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“Those who sow in tears will reap with songs of joy.”

--Psalm 126:5
Preface

The sixth edition of “Yale-G First Aid: Crush USMLE Step 2 CK & Step 3” has been significantly updated from the previous editions with the author’s persistent efforts. It is mainly based on

www.usmle.org*, www.uptodate.com, www.uworld.com, “Current Medical Diagnosis and Treatment,” Kaplan Medical books, and a large volume of supportive feedback from medical students and doctors in the US and around the world. According to such feedback, the book has collected the most up-to-date, comprehensive, and high-yield clinical knowledge for the USMLE Step 2 CK and Step 3 available in the USMLE market. This book can be the equivalent of “First Aid for the USMLE Step 1” for the USMLE Step 2 CK. **Important features of this book include:**

1. **Systematic, comprehensive, and concise reviews of all common and uncommon diseases and disorders that are frequently tested in the USMLE Step 2 CK and Step 3.** They are composed of important concepts, etiologies, essentials of disease diagnoses, differential diagnoses, treatments, and preventive measures.

2. Well-organized contents in easy-to-remember formats, abbreviations, and 100 tables and figures with summaries and differential diagnoses, and with necessary details of high-yield “orderly steps” in the diagnosis and management of major diseases. **Most important clinical points are in “PEARLS”, underlined, and bolded as priority contents and are more likely to be seen in the USMLE.**

3. **More than 700 fundamental high-yield clinical questions in USMLE format.**

4. **130 selected high-yield clinical images with brief diagnostic features** (in back of book to reduce printing cost) for deeper digestion and memory of the associated fundamental clinical knowledge.

After thorough digestion of this book followed by practice with the best Q-bank on uworld.com with note taking, you are ready to crush the USMLE Step 2 CK and Step 3 with **high scores!**

**This book is your ticket to the USMLE and ECFMG Certification!** It can also be a special aid in medical school education in the US and around the world. The author’s team takes all feedback and constructive corrections positively with rewards and maintains monthly updates in the new Kindle version on Amazon.com to make it the best clinical review book for USMLE.

Best wishes to all who appreciate this book!

Yale Gong, MD

Washington, D.C.

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*www.usmle.org: “USMLE Step 3: Examinees are seeing increased numbers of items that assess an expanded range of competency-based content, including foundational science essential for effective healthcare; biostatistics, epidemiology, and population health; literature interpretation; medical ethics; and patient safety.”*
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# Communication Disorders

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Chapter 1

INFECTIONOUS DISEASES

Infectious diseases are among the most common disorders that we encounter in our daily lives with significant losses. Nevertheless, most of them are preventable and curable. While the main pathogens causing particular diseases have not changed much over time, the sensitivity pattern of these microorganisms to antibiotics has changed significantly in recent years. Therefore, pathologic culture and drug sensitivity testing (C/S) is still the best clinical guide to treatment of infectious diseases.

**PEARLS—HIGH-YIELD CLINICAL POINTS OF ANTIBIOTICS**

1. Antibiotics for Gram\(^+\) Cocci

   —Staphylococcus (Staph) and Streptococcus (Strep) are the most common organisms.

   1. Drugs of choice
      
      (1) Strep-pneumoniae; Strep-hemolytic group A, B, C, G; Strep-viridans: penicillin (PCN) G, V/K.
      
      (2) Staph, non-penicillinase-producing: PCN; penicillinase-producing Staph: semisynthetic PCN.
      
      (3) Enterococcus faecalis: Ampicillin +/- gentamicin.
      
      (4) Enterococcus faecium: Vancomycin +/- gentamicin.

2. Semisynthetic or penicillinase-resistant penicillins (PRPs):

   **Mechanisms:** Blocking peptidoglycan cross-linking and inhibiting transpeptidases.

   IV: Oxacillin, nafcillin, or methicillin (rare); oral (PO): cloxacillin, dicloxacillin, etc. They are highly effective against Staph- and Strep-, and thus usually used in bacterial skin infections (cellulitis, impetigo), meningitis, endocarditis, osteomyelitis, and septic arthritis. **Main adverse effects** include anaphylaxis (rare) and interstitial nephritis (methicillin #1).

   Methicillin is rarely used now due to its renal toxicity. Thus, “Methicillin-resistant S. aureus” (MRSA) actually refers to “oxacillin- or nafcillin-resistant S. aureus”, and the drug of choice is vancomycin. However, for sensitive organisms, oxacillin/nafcillin is preferred to vancomycin.

   **Beta-lactamase inhibitors — “CAST”:** Clavulanic acid, Avibactam, Sulbactam, Tazobactam.

3. PCN-G, PCN-V/K, ampicillin, and amoxicillin: Effective against Strep (S. pyogenes, S. viridans, and S. pneumonia) and \textit{Gram}\(^+\) bacteria, but not against S. aureus. **Ampicillin and amoxicillin are penicillinase sensitive** and only effective against S. aureus when combined with sulbactam or clavulanate; both are effective against Enterococci and Listeria.

4. Replacement for penicillin (PCN) allergy:

   (1) Replacement for mild PCN allergy:
Cephalosporins (Cepha-): See below.

(2) Replacement for severe PCN allergy (hypersensitivity):

Fluoroquinolones, clindamycin, and macrolides.

Fluoroquinolones (ciprofloxacin, levofloxacin, gatifloxacin, moxifloxacin, ofloxacin):

Mechanisms: Inhibit DNA topoisomerase II and IV → bactericidal.

The new fluoroquinolones (moxifloxacin, levofloxacin, and gatifloxacin) are strongly effective against Pneumococcus, Mycoplasma, Chlamydia, and Legionella. Thus they are the first-line empiric antibiotics for pneumonia. Most quinolones are active against the Enterobacteriaceae. Only ciprofloxacin covers Pseudomonas.

Adverse effects: Abnormal bone growth and cartilage in children and pregnant women (contraindicated); tendonitis, Achilles tendon rupture, and prolonged QT on ECG. Gatifloxacin is rarely used now due to potentially abnormal glucose metabolism.

Macrolides (erythromycin, clarithromycin, azithromycin): Inhibit translocation during protein synthesis → bacteriostatic. They are mainly used for atypical pneumonia (Mycoplasma, Chlamydia, Legionella), STDs (Chlamydia), B. pertussis, and some patients infected with Gram+ cocci but severely allergic to PCNs.

Note: For life-threatening S. aureus (MRSA) and Strep-B infection with severe PCN allergy, strongest antibiotics are vancomycin, synergicid, and linezolid. Macrolides are never used for severe S. aureus infections.

II. Antibiotics for Gram¬ Bacteria

1. Drugs of first choice for Gram¬ Cocci

Moraxella catarrhalis: Cefuroxime;
Gonococcus: Cefixime, ceftriaxone;
Meningococcus: PCN or cefotaxime.

2. Drugs of first choice for Gram¬ Rods

Pseudomonas-A: It’s an opportunistic pathogen that mostly affects immunosuppressed patients. Drugs of choice: piperacillin-tazobactam (with beta-lactamase inhibition), ceftazidime, or cefepime, or a carbapenem +/- gentamicin.

Acinetobacter: imipenem, meropenem;
Bacteroids (GI strains): Metronidazole;
Campylobacter jejuni: Erythromycin or azithromycin;
Enterobacter: Ertapenem, imipenem, cefepime;
E. coli: Uncomplicated—fluoroquinolones, nitrofurantoin; sepsis—cefotaxime, ceftriaxone;
Haemophilus: General infection—**TMP-SMX (co-trimoxazole, bactrim)**; central nervous system (CNS) or serious infection—cefotaxime, ceftriaxone;

Helicobacter pylori: Amoxicillin + clarithromycin + proton pump inhibitors (PPI);

Klebsiella: A 3rd-generation cephalosporin;

Legionella species (pneumonia): Azithromycin or fluoroquinolones +/- rifampin;

Proteus mirabilis: Ampicillin.

For most multidrug resistant infections (including P. aeruginosa), colistin (polymyxin E) is the only option despite its nephrotoxicity and ototoxicity. Cefepime plus amikacin can be the second.

3. **PCNs (piperacillin, ticarcillin, azlocillin, mezlocillin):**

   They are strong against the large Enterobacteriaceae group (E. coli, Proteus, Enterobacter, Citrobacter, Morganella, Serratia, and Klebsiella) and Pseudomonas; usually applied for hospital-acquired pneumonia, intra-abdominal infections (cholecystitis, cholangitis, pyelonephritis), bacteremia, neutropenia with fever, etc.

4. **Cephalosporins:** See below.

5. **Fluoroquinolones:** See above.

6. **Aminoglycosides** (gentamycin, tobramycin, amikacin) and **monobactams** (aztreonam):

   **Mechanisms:** Block transpeptidation at 30S → bactericidal. (1) Effective against the same Gram’ bacilli as listed above. (2) Synergistic with beta-lactam antibiotics for enterococci and staphylococci. (3) Ineffective against anaerobes. (4) **Kidney and ear toxicity.**

7. **Carbapenems** (imipenem, meropenem):

   These are **broad-spectrum antibiotics on gram’ cocci, gram’ rods, and anaerobes, strongly active against Enterobacteriaceae and Pseudomonas (EXCEPT ertapenem), plus S. aureus.** Main adverse effects (S/E) include GI distress, CNS toxicity, and nephrotoxicity, which is preventable by using with cilastatin. Significant adverse effects limit use to life-threatening infections.

III. **Cephalosporins**

   **Mechanisms of actions:** Block peptidoglycan cross-linking and inhibit transpeptidases.

   1st-generation **cephalosporins** (Cepha-): Cefazolin, cefadroxil, and cephalaxin. They have good activity against most Gram’ bacteria (including S. aureus) plus Proteus, E. coli, and Klebsiella.

   2nd-generation **cephalosporins**: Cefuroxime, cefprozil, and cephamycins (cefoxitin, cefotetan).

   They are less effective against S. aureus but more against Gram’ bacilli (H. influenza, Bacteroides, E. coli, P. mirabilis, and Klebsiella) and **beta-lactamase producing** M. catarrhalis and Enterobacteriaceae. **Cefotetan** is the drug of choice for infection with diabetic ulcerations, but not with Pseudomonas.

   3rd-generation **cephalosporins**: Ceftazidime, cefotaxime, and ceftriaxone. They are strong against most Gram’ bacteria including **beta-lactamase producing organisms**. They are the drugs of choice for
pneumonia or meningitis caused by PCN-insensitive Pneumococci or Enterobacteriaceae because they can cross the blood-brain barrier. Ceftazidime also covers Pseudomonas.

4th-generation cephalosporins: Cefepime, cefozopran, and cefclidine. They are stronger than the 2nd- and 3rd-generation cephalosporins against most Gram and some Gram+ bacteria. Cefepime also covers S. aureus, Strep, and Pseudomonas.

5th-generation cephalosporins: Ceftaroline is similar to ceftriaxone but with unique coverage of penicillin-resistant Gram+ bacteria (Strep and MRSA), not active for Pseudomonas and Enterobacteriaceae. Ceftobiprole is active for penicillin-resistant bacteria and Enterobacteriaceae.

Adverse effects (S/E) of cephalosporins: Much less hypersensitivity than with PCN; 5-10% cross allergy with PCN; occasional GI symptoms, autoimmune hemolytic anemia, vitamin (Vit) K deficiency, and disulfiram-like reactions.

IV. Antibiotics for Anaerobes

1. Metronidazole (Flagyl) is best for abdominal and genital anaerobes.

2. Clindamycin: Blocks transpeptidation at 50S subunit; bacteriostatic. It’s best for chest anaerobes and invasive Strep-A. S/E: C. difficile (pseudomembranous) colitis, fever, diarrhea, etc.

3. Other choices: Carbapenems, piperacillin + tazobactam, ticarcillin + clavulanate, ampicillin + sulbactam, and amoxicillin + clavulanate are strong medicines for anaerobes. The 2nd-generation cephalosporins (cefoxitin and cefotetan) are also effective.

V. Antibiotics for Encapsulated Bacteria

These bacteria are more aggressive, including Pneumococcus (Pneumococ), H. Influenza (Hib), Meningococcus (Meningococ), and Klebsiella. Choices of antibiotics:

1. New quinolones (Levo-, Moxi-, and Gati-) or the 3rd-generation cephalosporins are the best for Pneumococcus, Meningococcus, and Klebsiella.

2. Ampicillin is the best choice for H. Influenza (and Listeria). Other bacteria that ampicillin covers include E coli, Proteus, and Salmonella. Ampicillin is recommended for initial otitis media, preventive use for dental infection or endocarditis, UTI with pregnancy, and limited Lyme disease.

VI. Special Antibiotics

1. Beta-lactam antibiotics—Penicillins, cephalosporins, carbapenems and aztreonam: More effective than most others in the same class.


3. Vancomycin (a glycopeptide): Strongly bactericidal against most Gram positives (including Staphylococci, MRSA), anaerobes, diphtheroids, and clostridium species. It’s usually saved for patients
allergic to beta-lactam antibiotics, with MRSA or persistent anaerobic infection. Vancomycin is combined with an aminoglycoside for a complex infection with both Gram⁺ and Gram⁻ bacteria.

