low and they may crash very rapidly despite initially stable pulse ox readings. Do not let pulse oximeter readings override clinical examination of a stridulous patient.

Tracheostomy

• Indications/workup:

  • The optimal timing for a tracheostomy is controversial; however, it is accepted that earlier intervention has beneficial effects for critically ill patients. Evaluation of the patient at 7 to 10 days after intubation is appropriate to assess for likelihood of extubation. If long-term intubation is probable, then a tracheostomy is justified. In some patients with neuromuscular disorders or severe neurologic injury in which long-term ventilatory support is anticipated, earlier tracheostomy may be indicated.

  • Workup for tracheostomy includes physical examination, evaluation of coagulation (PT/PTT, bleeding time, or PFA-100), peak airway pressures, and patient’s expected prognosis.

  - Patients on anticoagulation (including ASA) are at higher risk of both intraoperative and postoperative hemorrhage. Reverse or discontinue anticoagulation, if possible.

  - Patients with high peak airway pressures (numbers vary, but typically over 40 to 45 cm H₂O) are at higher risk for ventilatory complications due to leakage of air around the tracheostomy tube at high pressures and the need for excessive cuff pressures to maintain a seal.

  - Prognosis is a key component of discussion with the family regarding the purposes of a tracheostomy. Assistance with long ventilator weans is an appropriate indication, as is palliation of airway obstruction in end-of-life situations.
• Two types of tracheostomy may be performed: open surgical tracheostomy (performed in the operating room) or bedside percutaneous dilational tracheostomy (PDT). PDT avoids transporting a critically ill patient out of the ICU but may be prone to complications in some patients (e.g., obese or prior neck surgery). The choice of procedure is dependent on patient characteristics, as well as the experience and preference of the surgeon.

• Types of tracheostomy tubes: Tubes come in metal or plastic varieties. Shiley and Portex brand tubes are plastic and come with or without cuffs. Cuffs are necessary when ventilatory support is needed and are always used as the initial tracheostomy tube. Jackson tubes are metal and are not cuffed. After the tracheostomy, the tube is kept undisturbed for 3 to 7 days to allow for formation of a well-healed tract. At this point, the original tube is changed to either a similar cuffed tube or to a cuffless tube (if ventilatory support is not required).

• Post-tracheostomy care: ENT will perform the first tracheostomy tube change after 3 to 7 days to ensure a well-healed tract has formed. Further trach changes can be performed by nursing staff on the floors or by the patient/family after discharge. Frequent cleaning or changing of the inner cannula is recommended to prevent obstruction by crusting (typically at least tid). A patient with a tracheostomy should always wear a high-humidity trach collar to thin secretions and have bedside suction catheters available. All patients should have a spare trach in the room and should also have their obturator secured to the foot of the bed where it is easily accessible. The obturator is the metal or plastic inner piece that facilitates reinsertion of the trach. Nurses and physicians caring for these patients should know its location and have immediate access to it.

• Dislodged tracheostomy tube: If the trach tube comes out, first assess the stability of the patient.
  • If in respiratory distress or with stridor, call the ENT consult or emergency airway pager immediately for assistance. You may reinsert the tube with the aid of the obturator (which will be secured at the bedside as noted above) and resecure the collar to prevent further dislodgement. If this is not possible or unsuccessful and the patient is decompensating, transoral endotracheal intubation is an option for most patients. The exception is a patient with previous laryngeal surgery who may have difficult transoral access (or no access in the case of a patient after a total laryngectomy).
  • If the patient is stable and comfortable, you may attempt to reinsert the tracheostomy tube as noted above. Using a small Kelly clamp to retract the skin, along with a bright
light source, may give a better view of the tract. Occasionally, passing the trach tube over a Foley catheter or nasogastric tube will do the trick.

- If the patient is stridulous, talking, or breathing through the nose or mouth with the trach tube in place, it is likely in a false tract. If in proper position, most of the expired airflow should emanate through the tracheostomy tube and not the nose/mouth. Thus, you may feel for airflow through the tube. Also, passage of a tracheal suction catheter into the trachea via the tube should always elicit a cough and is a standard way of confirming proper tube placement. Removing the tube and reattempting with the methods above will usually allow for correct placement. If difficulty persists, call the ENT consult pager for assistance.

Epistaxis

- May be considered emergent, urgent, or routine based on volume of blood loss, hemodynamic stability, airway compromise, and whether the patient is currently bleeding. Be clear in defining the urgency of the consult when calling to elicit the appropriate rapidity of response.

- Pertinent history: Trauma (digital or facial)? Anticoagulant medications? Systemic diseases with bleeding diatheses (e.g., hemophilia, liver disease, von Willebrand disease, hereditary hemorrhagic telangiectasia)? Local nasal inflammation (e.g., rhinosinusitis, allergic rhinitis, digital trauma, foreign body)? Does the patient use nasally administered medications or nasal cannula oxygen?

- Pertinent physical exam: Ensure stable vitals. Systemic hypertension may initiate or perpetuate bleeding. Determine the source of bleeding (anterior vs. posterior and right vs. left naris). Identify any anatomic abnormalities such as septal deviation or septal perforation that may cause turbulent airflow and resultant epistaxis. Identification of most anterior sites can be aided by nasal speculum and light source (headlight or mirror).

- Workup: Check coagulation values including PT/PTT, bleeding time or PFA-100, and hematocrit.

- Treatment:
  - Patient should be seated completely upright to decrease risk of aspiration. For the same reason, head should be tilted slightly forward, not back.
  - **Simple nasal compression for 15 minutes** will stop most nosebleeds. To be effective, this must be maintained by the patient or staff without releasing pressure for the entire
15-minute period. Do not pack the nose with tissue or gauze. These will traumatize the nasal mucosa and result in further injury. If the initial round of compression is not successful, always try a second round of nasal compression.

- Control severe hypertension; however, most epistaxis is not directly caused by this.
- Application of topical vasoconstrictors (oxymetazoline hydrochloride 0.05% or phenylephrine hydrochloride 0.25%) may help to slow down bleeding. Follow this with nasal compression as described above.
- If a small anterior bleeder can be visualized, it can be cauterized with judicious use of a silver nitrate stick. Never cauterize both sides of the nasal septum, as this can lead to septal perforation.
- Remove nasal cannula O₂, which dries and irritates the nasal mucosa. Replace with humidified oxygen by face mask or tent and frequent use of nasal saline spray.
- **Nasal packing:** If the above measures fail to control bleeding, nasal packing will be required. Nasal packs are typically placed by the ENT service. This may be performed with Vaseline strip gauze or with commercially available nasal packs. When calling a consult, ensure that the patient has been positioned as above and anterior nasal compression is being maintained. Set up suction at the bedside. Anterior packs are usually well tolerated and require prophylactic anti-staphylococcal antibiotics to prevent toxic shock syndrome. If a standard nasal pack is unsuccessful, the patient may require placement of an Epistat balloon. This pack rests in the nasopharynx and, if placed, requires cardiac monitoring due to the possibility of vagally mediated bradyarrhythmias. All packs remain in place for 3 to 7 days and are removed by the ENT service. Stable, reliable patients can be discharged with a pack in place (except for those with Epistats) and return for removal as an outpatient. If packing fails, the patient will require embolization by interventional radiology or surgical ligation of bleeding vessels.

**Complicated Acute Sinusitis**

- This is an emergency when the infection has extended past the sinuses to involve intraorbital or intracranial structures. If this is suspected or confirmed, call a consultant immediately!
Approach to Consultation

• Pertinent history: Duration of sinusitis symptoms, vision changes, mental status changes, duration/route of antibiotic therapy, previous sinus surgery. Predisposing factors include malnutrition, diabetes, chemotherapy, long-term corticosteroids, allergic rhinitis, immunodeficiency states, environmental exposures, and presence of nasogastric tube.

• Pertinent physical exam: Meningeal signs and orbital signs (proptosis, chemosis, ophthalmoplegia, vision loss). These suggest extension of the infection beyond the sinus and necessitate immediate attention by an ENT consultant.

• Workup: High-resolution maxillofacial CT (with coronal cuts) with contrast to rule out subperiosteal/orbital abscess. Head CT with contrast may be indicated to look for intracranial involvement.

• Treatment:
  • An ophthalmology consult is required if there is suspected orbital involvement. They will be able to document pressures and visual acuity changes that determine the need for intervention.
  • IV antibiotics (including anaerobic coverage).
  • Copious use of saline (Ocean) nasal spray and 3 days of oxy-metazoline to aid in nasal drainage.
  • IV steroids (dexamethasone 10 mg or methylprednisolone 125 mg) to help diminish edema around orbits and reduce optic nerve damage.
  • Surgery (functional endoscopic sinus surgery or external surgical drainage) is definitive therapy to drain abscess and sinuses.
  • If optic nerve damage is imminent due to intraorbital abscess, then immediate lateral canthotomy with tendon cantholysis should be done to decrease intraocular pressure.

