

NR 566 / NR566 Advanced Pharmacology Care of the Family Midterm Exam | Rated A | Latest, 2020 / 2021| Chamberlain College

written by

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1. Pneumonia goals of treatment

- Return to baseline respiratory status
- Fever resolves in 2 to 4 days
- Leukocytosis resolves by day 4 of treatment
- Chest x-ray may take 4 weeks or more to return

2. Common bacterial pathogens of Adult pneumonia

- *S. pneumoniae*
- Patients with underlying lung disease
- Nontypeable *Haemophilus influenzae* and *Moraxella catarrhalis*
- *Staph aureus*: co-pathogen with influenza
- *Mycoplasma pneumoniae*
- Viral pneumonia

3. Common bacterial pathogens of Adult Community-Acquired Pneumonia

- Consult current treatment guidelines for the most recent treatment guidelines for community acquired pneumonia (CAP)

4. Common bacterial pathogens of CAP in pregnant women

- Main pathogens are *S. pneumoniae*
- *H. influenzae*, *M. pneumoniae*, and viruses
- Macrolides
- Pregnancy category B: erythromycin, azithromycin
- Pregnancy category C: clarithromycin
- Comorbid conditions or recent antibiotics:
- Beta-lactam plus a macrolide

5. Common pediatric pneumonia pathogens

- *S. pneumoniae* is the most common cause of bacterial pneumonia in patients of all ages
- Increase in viral pneumonia with PCV7 vaccine
- Infants 4 to 16 weeks
- Consider chlamydia
- Over 5 years through adolescence
- Consider mycoplasma
- Community-acquired methicillin-resistant *staphylococcus aureus*

- Virus

6. Clinical practice guidelines for treatment of CAP

- Children under age 5 years
- Bacterial pneumonia (*S. pneumoniae*)
- Amoxicillin: 80 to 90 mg/kg/day
- Ceftriaxone: 50 mg/kg/day until able to take oral antibiotics
- Penicillin allergy: clindamycin or a macrolide
- Infant with suspected chlamydial pneumonia
- Azithromycin 20 mg/kg/day for 3 days OR erythromycin (EryPed) 50 mg/kg for 14 days
- Children 5 Years or Older
- Mycoplasma or other atypical most likely
- Azithromycin: 10 mg/kg on day 1 and 5 mg/kg on days 2 through 5
- Clarithromycin: 15 mg/kg per day in two divided doses (maximum 1 g/day)
- Erythromycin: 40 to 50 mg/kg/day

7. CAP treatment in pregnancy

- abx treatment:
- 1st choice: Erythromycin or azithromycin cat B. or Clarythromycin cat C.

8. Radiologic findings during CAP treatment

- assist in confirming the dx of pneumonia vs other resp disorders such as lung abscess or tuberculosis

9. Treatment of chlamydial pneumonia

- the standard treatment for infants is erythromycin

10. Nicotine patch teaching

- Advise patients to dispose of used nicotine patches out of the reach of children or animals. Enough nicotine is left in a used patch to lead to toxic levels in a child or small animal.
- The transdermal nicotine system, or "patch," provides a slow, cutaneous absorption of nicotine over many hours. The patch is applied to clean, nonhairy skin on the upper body or upper arm when the patient wakes up. Peak nicotine levels occur in 2 to 6 hours (brand-dependent) and then gradually decrease. Once the patch is removed, nicotine levels in the blood reach a nondetectable level in 10 to 12 hours in nonsmokers.

11. Nicotine gum patient teaching

- Patients complain about the taste of the nicotine gum. Suggest that the patient try the flavored variety, which patients seem to tolerate better.
- The patient should not eat or drink for 15 minutes before or while the lozenge is dissolving in the mouth. There may be a tingling sensation in the mouth as the lozenge dissolves.
- Chewing too quickly causes an excess amount of nicotine to be released into the bloodstream, producing nausea, throat irritation, and hiccoughs. The patient should avoid

smoking while chewing nicotine gum because toxicity symptoms may occur (nausea, vomiting, and headache).

12. Common side effects associated with smoking cessation therapy

- Constipation.

13. Contraindications for smoking cessation therapy.

- Hypersensitivity to nicotine.
- Myocardial infarction.
- Life-threatening arrhythmias.
- Severe or worsening angina pectoris.
- Bupropion is contraindicated in patients with seizure disorders, bulimia, and anorexia nervosa and within 14 days of MAOIs.