4. **Doxycycline**: Effective against Chlamydia, limited Lyme disease, Rickettsia, primary and secondary syphilis patients allergic to penicillins, Mycoplasma, Borrelia, and Ehrlichia. **Adverse effects**: tooth discoloration (before 8 years of age), Fanconi syndrome (Type II RTA), photosensitivity, and esophagitis/ulcer.

---

**Pneumonia**

It’s a common infection of the pulmonary tissue characterized by inflammation of the lung parenchyma and abnormal filling of alveoli with fluid (consolidation and exudation). **Pneumonia is a leading cause of death from an infectious disease for patients of all ages.**

**PEARLS**

By etiology, pneumonia can be classified as “Typical” (lobar or bacterial pneumonia, about 50%) and “Atypical” (interstitial pneumonia, caused by Mycoplasma, Chlamydia, Legionella, Rickettsia, viruses, or Pneumocystis, etc). Predisposing factors include smoking, diabetes, alcoholism, malnutrition, lung cancer, and immunosuppression, etc.

1. **S. Pneumococcus**: It’s the most common cause of acute community-acquired pneumonia (“lobar pneumonia”). Choose **Macrolides, new quinolones or 3rd-generation cephalosporins**.

2. **Gram' bacilli** (E. coli, Pseudomonas or Enterobacter): Mostly causing hospital-acquired or ventilator-associated pneumonia. Choose **3rd-generation cephalosporins** or and carbapenems.

3. **S. aureus**: Usually following viral infection or bronchitis, especially influenza. Choose **semisynthetic penicillins** (oxacillin, nafcillin, etc.).

4. **Hib** (often in smokers, COPD) and **Klebsiella** (often in alcoholics): Choose a **2nd or 3rd generation of cephalosporins**.

5. **Mycoplasma**: More common in young and healthy patients. Choose **Macrolides**.

6. **Legionella**: A Gram’ bacterium, epidemic infection in older smokers or with special environment such as infected water sources and air-conditioning systems. Choose **Macrolides**.

7. **Pneumocystis carinii** (causing PCP): Often seen in HIV (+) patients with CD4 cells < 200/uL and not on antibiotic prophylaxis. Choose **TMP-SMX**.

8. **Coxiella burnetii** (Q-fever): From exposure to animals, particularly at the time they are giving birth. Treatment of choice is doxycycline. The second option is erythromycin.

9. **Chlamydia psittaci**: From bird’s feces and upper respiratory secretions. Choose **Macrolides**.

10. **Viruses**: Influenza A or B, adenovirus, parainfluenza virus, RSV, etc. **Oseltamivir or zanamivir** is effective for both influenzas A and B.

**Essentials of diagnosis**
1. **History of high fever**, cough, chest pain, tachypnea, and dyspnea (if severe). **Typical, bacterial pneumonia** mostly produces **purulent sputum**; **atypical pneumonia** commonly generates a **nonproductive or “dry” cough**. P/E shows respiratory rate increase and pulmonary rales.

2. **Lab diagnosis:**
   
   (1) Leukocytosis with left shift (bandemia); neutrophil dominant for “typical pneumonia” and usually lymphocytosis for “atypical pneumonia”.
   
   (2) **CXR (Image 12)** is the most important **initial test** to reveal if it’s lobar (bacterial, typically showing lobar consolidation and air bronchograms) or interstitial (other pathogens) pneumonia. The CXR should be considered a sensitive test—if the findings are not suggestive of pneumonia, do not treat patient with antibiotics.
   
   (3) **Sputum Gram stain and culture** is the most specific test to diagnose and distinguish the “typical” and “atypical” pneumonia, and thus should be obtained in all patients.

3. **Special pathogens—Lobar pneumonia:**

   Significant purulent sputum indicates **Pneumococcus (“rusty”), Klebsiella (“currant jelly”),** or Hemophilus. P/E usually reveals rales, rhonchi, and signs of **lung consolidation**; tachypnea and dyspnea indicate the severity of pneumonia.

4. **Special pathogens—“Atypical pneumonia”:**

   (1) **Mycoplasma**: Mild nonproductive dry cough and chest pain. Serologic antibody titer is the specific diagnosis if necessary. Usually it’s adequate to make diagnosis on clinical basis and to treat as an outpatient.
   
   (2) **Legionella**: Nonproductive dry cough, CNS symptoms (confusion, headache, and lethargy) plus GI symptoms (diarrhea and abdominal pain). Specific diagnostic test—Urine antigen test is the initial rapid tool. Other specific tests (take longer time) include specialized culture with charcoal yeast extract and direct fluorescent antibody (Ab) titers. WBC count can be normal or high with left shift.
   
   (3) **Chlamydia-pneumoniae, Chlamydia-psittaci, Coxiella, and Coccidioidomycoses**: All of these are diagnosed with specific antibody titers.

**Treatment**

It depends on the pathogen and severity, inpatients or outpatients. Early empiric treatment is crucial since specific pathogens usually cannot be determined at clinical diagnosis.

1. **Community-acquired pneumonia**—Empiric treatment against “typical” bacteria and “atypical” pathogens:
   
   (1) **Outpatient**: **First choice—macrolides** (erythromycin) cover pneumococcus, mycoplasma, and chlamydia; azithromycin or clarithromycin also covers Hib. **Alternatives**—new fluoroquinolones (levofloxacin, moxifloxacin, or gatifloxacin) are also good options.
   
   (2) **Inpatient**: **New fluoroquinolones (Levo, Moxi, Gati)** or 2nd/3rd generation of cephalosporins (cefuroxime or ceftriaxone) combined with doxycycline or a macrolide or beta-lactam/beta-lactamase
combination drug (ampicillin + sulbactam; ticarcillin + clavulanate; piperacillin + tazobactam) combined
with doxycycline or a macrolide.

2. Hospital-acquired pneumonia: Patient has increased risk of drug-resistant Gram^\text{\textregistered} bacilli
infection if staying > 5 days in the hospital, > age 60, or with COPD, diabetes (DM), cardiovascular
diseases (CVD), or renal disease, etc. Give empiric treatment with 3\textsuperscript{rd}-generation cephalosporins
(ceftazidime or cefotaxime), carbapenems (imipenem), or beta-lactam/beta-lactamase inhibitor Combo
(e.g., piperacillin + tazobactam).

3. Supportive therapies: Oxygen (O\textsubscript{2}) treatment by degree of severity and hypoxia: Oxygen
supplement is required with arterial PO\textsubscript{2} < 70 mmHg, O\textsubscript{2} saturation < 94% at room air, or RR > 24/min.
IV steroids and other medicines in hospital may help patients with serious disease improve further.

Complications

1. Pleural effusion: It can occur in about 50% of patients and usually resolve with antibiotic
treatment of the pneumonia. Empyema is rare.

2. Acute respiratory failure: It may occur if the pneumonia is severe.

**Preventive pneumococcal vaccination (PCV)**

Two types of pneumococcal vaccines are approved for use in the US:

1. PPSV23 (Pneumovax 23): including 23 purified pneumococcal polysaccharide antigens, used in
adults; (2) PCV13 (pneumococcal protein-conjugate vaccine): used in infants and children.

1. All children should receive four doses of vaccines (PCV13)—at 2, 4, 6, and 12-15 months of age.

2. Additional vaccination by PPSV23 is recommended for people with increased risk of pneumonia:
(1) Age 19-64 with intermediate risk (smoking, chronic heart/lung disease, diabetes); (2) Patients with
serious underlying disease or immunodeficiency [long steroid use, asplenic state, with cancer, or HIV
(+)], PCV13 followed by PPSV23 two months later; (3) All \geq 65: PPSV23 alone if vaccinated 5
years ago, or PCV13 followed by PPSV23 6-12 months later.

A single dose of PPSV23 is enough to confer life-long immunity for most people > 65 y/a. The
efficacy of the vaccine is about 70%. Re-dosing in 5 years is only considered for those with severe
immunodeficiency.

------

**Acquired Immunodeficiency Syndrome (AIDS)**

AIDS is an acquired immune deficiency syndrome caused by the human immunodeficiency virus
(HIV). HIV is a retrovirus that particularly targets and destroys CD\textsubscript{4}^+ T-cells, with a subtype HIV-1
(more common globally) and HIV-2 (endemic in West Africa). HIV can be latent for many years and
replicates rapidly, progressively decreases the number of CD4 cells, destroys cell-mediated immunity, and
increases the risk of developing dangerous opportunistic infections. HIV does not harm patients directly.

**Causes and risk factors**
IV drug use/abuse (IVDA) and unprotected sexual intercourse carry the highest risk of developing AIDS. The risk is 1/100 for each receptive anal intercourse, 1/1000 for vaginal and oral receptive intercourse, and 1/3000-10000 for insertive vaginal intercourse. Other risk factors include blood transfusion, needle sticks (1/300 risk), maternal HIV infection (30% risk without medication), etc.

There is usually a 10-year lag between catching HIV and developing initial symptoms, the time for a normal CD4 level (>700/uL) to the sick level of 200/uL or lower with rapid viral replication.

**PEARLS — Monitoring of the immune system changes**

1. **CD4 T-cell count**: It indicates the degree of immunosuppression, and is the most accurate method of determination of what infections or other diseases the patient is risky for, when to start prophylaxis and treatment, and how to adjust them. Without treatment, the CD4 T-cell count drops 50-75 cells every year. CD4 > 700/uL is considered normal. **HIV (+) with CD4 < 200/uL or cervical cancer can be diagnosed as AIDS.**

2. **Viral load testing (RT-PCR RNA level)**: It is used to (1) diagnose HIV in babies; (2) guide antiretroviral therapies, measure response to therapy, and determine the rate of disease progression.

**PEARS — Table 1-8: Important CD4 Counts and Associated Complications**

<table>
<thead>
<tr>
<th>CD4 (count/uL) / Infections and complications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>500-700</strong>: Lymphadenopathy or recurrent <em>vaginal candidiasis</em>; no treatment of AIDS is indicated yet.</td>
</tr>
<tr>
<td><strong>200-500</strong>: Oral thrush or <em>vaginal candidiasis</em>, varicella zoster, herpes simplex, pneumococcal pneumonia, pulmonary TB, Kaposi sarcoma, anemia, lymphoma (non-Hodgkin’s), cervical intraepithelial neoplasia (CIN), histoplasmosis, or coccidioidomycosis.</td>
</tr>
<tr>
<td><strong>100-200</strong>: Pneumocystis carinii pneumonia (PCP) or AIDS dementia complex.</td>
</tr>
<tr>
<td><strong>&lt;100</strong>: No.1: Toxoplasmosis; No.2: cryptococcus.</td>
</tr>
<tr>
<td><strong>&lt;50</strong>: CMV, <em>Cryptosporidiosis</em>, Mycobacterium avium complex (MAC), progressive multifocal leukoencephalopathy (PML), or CNS lymphoma.</td>
</tr>
</tbody>
</table>

**Essentials of diagnosis**

1. **With the above risk factors for months to years**, followed by **recurrent viral or fungal infections**, ill-defined febrile illness, flu-like symptoms (fever, malaise, rash, lymph node swelling), night sweats, weight loss, and cachexia.

2. **Important lab tests**
   1. **HIV screening for risky people**—*Third generation EIA or ELISA* (enzyme immunoassay) tests detect the presence of HIV-1 or/and HIV-2 antibody as early as three weeks after exposure to the virus. If it’s positive, 1-2 times of Western blot (WB) testing is required for confirmation of HIV-1 or/and HIV-2 antibody (IgG antibody to HIV-1 1-2 months after infection). EIA/ELISA has a high sensitivity but moderate specificity for HIV, whereas WB has the highest specificity but moderate sensitivity. A negative EIA/ELISA cannot exclude HIV infection and requires the “fourth generation HIV tests”.
(2) Fourth generation HIV tests—The combination antigen-antibody immunoassay is better able to identify acute or early HIV-1/ HIV-2 infection (defined as a 6-month period following HIV acquisition), compared with antibody-only (EIA) tests, since they can detect HIV p24 antigen earlier than the antibody. If it is positive, an HIV-1/HIV-2 antibody differentiation immunoassay is performed for confirmation. If the combo-test is negative, the person is considered HIV-negative and no further testing is needed for most patients in whom acute or early HIV infection is unlikely. However, in patients with a negative combo-test but suspected of having acute or early HIV infection, the viral load testing should be performed.

(3) Viral load testing: A recent HIV load test is considered more sensitive and specificity than EIA/ELISA. Acute or early HIV infection is diagnosed by a negative immunoassay in the presence of a positive virologic test. However, a viral RNA level < 10,000 copies/mL in a patient with a negative serology may represent a false positive viral test result, as patients with acute or early HIV infection typically have very high levels of viremia. Then the HIV load test should be immediately repeated on a new blood specimen. A second positive viral load test suggests HIV infection, which can be confirmed by a repeat serologic test several weeks later.

If both the immunoassay and virologic test are negative, HIV infection can be mostly excluded.

(4) Histology: A type of giant multinucleate cells called Warthin–Finkeldey cells are commonly found in hyperplastic lymph nodes early in the course of HIV infection and measles.