Vertigo

• Vertigo emergency:
  • Important vertigo emergencies are rare and include the following:
    • Wallenberg (lateral medullary) syndrome
    • Lateral pontomedullary syndrome
    • Cerebellar hemorrhage
    • Cerebellar infarction
    • Vertebrobasilar insufficiency
• History: Sensation and duration of dizziness, associated neurologic symptoms (headache, visual changes, unilateral weakness, dysarthria, or paresthesias), nausea, or vomiting.

• Physical exam: Associated neurologic findings (diplopia, dysarthria, drop attacks, vision loss, dysphagia, loss of pain/temperature sensation, loss of motor control), Horner’s syndrome, nuchal rigidity, papilledema, nystagmus of central origin (characterized by lack of fixation suppression, spontaneously upbeating or downbeating, or that which changes direction with changing gaze direction).

• Workup: Neurology consultation; CT/MRI or cerebral angiogram depending on suspected etiology.

• Treatment: Dependent on etiology, may include surgical decompression, anticoagulation, and/or supportive care.

• Vertigo (general):
  • Common etiologies:
    ▪ Benign paroxysmal positional vertigo (BPPV)
    ▪ Ménière’s disease
    ▪ Vestibular neuronitis
    ▪ Migraine-associated vertigo

  • History: It is important to rule out central from peripheral causes of vertigo (see above section) as well as differentiating true vertigo (abnormal perception of motion) from light-headedness or feeling “off-balance.” Note exacerbating factors (position changes, sudden head movement, noise, sound), other otologic symptoms (hearing loss, otalgia, otorrhea, tinnitus), general medical history, medications. Duration and frequency of episodes are crucial to making the correct diagnosis.

  • Physical exam: Nausea and vomiting (these tend to point to a peripheral cause), horizontal nystagmus, fixation suppression of nystagmus, Dix-Hallpike maneuver (BPPV).

  • Workup: Rule out medical causes including hypotension or hypertension, cardiac arrhythmias, endocrine abnormalities.

  • Treatment: Dependent on the exact etiology (e.g., BPPV treatment requires Epley maneuver for otolith repositioning). Short-term symptomatic treatment may include the following:
    ▪ Prochlorperazine suppositories, 25 mg q6h prn.
    ▪ Hydroxyzine, 12.5 to 25 mg PO q8h prn.
- Diazepam, 2 to 10 mg PO q6h prn.
- For severe cases, diazepam 5 to 10 mg IM or droperidol 2.5 mg IM.

**PSYCHIATRY**

**Before You Call a Consult**

- Respect patient autonomy and the right to refuse a consultation/treatment.
- Psychiatric consultation in and of itself may be stigmatizing.
- **Patients have the right to refuse consultation, unless:**
  - There is concern about the patient being a danger to himself/herself or to others.
  - There is concern about the patient’s decision-making capacity.
- Clinical pearl: The patient should always be told that a psychiatric consultant is coming to see him or her.

**Suicidality**

- When to suspect ideation: when the patient appears sad, depressed, or anxious; when there is a significant drug or alcohol history; when there is a history of domestic abuse; when psychosis is present.
- Before calling the consult, obtain the following information:
  - Key history: Age, gender, previous psychiatric treatment, presence of current suicidal ideation and suicide plan, presence of psychosis and command hallucinations, presence of anxiety, current meds, brief general medical history, and hospital course.
  - Key physical findings: Presence or absence of agitation, anxiety, overt psychosis.
- Treatment:
  - Keep patient safe; get a sitter until directed otherwise by psychiatry.
  - Do not let a suicidal patient leave without clearance from psychiatry; once medical issues are resolved, the patient may require transfer to psychiatry, possibly against the patient’s wishes.
- Clinical pearls:
  - Suicidal ideation is a symptom, not a diagnosis; a full psychiatric interview is necessary to determine the cause and direct treatment.
  - Never be afraid to ask about the presence of suicidality; you will not give the patients ideas they didn’t already have.
Violent Patients

• Critical diagnostic question: **Is delirium present** (i.e., does the patient have a fluctuating level of consciousness with altered mental status)?

• Before calling the consult, obtain the following information:
  - Key history: Age, gender, onset of symptoms, level of orientation, presence of psychosis, prior psychiatric treatment, current meds, brief medical history, and hospital course.
  - Key physical findings: Vital signs, overt psychosis, localized findings on neurologic exam.

• Workup: Directed at identifying the cause of the delirium; may include electrolytes, CBC, UA, UDS, LFTs, CSF studies.

• Treatment:
  - Protect the patient and staff; **if necessary sedate the patient with antipsychotics** (e.g., haloperidol IM in doses ranging from 0.5 mg in the frail and elderly to 5 mg in the younger and larger; better to use IM than IV due to higher risk of QT prolongation with IV administration); use restraints if necessary.
  - Identify and **treat the cause of the delirium**.
  - Family members can help reorient delirious patients and lessen their violence. Dimly lit, quiet rooms help, as do glasses and hearing aids for those who need them.

• Clinical pearls:
  - Common, less obvious causes of delirium are anticholinergic medications, benzodiazepines, undertreated pain, opiates, and steroids. Offending medicines should be tapered or discontinued as much as possible.
  - **Avoid using benzodiazepines for sedation in delirious patients unless the delirium is from alcohol or sedative withdrawal or phencyclidine intoxication**.
  - Do not put yourself in danger. Remove all possible items in the vicinity that could be used against you (e.g., stethoscope).
  - Have security with you.
  - Stand between the patient and an open door.
  - Using antipsychotics at high doses too frequently can sometimes lead to akathisia (an internal sensation of restlessness), which can increase agitation.
Competency/Decision-Making Capacity

- Definitions:
  - **Competence** is technically a legal term. Only a judge can declare someone incompetent (and appoint a guardian, for example).
  - **Decision-making capacity** refers to the ability of patients to give informed consent to medical care; psychiatrists can often assist in the assessment of capacity.

- Evaluation of decision-making capacity is usually an emergency or urgency as the patient typically requires emergent or urgent medical care for which the patient is unable or unwilling to give consent.

- Before calling the consult, obtain the following information:
  - Key historical information: Age, gender, proposed medical care and risks, benefits, and alternatives particular to the patient, medical history, current meds, presence of psychosis or depression, psychiatric history.
  - Key physical findings: Presence or absence of agitation, anxiety, overt psychosis.

- Demonstration of decision-making capacity requires all four of the following:
  1. The ability to communicate a choice.
  2. Understanding of the medical situation and likely outcome of no treatment.
  3. Understanding of risks and benefits of treatment options.
  4. Ability to manipulate information rationally and give a rational explanation for preferred treatment.

Some people add a fifth criterion, which is consistency of the choice over time, but this is more controversial.

- Clinical pearls:
  - Many consults to psychiatry result from patients not being adequately informed of the risks and benefits of the proposed treatment.
  - **Presence of psychosis does not necessarily mean that a patient lacks capacity** (e.g., belief that one is part of the intergalactic guard may have no bearing on understanding the risks and benefits of cardiac catheterization).
  - **Capacity is decision specific**; one may have capacity to take IV meds but not PO if one believes that all of the pills are sprayed with a poison.
**Capacity is time specific.** Demonstrating capacity today is no guarantee that one will be able to demonstrate capacity tomorrow should mental status fluctuate.

Psychiatric consultation can only help with determining if a patient lacks capacity to make a decision; the consult will not tell you who the decision-maker should be if the patient lacks capacity.

**Psychosis**

- Definition: Psychosis is a break with reality demonstrated by hallucinations, delusions, or bizarre behavior.

- Psychosis itself is not a psychiatric emergency. The psychiatric consult can wait until the morning. On a medical/surgical floor, **psychosis is often a symptom of delirium**, which can be a medical emergency.

- Critical diagnostic question: Is the patient delirious (i.e., does the patient have a fluctuating level of consciousness with altered mental status)? If so, see the section on the violent patient for further discussion of delirium.

- Before calling the consult, obtain the following information:
  - Key history: Age, gender, previous psychiatric treatment, nature of psychosis and symptom onset, presence of anxiety, current meds, brief general medical history and hospital course, presence of suicidal or homicidal ideas, presence of command hallucinations.
  - Key physical findings: Presence or absence of agitation, anxiety, thought disorder, bizarre behavior.