14. Tuberculosis Etiology

- Infectious disease caused by M. tuberculosis. Inhaled into the alveolus and spreads from lungs. M. tuberculosis grows slowly. Infection is spread almost exclusively by aerosolization of contaminated lung secretions

15. Rational drug selection for pregnancy for tuberculosis

- INH and RIF. EMB should be included unless INH resistance is unlikely. 6 month therapy. Pyridoxine (vit b6) 25 mg/d should be added to the regimen to decrease incidence of peripheral neuropathy assoc with INH.

16. Rational drug selection for children for tuberculosis

- INH and RIF are used for asymptomatic infection for 6-9 months. Multidrug regimens (INH RIF PZA EMB) are used for progressive disease. EMB may be used if risk of drug-resistant organisms is present. DOT should be used for all children.

17. Criteria for resistant TB diagnosis

- Primary resistance risk factors: exposure to a patient with drug-resistant TB, immigration from a country with a high prevalence of d-r TB, and greater than 4% incidence of d-r RB in the community.
- Acquired/Secondary risk factors: poorly or inadequately treated TB.
- DRUG RESISTANCE CAN ONLY BE PROVEN BY SUSCEPTIBILITY TESTING.
- Second-line treatment usually requires injectable medications, which complicates the treatment regimen. Fluoroquinolones such as levofloxacin, moxifloxacin, and gatifloxacin are all active against M. tuberculosis. Based on the evidence so far, levofloxacin is the preferred oral fluoroquinolone for treating drug-resistant TB or when first-line agents cannot be used because of intolerance

18. Risk factors for fatal hepatitis with INH use

- Pregnant and postpartum patients. Patients 50-64 years of age. Daily alcohol use, chronic liver disease, IV drug use. Black and hispanic women. INH has a black-box warning regarding the development of severe/fatal hepatitis, even after months of treatment.

19. Prophylactic TB treatment for high risk patients

- INH or via DOT with weekly INH/RPT. Standard anti-TB drugs ING and RIF used for asymptomatic infection for 6-9 months.

20. ACEIS and ARBS benefits in other conditions

- CAD, Post MI, heart failure.
- Clinical pearl: If you hear an abdominal bruit in a patient known to have vascular disease, give captopril, a short-acting ACEI, and measure serum creatinine prior to the dose and within 1 or 2 days after the dose. A rapid rise in the creatinine level suggests renal artery stenosis. A slower rise probably indicates a problem with poor hydration that can be corrected by rehydrating the patient and discontinuing or lowering the dose of any diuretics the patient is taking.

21. Management of ACEI side effects

- Education concerning hypotensive reactions.
- For ACEI-related cough, no effective treatment - may change to another ACEI or ARB.
- Change in taste resolves on its own within 8-12 weeks.

22. Amiodarone monitoring

- CXR, PFT every 3-6 mo.
- TSH, free T4 every 6 mo.
- Ophthalmic exam (slit lamp and fundoscopy) every 6 mo.

23. Flecainide monitoring

- ECG, liver function studies, serum drug levels.
- Watch for sinus node problems and AV block.
- Keep trough <1 mcg/mL.

24. Mexiletine monitoring

- Liver function studies, Aspartate aminotransferase (AST) elevations >3 times upper limit of normal have been observed.

25. Procainamide monitoring

- CBC and Antinuclear antibody (ANA) titer.
- At initiation of therapy for blood dyscrasias; at initiation for indication of lupus-like symptoms.

26. Propafenone monitoring

- Liver function studies, CBC, renal and liver function studies

27. Sotalol monitoring

- Fasting blood glucose

28. Cardiac glycosides monitoring

- In general, testing should be done when any of the following occurs:
 - The patient is taking other drugs that may alter the pharmacokinetics of digoxin.

- Steady state has been achieved (4 to 5 half-lives or 1 to 2 weeks) after starting a new dose.
- Toxicity is suspected.
- Confirmation of adequacy of maintenance dose is needed in situations of poor therapeutic response or patient adherence.
- A reference point is needed in adjusting a dose.
- The patient has progressive renal function decline.

29. Myocardial oxygen supply

- Supply is reduced by the following:
 - Hemodynamic factors such as increased resistance in coronary vessels, hypotension, and decreased blood volume. ACE inhibitors, beta blockers, direct renin inhibitors, and the dihydropyridine CCBs decrease peripheral resistance through their vasodilatory actions.
 - Cardiac factors such as decreases in diastolic filling time, increases in heart rate, and valvular incompetence. Beta blockers and non-dihydropyridine CCBs decrease heart rate. The beta blockers have the further advantage of preventing the recurrence of MIs.
 - Hematological factors such as the oxygen content of the blood, the acid-base status of the blood, and anemia.
 - Systemic disorders, such as shock, which reduce blood flow or the availability of oxygen.