**PEARLS**—Important evaluations recommended for HIV (+) persons

1. Detailed history and physical examination!
2. Routine chemistry and hematologic tests.
3. CD4 lymphocyte count and two plasma RNA tests for HIV load.
4. Screening for syphilis (VDRL/RPR) and PPD test. Syphilis (+) patients with risk factors for AIDS should take screening HIV-ELISA. If PPD is (+) (induration > 5 mm), patient is treated with INH for 9 months.
5. Anti-toxoplasma titer test.
6. Hepatitis tests: HAV and HBV serology tests; if (-), vaccination is given. If HAV or HBV antigen is already (+), vaccination is not needed. If both HBV and HCV tests are (+), only HAV vaccine is given.
7. Pneumococcal vaccination (unless CD4 < 200/uL): Offered to all HIV (+) people; boosted every 5 years.
8. Mini mental status exam (MMSE).
9. HIV counselling to possibly infected people.

**Differential diagnosis**

Mononucleosis (due to EBV or CMV), toxoplasmosis, measles, rubella, syphilis, viral hepatitis, disseminated gonococcal infection, and other viral infections.

**Treatment**

1. Drug resistance testing: For all patients with early HIV infection, drug resistance testing should be performed after the initial diagnosis has been established, regardless of whether treatment is being considered.
2. **Most patients**: Start antiviral treatment when CD₄ < 500/uL with symptoms; for asymptomatic patient, when CD₄ < 350/uL, viral load > 55 x 10⁴/uL, or opportunistic infection occurs. **Medical therapies** should include a combined nucleoside, non-nucleoside, and a protease inhibitor (Inh). The best initial combination is **Atripla** (emtricitabine-tenofovir-efavirenz) or alafenamide-emtricitabine-dolutegravir. **Goal**: Viral load < 400/uL.

**New anti-HIV medicines (mostly against HIV-1):**

(1) **Nucleoside reverse transcriptase inhibitors (NRTIs)**: Tenofovir (TDF), abacavir, lamivudine (3TC), emtricitabine, azidothymidine (AZT)/zidovudine (ZDV), didanosine (DDI), etc. AZT or ZDV can be used for general prophylaxis and during pregnancy to reduce risk of fetal transmission. **Adverse effects** include neuropathy, pancreatitis, anemia, diabetes insipidus, and bone marrow suppression, which can be reversed by G-CSF (granulocyte colony-stimulating factor) and erythropoietin.

(2) **Non-nucleoside reverse transcriptase inhibitors (NNRTIs)**: Efavirenz (EFV), delavirdine, etc. Adverse effects include CNS toxicity, rash, hyperlipidemia, and elevated hepatic transaminases.

(3) **Protease inhibitors (PIs)**: Lopinavir, darunavir, atazanavir, etc., typically administered with an NRTI combination, not alone. Adverse effects include hyperglycemia, GI, liver, and kidney toxicity.

(4) **Fusion inhibitor**: Enfuvirtide (T-20), not effective on HIV-2. **Mnemonic**: “EnFUVirtide inhibits Fusion.”

(5) **Chemokine coreceptor 5 (CCR5) antagonist**: Maraviroc (MVC).

(6) **Integrase inhibitors**: Raltegravir, elvitegravir and dolutegravir are new drugs with potent anti-HIV-2 activity. The main adverse effect is increased creatine kinase.

3. **Pregnant patients**

All children at birth will carry the maternal HIV antibody and have ELISA (+) testing, but only 25-30% will remain truly infected. Pregnant women with low CD₄ or high viral load should be treated fully for their HIV as above. Cesarean delivery for HIV (+) mothers is performed to prevent transmission of the virus if the CD₄ is < 350/uL or the viral load is > 1000/uL. Obtain best control of HIV with medications by the time of parturition.

4. **Post-exposure prophylaxis**

**Indications**: All persons with direct exposure to the blood or body fluids of HIV (+) patients.

**Preventive drugs**: zidovudine with and without other three combo-drugs for 4 weeks. Statistic data show that zidovudine alone can decrease the risk by 80%.

**Vaccinations**: All HIV (+) patients should take vaccines for Pneumococcus, Influenza, and HBV.
Chapter 1: Infectious Diseases

Chapter 1: High-yield Questions (HYQ)

1. A 10-year-old (y/o) girl presents with fever, intermittent abdominal pain, and bloody diarrhea for the past 10 days after a local camping trip. She feels weak and fatigued. Stool analysis reveals some WBCs and RBCs but no bacteria, ova, or parasites. Two stool cultures on enteric pathogens are negative. Physical examination (P/E) finds $T = 38.5^\circ C$ and a soft abdomen with mild RLQ tenderness. CBC results are normal (Nl). What’s the most likely diagnosis (Dx)?
   A. Traveler’s diarrhea  
   B. Cryptosporidiosis  
   C. Giardiasis  
   D. Crohn disease  
   E. Dysentery

2. A 55 y/o woman presents with malaise, fever, and nausea for the past 2 days. She has been on chemotherapy for breast cancer for the past 3 months. She denies headache, chill, cough, and bone pain. P/E finds $T = 38.7^\circ C$, HR = 90/min; other results are unremarkable. CBC reveals pancytopenia with WBC = 1,200/uL. Her CXR and urine analysis are normal. Blood and urine samples are taken for pathogen cultures. The best next step of treatment (Tx) is
   A. IV ceftazidime and vancomycin  
   B. IV cefepime and vancomycin  
   C. IV cefepime  
   D. IV gentamycin and vancomycin  
   E. IV piperacillin  
   F. IV amphotericin B

3. A 65 y/o man presents with an abrupt fever, headache, dry cough, diarrhea, and abdominal pain for the past 10 hours. He lives alone in an old apartment with unsanitary conditions. P/E results are normal except for $T = 38.5^\circ C$. CXR reveals bilateral infiltrates. WBC = 9,000/uL with left shift (increased ratio of immature neutrophils). There are no other abnormal findings. What’s the most likely cause?
   A. H. influenzae (Hib)  
   B. Mycoplasma  
   C. Legionella  
   D. Chlamydia  
   E. TB bacilli

4-5:  4. A 27 y/o woman comes to the hospital one week after a trip to the Northeast (U.S.), and presents with mild fever, headache, and an expanding rash on the right foot. P/E finds $T = 38^\circ C$, stable vital signs, and a 3-cm circular, erythematous, nontender rash with central clearing on the right foot. ECG reveals 1° A-V block. There are no other abnormal findings except for a 3-month pregnancy. What’s the most likely diagnosis?
   A. Cellulitis  
   B. Rocky Mountain spotted fever (RMSF)  
   C. Lyme disease  
   D. Tularemia  
   E. “Cat-scratch disease”

5. For the above patient (in Q4) who wishes to go home after an immediate therapy, the most appropriate antibiotic to use in hospital is
   A. oral azithromycin  
   B. oral doxycycline  
   C. oral amoxicillin  
   D. IV PCN-G  
   E. IV ceftriaxone
6-8: A 60 y/o woman is brought to the emergency room (ER) with fever, RUQ abdominal pain, nausea, and mild vomiting for the past 5 hours. P/E results: T = 39.5°C, HR = 90/min, marked RUQ tenderness on palpation and rebound tenderness. CBC reveals WBC = 18 x 10³/uL with predominant neutrophils and bands. Ultrasonography shows gallstones with dilated bile ducts and fluid. Her vital signs are stable. What’s the best initial treatment?

A. Oral quinolones  B. IV quinolones  C. IV ceftazidime
D. IV ampicillin  E. Support and observation in hospital

7. Continued with Q6: The patient still has persistent symptoms after 5 hours of the appropriate antibiotic treatment. What’s the best next step now?

A. Fluid and blood cultures  B. IV quinolones  C. IV cefepime
D. IV ampicillin  E. ERCP

8. Continued with Q6-7: For the above patient, the proper tests show mixed Gram’ bacteria and anaerobes. At this time, the most appropriate antibiotic to be included is

A. clindamycin  B. quinolones  C. gentamycin
D. ampicillin  E. metronidazole

9-10: A 68 y/o man is brought to the ER for high fever, headache, nausea, vomiting, and confusion. He had a small dental surgery 3 days ago. P/E results: Clouding of consciousness, T = 39.5°C, HR = 90/min, RR = 25/min, normal BP, neck stiffness; Kernig’s sign is suspicious. Sensation seems normal. Eye exam shows equal-sized, mildly dilated pupils and papiledema (by fundoscopy). Blood is taken for culture and sensitivity testing. What’s the most appropriate next step?

A. A head MRI scan  B. CSF assay with culture-sensitivity
C. A head CT scan  D. An empiric antibiotic  E. IV steroid

10. For the above patient, the most appropriate management has been made. Now the best medical treatment is intravenous administration of

A. large doses of PCN-G  B. ceftazidime + vancomycin  C. ceftriaxone + vancomycin
D. cefotaxime + ampicillin  E. ceftriaxone + ampicillin + vancomycin

11. A 70 y/o man is brought to the ER for fever, headache, and confusion for the past 2 days. He lives alone with poor living conditions. P/E results: Alert, T = 38°C, HR = 88/min, normal RR and BP, neck stiffness; Kernig’s sign is suspicious. Eye exam is normal. CBC: WBC = 15 x 10³/uL, with 50% of lymphocytes (LC). CSF: Opening pressure = 200 mmH₂O, LC = 60%, neutrophils = 40%, protein = 55 mg/dL, and glucose = 30 mg/dL. This patient most likely has

A. pneumococcal meningitis  B. viral meningitis  C. TB meningitis
D. TB encephalitis  E. viral encephalitis  F. fungal meningitis

12. A 16 y/o girl is brought to the ER for eye pain and blurred vision for the past 12 hours. She uses contact lenses daily and follows the sanitary procedures most of the time. Eye exam shows a hazy cornea with central ulceration and adjacent stromal abscesses. Eye movement is normal. What’s the most likely diagnosis?

A. Bacterial keratitis  B. Fungal keratitis  C. CMV retinitis
D. HSV retinitis  E. Orbital cellulitis

13. A 40 y/o man complains of intermittent abdominal discomfort, decreased appetite, and 5-kg weight loss for the past 3 months. He has a history of smoking and alcohol drinking for 5 years, and two previous blood transfusions. P/E results are mostly normal. Ultrasonography shows a normal liver image without any mass. Serology results show that liver function tests (LFTs) are normal; HBsAg, HBeAg, and anti-HBs, anti-HBc, and anti-HBe IgGs are all positive. What’s the best explanation?

A. Liver cell carcinoma  B. Chronic Hepatitis B, with low viral replication
C. Chronic Hepatitis B, with active viral replication  D. Recovery from Hepatitis B, with immunity
E. Chronic hepatitis B, with heterotypic Anti-HBs

14. A 25 y/o man suffered from a puncture wound of the right foot 3 days ago and now presents with fever and increased foot pain. P/E shows a swollen wound on the right ankle with tenderness on palpation. T = 39 °C, HR = 90/min, RR = 26/min; BP is normal. CBC shows WBC = 15 x 10^3/uL, with predominant neutrophils and bands. ESR = 120. Tech is scheduled and the blood is taken for culture and sensitivity testing. What’s the best initial therapy?

A. Oxacillin  B. Vancomycin  C. Ceftriaxone
D. Cefotetan  E. Cefepime

15-16: 15. A 20 y/o sexually active man presents with fever, a sore throat, swollen lymph nodes on the neck, and abdominal pain for the past 2 days. He developed a generalized skin rash after taking ampicillin. He reports having used allopurinol for gout before the onset of these symptoms. P/E shows enlarged tonsils, cervical lymph nodes, and spleen, and a maculopapular rash all over the body. T = 39°C. Vital signs are normal. More tests are scheduled. What’s the best explanation for his conditions?

A. Ampicillin allergy  B. Acute upper respiratory infection
C. Allopurinol allergy  D. Infectious mononucleosis
E. AIDS  F. Chlamydia infection

16. CBC for the above patient reports: hematocrits (HCT) = 44%, WBC = 8500/uL with many atypical cells, platelets = 85 x 10^3/uL. Monospot test is (+). Apart from bed rest, what’s the most appropriate next treatment?

A. Acyclovir  B. Ganciclovir  C. Steroids
D. PCN-G  E. Early exercise

17. A 20 y/o sexually active female presents with lower abdominal pain, dysuria, and increased, purulent vaginal secretions without odor. She has no fever or other symptoms. Pelvic exam shows a red cervix with mucus, and cervical motion tenderness. Urinalysis shows WBCs and protein. What’s the best next step?