- Clinical pearls:
  - Visual hallucinations usually result from delirium or intoxication.
  - Auditory hallucinations are the most common form in psychiatric disorders.
  - Olfactory and gustatory hallucinations are usually seen in the aura of a seizure.
  - Tactile hallucinations can result from drug withdrawal.
  - Psychosis is a symptom, not a diagnosis; a full psychiatric interview is necessary to determine the cause and direct treatment.
  - Psychosis can be related to dementia as well as delirium. Of note, elderly dementia patients on antipsychotics have increased mortality risk; risk/benefit ratio must be examined to determine treatment.
Approach to Consultation

Domestic Violence, Rape, and Psychiatric Trauma

- Legal reporting requirements:
  - **Physicians in every state are required to break confidentiality and report suspected cases of child abuse** to local authorities, usually called the Division of Family Services or Child Protective Services.
  - Many states also require that suspected elder abuse be reported.
  - There are no such legal requirements for spouse abuse.
- Rape victims should be referred to obstetrics and gynecology for collection of evidence, treatment of physical trauma, evaluation of exposure to STDs, and referred for follow-up counseling.
- Psychiatric consultation may help with the treatment of depression, anxiety, substance abuse, posttraumatic stress disorder, and personality disorders that are all commonly found in the victims of domestic violence and rape. Perpetrators of domestic violence also frequently have many of these problems.
- Before calling the consult, make sure the patient is willing to see a psychiatrist. Include the following information:
  - Key history: Age, gender, previous psychiatric treatment, nature of symptoms, presence of anxiety and depression, current meds, brief general medical history and hospital course, presence of suicidal or homicidal ideas.
  - Key physical findings: Presence or absence of agitation, anxiety, overt psychosis.
- Diagnosis: Questions regarding domestic abuse should be asked as a routine part of the social history in every patient.
- Treatment:
  - Should begin with **referral to a specific rape/domestic violence support program** if one is available locally.
  - Will depend on the patient’s individual symptoms.
  - Generally includes allowing the patient to tell and retell the story of the trauma in a safe, supportive environment so that the associated anxiety lessens over time.
- Clinical pearls:
  - If in doubt, call protective services regarding child abuse for more guidance.
  - Patients rarely volunteer information on being a victim of domestic violence; the first step toward helping them is to ask.
Never tell victims of domestic violence that they must leave their current living situation immediately. Such a proclamation could subsequently result in a lethal attack.

**Withdrawal/Chemical Dependency**

- **Minor alcohol withdrawal:**
  - **Diagnosis:**
    - Tremors, headache, nausea, sweating, and autonomic instability occurring approximately 12 hours after the last drink and lasting up to 5 days if untreated.
    - No hallucinations, seizures, or delirium.
  - **Treatment:**
    - **Benzodiazepines** (e.g., lorazepam 0.5 to 2 mg q6-8h or chlordiazepoxide 25 mg qid) given scheduled and/or prn to keep vital signs stable, with a gradual taper over approximately 4 days. Need only to treat for objective symptoms of alcohol withdrawal. For severe withdrawal, consider giving more frequent doses of lorazepam than above depending on the clinical course, based on objective changes in autonomic tone (HR, BP).
    - Frequent monitoring of vital signs.
    - Adequate hydration.
    - Adequate replacement of electrolytes, particularly potassium and magnesium, as needed.
    - Replacement of vitamins, especially vitamin C, folate, and thiamine.
    - Seizure prophylaxis in those with a history of seizures.
    - The patient should be encouraged to allow psychiatric consultation for diagnosis and treatment of a possible chemical dependency.
- **Major alcohol withdrawal** (aka rum fits and DTs).
  - This should not result in a straight psychiatric consultation. Consultation with a medpsych service or internal medicine may be more appropriate, and such consultation may be emergent.
  - **Diagnosis:**
    - See the criteria under minor alcohol withdrawal.
    - Between 3 days and 2 weeks after the last drink, minor withdrawal symptoms become accompanied by hallucinations, seizures, or delirium. Autonomic instability worsens.
• Treatment:
  - Same as for minor alcohol withdrawal, but monitoring of vital signs and supportive treatment are even more important; severe cases may require transfer to the ICU.
  - Haloperidol added to the benzodiazepine can help treat hallucinations.
  - The patient should be encouraged to allow psychiatric consultation for diagnosis and treatment of a possible chemical dependency.

• Cocaine and opioid withdrawal:
  - Diagnosis:
    - In opioid withdrawal: nausea, muscle aches, rhinorrhea, diarrhea, piloerection, and craving.
    - In cocaine withdrawal: fatigue, agitation, increased appetite.
  - Treatment:
    - While both conditions are unpleasant for the patient, they are rarely medically serious.
    - Opioid withdrawal can be treated with clonidine, 0.1 mg PO tid or a methadone taper. Can treat other symptoms prn (e.g., ibuprofen for aches).
    - The patient should be encouraged to allow psychiatric consultation for diagnosis and treatment of a possible chemical dependency.

• Chemical dependency:
  - Before calling the consult, make sure the patient is willing to see a psychiatrist. Include the following information:
    - Key history: Age, gender, previous psychiatric treatment, amount of use, route of use, withdrawal symptoms, presence of anxiety and depression, current meds, brief general medical history and hospital course, presence of suicidal or homicidal ideas, presence of psychosis.
    - Key physical findings: Presence or absence of agitation, anxiety, overt psychosis, withdrawal signs.
  - Diagnosis: Questions regarding alcohol and substance use should be asked as a routine part of the social history in every patient.
  - Criteria revolve around tolerance, withdrawal, and inability to control use.
If two of the CAGE questions are positive, the patient should be encouraged to allow psychiatric consultation for more definitive diagnosis. Other patients, of course, may also be appropriate for referral.

1. Ever tried to Cut down?
2. Have people Annoyed you by criticizing your drinking?
3. Felt Guilty about drinking?
4. Had an Eye opener (morning drink) to avoid withdrawal?

Treatment:

- Support groups such as AA.
- Anticraving medication such as naltrexone, nalmefene, or ondansetron for alcoholism.
- Methadone maintenance (from specially licensed clinics) for severe opioid dependence. Suboxone is also used for maintenance treatment of opioid dependence, but is often not readily available in nonpsychiatric inpatient units due to restricted distribution.
- Psychotherapy aimed at relapse prevention.
- Treatment of comorbid depression and anxiety disorders, which are very common.

Clinical pearls:

- Sedative withdrawal has the same clinical picture as alcohol withdrawal.
- The shorter the half-life of the benzodiazepine (e.g., alprazolam), the more likely the withdrawal.
- Untreated DTs have a mortality of over 15%.
- Drug use often accompanies STDs, physical trauma, and other medical conditions.

GENERAL SURGERY

Key Points

- The following recommendations apply to almost all instances of calling for a consultation from essentially any service.
- Identify yourself and the patient that needs a consult and then clearly identify the questions you need answered.
- Give an indication of the urgency of the consult (i.e., stat, a few hours, or sometime today).
- Present the crucial information for the problem.
• State whether important radiographs have been obtained. If they are not accessible to the consultant electronically (i.e., if they were done at another hospital), state their location.

• State whether the patient has had a recent operation, and who performed it, or whether the patient has ever been operated on for a similar or related problem, and by whom.

• Time-efficient communication is crucial.

For example: Hi, this is Mike Smith. I am a medicine resident. I have an urgent consult regarding the management of a patient with a pulseless and cold right foot. The patient is Mr. Smith, his DOB is 5/5/45 and he is located in room 6443. He is a 62 y.o. diabetic male with CAD and severe PVD who ... 

Hernia

• A strangulated hernia is a surgical emergency, call a consult immediately!
  • A reducible hernia is one that can easily return through its fascial defect.
  • An incarcerated hernia is one that is irreducible (impossible to push back through the fascial defect).
  • A strangulated hernia is one in which the blood flow of the hernia’s contents is compromised leading to necrosis of the contained structures. The signs of this are a fever, leukocytosis, hypotension, erythema of the overlying skin, or extreme pain with light palpation of the hernia.

• Pertinent information: Location and duration of the hernia, scar overlying hernia, patient’s surgical history, associated symptoms, status (strangulated, incarcerated, or reducible), time of patient’s last bowel movement, fever, leukocytosis, or erythema of the skin overlying the hernia. Is the patient immunocompromised?

• Physical exam: Diagnosis of a hernia is made by examining the patient in a standing position with the patient performing Valsalva maneuver or coughing. A mass that protrudes is a hernia until proven otherwise.

• Treatment:
  • Attempt to reduce an incarcerated, nonstrangulated hernia that does not protrude inferior to the inguinal ligament. Do NOT attempt to reduce a hernia that you suspect is strangulated.
  • Place the patient supine in the Trendelenburg position and slowly apply firm, constant, circular pressure with the palm of the hand to the hernia.
• If the hernia reduces, then perform an abdominal examination an hour later to prove that ischemic bowel was not reduced.

• If the patient has abdominal pain and you suspect ischemic bowel from the hernia, then call surgery urgently.

• Most hernias require an operation if the patient can tolerate the risks of anesthesia. Therefore, a nonurgent surgery consult should be called even if the hernia is reduced (the consult can wait until morning).

• Trusses or binders are usually not effective in the treatment of hernias.

• Clinical pearls: Hernias are classified by both anatomy and status. Over 75% of hernias occur in the inguinal region, 10% are incisional or ventral hernias, 3% are femoral, and the rest are unusual types. The location is of less concern than the status of the hernia. Not all incarcerated hernias are strangulating.

Small Bowel Obstruction

• Small bowel obstruction is an urgent consult. The most common causes are adhesions from previous abdominal operations (50% to 70%), incarcerated hernias, and carcinoma. As soon as a small bowel obstruction is diagnosed, intravenous fluids should be started, and a Foley catheter and nasogastric tube should be placed.