30. Myocardial oxygen demand

- High systolic blood pressure, which increases the work the heart has to do to move blood from the left ventricle to the systemic circulation. One focus of anginal management is control of blood pressure. ACE inhibitors, beta blockers, direct renin inhibitors, and both types of CCBs decrease blood pressure.
 - Increased ventricular volume, which increases the work the heart has to do because the left ventricle must move more blood. ACE inhibitors reduce sodium and water retention.
 - Increased thickness of the myocardium (ventricular hypertrophy). The same mechanism that facilitated growth of the vessel walls in atherosclerosis also increases the thickness of the myocardium. ACE inhibitors play a major role here to decrease the remodeling. Beta blockers can assist in prevention of ventricular hypertrophy but play a smaller role.
 - Increased heart rate resulting from exercise, stress, hyperthyroidism, fever, anemia, hyperviscosity of the blood, or negative feedback systems' response to decreased cardiac output. Beta blockers can assist in decreasing heart rate resulting from conditions such as hyperthyroidism and from negative feedback patterns secondary to decreased cardiac output.
 - Conditions that heighten the myocardium's contractile response. Beta blockers and CCBs both have negative inotropic effects.

31. Bioavailability of bisphosphonate drugs and appropriate patient education

- Histamine2 blocking agents double alendronate bioavailability, but the impact is unknown. Aspirin may decrease the bioavailability of tiludronate by up to 50% when taken 2 hours after the tiludronate. Although indomethacin increases the bioavailability of tiludronate by 2- to 4-fold, the bioavailability is not significantly altered by diclofenac; therefore, each NSAID must be considered individually.

32. Adverse effects associated with long-term use of bisphonates

- Etidronate has also been associated with fractures in patients with Paget's disease when they are given high doses or when therapy lasted longer than 6 months. These patients must be carefully monitored with x-rays and laboratory work to assess for these lesions. The development of a rare form of subtrochanteric femur fracture in non-Paget's patients using bisphosphonates is under close scrutiny and has contributed to movement away from osteopenia prevention care to only osteoporosis therapy (FDA, 2010a).

33. Specifics about administration and education regarding pancreatic enzymes

- All doses are taken immediately before or with meals or snacks with a fatty component. Fruit, hard candy, fruit juice like drinks, tea or coffee, or popsicles do not require enzymes (CFF, 2009). Capsules may be opened and sprinkled on food. Capsules with enteric-coated beads should not be chewed. They may be sprinkled on soft acidic food that is not hot and that can be swallowed without chewing, such as applesauce or gelatin. Swallow immediately because the proteolytic enzymes may irritate the mucosa. Following with a glass of water or juice or eating immediately after taking the drug helps to ensure that the medication is swallowed and does not remain in contact with the mouth and esophagus for long periods. Pancrelipase is destroyed by acid. Proton pump inhibitors, sodium bicarbonate, or aluminum-based antacids may be used with preparations without enteric coating to neutralize gastric pH. Calcium- and magnesium-based antacids should not be used for this purpose because they interfere with drug action. Enteric-coated beads are designed to withstand the acid pH of the stomach. Enteric-coated formulations should not be mixed with alkaline food or the coating will be destroyed.

34. Common adverse effects with aromatase inhibitors

- Adverse effects for the drug class include various pain syndromes, vertigo, insomnia resulting in daytime sleepiness and confusion, increased risk of blood clots, and hair loss. A key concern is the loss of bone mass. Bone loss can be significant when considering the concurrent osteoporotic risks of postmenopause. Closer monitoring is required. All patients should be on calcium and vitamin D supplementation. A relative leukopenia can occur, but the incidence of viral and bacteria infections is not considered greater than matched groups (about 10%). Hypertension occurs in 10% of patients. A life-threatening increase in blood clotting can result in MI, stroke, or pulmonary embolus. Hot flashes can be intense.