A. Secretion test for chlamydia  B. Thayer-Martin for gonococcus
C. VDRL test for syphilis  D. KOH test for candida
E. Smear for Trichomonas

18. Continued from Q15: Fluorescent antibody test for chlamydia in Q15 has come out with (+), and Thayer-Martin for gonococcus is (+). Given this, the best treatment now is:

A. a single dose of ceftriaxone and azithromycin
B. a single dose of ceftriaxone
C. a single dose of ceftriaxone and doxycycline
D. IV cefoxitin + oral azithromycin
E. Ampicillin + gentamycin + metronidazole + clindamycin for 5 days

19. A 17 y/o girl presents with fever, headache, dry cough, and weakness for the past 3 days. P/E is normal except for T = 38.5°C. The urine dipstick testing reveals protein (++) but no bacteria, RBCs or WBCs. What’s the best next step?
A. Serum BUN level testing
B. 24-hour collection of urinary protein
C. Urine culture for pathogens
D. Reassurance: “It’s common and benign.”
E. Repeating the dipstick test

20. A 60 y/o man presents with increased urinary frequency and urgency, and a sensation of suprapubic fullness but difficulties in voiding for the past 3 days. P/E finds an enlarged urinary bladder and an indurated, enlarged prostate with tenderness. Urinalysis is normal. Analysis for prostatic secretions reveals 18 WBCs/HPF (normal reference is <10), but cultures for bacteria are (-). Other results are unremarkable. A urinary catheter is inserted and 300 mL of urine is removed. What’s the most appropriate next step?
A. Repeating the secretion culture
B. Empirical TMP-SMX for E coli
C. Use of an alpha-R blocker
D. Test of prostate specific antigen (PSA)
E. Fine needle aspiration of the prostate

21. A 58 y/o man presents with fever, chills, right flank pain, and dysuria for the past 5 hours. He occasionally smokes and drinks alcohol. P/E finds T = 38.5°C, HR = 110/min, and percussion tenderness over the right renal area. CBC shows WBC = 15 x 10³/uL with left shift. Urinalysis reveals WBC and protein. Urine is sent for culture and sensitivity test. He went home with prescribed oral ciprofloxacin. He comes back 3 days later with T = 38.3°C. What’s the most appropriate next step?
A. Increase the dose of oral ciprofloxacin
B. IV ciprofloxacin
C. IV cefepime
D. IV ampicillin + gentamycin
E. Renal CT scan

22. A 30 y/o man has his second onset of fever, chills, cough with sputum, and chest pain for the past month. He has a history of risky sexual behavior with both males and females for “several years.” P/E shows T = 39°C, HR = 110/min, and diffuse rales in the lungs. CXR reveals multiple lobar infiltrates. CBC shows WBC = 1200/uL and CD4 = 200/uL. Blood and sputum are taken for examination of pathogens. What’s the best initial treatment?
A. Azithromycin or erythromycin
B. Levofoxacin + erythromycin
C. Cefotaxime + imipenem
D. TMP-SMX
E. Ceftazidime + TMP-SMX

23-26: Match the following clinical scenarios with the most likely diagnosis.
A. Bacterial meningitis
B. Subacute sclerosing panencephalitis
C. Viral meningitis
D. AIDS encephalopathy
E. PML
F. Herpes encephalitis
G. Brain abscess
H. Malaria
I. Fulminant viral encephalitis      J. Fulminant hepatitis

23. A 25 y/o man is hospitalized for decreased memory and changes in mood for the past 3 weeks, with occasional right arm clonus. He had a history of fever and headache 4 weeks ago but no trauma. P/E results: T = 39°C, alert. Neurologic examination reveals decreased recent memory, speech difficulties, and right hemiparesis. Lab tests: increased WBC and LC ratio. CSF: opening pressure = 220 mmHg, LC = 60%, neutrophils = 40%; culture (-). EEG: spike-and-wave discharges originating from the temporal lobe.

24. A 25 y/o man is hospitalized for a chronic bleeding disease and progressive memory loss. Severely low platelet counts have forced him to receive four times of urgent blood transfusions in a poorly equipped hospital over the past 5 years. Neurologic examination reveals poor recent and remote memory, decreased vision, gait ataxia, limb hyper-reflexia, and changes in mood and personality. T = 38.5°C. Head MRI scan is unremarkable.

25. A 25 y/o man is hospitalized for a month’s history of headache, fever, right arm clonus, memory loss, and changes in mood. He has been HIV (+) for the past 5 months. Neurologic examination shows decreased recent memory, speech difficulties, and right hemiparesis. T=39°C, CSF-P = 220 mmH2O; culture is (-). CT with contrast reveals a mass in the left temporal lobe.

26. A 45 y/o man is back from a trip to the countryside with malaise, headache, confusion, periodic high fever, chills, and sweating for the past 3 days. P/E reveals T = 41°C, confused status, neck and limb stiffness, generalized rash and lymph node swellings, and hepatosplenomegaly. CBC reveals anemia, leukopenia, and reticulocytosis. Blood samples are taken for special tests.

27. Diagnosis of pathogen for the above patient in Question 23 is confirmed by immunoassay of the CSF. IV acyclovir was administered for the past 3 days and re-examination of the patient shows no changes in his conditions. What’s the most appropriate next step now?
   A. Increase the dose of acyclovir          B. Change to ceftriaxone
   C. Change to foscarnet             D. Change to famciclovir
   E. Change to amphotericin B

28. A 65 y/o man has been undergoing 2-week’s chemotherapy for lymphoma. P/E finds T = 39°C and other results are (-). CBC shows WBC = 500/uL without bands. The most appropriate next step is to
   A. wait for the results of blood culture and sensitivity test to give the correct antibiotics
   B. give oral agents to prevent bacterial and fungal infections
   C. give IV agents to prevent bacterial and fungal infections
   D. give broad-spectrum antibiotics to cover Gram- bacteria, Pseudomonas, and S. aureus
   E. take blood samples for culture and sensitivity test

29. A 25 y/o female working in a day care center develops a pruritic rash in crops over her whole body except the palms and soles, with fever, headache, cough, and dyspnea for the past 3 days. She claims that she has received all of the appropriate pediatric immunizations. P/E finds T = 39°C, generalized small vesicles on erythematous bases with crusting, and rales heard over the lungs. CBC report is awaited. This patient most likely
   A. has about normal CXR result          B. will infect her husband soon
   C. has leukocytosis                  D. will have life-time immunity after recovery
   E. has missed a vaccine in the childhood
30. A 15 y/o girl has just returned from a spring camping trip and presents with irritating red eyes and copious watery discharge from the eyes and nose. P/E reveals a mild fever, tachycardia, and congested conjunctiva and nasal membranes. There are no other abnormal findings. The most likely cause is
A. allergy  B. bacterium  C. chlamydia
D. virus  E. foreign body

31. A 16 y/o boy has just returned from a spring camping trip and presents with painful red eyes with copious watery discharge. P/E reveals a mild fever, tachycardia, and congested conjunctiva. Fundoscopy shows retinal pallor and ulceration. There are no other abnormal findings. The most likely etiology is
A. CMV  B. HSV  C. chlamydia
D. candida  E. allergy

32. A 25 y/o man presents with headache and a painful swelling localized to the left eyelid with tenderness, which is not associated with eye movement. There is no conjunctival congestion or discharge, nor other abnormal findings. He claims he has “two girl-friends.” The most likely diagnosis is
A. orbital cellulitis  B. post-orbital cellulitis  C. dacyrocystitis
D. hordeolum  E. chalazion

33. A 45 y/o man presents with fever, abdominal pain, nausea, and loose stools of yellowish color for the past 3 days, beginning after he eat a large meal with friends. None of his friends has similar symptoms. He has a 5-year history of smoking, alcohol consumption, and decreased weight. P/E finds a moderate fever and a firm, distended abdomen with decreased bowel sounds, tenderness on deep palpation, and with rebound tenderness. Ascites is (+) and the spleen is enlarged. What’s the most important next test to determine the etiology?
A. Abdominal ultrasound  B. CBC  C. Ascites analysis
D. Abdominal CT scan  E. Stool analysis

34. A 25 y/o female presents with fatigue, decreased appetite, nausea, and yellow urine for the past week. Her last menstrual period (LMP) was 4 weeks ago. P/E finds normal vital signs, jaundice, flat and soft abdomen, and an enlarged liver. Serum ALT and bilirubin are elevated; beta-hCG is (-); anti-HAV IgM and HBsAg are (-); anti-HAV IgG, anti-HBc IgM, and HBeAg are all (+). What’s the most likely diagnosis?
A. Acute hepatitis A with HBV carrier  B. Acute hepatitis B with HAV immunity
C. Acute hepatitis A and B  D. Acute hepatitis B with HAV carrier
E. HBV carrier with HAV immunity

35. A 40 y/o man presents with low-grade fever, sweats, coughs with blood-tinged sputum, right chest pain, and decreased weight for the past month. He has a history of smoking and alcohol use for the past 5 years. P/E finds T = 38°C, normal vital signs, decreased respiratory sounds, and dullness on percussion of the right lower chest. CBC reveals anemia and increased WBC counts and lymphocyte (LC) percentage. CXR shows diffuse infiltrates in the right lower lung. The most appropriate next test for diagnosis is
A. serologic tests  B. chest CT  C. sputum culture
D. PPD test  E. sputum stain for acid fast bacilli
36-41: Match the following clinical scenarios with the most likely etiology.

A. Klebsiella granulomatis  
B. H. ducreyi  
C. Chlamydia  
D. HSV  
E. HPV  
F. Poxvirus  
G. Candida  
H. T. pallidum  
I. Gonococcus  
J. Allergy

36. A sexually active female presents with multiple soft, fast growing, pedunculated, and pink papules of 3-4 mm in size on the vulva for the past week. There are no other abnormal findings.

37. A sexually active female presents with multiple red, painful, and itchy vesicles with circular, scarring ulcers on the vulva for the past 2 weeks. Tissue is taken for Tzanck test and culture.

38. A sexually active female presents with a week of multiple painless, shallow, circular ulcers on the vulva, with low-grade fever, dysuria, tender swelling of the left inguinal lymph nodes, and a purulent draining sinus. A sample from the ulcer is taken for smear stain and fluorescent tests.

39. A sexually active female presents with a week of multiple painful, irregular, deep papules and ulcers on the vulva with a bad odor, and suppuration of the left inguinal lymph nodes. There are no other abnormal findings. A sample from the ulcer is taken for a Gram stain.

40. A sexually active female presents with a week of multiple raised, red, painless papules (0.5-1cm) with granulomatous ulcers on the vulva. There are no other abnormal findings. A sample from the ulcer is taken for a pathologic stain.

41. A sexually active man presents with low-grade fever, diffuse and symmetric pink papules, and painless swelling of lymph nodes in both inguinal areas. The patient reports finding a small painless smooth ulcer on his penis one month ago, which has now disappeared. Specific serology confirms the diagnosis.

42. A 17 y/o girl is brought to the ER with fever, nausea, vomiting, dizziness, abdominal pain, knee pain, and weakness. She travelled in another city 5 days ago with her boyfriend and reveals that her last menstrual period (LMP) occurred during the trip. P/E results: Alert, T = 39°C, HR = 95/min, BP = 90/55 mmHg; soft neck, desquamative rash on hands and feet, and tenderness on the middle abdomen and both knees without swelling. IV fluid is started. Urine sample is taken for analysis and culture. The most appropriate next step is

A. IV nafcillin  
B. IV ceftriaxone  
C. blood culture and sensitivity test  
D. joint fluid aspiration  
E. abdominal ultrasound

43. A 30 y/o female with multiple pets presents with fever, dry cough, chest pain, and shortness of breath for the past 3 days. P/E results: Alert, T = 39°C, RR = 28/min, HR = 90/min, normal BP, rough respiratory sounds. WBC is 12,000/uL. CXR reveals interstitial infiltrates. What’s the best initial treatment?

A. Erythromycin  
B. Doxycycline  
C. Levofoxacin  
D. Azithromycin  
E. Amoxicillin

44. A 60 y/o female presents with fever, headache, dry cough, and loss of appetite for the past 3 days after attending a party. Two other friends from the party also have similar symptoms. P/E shows T = 38.5°C and there are no other abnormal findings. What’s the most appropriate next step?

A. Amantadine for 4-5 days  
B. Oseltamivir for 4-5 days  
C. Symptomatic treatment
D. Annual flu vaccination

E. Blood culture and sensitivity test

45-50: Match the following clinical scenarios with the most likely cause.

A. Campylobacter jejuni
B. E. coli (O157:H7)
C. S. aureus
D. Shigella
E. Salmonella
F. Enterotoxigenic E. coli
G. Giardia
H. Bacillus cereus
I. Clostridium perfringens
J. Clostridium difficile
K. Clostridium botulinum
L. Vibrio parahaemolyticus
M. Yersinia enterocolitica
N. Proteus
O. Klebsiella

45. A 17 y/o boy ingested a cup of leftover milk at home. Four hours later, he has severe lower abdominal cramps and loose stools with sparse blood and mucus. There is no vomiting. P/E finds T = 38°C and mild tenderness in the middle abdomen. Stool analysis reveals WBCs and RBCs. CBC results are normal.

46. A 10 y/o boy had a meal of reheated rice in a friend’s home. Two hours later, he has severe nausea, vomiting, and upper abdominal cramps. He has one relatively loose stool during the day. P/E finds no fever or other abnormal results. Stool analysis and CBC results are normal.

47. A 16 y/o boy joined a lunch with all kinds of foods (including seafood) in an unsanitary restaurant. Several hours later, he presents with severe, recurrent lower abdominal cramps and loose stools with blood and mucus for a few hours. He has nausea and sensation of urgently passing stools. There is no vomiting. P/E finds T = 39°C and the lower abdomen is soft with tenderness. Stool analysis shows WBCs and RBCs. CBC reveals leukocytosis.

48. A 15 y/o boy had a lunch in an unsanitary restaurant. Several hours later, he has abrupt profuse, watery diarrhea. There is no vomiting, abdominal cramp, or fever. He’s been using amoxicillin for otitis media for the past 10 days. P/E results are unremarkable. Stool toxin test is (+). CBC is normal.