• Pertinent information: The hallmarks of diagnosis are abdominal distension, nausea, vomiting, waves of abdominal pain progressing to constant pain (an ominous sign), and cessation of flatus and bowel movements.
  • Which symptoms are present? How long have they been present?
  • When was the patient’s last bowel movement? Last flatus?
  • Is the patient febrile (and do they have a leukocytosis)?
  • Are there any hernias?
  • Have they had any previous abdominal or pelvic operations?
  • What is the output from the nasogastric tube and Foley catheter? What are the patient’s vital signs and fluid status? With a persistent obstruction, hypovolemia often results due to impaired absorption, third spacing, and vomiting.
  • Does the digital rectal exam reveal impacted stool or a rectal mass?

• Physical exam: Check for hernias in the groin and umbilicus and all scars for incisional hernias. Are the hernias incarcerated or strangulated? If peritoneal signs are present, urgent consult is indicated.
• Diagnosis: An obstructive series (KUB, right lateral decubitus abdominal film, and CXR) should be obtained. What does the obstructive series show? Is there colonic or rectal air? Are there air-fluid levels? Most importantly, does the radiograph demonstrate any free air? A CT scan with intravenous contrast can be helpful to identify a transition point and subtle signs of intestinal ischemia.

• Treatment options:
  • Patients with complete bowel obstruction or peritonitis generally require prompt surgical intervention. These are often associated with strangulation of bowel.
  • Most patients with partial small bowel obstruction can be managed expectantly with nasogastric tube decompression, fluid resuscitation, serial abdominal exams, and daily abdominal plain films.

Hints for Diagnosis of an Acute Abdomen

• An acute abdomen warrants immediate surgical intervention. These hints are not rigid rules because the diagnosis of an acute abdomen can require much judgment. The most common signs indicating an acute abdomen are peritoneal signs due to peritoneal inflammation. If at all there is any doubt, call ….

• Signs:
  • Rebound: This should never be tested for by pushing into the patient’s abdomen and releasing, since it can cause excruciating pain in the patient with peritonitis. Instead, be gentle: pain with percussion on the anterior abdominal wall is the best test. Tapping on a patient’s foot can also transmit vibrations to the abdominal cavity. Patients with an acute abdomen will not tolerate this.
  • Guarding: Involuntary guarding is another finding in peritonitis. To distinguish involuntary guarding from voluntary guarding, apply constant pressure to the abdominal wall in a location far away from the point of maximal pain and ask the patient to take a deep breath and relax. If the muscles remain spastic despite this, involuntary guarding is likely present.
  • Sitting up for posterior chest auscultation or rolling over for a rectal examination: Most patients with peritoneal signs will not do this.

Abdominal Pain with a Pulsatile Abdominal Mass

• Abdominal pain with a pulsatile abdominal mass is an incredible emergency!
Abdominal pain with a pulsatile abdominal mass, suggesting an AAA, is the easiest problem you will ever evaluate:

- **Call surgery for a stat consult!**
- Type and cross the patient for six units of blood.
- Ensure there is adequate intravenous access, at least two large-bore catheters.

### Ischemic Lower Extremity

- **An ischemic extremity is an emergency, call a consult immediately!**
- Symptoms of acute arterial insufficiency can occur abruptly. On exam, look for the 6 P’s: pain, pallor, pulselessness, paresthesias, paralysis, and poikilothermia.
- An ischemic extremity may be due to acute events (embolic disease) or chronic disease (atherosclerosis). The acute event is an emergency because perfusion must be reestablished within 6 to 8 hours. Unlike patients with chronic disease, many patients with acute obstruction have not developed collateral circulation to supply the lower leg. If unsure whether the ischemia is acute or chronic, do not hesitate to call an immediate surgical consult.

- **Pertinent information:**
  - Suspected source: embolism (atrial fibrillation/arrhythmia, LV aneurysm, AAA, or popliteal aneurysm) or chronic disease (atherosclerosis).
  - Did the pain come on suddenly? Is the pain unilateral?
  - Status of the vascular system: Has this patient had vascular surgery? If so, where does the bypass start and end? Where are the scars, and who was his or her surgeon?

- **Physical exam:**
  - On exam, look for the 6 P’s: pain, pallor, pulselessness, paresthesias, paralysis, and poikilothermia.
  - Status of collateral flow: Palpate or perform a Doppler examination in the femoral, popliteal, dorsalis pedis (DP), and posterior tibial (PT) arteries. Be able to tell the consultant if there is a temperature difference in the extremities and at what level (foot, shin, thigh, or whole leg).
  - Severity of ischemia: The peripheral nerve is the tissue that is most sensitive to ischemia. The most sensitive test to determine if the foot is viable is to test for proprioception of the toes. This will diminish within 5 minutes of cessation of blood flow. Next, test motor function and light touch.
Treatment options:

• Most patients are started on intravenous heparin therapy.
• Possible interventions include surgical bypass, surgical or interventional radiographic thrombectomy, or locally delivered intravascular thrombolytics.

Ischemic Ulcer of the Lower Extremity

• Ischemic ulcer of the lower extremity is an elective consult.
• Pertinent information:
  • These ulcers are commonly found on the first metatarsal head or tips of the toes and are due to a combination of unrecognized trauma, poor circulation, and infection. These are distinguished from venous stasis ulcers by location (usually found near the medial malleolus), appearance (heaped up, engorged edges), and sensitivity (very painful).
  • Status of the vascular system: Has this patient had vascular surgery? If so, where does the bypass start and end? Where are the scars?
  • Have any AAIs been performed? If not, use a Doppler and portable blood pressure cuff to do bedside AAIs. Place the blood pressure cuff on the arm and then using the Doppler, find a pulse in the radial artery. Inflate the cuff until the Doppler signal disappears. Record the pressure at which the signal disappears (the systolic blood pressure, SBP). Repeat on the opposite arm. Next, place the cuff on the calf. Using the Doppler, find the DP or PT pulse distal to the cuff and inflate until the signal is again lost. Divide the SBP of the left foot by the SBP in the left arm for the AAI. Repeat on the right side.
  • Plain radiographs should be obtained to document osteomyelitis of the underlying bone. Any exposed bone is assumed to have osteomyelitis until proven otherwise.
  • Is the patient a diabetic? Are his blood sugars well controlled? Diabetics can have calcified vessels that make AAIs unreliable. Some diabetics develop ulcers secondary to microvascular disease without obvious atherosclerosis in the larger vessels. Diabetics can also have poor wound healing if their blood sugars are poorly controlled.
  • Pertinent physical exam: Palpate or perform a Doppler examination of the pulses in the femoral, popliteal, dorsalis pedis, and posterior tibial arteries. Look for signs of infection (e.g., erythema, pain, fluctuance, pus discharge with palpation).
• Treatment options:
  • Arterial: Amputation, debridement, or any procedure to increase vascular inflow in arterial disease and promote wound healing. Often these patients will need an imaging study such as a CT angio, MRA, or angiogram to determine the status of the vascular supply.
  • Venous leg elevation, Unna boots, or compression stockings to encourage venous drainage.

Retroperitoneal Bleeding
• Retroperitoneal bleeding is an urgent consult.
• Retroperitoneal bleeding is most commonly seen in patients after coronary angiography as a complication from arterial puncture of the femoral artery. The frequent use of anticoagulant and antiplatelet medications contributes to the incidence of retroperitoneal bleeding in this patient population. The hallmark for this diagnosis is a decreasing hematocrit in a patient complaining of flank, back, or abdominal pain.

  Pertinent information: What procedure was performed? What anticoagulants and antiplatelet agents were used, and have they been stopped? How much and how quickly has the hematocrit decreased? On the CT scan (go see it yourself; do not trust the radiologist), how large is it (in centimeters)? Does it compress the urinary system causing hydronephrosis or hydroureter, or compress the renal vein? These last two findings may necessitate urgent percutaneous nephrostomy tubes or surgery, respectively.

  Pertinent physical exam: Neurologic status of the patient’s ipsilateral leg? Test this by asking the patient to perform a straight leg lift and then test light touch on the medial and lateral upper thigh. If these senses are diminished, the patient may need urgent operative decompression of the hematoma.

• Diagnosis: Abdominal CT scan secures the diagnosis.

• Treatment options:
  • Variable treatment depending on situation.
  • Serial CBC and coags (PT/PTT/INR).
  • Reverse anticoagulation with vitamin K and FFP as needed. Careful coordination with cardiologists will be necessary if an intervention such as angioplasty or stenting has taken place.
  • Supportive measures (fluids, blood) and/or operative intervention.
Femoral Artery Pseudoaneurysm

- **Femoral artery pseudoaneurysm is an urgent consultation in most cases.**
- Pertinent information: This complication of arterial puncture occurs in the same patient population as retroperitoneal hemorrhages. What procedure was performed? What anticoagulants and antiplatelet agents were used, and have they been stopped? How large is the pseudoaneurysm by ultrasound and does it have a long, thin neck? These pseudoaneurysms are more likely to spontaneoulsy thrombose or be amenable to ultrasound-guided compression.
- Physical exam: The hallmark of diagnosis is a thrill or bruit over the puncture site. Is there any evidence of emboli to the ipsilateral foot? Look at the tips of the toes and search for petechiae or new larger purple or black spots. If present, the patient may need immediate operative intervention. Is there any evidence of compression of the femoral nerve? Test motor function and light touch sensation in the leg. If absent, the patient may need immediate operative intervention.
- Diagnosis: Ultrasound confirms the diagnosis.
- Treatment options vary from expectant management to ultrasonic compression, US-guided thrombin injection, or operative closure of the arteriotomy and evacuation of the hematoma.