35. Drugs associated risk for bone loss which should be monitored

- Aromatase inhibitors
- Thyroid hormones
- Glucocorticoids
- PPIs
- SSRIs

36. Clinical signs and symptoms DM

- Increased thirst
- Frequent urination
- Extreme hunger
- Unexplained weight loss
- Presence of ketones in the urine (ketones are a byproduct of the breakdown of muscle and fat that happens when there's not enough available insulin)
- Fatigue
- Irritability
- Blurred vision
- Slow-healing sores
- Frequent infections, such as gums or skin infections and vaginal infections

37. Risk factors & associated complications of DM

- Complications: stroke, heart attack, peripheral artery disease, diabetic retinopathy, cataracts, glaucoma, diabetic nephropathy, peripheral neuropathy, diabetic foot.
- Risk factors: >45 years old, physical inactivity, 1st degree relative with DM, high risk ethnic group (african american, hispanic, native american, asian american, and pacific islander), hx of gest DM, htn, HDL < 35, triglycerides >250, polycystic ovarian syndrome, acanthosis nigricans, hx of cardiovascular disease.

38. Diagnostic criteria of DM

- Acute symptoms of diabetes plus casual plasma glucose concentration ≥ 200 mg/dL.
- *Casual is defined as any time of day without regard to time since last meal. The classic symptoms of diabetes are polyuria, polydipsia, and unexplained weight loss.
- Fasting plasma glucose ≥ 126 mg/dL. * Fasting is defined as no caloric intake for at least 8 h.
- 2-h postload plasma glucose in an oral glucose tolerance test ≥ 200 mg/dL. The test uses a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.
- Hb A1c $\geq 6.5\%$.
- PRE-DIABETES:
 - o Fasting plasma glucose 100-125 mg/dL (IFG) or
 - o plasma glucose 140-199 mg/dL (IGT) 2 hr post-ingestion of standard glucose load (75 g) or
 - o Hb A1c 5.7%-6.4%

39. Criteria for screening asymptomatic adults

- Individuals ≥ 45 yr and who have a BMI ≥ 25 kg/m² should be tested. If normal, the test should be repeated at 3 yr intervals.

- Individuals <45 yr and who have a BMI ≥ 25 kg/m² and have additional risk factors should have more frequent testing.
- Additional risk factors are the following:
 - o Physically inactive
 - o First-degree relative with diabetes
 - o Members of high-risk ethnic group (African American, Hispanic, Native American, Asian American, Pacific Islander)
 - o Delivered a baby weighing >9 lb or previously diagnosed with GDM
 - o Hypertensive (B/P $\geq 140/90$ mm Hg)
 - o HDL cholesterol ≤ 35 mg/dL and/or triglyceride level ≥ 250 mg/dL
 - o Have polycystic ovary syndrome (PCOS)
 - o IGT or IFG on previous testing
 - o Have other clinical conditions associated with insulin resistance (PCOS or acanthosis nigricans)
 - o History of CVD

40. **Rapid Acting Insulin**

- Humalog, Novolog, Apidra

41. **Short Acting Insulin**

- Regular (Humulin R, Novolin R)

42. **Intermediate Acting Insulin**

- Isophane (NPH, Humulin N)

43. **Long Acting Insulin**

- Lantus, Levimir

44. **Fixed Combo Insulin**

- 70/30 (NPH/regular ratio)
- 50/50 (NPH/regular ratio)
- 75/25 (NPH/lispro)
- 70/30 (NPH/aspart)

45. **A1C Treatment Goal**

- Less than 7%

46. **Daily dose of insulin for initiation**

- 0.1/kg or 10 units

47. **Insulin Treatment Algorithm for Type 1 DM**

- Total daily insulin requirement is 0.3 to 0.5 units/kg body weight/d with titration to glycemic targets. Higher doses for acute illness. Adjustments made after reviewing patterns over 3 days. Hypoglycemia addressed first, then hyperglycemia. Adjustments up or down done in increments of 1 unit.

48. A1C monitoring during oral or insulin diabetes management

- Because Hb A1c reflects mean glycemia over the preceding 2 to 3 months, it should be measured at least twice a year if patients are meeting treatment goals or have stable glycemic control; it should be measured every 3 months if therapy has changed or if patients are not meeting treatment goals

49. Correlate mean plasma glucose level according to A1C

- Hemoglobin A1c Levels
- Mean Plasma Glucose (mg/dL)
- 6=
- 126
- 7=
- 154
- 8=
- 183
- 9=
- 212
- 10=
- 240
- 11=
- 269
- 12=
- 290

50. Clinical manifestations of diabetic autonomic neuropathy

- Resting tachycardia, exercise intolerance, orthostatic hypotension, constipation, gastroparesis, erectile dysfunction, sweat gland dysfunction, impaired neurovascular function, and the potential for autonomic failure in response to hypoglycemia.