49. A 17 y/o girl joined a friend’s lunch in a restaurant with fresh raw fish. Several hours later, she has fever, nausea, severe lower abdominal cramps, and diarrhea with blood and mucus. P/E finds T = 38.5°C and a soft abdomen with RLQ tenderness. Stool analysis reveals WBCs and RBCs. CBC reveals leukocytosis.

50. A 20 y/o newly married woman presents with urinary frequency, urgency, and burning sensation for a day. She denies any fever, flank pain, or abnormal vaginal discharge. She also denies any history of UTI or STD. P/E results are about normal. Urine analysis reveals WBCs and alkalosis. CBC is normal.

51. A 22 y/o woman comes to the clinic for a general health exam. She is confirmed with 5-week’s gestation by a positive blood hCG test. She took rubella immunization 6 weeks ago and has been using contraception to her best efforts because the physician has advised her to avoid pregnancy within 3 months. She is generally healthy and asks about the options to avoid harm to the fetus. What’s your best next step of management?

A. Give advice on abortion
B. Give reassurance
C. Give IV immunoglobulin
D. Perform pelvic ultrasonography
E. Explain the risks and benefits of abortion and let the patient decide
52. A 30 y/o sexually active man presents with fever, fatigue, and skin rash for the past few days. He was HIV (+) two years ago but has had no obvious symptoms until now. P/E shows T = 38.5°C, normal vital sign, and multiple non-tender, 1-1.5 cm, round, reddish, vascular papules on both arms. What’s the most likely diagnosis?
A. Molluscum contagiosum (MC)  B. Common warts  C. Herpes simplex (HS)
D. Kaposi sarcoma (KS)  E. Bacillary angiomatosis

53. A 25 y/o woman presents with painful, swollen left knee for the past 3 days. She cannot think of any significant events related to it. She denies any abnormal urinary or vaginal discharges, or history of trauma, diseases or drug use. Careful history taking reveals that she has several sexual partners and she uses condoms most of the times. P/E finds low fever, tachycardia, and a swollen left knee with tenderness and limited range of motion. Arthrocentesis: WBC = 85 x 10^3/μL with 88% neutrophils. Gram staining of the joint aspirate is (-). Urinalysis is normal. What’s the best next step for diagnosis?
A. Blood culture  B. Urine culture  C. Culture of the joint aspirate
D. Culture of the vaginal discharge  E. Culture of the urethral swab

54. A 60 y/o man has been hospitalized for the treatment of diabetes for the past 2 weeks. He has a 10-year history of smoking and alcohol drinking. Symptoms: fever, cough with yellowish sputum, chest pain, tachypnea, and dyspnea. P/E results: T = 38.8°C, RR = 28/min, HR = 90/min; BP is normal; respiratory rales are (+) in both lungs. CBC reveals HCT = 40%, WBC = 12 x 10^3/μL, neutrophils = 88%. CXR shows multiple infiltrates in both lungs. Sputum is taken for Gram stain and culture. Which of the following is NOT a common pathogen for this patient?
A. Pneumococcus  B. S. aureus  C. E. coli
D. Pseudomonas  E. Hib  F. Klebsiella

55. A 30 y/o man presents with malaise, general muscle pain, and decreased appetite for the past month. He has a 5-year history of smoking, alcohol drinking, and IV drug abuse. P/E results are unremarkable. CBC reveals HCT = 38%, WBC = 6,000/μL, LC = 44%, platelets = 100 x 10^3/μL. Anti-HCV is (+) and LFTs are normal. Test of HCV RNA load by PCR is started. Which of the following is NOT commonly associated with the patient’s disease?
A. Mixed cryoglobulinemia  B. Polyarteritis nodosa  C. Sjogren syndrome
D. Hashimoto thyroiditis  E. Membrane glomerulonephropathy
F. Idiopathic thrombocytopenic purpura  G. Plasmacytoma  H. T-cell lymphoma

56. In a pre-term exam, an asymptomatic pregnant patient has been found that the HBsAg, anti-HBe and anti-HBc IgGs are (+), and HBeAg and anti-HBs IgG are (-). There are no other abnormal findings. For this patient’s conditions, all the following should be administered immediately after delivery EXCEPT
A. IV HBIG to the newborn  B. HBV vaccine to the newborn
C. HBV vaccine to the mother  D. Ribavirin to the mother
E. Alpha-interferon to the mother

57. A 55 y/o man with immunodeficiency presents with malaise, fever, night sweats, and cough with yellow sputum over the past week. He has been on antibiotic prophylaxis for the past 2 months. P/E results: Alert, T = 39°C, RR = 28/min, HR = 90/min; BP is normal; respiratory sounds are rough. CBC reveals HCT = 32%, WBC = 8,000/ul, neutrophils = 85%, LC = 9%. Skin PPD is 9 mm induration. CXR shows left lobe cavitation. Sputum smear reveals weakly acid-fast filamentous branching rods. What’s the most likely cause of the disease?
Chapter 1: Infectious Diseases

58. A 10 y/o boy is brought to the clinic an hour after he was bitten by a neighbor’s dog due to his provocation. The dog did not get immunization for rabies and is not showing any abnormal symptoms. P/E finds a tender swollen lesion without bleeding on the left forearm of the boy. His wound is cleaned with iodine. The best next step is to
A. observe the dog for 10 days
B. kill the dog and perform the brain biopsy
C. give the boy IV immunoglobulin
D. give the boy active rabies immunization
E. give the boy active and passive immunization
F. give reassurance

59. During a fight at school, a 9 y/o boy received a bite on the right forearm by another boy, and is brought to the clinic an hour later. His records show up-to-date immunizations. P/E finds a swollen tender lesion with tiny bleeding on the right forearm. There are no other abnormal findings. Apart from cleaning the wound, the best next treatment is
A. observation
B. amoxicillin
C. ampicillin and clavulanate
D. amoxicillin and clavulanate
E. clindamycin
F. erythromycin

60. In medical records, human bites have been shown to transmit all the following infections EXCEPT
A. hepatitis B
B. hepatitis C
C. herpes simplex
D. syphilis
E. TB
F. actinomycosis
G. tetanus
H. AIDS

61. A 40 y/o man with a history of alcoholism presents with high fever, cough with “currant jelly” sputum, chest pain, tachypnea, and dyspnea. A CXR confirms the diagnosis. What is the most likely pathologic organism and the best treatment in hospital?

62. A 15 y/o boy stepped on a rusted nail with bleeding that stopped automatically. Three days later, he presents with “lockjaw”, neck stiffness, lower extremity extension, dysphagia, headache, and irritability. What are the therapeutic points?

63. A 70 y/o man presents with sinusitis followed by high fever, photophobia, headache, nausea, vomiting, confusion, and neck stiffness. CSF testing confirms the diagnosis. What’s the most likely organism that caused the infection?

64. A pregnant woman with pets presents with high fever, headache, altered mental status, and neck stiffness. CSF testing confirms the diagnosis. What’s the most likely organism that caused the infection and the best therapy?

65. A 65 y/o woman has an oral infection with testing showing branching rods. What’s the most likely organism that caused the infection?

66. What is the most likely organism and the best antibiotics for infections in burns, malignant external otitis, or osteomyelitis from a foot wound puncture?

67. What are the three most common causes of fever of unknown origin (FUO) in adults?

68. What are the common AIDS-defined illness?
69. What are the clinical features of Rocky Mountain spotted fever and the best treatment?
70. What are the clinical features of systemic inflammatory response syndrome (SIRS) and sepsis?

**Answers and Explanations**

1. (D). Regional enteritis (Crohn disease), ulcerative colitis, dysentery and invasive E. coli (O157:H7) may all cause bloody diarrhea and fever. Two negative stool cultures in this case may exclude infections. Traveler’s diarrhea, cryptosporidiosis and giardiasis usually cause small bowel infection with watery diarrhea.

2. (C). IV cefepime. It’s an urgent case of febrile neutropenia induced by chemotherapy, which requires immediate antibiotics in hospital. Empiric treatment includes monotherapy of ceftazidime, cefepime, or a carbapenem, or combined therapy of piperacillin plus gentamycin, to cover Gram` bacteria (especially Pseudomonas). Vancomycin is usually added for resistant S. aureus or Pneumococcus or severe skin/mucosa infections. If fever persists despite above treatment, amphotericin B is added to cover suspected systemic fungal infection. Piperacillin alone is inadequate.

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Chapter 2
CARDIOVASCULAR DISEASES

Cardiovascular diseases (CVDs) carry a high morbidity and the highest mortality rate among all diseases in most countries. CVDs are closely associated with human life styles and thus can be reduced significantly by regular health examinations, consultations, and life style modifications.

**PEARLS — IMPORTANT DIFFERENTIATIONS OF CHEST PAIN**

Chest pain is the most common symptom for most cardiovascular diseases, respiratory diseases, and some upper abdominal disorders. Thus, it’s important to grasp the differential diagnosis associated with it.

**Angina and myocardial infarction (MI):** See details on the same topics below.

**Myocarditis:** It is usually preceded by a viral disease and associated with a vague chest pain. Creatine kinase (CK)-MB is often increased. ECG (EKG) will show abnormal conduction or Q waves.

**Pericarditis:** It may be preceded by a viral illness. Chest pain is sharp, pleuritic, and positional—worse with lying down and relieved by sitting up. Pericardial rub often exists. ECG usually shows diffuse ST elevation without Q waves. CK is mostly normal. It responds well to anti-inflammatory drugs.

**Pleuritis:** Mostly after lung infection, with sharp chest pain that is worse on inspiration and certain position; tenderness, friction rub or dullness may be present. CXR or CT scan is the best diagnostic test.

**Pneumonia:** Moderate chest pain with fever, cough, sputum, and hemoptysis. CXR is the best test.

**Pneumothorax:** Typically sudden, sharp, pleuritic chest pain and dyspnea; absent breath sounds; mediastinum shifted to the opposite site—suspect of tension pneumothorax—requiring urgent intercostal needle puncture. Non-tension pneumothorax can wait for CXR confirmation and natural relief.

**Aortic (aneurysm) dissection:** Typical severe, sharp, tearing chest pain radiating to the back; loss of pulses, unequal BP between arms, or aortic insufficiency; neurologic signs; mediastinum widened on CXR. MI may occur if dissection extends into coronary artery (Cor-A). Diagnosis is confirmed by transesophageal echocardiography (TEE), CT scan, or aortography.

**Pulmonary embolism (PE):** Sudden chest pain, dyspnea, tachycardia, cough, and hypoxemia, usually 3-5 days following a surgery or long immobility. The chest pain is usually pleuritic but may resemble angina. **CT pulmonary angiography** has supplanted V/Q scanning as the preferred means of diagnosis.

**Mitral valve prolapse:** Transient chest pain with a typical midsystolic click murmur.

**Pulmonary hypertension (HTN):** Dull chest pain with symptoms and signs of right ventricular (RV) failure.
**Costochondritis:** Chest pain is usually stabbing, localized, exacerbated with inspiration or chest palpation (reproducible). ECG is normal (NI).

**Gastric diseases:** GERD (burning chest pain, acid reflux, bad taste, relief with antacids); stomach spasm; PUD (epigastric pain before or after eating).

**Pancreatitis:** Post-meal persisting sharp epigastric pain radiating to the back, with nausea, vomiting, fever, and increased amylase and lipase levels.

**Gallbladder disease:** Post-meal right upper quadrant (RUQ) abdominal pain with tenderness, nausea, vomiting, and/or jaundice usually in a middle-aged obese woman.

**Hiatal hernia:** Burning chest or epigastric pain; nausea, vomiting; reflux of food; relief with antacids.

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**ISCHEMIC HEART DISEASE (IHD)**

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**CORONARY ARTERY DISEASE (CAD)**

**Etiology and pathogenesis**

The basic mechanisms of CAD are that coronary O\textsubscript{2} demand surpasses supply due to decreased blood flow secondary to atherosclerotic narrowing of the coronary artery (Cor-A), leading to cardiac dysfunctioning. It’s fatal if there is narrowing of 1-2 major coronary arteries that causes > 75% decrease in cross-sectional area (or 50% decrease in diameter).

**Factors that increase O\textsubscript{2} demand:** Physical exertion or stress, emotional or mental stress (including anxiety), large meals, etc.

**Factors that lower the O\textsubscript{2}-carrying capacity of the blood:** Anemia, carbon monoxide (CO) poisoning, platelet microthrombi at the site of coronary stenosis (focal damage), etc.

**Major risk factors**

**Age** (male > 45, female > 55 y/a), **male** gender, **smoking, hypertension, diabetes**, heredity (including race, family history < 55 y/a), **atherosclerosis, hypercholesterolemia** (Hyper-Chol, LDL > 200 mg/dL, HDL < 40 mg/dL), physical inactivity, obesity and overweight, stress, excess alcohol use, and postmenopausal women.

**The metabolic syndrome (Insulin resistance syndrome):** It refers to any combination of hyperglycemia, hyperlipidemia, hyperuricemia, and hypertension. Genetic predisposition, lack of exercise, and body fat distribution may increase the likelihood of developing diabetes and CVD.