Pneumothorax

- **Pneumothorax is usually an urgent consult; it is an emergent consult if the patient is receiving positive pressure ventilation or if there is suspicion of a tension pneumothorax.**
- Common symptoms include dyspnea and chest pain. Careful examination for signs of tension pneumothorax (deviation of the trachea to the opposite side, muffled or absent breath sounds on the affected side, and hypotension) must be performed. If there are no signs of tension pneumothorax, an upright chest X-ray should be obtained.
- Pertinent information:
  - What is the patient’s exam and how does he/she look?
  - Which side is affected?
  - Is there a CXR?
  - Is the patient on positive pressure ventilation?
  - Is this a tension pneumothorax?
• Treatment: Options vary as to the size and the physiologic impact of the pneumothorax:
  • If a tension pneumothorax is found and the patient is decompensating, immediately place a large-bore (14G or 16G) angiocatheter in the second interspace above the rib at the midclavicular line of the affected side. A chest tube tray and a 28 French chest tube should be brought to the bedside so that the consultant may place the tube as soon as they arrive.
  • If the patient is on positive pressure ventilation, the patient should be monitored closely until tube thoracostomy is performed. If the patient decompensates, immediately place a large (14G or 16G) angiocatheter in the second interspace at the midclavicular line of the affected side.
  • Expectant management with serial chest radiographs may suffice in a young, asymptomatic patient with a small pneumothorax who is not on positive pressure ventilation.
  • Most patients may require open tube thoracostomy or percutaneous tube thoracostomy (a 16 French Thal-Quick tube).

Pleural Effusion

• Pleural effusions are generally an elective or semi-urgent consult, unless the patient is short of breath, in which case an emergent consult is warranted.
• Common symptoms include dyspnea and chest pain. Effusions can result from a wide spectrum of benign, malignant, and inflammatory conditions.
• Pertinent information:
  • Is the patient short of breath and/or having trouble breathing? Depending on the size of the effusion and the patient’s pulmonary status, patients will differ in their symptomatology.
  • Are there any CXR, chest CT, or US studies that document the effusion?
  • Is it getting bigger?
  • Has a thoracentesis been done? If so, what did the Gram stain/culture, pH, glucose, amylase, lactate dehydrogenase, protein levels, cell count, and cytology show of the fluid?
• Treatment: Options vary depending on the etiology and the character of the pleural effusion.
  • Pleural effusions generally need to be drained via thoracentesis, open tube thoracostomy, or placement of an implantable
Approach to Consultation

long-term catheter that allows repeated drainage of recurrent pleural effusions.

- Fluid should be sent off for Gram stain and culture, cytology, cell count, and biochemical analyses (pH, glucose, amylase, LDH, protein) to help discriminate between an exudative and a transudative effusion.

- Additional options include streptokinase for loculated effusions or pleurodesis for recurrent effusions.

Perirectal Abscess

- Generally this is an urgent consult unless the patient is septic, then it is an emergency! Remember that there is no such thing as an unimportant abscess. It should always be evaluated by a surgeon.

- Pertinent information: Is the patient diabetic or immunosuppressed? If so, then they are far more likely to die or to have greater morbidity from this disease. Is the patient febrile, and do they have a leukocytosis?

- Physical exam:
  - Where is it (relative to the scrotum/vagina and anus)?
  - How far does the erythema and induration extend? If the stigmata of infection extend out from the anus, then the patient may have Fournier gangrene, which is a surgical emergency!
  - Has a CT scan been obtained?

- Treatment options:
  - Incision and drainage either at the bedside or in the operating room.
  - IV antibiotics.
  - Fournier’s gangrene will necessitate wide debridement in the operating room with massive irrigation of the affected areas.

PRESSURE ULCERS

- Generally these are elective or urgent consults unless the patient is septic.

- Pressure ulcers result from prolonged pressure to soft tissue over bony prominences. Most commonly they occur in immobile patients over the occiput, sacrum, greater trochanter, and heels.

- Pertinent information:
  - Is the patient septic?
  - Is the patient diabetic or immunosuppressed? Has the area been irradiated in the past? What is the patient’s nutritional status?
• Is the patient immobile? What is the extent and etiology of the patient’s immobility?
• What is the duration of the ulcer?
• What is their current wound care management?
• Physical exam: How does it look? Where is it? How deep is it? Is there any erythema, induration, or fluctuance around it? Do you see any exposed bone?
• Treatment options:
  • Most superficial pressure ulcers heal spontaneously when the pressure is relieved; however, this can be a lengthy process requiring over 6 months.
  • Local wound care and optimization of nutrition are key for ulcer healing. Urinary and fecal continence needs to be maintained to prevent maceration and skin breakdown.
  • Simple closure, split-thickness skin grafting, or musculocutaneous flaps are possible, but often not successful unless the pressure can be removed.
KEYS TO INTENSIVE CARE UNIT SURVIVAL

• Transfer notes with key details of the patient’s past medical history and course are always helpful. The primary physician, receiving physician, and the patient’s family members should be notified. Note any details or special situations that need attention and/or follow-up. This should always be in addition to a conversation between physicians.

• Admitting a patient to an ICU can be intimidating, but keep in mind the ABCs (Airway, Breathing, Circulation) and focus on stabilizing the patient.

• Be nice to the nurses, respiratory therapists, and all other ancillary staff during your stay. They can often make very useful suggestions, catch things you miss, and be immensely helpful to you in critical situations. They can be the difference between an enjoyable and miserable experience!

• Ask (and keep asking) if you have questions or problems. Mistakes from inexperience in critically ill patients can have catastrophic consequences.

• Always treat the patient, not the numbers.

• It’s always a good idea to make rounds on patients and follow up on labs several times a day even if things seem stable.

• Daily ICU notes should include ventilator settings, I/Os, pulmonary artery catheter measurements, medications and drips (e.g., antibiotics, sedatives, vasopressors, inotropes), nutritional status, and documentation of all hardware (e.g., central venous catheters, arterial lines, endotracheal tube, feeding tubes).

• References to have nearby at all times:
Ventilators

Suggestions for Initial Ventilator Settings

**Basic Settings**
- **Mode:**
  - AC/VC: Volume control. Minimal minute ventilation is set, often best mode to start with. Tidal volume is set. Airway pressures are dependent on compliance.
  - SIMV + PSV (i.e., synchronous intermittent mandatory ventilation with pressure support ventilation): Similar to AC except patient-triggered breaths are delivered with pressure support instead of full tidal volume.
  - PCV: Pressure-controlled ventilation. Airway pressures are set and the tidal volume delivered depends on compliance.
- **Tidal volume:** 6 mL/kg IBW for lung protective ventilation in ARDS patients; otherwise start with 6 to 9 mL/kg.
- **Rate:** 10 to 15 breaths/min.
- **FiO₂:** 1.00, then titrate down (goal to get FiO₂ to ≤0.60 as quickly as possible).
- **PEEP:** 5 cm H₂O (monitor for auto-PEEP, especially with obstructive lung disease).

**Advanced Settings**
- Ask for help before you change these.
- **Inspiratory flow:** 50 to 60 L/min. With COPD may need >100 L/min.
- **I:E ratio:** 1:2–1:3. This must be set with PCV.
- **Peak and plateau pressures:** Goal plateau pressure <32 cm H₂O; prefer peak pressure <45 cm H₂O.

**Ventilator Adjustments**
- PO₂ of 60 mm Hg or greater is generally sufficient. Oxygenation is most affected by mean air pressure. Adjustments to FiO₂ and
PEEP can help increase oxygenation. Remember that the oxygen saturations and $\text{PO}_2$ can be discordant, need to verify that they are consistent.

- $\text{PCO}_2$ is regulated by minute ventilation. Increasing the respiratory rate or tidal volume increases minute ventilation and decreases $\text{PCO}_2$.
- See Table 22-1 for suggested adjustments.

### Worsening Oxygenation

The knee-jerk impulse is to turn up the $\text{FiO}_2$. **Don’t panic!** Approach the problem in a stepwise manner:

- Is there a ventilator problem?
- Is the ET tube in the correct position or has it migrated? Check the positioning (e.g., 25 cm at the lip) of the ET tube and look at the most recent chest radiograph.
- Is there a cuff leak or kink in the ET tube? The respiratory therapist can help evaluate.
- Recheck the ventilator settings. Have there been inadvertent changes?
- Is there an obstruction in the ET tube (e.g., mucous plug)? Pass the suction catheter to evaluate.
- Is it a patient-related problem (e.g., biting, agitation)? Sedation may be needed.
- Always consider pneumothorax. Listen on both sides and consider a CXR.
- Is the underlying problem worsening? Is the patient fluid overloaded or is there bronchospasm? Is PE a possibility? Has ventilator-associated pneumonia developed?
- Is the patient oversedated? Do you need to rethink the mode of ventilation? Check the PEEP at end expiration. Hypoxemia can result from a loss of PEEP.

### TABLE 22-1

<table>
<thead>
<tr>
<th>$\text{PCO}_2$</th>
<th>$\text{PO}_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>High</td>
<td>$\downarrow$Tidal volume $\downarrow$Respiratory rate</td>
</tr>
<tr>
<td>Low</td>
<td>$\downarrow$Tidal volume $\downarrow$Respiratory rate</td>
</tr>
</tbody>
</table>

*aVery general recommendations; must be individualized for each patient.*
Weaning Parameters

The method of weaning is not as relevant as knowing the appropriate time to wean. See Table 22-2.

ICU SEDATION/PARALYSIS

- Ventilated patients generally require sedation. This is usually achieved through continuous IV infusion of sedatives. See Table 22-3.
- Achieve the desired level of sedation with boluses before starting a continuous infusion. Specify the desired level of sedation (Table 22-4). If the patient becomes agitated, rebolus to desired level of sedation and then make small incremental changes in the drip rate.
- Titrate to minimum effective dose and **reassess the need for continuous sedation daily**.
- Consider adding paralytics (Table 22-5) for patients with very poor oxygenation or if patient–ventilator desynchronization persists despite adequate sedation causing difficulty with ventilation. **Ensure the patient is completely sedated before adding paralytics.**

### TABLE 22-2 GUIDELINES FOR ASSESSING WITHDRAWAL OF MECHANICAL VENTILATION

<table>
<thead>
<tr>
<th>Criterion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient’s mental status: awake, alert, cooperative</td>
</tr>
<tr>
<td>$P_{O_2} &gt; 60$ mm Hg with an $F_{I{O_2}} &lt; 0.5$</td>
</tr>
<tr>
<td>$P{E E P} \leq 5$ cm H$_2$O</td>
</tr>
<tr>
<td>$P{C O_2}$ and pH acceptable</td>
</tr>
<tr>
<td>Spontaneous tidal volume $&gt; 5$ mL/kg</td>
</tr>
<tr>
<td>Vital capacity $&gt; 10$ mL/kg</td>
</tr>
<tr>
<td>Minute ventilation $&lt; 10$ L/min</td>
</tr>
<tr>
<td>Maximum voluntary ventilation double of minute ventilation</td>
</tr>
<tr>
<td>Maximum negative inspiratory pressure $\geq 25$ cm H$_2$O</td>
</tr>
<tr>
<td>Respiratory rate $&lt; 30$ breaths/min</td>
</tr>
<tr>
<td>Static compliance $&gt; 30$ mL/cm H$_2$O</td>
</tr>
<tr>
<td>Rapid shallow breathing index $&lt; 100$ breaths/L(^a)</td>
</tr>
<tr>
<td>Stable vital signs following 30 min–1 h spontaneous breathing trial</td>
</tr>
</tbody>
</table>

\(^a\)Rapid shallow breathing index = Respiratory rate/tidal volume in liters.

TABLE 22-3  RICHMOND AGITATION AND SEDATION SCALE

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>+4</td>
<td>Overtly combative, violent, immediate danger to staff</td>
</tr>
<tr>
<td>+3</td>
<td>Pulls or removes tubes or catheters; aggressive</td>
</tr>
<tr>
<td>+2</td>
<td>Frequent non-purposeful movement, fights ventilator</td>
</tr>
<tr>
<td>+1</td>
<td>Anxious but movements not aggressive or vigorous</td>
</tr>
<tr>
<td>0</td>
<td>Alert and calm</td>
</tr>
<tr>
<td>−1</td>
<td>Not fully alert, but has sustained awakening (eye opening/eye contact) to voice ≥10 s</td>
</tr>
<tr>
<td>−2</td>
<td>Briefly awakens with &lt;10 s eye contact to voice</td>
</tr>
<tr>
<td>−3</td>
<td>Movement or eye opening to voice (no eye contact)</td>
</tr>
<tr>
<td>−4</td>
<td>No response to voice, movement or eye opening to physical stimulation</td>
</tr>
<tr>
<td>−5</td>
<td>No response to voice or physical stimulation</td>
</tr>
</tbody>
</table>


- The degree of paralysis is usually monitored by peripheral nerve stimulation and the train-of-four method. Complete paralysis is unnecessary for many patients.
- **Paralysis should be discontinued daily to determine the continuing need for paralysis and to assess for adequate sedation.**

**CARDIAC PARAMETERS**

- Normal cardiac output: 5 ± 1 L/min
- Normal cardiac index: 3 ± 0.54 min/m²
- Normal filling pressures:
  - Right atrial pressure: 0–8 mm Hg
  - Right ventricular pressure: 5–30/0–8 mm Hg
  - Pulmonary artery pressure: 15–30/3–12 mm Hg
  - Pulmonary wedge pressure: 3–12 mm Hg
<table>
<thead>
<tr>
<th>Drug</th>
<th>Bolus Dosing</th>
<th>Onset (Single Dose)</th>
<th>Duration (Single Dose)</th>
<th>Continuous Dilution</th>
<th>Maintenance Dose Range</th>
<th>Titration Increment</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fentanyl</td>
<td>25–100 μg, max 300 μg in 15 min</td>
<td>1–2 min, peak 2–5 min</td>
<td>30–60 min</td>
<td>2500 μg/50 mL</td>
<td>50–200 μg/h</td>
<td>50 μg/h</td>
<td>Possible bradycardia with bolus doses; prolonged effect in renal and hepatic failure</td>
</tr>
<tr>
<td>Morphine</td>
<td>10–15 mg</td>
<td>5–10 min, peak 20 min</td>
<td>3–4 h</td>
<td>100 mg/100 mL</td>
<td>1–50 mg/h</td>
<td>2–5 mg/h</td>
<td>Possible hypotension due to histamine release; prolonged effect in renal and hepatic failure</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>2–4 mg</td>
<td>20–40 min</td>
<td>3–6 h</td>
<td>40 mg/40 mL</td>
<td>0.5–4 mg/h</td>
<td>0.25 mg/h</td>
<td>Prolonged effect in renal and hepatic failure; associated with acute tubular necrosis, lactic acidosis, and hyper-osmolar states with prolonged infusion</td>
</tr>
<tr>
<td>Drug</td>
<td>Dose/Rate Information</td>
<td>Administration</td>
<td>Dilution</td>
<td>Rate</td>
<td>1 mg/h</td>
<td>Duration</td>
<td>10% Lipid kcal/mL</td>
</tr>
<tr>
<td>--------------</td>
<td>------------------------------------------------------------</td>
<td>----------------</td>
<td>----------</td>
<td>------</td>
<td>--------</td>
<td>----------</td>
<td>--------------------</td>
</tr>
<tr>
<td>Midazolam</td>
<td>1–5 mg, max 15 mg in 15 min</td>
<td>1–4 min</td>
<td>30–60 min</td>
<td>50 mg/50 mL</td>
<td>1–8 mg/h</td>
<td>1 mg/h</td>
<td>Possible hypotension with bolus doses; prolonged effect in renal and hepatic failure</td>
</tr>
<tr>
<td>Propofol</td>
<td>Not recommended</td>
<td>1–2 min</td>
<td>30 min</td>
<td>1000 mg/100 mL</td>
<td>25–50 μg/kg/min</td>
<td>10 μg/kg/min</td>
<td>10% lipid = 1.1 kcal/mL; possible hypotension, bradycardia, hypertriglyceridemia, pancreatitis, and propofol-related infusion syndrome</td>
</tr>
<tr>
<td>Dexmedetomidine</td>
<td>Not recommended</td>
<td>10 min</td>
<td>30 min</td>
<td>400 μg/100 mL</td>
<td>0.2–0.7 μg/kg/h</td>
<td>0.1 μg/kg/h</td>
<td>Possible hypotension, bradycardia; doses up to 1.5 μg/kg/h have been safely used for up to 30 days</td>
</tr>
</tbody>
</table>