**(Cardiac) syndrome X:** It refers to angina pectoris (ischemia) occurring in patients with normal coronary arteriogram.

**Prognostic indicators**

1. **Left ventricular function—Ejection fraction (EF):** Normal > 50%; if < 50%, associated with increased mortality.
2. **Severity—Vessels involved:** Left main coronary artery or > 2 arteries indicating worse prognosis.
Angina Pectoris

It mostly refers to “stable angina”, a paroxysmal chest pain resulting from cardiac ischemia—an imbalance between oxygen supply and demand, and is most commonly caused by the inability of atherosclerotic coronary arteries to perfuse the heart under conditions of increased myocardial oxygen consumption (exercise, stress). Stable angina is the type when the chest pain is precipitated by predictable factors (exercise, exertion, etc.). Unstable angina is angina that occurs at any time.

Essentials of diagnosis
I. Clinical features
1. Nature of the pain: Heavy, pressing, or squeezing.
2. Location: Substernal or precordial.
3. Radiation: Commonly to the left jaw or arms.
4. Duration: 15 sec—15 min.
5. Precipitating factors: Exertion, anxiety, meals, and coldness. Risk factors are the same as above.
6. Associated symptoms: Shortness of breath (SOB), anxiety, fatigue, nausea and vomiting (N/V), palpitations, and diaphoresis.
7. Pain relief: Nitroglycerin (in a few min), resting (standing or sitting).
9. ECG: ST-T depression.

II. Types of angina
1. Chronic stable angina: It occurs during exertion, can be relieved by rest or nitrates, and can recur. ECG usually shows evidence of ischemia during pain or stress testing. Angiography mostly demonstrates significant obstruction of major coronary arteries.

2. Unstable angina: New onset of chest pain that occurs at rest or with less exertion, or requires more medicines to be relieved. It follows a worsening pattern in frequency, duration, or/and severity of symptoms. It should be considered and managed as a form of acute coronary syndrome. Diagnosis is based on presence of severe angina in those patterns without ST-elevation on ECG. It often progresses to myocardial infarction, and thus should be stabilized with aggressive management (aspirin, beta blockers, LMWH, nitrides, etc.) before stress testing or cardiac catheterization is performed.

3. Prinzmetal (variant) angina: Chest pain occurs at rest or stress without the usual precipitating factor of exertion, due to coronary artery spasm. It may result from an altered autonomic neurologic control of the coronary arteries or altered artery contraction. ECG mostly shows ST-T elevation. Cardiac catheter will show no atherosclerosis. Management is mainly a diagnostic therapy of a Ca-channel blocker (the best diagnostic treatment, which usually relieves symptoms immediately), or a nitrate.

III. Lab diagnosis
1. **ECG**: **It is the best initial test for all forms of chest pain.** It can exclude previous myocardial infarction (MI) or obvious arrhythmia; also to evaluate the use of regular stress test versus thallium testing.

2. **Exercise ECG/echocardiography or treadmill (stress) test**: When ECG is normal or inconclusive, stress testing is the best diagnostic test to confirm angina (ischemia), determine the severity of disease, and evaluate post-MI conditions. Echocardiography is more sensitive than ECG. Stress test is (+) if any of the chest pain, ST-segment depression, hypotension, or significant arrhythmia is induced by exercise.

3. **Thallium (scan)-treadmill test**—similar to exercise echocardiography but focusing more on the coronary arteries) — **Indications:**
   - (1) Patients with inconclusive regular treadmill testing.
   - (2) Patients with mitral valve prolapse.
   - (3) Patients with WPW syndrome.
   - (4) Patients with left bundle branch block.
   - (5) Young women with high false positive results on regular stress testing.
   - (6) Patient with uncertain acute ischemic changes on ECG (nonspecific ST-wave changes, inability to read the ECG, etc.).
   - (7) Patient requiring quinidine, procainamide, or digitalis.

   **Ischemia versus infarction**: In ischemia, it will show a reversal of the decrease in thallium uptake or wall motion that will return to normal after a period of rest.

4. **For patients unable to exercise**: Perform dipyridamole-thallium, adenosine-thallium, or stress/dobutamine echocardiography (Echo).

5. **Contraindications for stress testing**: Unstable angina, aortic stenosis, idiopathic hypertrophic subaortic stenosis, severe COPD, acute CHF, acute ischemic changes on ECG, aortic dissection, and severe hypertension.

6. **Coronary angiography**: This is the most accurate method of detecting CAD, to detect the presence of narrowing that is best treated with surgery or angioplasty—usually when more than 70% stenosis exists. Coronary angiography is often used when ECG or stress testing results are equivocal.

7. Holter monitoring: It’s a continuous ambulatory ECG monitoring that records the rhythm, usually used for arrhythmias and for a 24-hour period (but may extend to 48-72 hours). It does not detect ischemia because it’s not accurate for ST segment evaluation.

8. **Cardiac catheterization**: Usually applied in patients poorly controlled with drugs or positive stress test, to help determine the need for angioplasty—triple vessel disease or left main coronary artery disease. Occupational workers for public safety (bus driver, airline pilot) with any CAD symptoms require cardiac catheterization.

**Differential diagnosis**
See “Important Differentiations of Chest Pain” above.

**Treatment**

1. **Medical therapies for angina:**

   **Drugs that can reduce mortality rates for patients with chronic angina include aspirin, beta-R blockers, and nitrates** (nitrates did not show decreased mortality in acute MI).

   (1) **Nitrates**: They are the first-line therapy for acute angina. Low doses of nitrates increase vein-dilation and decrease preload; high doses of nitrates increase small-artery and coronary artery dilatation and O₂ supplement, and decrease both afterload and preload.

      **Adverse effects (S/E)**: Vasodilation can lead to orthostatic hypotension, reflex tachycardia, throbbing headache, and blushing. It’s contraindicated if systolic BP < 90 mmHg (to avoid syncope). It’s important to have a > 8-hour window-free period to reduce the incidence of tachyphylaxis (usually 12am—6am).

   (2) **Beta-R blockers**: They decrease HR, contractility, and blood pressure, and thus decrease myocardial O₂ requirement. They are **contraindicated in severe asthma as well as vasospastic** or variant (Prinzmetal) angina to avoid induction of coronary vasospasm from unopposed alpha-receptor activity. **Long maintenance therapy has been shown to reduce mortality in acute MI and CHF and risk of reinfarction.**

      **Adverse effects**: Fatigue, bronchoconstriction, depression, hallucinations, sexual dysfunction, insomnia, dyslipidemia, Raynaud phenomenon. A nonselective beta-R blocker (propranolol) may mask hypoglycemic symptoms in diabetes (IDDM) and is not routinely used in CAD.

      **Selective beta₁ blockers** (with less adverse effects): Atenolol, metoprolol, or acebutolol is most commonly used in angina pectoris and MI (and CHF).

      **New nonselective beta-R blockers**: Penbutolol and carteolol are used for hypertension.

   (3) **Ca-channel blockers**: They decrease preload and afterload. It may be harmful during the post-infarction period, especially if patient has left ventricular failure. Its efficacy in angina is limited.

      **Adverse effects**: Cardiac—reflex tachycardia, hypotension, and dizziness, CHF; noncardiac—flushing, headache, weakness, nausea, constipation, wheezing, and peripheral edema.

**PEARLS**—Specific Ca-channel blockers with various selectivity

<table>
<thead>
<tr>
<th>Strong on heart: Verapamil</th>
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<tbody>
<tr>
<td>Strong on peripheral vessels: Nifedipine</td>
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<tr>
<td>Intermediate: Diltiazem</td>
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**Verapamil**: It causes significant A-V block in ECG; moderate decrease in coronary blood flow and cardiac contractility; hypotension, and ankle edema. It’s contraindicated in sick sinus syndrome, A-V node block and ejection fraction (EF) < 35%.

**Diltiazem**: It causes moderate A-V block and increase in coronary blood flow, mild decrease in contractility, and mild hypotension.
Nifedipine: It has minimal A-V block; mild decrease in cardiac contractility; significant hypotension; increase in coronary blood flow, ankle edema, and headache. It’s contraindicated in aortic stenosis and unstable angina.

(4) **Newer therapies: Ranolazine**, a late Na-channel blocker, is used either in combination with a beta blocker or as a substitute in patients who cannot receive one.

2. **Treatment of unstable angina:**

   (1) Hospitalize the patient and treat with aggressive medications—aspirin, nitrates, beta-R blockers, heparin, and lipid-lowering agents as described above. Heparin (IV or SC) or low molecular weight heparin (LMWH) is the major therapy because of its high efficacy.

   (2) Glycoprotein inhibitors with angioplasty and stent placement are very effective, but thrombolytics are not.

   (3) **Revascularization:**

      (a) **CBG (Coronary bypass graft)**: Very useful in those with major left coronary disease or 3-vessel disease and left ventricular (LV) dysfunction. It’s indicated in cases with symptoms despite medical treatment or with severe adverse effects from therapies. It’s more beneficial in those with diabetes or low ejection fraction, although the performance carries more risk.

      (b) **PTCA (Percutaneous transluminal coronary angioplasty)**: It is indicated in significant cardiac lesions not eligible for CBG. It’s an easier procedure with more risk of re-stenosis. Stent placement is now a standard procedure. Glycoprotein 2b/3a inhibitors (abciximab, tirofiban, or eptifibatide) are usually used with the procedure, followed by aspirin plus ticlopidine or clopidogrel.

3. **Preventive therapies:**

   (1) **Lifestyle modification for risk reduction (highly important!)**: Smoking cessation; reduction of stress, weight, LDL-C, and triglyceride; regular exercise; treatment of diabetes, hypertension, anemia, and COPD. Omega-3 fatty acids (DHA and EPA) and nuts intake can lower risk of CVDs.

   (2) **Antiplatelet therapy**: Low-dose aspirin daily is very effective in prevention of angina. New antiplatelet drug—ticlopidine or clopidogrel is an alternative to aspirin in patient who cannot tolerate aspirin. Note that ticlopidine can cause adverse neutropenia.

   (3) **Lipid management**: See “HYPERLIPIDEMIA”.

**Myocardial Infarction (MI)**

MI is ischemic myocardial necrosis as a result of an abrupt reduction in the coronary blood flow to a segment of myocardium, usually due to a thrombotic occlusion of a coronary artery previously narrowed by atherosclerosis. MI is associated with a 30% mortality rate and 50% pre-hospital deaths.

**Etiology**
Atherosclerosis by all causes is the main pathologic basis. Most cases are due to acute coronary thrombosis—atheromatous plaque ruptures into the vessel lumen and thrombosis forms on top of the lesion causing the vascular occlusion.

Risk factors: Same as those for CAD (above).

Non-atherosclerosis causes: Vasculitis, SLE, polyarteritis nodosa, Takayasu arteritis, mucocutaneous lymph node (LN) syndrome (Kawasaki disease, Image 36), coronary spasm, variant angina, cocaine abuse, coronary artery embolus, atrial myxoma, atrial or ventricular thrombus, polycythemia vera, thrombocytosis, and anomaly of coronary arteries.

Pathogenesis

Acute MI is mostly “ST-elevated MI” and localized to the left ventricle (LV) and in one of the two forms below:

1. Transmural infarct: It’s more often associated with Q waves.
2. Subendocardial infarct: It’s mostly “non-Q-wave MI”, confined to the inner 1/2 to 1/3 of the LV wall. The LV subendocardium region is most susceptible to ischemia, because of tenuous oxygen supplement. Diltiazem use can reduce the risk of recurrence.

PEARLS—Coronary artery anatomy and MI (Images 1-10)

LCA and LAD: They supply most of the LV and the anterior interventricular septum. They account for the most common occlusion in CAD, causing LV anterior wall MI.

CFX: Circumflex artery, supplies the left lateral wall. Its occlusion causes lateral wall MI.

RCA: Supplies the SA, AV nodes, and most of the inferior portion of the LV. Its occlusion causes inferior MI.

Essentials of diagnosis

1. Symptoms: Characteristic chest pain—severe, crushing, prolonged (usually > 20 min) chest pain, similar in quality to but more severe than angina; associated with dyspnea, anxiety, diaphoresis, nausea, vomiting, weakness, low fever, sense of impending doom, and syncope (in elderly). Painless and atypical MI can be up to 1/3 cases and more likely in postoperative or diabetic patients and the elderly. Sudden cardiac death can occur due to ventricular fibrillation (V-fib).

2. Signs: Congestive heart failure (CHF) —arrhythmias (mostly tachycardia; inferior MI may have bradycardia), S4 gallop, JVD, and dyskinetic left ventricular (LV) impulse. Cardiogenic shock signs are seen with > 40% of myocardial infarction—BP decrease, S3 gallop, and rales. Systolic murmur of papillary muscle rupture, ventricular septal rupture or pericardial friction rub (usually with transmural infarction) on the 3rd-4th day may be heard.

3. PEARLS—ECG (Images 4-10): It’s the best diagnostic test within 6 hours of onset and represented by ventricular wall hypokinesia, peaked T waves (early), ST-segment elevation (transmural infarct) or depression (subendocardial lesion), new left bundle branch block (LBBB), or Q waves (necrosis, late).