Adapted from Casabar E, Portell J, eds. Tool Book: Drug Dosing and Usage Guideline. 10th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital, Washington University Medical Center; 2012.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Bolus Dosing (mg/kg)</th>
<th>Onset (Single Dose, min)</th>
<th>Duration (Single Dose, min)</th>
<th>Continuous Dilution</th>
<th>Maintenance Dose Range (μg/kg/min)</th>
<th>Titration Increment (μg/kg/min)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pancuronium</td>
<td>0.05–0.1</td>
<td>2–4</td>
<td>2–4</td>
<td>50 mg/50 mL</td>
<td>0.5–1.5</td>
<td>0.25</td>
<td>May cause mild tachycardia; effects prolonged in renal and hepatic failure</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>0.05–0.1</td>
<td>2–4</td>
<td>35–45</td>
<td>50 mg/100 mL</td>
<td>0.5–1.5</td>
<td>0.25</td>
<td>Effects prolonged in renal and hepatic failure</td>
</tr>
<tr>
<td>Atracurium</td>
<td>0.3–0.5</td>
<td>2–3</td>
<td>25–35</td>
<td>500 mg/100 mL</td>
<td>5–25</td>
<td>5</td>
<td>Reserve for patients with renal or hepatic in whom train of four cannot be obtained; may cause histamine release; dose may escalate over time</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>0.6–1</td>
<td>1–2</td>
<td>30</td>
<td>200 mg/200 mL</td>
<td>8–12</td>
<td>0.8–1.2</td>
<td>Effects prolonged in renal and hepatic failure</td>
</tr>
<tr>
<td>Cisatracurium</td>
<td>0.1–0.2</td>
<td>2–3</td>
<td>45–60</td>
<td>200 mg/100 mL</td>
<td>2–10</td>
<td>2</td>
<td>Reserve for patients with renal or hepatic in whom train of four cannot be obtained and histamine release would not be tolerated</td>
</tr>
</tbody>
</table>

Adapted from Casabar E, Portell J, eds. Tool Book: Drug Dosing and Usage Guideline. 10th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital, Washington University Medical Center; 2012.
TABLE 22-6  HEMODYNAMIC PROFILES ASSOCIATED WITH SHOCK

<table>
<thead>
<tr>
<th>Type</th>
<th>CVP</th>
<th>CI/CO</th>
<th>SVR</th>
<th>SvO₂</th>
<th>PCWP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic (e.g., hemorrhagic)</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Cardiogenic (e.g., MI, CHF, tamponade)</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Distributive (e.g., septic)</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
<td>N–</td>
<td>N–</td>
</tr>
</tbody>
</table>

CVP, central venous pressure; CI, cardiac index; CO, cardiac output; SVR, systemic vascular resistance; SvO₂, mixed venous oxygen saturation; PCWP, pulmonary capillary wedge pressure; N, normal.

SHOCK

Hemodynamic Profiles Associated with Shock
The hemodynamic parameters associated with the major forms of shock are presented in Table 22-6.

Treatment of Shock

- Determine the type of shock you are dealing with.
- **Fluid resuscitation** is vital, especially for hypovolemic shock. Crystalloid (normal saline or lactated Ringer’s) should be started immediately. For hemorrhagic shock, blood products should be administered.
- Use of **vasopressors and/or inotropes** may be necessary. Vasoactive agents are generally titrated to a mean arterial pressure of ≥60 to 65 mmHg. Afterload reduction may be helpful in cardiogenic shock. See Table 22-7 for dosages.

DRIPS

Other intravenous drips commonly used in the ICU are presented in Table 22-7.
<table>
<thead>
<tr>
<th>Table 22-7: Commonly Used ICU Drips</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vasopressors</strong></td>
</tr>
<tr>
<td>-------------------------------------</td>
</tr>
<tr>
<td>Dopamine</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Epinephrine</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Norepinephrine</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Vasopressin</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Drug</td>
</tr>
<tr>
<td>-------------</td>
</tr>
</tbody>
</table>
| Milrinone   | PDE III inhibitor resulting in reduced degradation of cAMP resulting in increased calcium influx | Loading dose: 50 μg/kg over 10 min  
Maintenance dose: 0.25–0.75 μg/kg/min | Inotrope, direct peripheral vasodilator  
Decrease dose in renal dysfunction |
| Nitroglycerine | Converted to NO that activates guanylyl cyclase resulting in cGMP production that causes vascular smooth muscle relaxation | Start at: 5–10 μg/min  
Titrate: 10–20 μg/min every 5 min until desired effect | Tachyphylaxis can occur  
At high doses, reflex tachycardia can occur |
| Nitroprusside | Same as nitroglycerine                                                  | Start at: 0.25 μg/kg/min  
Titrate: to desired effect  
Usual max dose: 10 μg/kg/min | Signs of toxicity include metabolic acidosis, tremors, seizures, and coma  
Check sodium thiocyanate levels with prolonged use  
Do not use in renal failure |

Adapted from Casabar E, Portell J, eds. Tool Book: Drug Dosing and Usage Guideline. 10th ed. St. Louis, MO: Department of Pharmacy, Barnes-Jewish Hospital, Washington University Medical Center; 2012.
SUGGESTIONS FOR PROPHYLAXIS

• DVT: see DVT Prophylaxis section in Chapter 10.
• GI: see GI Prophylaxis section in Chapter 10.
• Decubitus ulcers: turning patient several times a day, vigilant skin care, egg crate mattress, flotation bed, and ensuring adequate nutrition.
• Deconditioning: physical therapy and nutritional support.
• Aspiration precautions: elevate head of the bed >30° and adequate suctioning.
• Seizure or fall precautions: as appropriate.
• Infection: maintain oral hygiene, keep track of all lines (peripheral IV, central line, NG tube, feeding tubes, Foley catheters), and remove as soon as no longer needed. Target or discontinue antibiotic therapy per guidelines to reduce induction of resistance and development of Clostridium difficile colitis.
• Follow isolation (respiratory or contact) precautions at all times and practice scrupulous hand hygiene.

TOTAL PARENTERAL NUTRITION

• Consider this option if the GI tract is unusable for at least 7 to 10 days. Sterile vascular access is needed.
• Administered through the distal port of a central venous catheter. Reserve this port if you think initiating total parenteral nutrition (TPN) is a possibility—TPN cannot be given through a port that has already been used.
• For initial orders and questions, a nutritional support consult will be valuable for information, advice, other options, and help with calculating projected nutritional needs.
• TPN orders must be written daily and received by a certain time— make sure this is done before signing out.
• Monitor vital signs, daily weight, I/Os, Accu-Cheks, and routine labs (CBC, electrolytes, BUN, Mg, phos) frequently. Monitor triglycerides and hepatic function at least once a week.
• May add H2 blockers, steroids, insulin, and vitamin K to TPN if so desired.
• If TPN must be stopped, monitor blood sugar and administer IV fluids (e.g., D10) at the same rate.
**VASCULAR ACCESS**

**Ultrasound-Guided Central Venous Access**

- The use of ultrasound guidance for the placement of central venous catheters is superior to the landmark-guided technique due to an improvement in average access time, reducing the number of attempts, and minimizing rates of complications.
- Ultrasound guidance should be the method of choice for venous catheterization, especially in select populations (e.g., obese, critically ill, or history of multiple prior central venous catheters).
- **Indirect guidance** refers to assessing the vascular structures using 2D ultrasound prior to performing needle puncture and venous canalization.
- **Direct guidance** refers to the use of real-time ultrasound images during the needle puncture. The view can be either transverse (a cross section of the vein) or longitudinal (visualizing the vein on its long access). The transverse technique, which has been shown to be easier to learn by inexperienced physicians, will be described here.
- It is possible to distinguish veins from arteries using ultrasound.
  - Veins, in contrast to arteries, are more easily compressible with application of anterior–posterior pressure with the ultrasound probe. Central veins tend to be larger and less circular than adjacent arteries, but this can be misleading (e.g., patients with low intravascular volume).
  - The relationship of the vein to the artery can also be useful: The internal jugular is typically anterolateral to the carotid artery. The femoral vein is typically medial to the femoral artery.
  - Doppler ultrasonography with color flow can help identify arteries on the basis of pulsatile flow, but this can be misleading, as well (e.g., patients with severe tricuspid regurgitation).
- See Table 23-1 for all you need to know about vascular access.
### TABLE 23-1  
ALL YOU REALLY NEED TO KNOW ABOUT VASCULAR ACCESS

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
<th>Common Uses</th>
<th>Duration of Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triple-lumen catheter</td>
<td>Three separate lumens. Placed via the Seldinger technique usually at the bedside. Subclavian or internal jugular veins preferred, femoral vein can be used.</td>
<td>When peripheral access is exhausted and in emergency situations. Blood may be drawn from the catheter.</td>
<td>Short-term use (~7 days). Replace femoral lines more frequently (~3 days).</td>
</tr>
<tr>
<td>Hickman catheter</td>
<td>Surgically placed (single or multi-lumen). Subcutaneously tunneled. Dacron cuff at the skin entry site. Located in subclavian vein (tip is located near the right atrium).</td>
<td>Long-term intravenous medications and/or fluids. Blood may be drawn from the catheter.</td>
<td>Long-term use. May be left in place indefinitely as long as functioning properly.</td>
</tr>
<tr>
<td>Hohn catheter</td>
<td>Single- or double-lumen Silastic catheters. Placed via Seldinger technique by interventional radiology or surgery without a subcutaneous tunnel.</td>
<td>Placed when peripheral access is exhausted. Administration of medications and fluids (when double-lumen). Blood may be drawn from the catheter.</td>
<td>Intermediate-term use (up to 6 weeks)</td>
</tr>
<tr>
<td>Device Type</td>
<td>Description</td>
<td>Uses</td>
<td>Notes</td>
</tr>
<tr>
<td>-----------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------</td>
</tr>
<tr>
<td>Implanted venous access device</td>
<td>Antimicrobial cuff at the skin insertion site. Located in subclavian or internal jugular veins. Placed subcutaneously by a surgeon or interventional radiology. Single- or double-lumen. Specialized right-angle needle is required to access the portal chamber. Located in subclavian vein; tip is located near the right atrium.</td>
<td>Long-term intravenous medications and/or fluids, especially chemotherapy. Blood may be drawn from the catheter.</td>
<td>Intended for indefinite use.</td>
</tr>
<tr>
<td>Tunneled cuffed catheters (Ash, DuraFlow, Tesio, etc.)</td>
<td>Dual-lumen Silastic catheters. Placed by interventional radiology. Can be placed in the internal jugular vein or subclavian vein.</td>
<td>Used for hemodialysis. Do not use catheter for any other reason without checking with nephrologist.</td>
<td>Intermediate-term use to allow graft or fistula maturation, if patients refuse permanent access, or if graft or fistula is contraindicated.</td>
</tr>
<tr>
<td>Midline catheter</td>
<td>Kink proof material. Peripherally placed by trained nursing personnel.</td>
<td>Intermediate-term intravenous medications are planned (e.g., a several week course of antibiotics).</td>
<td>Intermediate-term use (1–6 weeks).</td>
</tr>
</tbody>
</table>

(Continued)
<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
<th>Common Uses</th>
<th>Duration of Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>PICC</td>
<td>Located in antecubital vein. Consider the possibility of midline catheter placement early before potential peripheral vessels are damaged.</td>
<td>Not intended for TPN or chemotherapy. Blood drawing is discouraged (causes fibrin deposition at the tip and eventual catheter failure). Long-term intravenous medications are planned. TPN and more irritating medications provided the tip is in the superior vena cava. Blood may be drawn from the catheter.</td>
<td>Heparin flushing should be performed when the catheter is not being used for therapy at least twice a day. Long-term use. May be left in place indefinitely as long as functioning properly.</td>
</tr>
<tr>
<td></td>
<td>17-inch Silastic catheter placed by trained nursing personnel. Interventional radiology can place PICC lines under fluoroscopy if necessary. Located in basilic, cephalic, or median cubital vein.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Complications from catheter placement may include bleeding, pneumothorax, infection, and thrombosis. Some clotted catheters may be opened with alteplase (Cathflo Activase). The various catheters require that specified amounts of the appropriately diluted solution be instilled to just fill the lumen, ensuring that the thrombolytic is not injected systemically. Your hospital will likely have specified procedures for declotting catheters, so do not attempt without knowing your local policy. Also, it may be advisable to check with the service that actually put the catheter in.
Equipment

- This is in addition to what is typically needed for central venous access
- A real-time 2D ultrasound machine with transducer: Make sure it is fully charged!
- Sterile plastic transducer sheath
- Sterile and nonsterile ultrasound gel (sterile gel is often included with the plastic transducer sheath kit)
- A nonsterile assistant

Procedure

1. First and foremost, track down and obtain a fully charged ultrasound machine.

2. It is usually helpful to visualize the vascular structures at your access site prior to sterilization of the procedure site. This can be done with nonsterile ultrasound gel or surgical lubricant.
   - Note the depth and caliber of the vein.
   - Evaluate for vein patency and compressibility.
   - Identify adjacent structures. Remember, the vein is typically anterolateral to the artery for the IJ and medial to the artery for the femoral.
   - Look for an alternate site if multiple collateral vessels without a single large lumen or if a central thrombus is visualized.

3. Cleanse and drape the patient as you would for sterile procedures.

4. Apply sterile ultrasound gel to the interior of the plastic transducer sheath (alternatively, your nonsterile assistant may apply nonsterile gel to the ultrasound transducer).

5. With the aid of your nonsterile assistant, carefully lower the ultrasound transducer into the opening of the plastic sheath (ensure that the transducer does not contact the outer surface of the sheath). The sheath should be pulled by the assistant to cover the length of the transducer cord that may contact the sterile field.

6. Place sterile ultrasound gel on the patient at the selected access site.

7. Once again locate the vein at the selected entry site. Rotate the ultrasound probe to obtain the transverse view (perpendicular to the course of the vein). As for the landmark technique, you should err initially on aiming your needle away from the artery (e.g., laterally or toward the ipsilateral nipple for IJ access, and
medially for femoral venous access). Before beginning, align your ultrasound view perpendicular to your intended needle path.

8. After anesthetizing the area, insert your introducer needle from the central venous access kit at a 45° angle to the skin at a distance away from the transducer that is approximately equal to the depth of the vein (as previously measured [see Figure 23-1]).

When starting out, it is best to have a sterile assistant hold the ultrasound transducer for you to free both hands for the line placement.

9. Once venous return is obtained, the ultrasound probe may be left to the side on the sterile field.

10. The other procedural aspects of central line placement are covered in detail on the accompanying procedure card.

OTHER PROCEDURES

All other procedures (i.e., arterial line placement, peripheral IV, thoracentesis, paracentesis, arthrocentesis, and lumbar puncture) are described in detail on the accompanying procedure card.

GUIDELINES FOR OCCUPATIONAL EXPOSURES

- If an exposure to blood or other bodily fluids occurs:
  - Stop what you are doing immediately! Take a deep breath; don’t panic.

![Figure 23-1. Needle insertion site using ultrasound guidance.](image-url)
• Cleanse wound with soap and water. For mucous membrane exposures, rinse with copious amounts of water.

• Call the hospital’s exposure hotline to report the exposure and get further instructions. Each hospital has its own procedures on handling occupational exposures, you likely heard about them in your orientation. Be sure to follow them. In reporting an incident, you will need the following information:
  - Date and time of exposure.
  - Details of procedure being performed, amount of fluid or material exposed to, severity of exposure, type of needle used (e.g., hollow bore).
  - Details of exposure source—e.g., known HIV, HBV, HCV positive? If source has known HIV, obtain the names and dosages of medications the source is taking.
  - You and the source patient will need to be evaluated for HIV, hepatitis B, and hepatitis C. Follow instructions from employee/occupational health for testing and follow-up.

• The risk of transmission of a bloodborne pathogen depends on the pathogen involved, the type of exposure, amount of blood involved in the exposure, and amount of virus in the patient’s blood at the time of exposure.

• If you have been exposed, you should avoid exchange of bodily fluids with other persons until follow-up is complete, including using condoms with sexual partners until the results of the HIV test from the source patient are known.

• For more information on postexposure risk and therapies:
  - National Clinicians’ Postexposure Hotline 1-888-448-4911 or http://www.nccc.ucsf.edu/about_nccc/pepline/ (last accessed July 30, 2012).
1. When in doubt, ask and ask again. Call someone (wake up someone if you need to), preferably someone who knows more than you do.
2. Stay organized and prioritize your tasks.
3. Real patient care first, documentation later.
4. When in doubt, it’s always better (albeit more painful) to go see the patient.
5. The right thing to do usually involves more time expenditure.
6. Never but never make it up! If you don’t know, you’d better say so. Even stretching the truth is dangerous.
7. Nurses are almost always right. If they are wrong, be selective about pointing this out.
8. Walk if you don’t need to run. Sit down if you don’t need to stand. Lie down if there’s a bed nearby. Answer all of nature’s calls. There is almost always time to refuel (i.e., eat).
9. Take primary responsibility for your patients—you are their doctor.
10. Listen to your patients. They’ll usually tell you what you need to know.
11. Resist the temptation to discuss patient care in public areas; no good can come of it.
12. A healthy amount of compassion and compulsion makes it difficult to harm patients.
13. See one, do one, teach one. You’ll be expected to assume more teaching responsibilities as time goes on. Start developing your own teaching style and discuss expectations clearly with all learners.
14. Choose your battles carefully! Even in the name of patient care, ugly behavior is an ugly thing. You will be remembered for violations—don’t get a reputation.
15. Help out your colleagues. If you finish your work early, check with other members of your team or the cross-covering intern.
to see if they need anything; they can return the favor when you need it most.

16. Before going home for the day, make sure your patients are tucked in and check out with your resident. A complete sign-out is vital—make sure to include any information (studies, consults, procedures) that may be needed to make major therapeutic decisions.

17. Worthy goals for internship include learning to distinguish the life-threatening issues from the acute ones from the stable ones; mastering the interpretation and proper usage of diagnostic tests; learning procedural skills; refining the ability to ask specific questions for every consult you request.

18. Fear and anxiety are normal. Take a deep breath and plunge in—there are people around to help you. If you are feeling overwhelmed by fear, anxiety, or other emotions, seek help—don’t be a hero!

19. There is no magic spell on the last day of internship that will turn you into a resident. Trust that if you do and learn the right things during internship, you will be prepared to rise to the challenges of residency.

20. Residency, too, shall pass.
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