LV anterior wall MI (#1 common): ST-segment elevation (+ Q wave) in anterior leads (V1-4).
Posterior MI: ST-segment depression (+ large R wave) in inferior leads (V1-2).
Inferior MI: ST-segment elevation (+ Q wave) in inferior leads (II, III, and aVF).
Lateral wall MI: ST-segment elevation (+ Q wave) in leads I, aVL, and V5-6.

4. Cardiac enzymes (Figure 2-1):
   (1) **CK-MB** is both highly sensitive and specific for MI when measured within 36 hrs of chest pain. It begins to elevate at 4-6 hrs after MI, reaches a peak at 12-24 hrs and is back to normal in 72 hrs. CK levels may increase following cardioversion, defibrillation, cardio-pulmonary resuscitation, or muscle trauma, but the MB fraction will only increase with certain extent of myocardial death.

   (2) **Troponin** is most specific but moderately sensitive; it begins to rise 2-4 hrs after the start of the chest pain, and remains high for 7-10 days. It’s a more valuable biomarker for MI with the chest pain within 8 hrs and after 36 hrs.

   (3) **Lactate dehydrogenase** (LDH): It’s non-specific and not used for diagnosis of acute MI, but useful for re-infarction. In acute MI, LDH increases after 12 hrs of chest pain and peaks in 24-72 hrs and remains high for 10-14 days after MI. LDH-1/LDH-2 ratio > 1.0 supports MI.

5. **CBC**: Leukocytosis of 10-20 x 10³/uL.

6. **Thallium-201 (Tl-201) and Technetium-99m (Tc-99m) scan**: Tl-201 scan is sensitive but not very specific because it cannot distinguish between zones of severe ischemia (“cold spots”) and infarction. Tc-99m scan provides better resolution for the same function.

7. **Special type of MI**: It may be clinically silent or present as congestive heart failure (CHF) or dysrhythmia in the absence of chest pain, especially in elderly, postoperative, hypertensive, or diabetic patients.

**Figure 2-1: Biomarkers of Acute MI** (Courtesy of www.onlinejacc.org)
Differential diagnosis

Angina, pulmonary embolism (PE), pneumothorax, pneumonia, aortic dissection, pericarditis, costochondritis, etc.

Treatment

1. “ABC” first—airway, breathing, and circulation. Supplemental oxygen has shown a reduction in the risk of death.

2. Treat sustained ventricular arrhythmia or heart failure rapidly.

3. Beta-1-R blockers: They’ve clearly shown reduced post-MI mortality rate if no contraindications exist (bradycardia, A-V block, hypotension, or COPD). A beta blocker (metoprolol) is a good early start (IV injection every 5 min) after an acute MI and also good maintenance therapy.

4. Nitrates (PO or IV): They can alleviate pain, lung congestion, and left heart failure, but did not clearly reduce post-MI mortality.

5. Antiplatelet therapy: Aspirin (PO) can reduce coronary reocclusion by inhibiting platelet aggregation on top of the thrombus and clearly reduce post-MI mortality. It’s used as part of the maintenance therapy. Clopidogrel, ticlopidine (less S/E), or prasugrel is indicated in (1) aspirin intolerance (such as allergy); (2) recent angioplasty with stenting. Prasugrel has more risk of hemorrhagic stroke in elder patients. Other NSAIDs should be avoided or stopped in patients with MI.

6. Thrombolytic therapy: Best within 6-12 hrs for ST-segment-elevation MI; the earlier, the better outcome. Indications include typical chest pain of acute MI <12 hrs and new LBBB. t-PA (with more tissue selection), streptokinase (with stronger effect), reteplase, or anistreplase is among the good options. Beyond 24 hrs of symptom onset, it is usually ineffective and contraindicated.

Complications of thrombolysis:

(1) Overuse: hemorrhage, more common with tissue plasminogen activator.

(2) Re-perfusion arrhythmias.

Contraindications to thrombolytic therapy:

(1) Active bleeding disease;

(2) Dissecting aortic aneurysm (suspect);

(3) Uncontrolled hypertension > 180/110 (First control BP, then give thrombolytics);

(4) Known traumatic CPR;

(5) Recent head trauma or stroke (< 3 months);

(6) History of major trauma or surgery (< 3 years).

7. Analgesics: IV opiates (morphine) are important to relieve pain, to supply relaxation and sedation, and to alleviate CVS and respiratory stress effectively.

8. ACE inhibitors (Angiotensin-converting enzyme inhibitors, ACE-I): It has shown to reduce post-MI mortality. It’s best beneficial for post-MI patients with CHF, LV dysfunction with an EF (ejection
(fraction) < 40%, or regurgitant disease. It should be started early and in the maintenance therapy. It’s also used in any anterior wall MI and should be stopped after 6 weeks. Dry cough is the most common S/E. If it’s intolerable to the patient, the ACE-Inh should be ceased and another agent be considered.

9. Hypolipidemic therapy: Atorvastatin should also be started early and before percutaneous coronary intervention (PCI).

10. Anticoagulation: Heparin—IV bolus initially and then continuous infusion to keep the PTT 1.5-2 times the normal value. It’s useful for unstable angina and as a follow-up treatment for t-PA use.

11. Coronary angiography and angioplasty—indications:

(1) Patients with typical and persistent symptoms with new left bundle branch block; (2) Acute MI when thrombolytics are contraindicated or patient is in a well-equipped hospital; (3) Clinical CHF, post-MI patient with CHF, EF < 40%, recurrent ischemia and ventricular arrhythmias, or failed thrombolytic therapies.

For most patients, clinical trials have demonstrated superiority of primary PCI, irrespective of whether balloon angioplasty or stenting is performed. Bypass surgery—Coronary artery bypass graft surgery (CABG) is infrequently performed in patients with STEMI. The main indications are urgent CABG related to failure of fibrinolysis or PCI, or hemodynamically important mechanical complications. The benefit of revascularization must be weighed against the increase in mortality associated with CABG in the first three to seven days after STEMI. Thus, if the patient is stabilized, surgery should be delayed to allow myocardial recovery. Patients with the above critical conditions should undergo CABG during the initial hospitalization.

Contraindications to full-dose anticoagulation:

(1) Active bleeding disease;
(2) Recent major surgery;
(3) Severe hypertension (sustained BP > 190/110 mmHg);
(4) Hemorrhagic diathesis (congenital, hepatic, or drug-induced);
(5) Presence of purpura;
(6) Infectious endocarditis;
(7) Anticipated invasive bedside procedures (e.g., thoracentesis, arterial or venous line insertion).

10. Erythropoietin: Its non-erythropoietic effects including anti-inflammatory, antiapoptotic, and angiogenic properties may be cardioprotective in patients with acute ST-elevated MI.

11. Post-MI management:

(1) Stress testing: All post-MI patients should have a submaximal stress test (70% target load) after 5-7 days or a maximal stress test (85% target load) after 2-3 weeks.

(2) Postinfarction angina or ischemia on stress test: Angiography is recommended to determine the need for angioplasty or bypass surgery.

(3) Medical treatment: A beta-R blocker and aspirin should be given to all post-MI patients without a specific contraindication. ACE-I (inhibitor) should be used in cases with EF < 40%. Lipid lowering drugs should be used to maintain LDL < 100 mg/dL. Smoking and alcohol abstinence is necessary.
Complications of acute MI

1. Arrhythmias

   (1) Sinus bradycardia: Commonly seen in early stages of (inferior) MI due to sinus or A-V junctional block and may be protective. Usually no treatment is needed. If it’s severe, atropine or temporary pacing can be applied.

   (2) Premature atrial or ventricular contractions (PVC): Observation.

   (3) Tachyarrhythmias: Supraventricular (SVT) — adenosine is the #1 choice; ventricular tachycardia (V-tach) — lidocaine is the #1 drug (smaller dose for the elderly, and never used as prophylaxis because lidocaine can induce ventricular tachycardia or ventricular fibrillation by itself); ventricular fibrillation (V-fib) or asystole — immediate unsynchronized defibrillation and CPR to save life!

   (4) Temporary transvenous pacing — indications: Complete A-V block; 2nd degree A-V block (type 2); sinus bradycardia despite atropine use; LBBB during MI; new bifascicular block; with hypoperfusion.

2. Pump dysfunction: Left or right ventricular or bi-ventricular failure; ventricular aneurysm; infarct expansion. Severe left or bi-ventricular failure is an indication for intra-aortic balloon counterpulsation. This can increase cardiac output and perfusion through the coronary artery.

3. Mechanical disruption: Papillary muscle dysfunction or rupture (causing mitral regurgitation, with systolic murmurs at apex radiating to the left axilla), ventricular septal rupture (within 10 days, repairable), free wall rupture (causing cardiac tamponade, with 90% mortality), or pseudoaneurysm (risk of rupture). Treatment requires emergent surgical repair.

4. Acute pericarditis—Dressler syndrome—Post-MI syndrome: It’s immunologically based, with fever, malaise, pericarditis, pleuritis, and leukocytosis; usually late onset, 2-4 weeks post-MI. Aspirin is the effective therapy. Steroids should be avoided because they may hinder myocardial scar formation.

5. Thromboembolism: Mural thrombus with systemic embolism or DVT with prolonged immobilization. Frequent movements are the best prevention and treatment.

6. Postinfarction angina: If it’s after thrombolytic therapies, it should be treated with angioplasty or bypass surgery.

7. Recurrent infarction: It includes extension of existing infarction and re-infarction, with high mortality. Diagnosis is difficult but should be suspected if there is a persistent elevation or re-elevation of ST-segment and high CK-MB after 36 hours. Treatment is repeating thrombolysis or urgent cardiac catheterization and PTCA, along with standard medical therapies for MI.

8. Sudden cardiac death: Mostly due to ventricular fibrillation or/and asystole.

Right Ventricular Myocardial Infarction (RVMI)

RVMI mainly results from occlusion of the proximal right coronary artery, accompanying about 30% of the inferior LV-MI. The patient usually shows a typical right-sided infarction and heart failure, with the classic physical triad of hypotension, jugular vein distention (JVD), and clear lungs on auscultation. ECG usually reveals ST-segment elevations in an inferior and a posterior distribution (I, III, aVF, V4R).
**Diagnosis:** Based on the above “Triad”, increased cardiac enzymes, and abnormal ECG results (ST-elevation) in right ventricular leads (V4R).

**Treatment:** The primary treatment is *maintenance of the RV preload—fluids, NOT diuretics;* normal saline but not nitrates or opioids, and augmentation of the cardiac contractility—dopamine. Patients with predominant RVMI usually do not benefit from afterload reducing treatment with either an intraaortic balloon pump or vasodilating agents. It’s critical for early effective corrections to restore perfusion.

**Acute Coronary Syndrome (ACS)**

ACS refers to *any group of symptoms attributed to obstruction of the coronary arteries.* ACS usually occurs as a result of one of three conditions: *ST-elevation myocardial infarction (30%), non-ST-elevation myocardial infarction (25%), or unstable angina (38%).*

It is difficult to determine the precise etiology from its history and P/E alone. **The risk factors for ACS are the same as for CAD.**

**PEARLS—Diagnostic guidelines for ACS**

1. The most common symptom prompting diagnosis of ACS is *pressure-like chest pain (> 30 min with infarction),* often radiating to the left arm, and associated with anxiety, nausea, and diaphoresis.

2. **Lab diagnosis:** (1) ECG is abnormal immediately at onset of typical chest pain. **ST-T elevation** progresses to Q-waves or left branch block over up to 7 days. (2) Abnormal myoglobin starts 1-4 hrs after chest pain and lasts 1-2 days; **CK-MB** starts 4-6 hrs and lasts 3 days; troponin starts 2-4 hrs and lasts 7-10 days. **Troponin** cannot distinguish a reinfarction occurring several days after the first onset. Renal inefficiency can result in a false increase in troponin.

3. **Reinfarction:** If a patient presents with a new chest pain within a few days of the first infarction or attack, perform an ECG to detect new ST segment abnormalities. Elevated CK-MB levels after several days indicate new infarction.

**PEARLS—Therapeutic guidelines for ACS**

1. **ST-elevation MI:** Oxygen, aspirin and beta1 blockers should be started ASAP for best benefits. Primary PCI within 90 min of first medical contact is the goal. Thrombolysis within 30 min in hospital and 6-12 hours of onset of symptoms reduces mortality.

2. **Post-MI take-home medications:** Aspirin (or clopidogrel if aspirin-intolerant), beta1 blockers (metoprolol), ACE-I (or ARB—angiotensin-R blockers if cough-persistent), and statins.

3. **Glycoprotein IIb/IIIa inhibitors (abciximab, tirofiban, eptifibatide):** Useful in ACS with ST depression (non-ST elevation MI) and patients to undergo angioplasty and stenting. tPA is beneficial only with ST elevation MI. Heparin is best for non-ST elevation MI.
4. **In non-ST elevation ACS**, if patient is not better (persistent pain, S3 gallop, worse ECG changes, and rising troponin levels) after using all given medications, urgent angiography and possibly angioplasty (PCI) should be performed.

5. **The No.1 common cause of death is ventricular arrhythmia**—tachycardia and fibrillation. Always get ready to perform immediate electrical cardioversion or defibrillation.

   **Contraindications:** Do not use any “prophylactic antiarrhythmic medications for ventricular tachycardia or fibrillation” because it increases ventricular arrhythmia and mortality.

   Do not use nitrates and sildenafil together to avoid severe vascular complications.

   **Post-MI impotence:** Erection problem is mostly due to anxiety. Sexual activity can be recovered when the patient is asymptomatic.
PEARLS — Table 14-2: Toxicity and Specific Treatment of Common Drugs

<table>
<thead>
<tr>
<th>Drug / Toxicity and Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acyclovir</strong>: Renal tube crystal and obstruction, may lead to renal failure. <strong>Tx</strong>: Large fluid intake.</td>
</tr>
<tr>
<td><strong>ACE inhibitors (Captopril, etc.)</strong>: Cough (#1 common), rash, proteinuria, angioedema, taste changes, hypo-Na, and hyper-K.</td>
</tr>
<tr>
<td><strong>Aminoglycosides</strong>: Ototoxicity, nephrotoxicity, etc.</td>
</tr>
<tr>
<td><strong>Amiodarone</strong>: Pulmonary fibrosis (also with bleomycin), arrhythmia (prolonged Q-T as with quinidine), peripheral deposit (skin discolor, photosensitivity), hypo/hyper-thyroidism</td>
</tr>
<tr>
<td><strong>Amphotericin</strong>: Fever, chills, and nephrotoxicity; Mnemonic: “Ampho-terror”.</td>
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<tr>
<td><strong>Amantadine</strong>: Ataxia and livedo reticularis.</td>
</tr>
<tr>
<td><strong>Antimuscarinic, anticholinergic agents</strong>: Urinary retention, constipation, sedation, orthostatic hypotension, and paralysis. <strong>Antidote</strong>: Physostigmine.</td>
</tr>
<tr>
<td><strong>Antipsychotics</strong>: Extrapyramidal symptoms (dystonia, akathisia, and convulsion) — Treat with propranolol or diazepam; neuroleptic malignant syndrome (NMS) — Treat with dantrolene +/- bromocriptine or amantadine; anticholinergic effects — Treat with physostigmine.</td>
</tr>
<tr>
<td><strong>Azathioprine</strong>: Dose-related diarrhea, liver toxicity, and WBC decrease.</td>
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<tr>
<td><strong>AZT</strong>: Thrombocytopenia and megaloblastic anemia.</td>
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<tr>
<td><strong>Benzodiazepines (BZD)</strong>: Psychological and physical dependence; addictive effects with other CNS depressants. <strong>Antidote</strong>: Flumazenil. Caution: Flumazenil can cause seizures in chronic BZD-dependent patients.</td>
</tr>
<tr>
<td><strong>Beta-R-blockers (-olol)</strong>: Asthma exacerbation, A-V block, CHF, masking of hypoglycemia, and impotence. <strong>Antidote</strong>: Glucagon.</td>
</tr>
<tr>
<td><strong>Bile acid resins</strong>: GI upset and malabsorption of lipid-soluble vitamins and medicines.</td>
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<tr>
<td><strong>Ca-blockers</strong>: Cardiac depression, peripheral edema, and constipation.</td>
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<tr>
<td><strong>Carbamazepine</strong>: Agranulocytosis, aplastic anemia, and induction of p450.</td>
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<tr>
<td>Drug</td>
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<td>-----------------------------</td>
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<tr>
<td>Chloramphenicol</td>
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<td>Clonidine</td>
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<td>Clozapine</td>
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<tr>
<td>Corticosteroids</td>
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<tr>
<td>Cisplatin</td>
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<tr>
<td>Colchicine, meclocline, and lithium</td>
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<tr>
<td>Cyclophosphamide</td>
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<tr>
<td>Cyclosporine</td>
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<tr>
<td>Doxorubicin</td>
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<tr>
<td>Fluorquinolones (quinolones)</td>
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<td>Fluconazole (-azoledes)</td>
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<tr>
<td>Furosemide</td>
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<tr>
<td>Gemfibrozil, -statins</td>
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<tr>
<td>Halothane</td>
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<tr>
<td>Hydralazine</td>
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<td>Isoniazid (INH)</td>
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<td>Iron salts</td>
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<td>MAOIs</td>
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<td>Metyldopa</td>
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<td>Metronidazole</td>
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<td>Penicillin</td>
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<td>Phenytoin</td>
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<td>Prazosin</td>
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<td>Quinidine</td>
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SSRIs: More sexual dysfunction, GI stimulation (nausea/vomiting) and CNS toxicity (headache, insomnia, and tremor) than TCAs. Toxicity increases with coexistent MAOIs or TCAs. **Treatment of overdose:** Use BZD (lorazepam) for agitation, tremor, or seizures; NaHCO₃ for arrhythmia. Avoid serotonergic medications.

TCAs: More serious adverse effects than SSRIs—“3 Cs”: Cardiotoxicity (typical QRS widening in ECG), Convulsion, and Coma; anticholinergic effects (dry mouth, constipation, and urinary retention). **Tx:** NaHCO₃ is effective in treating arrhythmia (but not increasing excretion of TCAs). Use lorazepam for seizures.

Theophylline: Ventricular arrhythmia, GI upset, hyper-ventilation, convulsion, hypo-K, hypo-Mg, hypo-P, hyper-Ca, and hyperglycemia. **Tx:** 1. Active charcoal; 2. hemodialysis; lorazepam for convulsion; amiodarone for ventricular arrhythmia. Avoid lidocaine.


TPA, streptokinase: Bleeding tendency. **Antidote:** Aminocaproic acid.

Valproic acid: Neural tube defects (congenital) and hepatotoxicity (rare).

Vancomycin: CN8 toxicity, nephrotoxicity, and “red man syndrome” (due to histamine release, not allergy).

Warfarin: Bleeding tendency, teratogen, and drug-drug interactions. **Antidote:** Vitamin K or FFP (fresh frozen plasma), recovering in 1-2 days.
Chapter 15

SURGERY

PEARLS

Table 15-6: Important Differentiations of Abdominal Pain

<table>
<thead>
<tr>
<th>I. Left upper quadrant (LUQ)</th>
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<tbody>
<tr>
<td><strong>Gastritis</strong>: LUQ or epigastric pain or discomfort, heartburn, nausea, vomiting, and hematemesis.</td>
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<tr>
<td><strong>Gastric ulcer</strong>: LUQ or epigastric pain or discomfort associated with food intake.</td>
</tr>
<tr>
<td>Others: splenic rupture, infarct, abscess; splenomegaly; IBS (splenic flexure syndrome).</td>
</tr>
</tbody>
</table>

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<tr>
<th>II. (Middle) epigastrium</th>
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<tbody>
<tr>
<td><strong>Acute pancreatitis</strong>: Acute, persistent upper abdominal pain radiating to the back; usually after big meals/alcohol. <strong>Chronic pancreatitis</strong>: Chronic epigastric pain radiating to the back; associated with pancreatic insufficiency. <strong>Peptic ulcer disease</strong>: Epigastric pain or discomfort; occasionally localized to one side. <strong>GERD</strong>: Epigastric pain associated with heartburn, regurgitation, and dysphagia. <strong>Gastritis/gastropathy</strong>: Abdominal discomfort/pain, heartburn, nausea, vomiting, and hematemesis; may have history of ingestion of alcohol or NSAIDs. <strong>Functional dyspepsia</strong>: The presence of one or more of the following: postprandial fullness, early satiation, epigastric pain, or burning; no evidence of structural disease. <strong>Gastroparesis</strong>: Nausea, vomiting, abdominal pain, early satiety, postprandial fullness, and bloating. Most causes are idiopathic, diabetic, or postsurgical.</td>
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</tbody>
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<th>III. Right upper quadrant (RUQ)</th>
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<tr>
<td><strong>Biliary colic</strong>: Intense, dull pain located in the RUQ or epigastrium; associated with nausea, vomiting, and diaphoresis; usually lasting &gt; 30 min and alleviated within 1 hour; generally benign P/E results. <strong>Acute cholecystitis</strong>: Prolonged (&gt; 4 hours) RUQ or epigastric pain typically following fatty foods; fever. Patients will have abdominal guarding and Murphy’s sign. <strong>Acute cholangitis</strong>: Triad of fever, jaundice, RUQ pain; may have atypical presentation in older or immunosuppressed patients. <strong>Sphincter of Oddi dysfunction</strong>: RUQ pain similar to other biliary type pain without other apparent causes. <strong>Acute hepatitis</strong>: Dull RUQ pain with fatigue, malaise, nausea, vomiting, and anorexia; +/- jaundice, dark urine, and light-colored stools. Causes include hepatitis A, alcohol, and drug-induction. <strong>Perihepatitis (Fitz-Hugh-Curtis syndrome)</strong>: RUQ pain with a pleuritic condition; pain may radiate to the right shoulder; aminotransferases are usually normal or only slightly elevated. <strong>Budd-Chiari syndrome</strong>: Fever, abdominal pain and distention (from ascites), lower extremity edema, jaundice, gastrointestinal bleeding, and/or hepatic encephalopathy. <strong>Portal vein thrombosis</strong>: RUQ pain, dyspepsia, or gastrointestinal bleeding; most commonly associated with cirrhosis. Others: duodenal ulcer (perforation), hepatic abscess.</td>
</tr>
<tr>
<td>IV. Right lower quadrant (RLQ)</td>
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<tr>
<td><strong>Appendicitis</strong>: Periumbilical pain initially that radiates to the right lower quadrant; associated with anorexia, nausea, and vomiting.</td>
</tr>
<tr>
<td><strong>Cecal diverticulitis</strong>: Constant RLQ pain and low fever for several days; may have nausea and vomiting but no lower GI bleeding. Others: ectopic pregnancy, ovarian torsion.</td>
</tr>
<tr>
<td>V. Left lower quadrant (LLQ)</td>
</tr>
<tr>
<td><strong>(Sigmoid) diverticulitis</strong>: Constant LLQ pain and low fever for several days; may have palpable sigmoid mass but no lower GI bleeding.</td>
</tr>
<tr>
<td><strong>(Sigmoid) diverticulosis</strong>: LLQ colicky pain and relieved by defecation; may also have typical painless rectal bleeding or hematochezia (melena). Others: sigmoid volvulus, ectopic pregnancy, ovarian torsion.</td>
</tr>
<tr>
<td>VI. Lower abdomen (left or right)</td>
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<tr>
<td><strong>Infectious colitis</strong>: Diarrhea and associated abdominal pain +/- fever. Clostridium difficile infection can show acute abdomen and peritoneal signs in the setting of perforation and fulminant colitis.</td>
</tr>
<tr>
<td><strong>Nephrolithiasis</strong>: Usually mild to severe flank pain (left or right); may have back or abdominal pain.</td>
</tr>
<tr>
<td><strong>Ectopic pregnancy</strong>: Triad of amenorrhea, unilateral lower abdominal pain, and spotting vaginal bleeding (1-2 weeks after LMP). Others: twisted/ruptured ovary cyst, endometriosis, intestinal obstruction, abdominal abscess.</td>
</tr>
</tbody>
</table>
HIGH-YIELD CLINICAL IMAGES

(Courtesy images reorganized from www.images.google.com & related websites for public sharing)

Image 1-2. Normal heart anatomy and ECG (EKG): SA and AV nodes are supplied mostly by the right coronary artery (RCA), which supplies the inferior portion of the LV (via the posterior descending artery). Most coronary A. occlusions occur in the LAD (L-anterior descending A.), which supplies the anterior interventricular septum.

3a. A-V block: 1°: P-R interval >0.02s; 2°: P wave occurs without QRS; 3°: No P before QRS, escape rhythm.

4. Hypo-K and hyper-K: flat T wave vs peak T wave. QRS complex can be irregular with severe hyper-K.
5. Atrial fibrillation: wavy baseline without discernible P waves; variable QRS response
6. Atrial flutter: regular rhythm; “sawtooth” P waves; varying A-V conduction (5:1 and 4:1)
7. Ventricular tachycardia: ≥ 3 consecutive PVCs; regular, rapid wide-complex rhythms
8. Ventricular fibrillation: totally erratic tracing, requiring emergent defibrillation to save life
9. Anterior wall MI: ST-elevation in leads V1-V5 with reciprocal ST-T in the inferior leads (aVR, aVL)
10. Inferior wall MI: ST-elevation in leads II, III, and aVF with reciprocal ST-T in the anterior leads (V1-V3)
11. Chronic bronchitis: thickening of bronchial walls, increased linear markings + enlarged cardiac shadow
12. Lobar pneumonia: lobar consolidation and air bronchograms
13. Asthma, emphysema, and bronchiectasis: hyperinflated lungs and flattened diaphragm indicating asthma and emphysema; streaky shadowing and bronchial wall thickening in both lungs indicating bronchiectasis
14. Cardiogenic pulmonary edema (CHF): increased vascular shadows in all lobes + enlarged left atrium
15. Pneumoconiosis: multiple small irregular opacities and interstitial densities

16. Sarcoidosis: bilateral hilar adenopathy
17. Small-cell lung cancer with lymphadenopathy: CT confirms enlarged left hilum and mediastinum

18. Orbital cellulitis: proptosis, painful eyes, decreased eye movement, and red swollen eyelids
19. Chalazion: meibomian gland lipogranuloma
20. Hordeolum (Stye): “Horrible Staph”—eyelid infection
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