51. Hypoglycemia treatment (amount of carbohydrates and examples)

- They should take 15 gm of carbohydrate and recheck their sugars in 15 minutes.

52. Drug monitoring with metformin

- Monitor B12 levels

53. Antidiabetic medications associated with photosensitivity

- Sulfonylureas

54. Antidiabetics to avoid in the elderly & why

- Sulfonylureas produces severe hypoglycemia.
- Glimepiride produces hypoglycemia.
- Glyburide is the most likely to cause hypoglycemia.
- Metformin due to older adults often have renal insufficiency or heart failure.
- Alpha-glucosidase inhibitors are not well tolerated.
- All meds should be started at the lowest possible dose.

55. Improving patient compliance with diabetes treatment

- Nonadherence to the treatment regimen may result in increased risk for complications and reduced life expectancy. Healthcare providers should be aware of potential problems with nonadherence, discuss the importance of adherence at each follow-up visit, and assist patients in removing barriers to adherence such as lack of social support and cost of the treatment regimen. A team approach with the patient as an active partner should be maximized. Ways to deal with nonadherence are discussed in Chapter 6. Patient education booklets are available from the ADA, which can be accessed on the Internet at www.diabetes.org.

56. Diabetic medications to avoid when taking digoxin

- Metformin - dig may increase the effect of metformin leading to lactic acidosis.

57. Diabetic medications with need for renal dose adjustment

- Metformin

58. Diabetic medications associated with increased risk for genital mycotic infections

- Selective Sodium Glucose Co-transporter 2 (SGLT-2)

59. Time anticipated for total reversal of hyperthyroid symptoms with methimazole

- A treatment typically requires 6 to 12 months for total reversal of hyperthyroid symptoms.

60. Routine testing with drug therapy

- TSH and free T4 levels
- Every 4 to 8 weeks until euthyroid
- During pregnancy evaluate at 8 weeks' and 6 months' gestation

61. Recommend dietary iodine intake

- 100-150 mcg/day for normal thyroid function

62. Drugs that increase metabolism of T4

- Carbamazepine, Phenytoin

63. Symptoms of Hyperthyroidism

- increased CO, decreased peripheral vascular resistance, tachycardia at rest, arrhythmias, dyspnea and reduced vital capacity, increased appetite with weight loss, diarrhea, nausea, vomiting, abdominal pain, sweating, flushing warm skin, hair loss, nails grow away from nail beds, oligo/amenorrhea, impotence/decreased libido in men, restlessness, short attention span, fatigue, insomnia, emotional lability, enlarged gland.

64. Symptoms of Hypothyroidism

- reduced stroke volume and HR, increased peripheral resistance to maintain BP, bradycardia, macrocytic anemia assoc. With B12 deficiency, dyspnea, hypoventilation, CO₂ retention, decreased appetite, constipation, weight gain, fluid retention, dry flaky skin, dry hair, slow wound healing, cool skin, decreased libido, confusion, slow speech, memory loss, clumsy movements.

65. Hyperthyroid drugs with risk for hepatic toxicity

- propylthiouracil

66. Bile acid sequestrants absorption and administration

- affect LDL-C with a modest increase in HDL-C. They are not commonly prescribed to treat dyslipidemias in patients with diabetes. Not only do they increase TGs but they may pose problems for patients with diabetic gastroparesis. The increase in TG is especially of concern in diabetics because the pancreas is already under stress.

67. Levothyroxine administration instructions

- Take first thing in the morning at least 30, preferably one hour before eating. On an empty stomach with only water. Achieve consistency in taking the med to avoid fluctuating thyroid levels.

68. Differentiate between primary and secondary hypothyroidism

- Primary disorders include the following:
 - o Defective hormone synthesis resulting from autoimmune thyroiditis, endemic iodine deficiency, or antithyroid drugs that were used to treat hyperthyroidism
 - o Congenital defects or loss of tissue after treatment for hyperthyroidism
- Secondary causes of hypothyroidism, which are less common, include conditions that cause either pituitary or hypothalamic failure. In secondary disorders, the TSH response is inadequate so that the gland is normal or reduced in size, with both T₃ and T₄ synthesis equally reduced.

69. Differentiate between primary and secondary hyperthyroidism

- Primary is the term used when the pathology is within the thyroid gland.
- Secondary hyperthyroidism is the term used when the thyroid gland is stimulated by excessive TSH in circulation.

70. Precautions and testing for xanthine derivatives

- Monitored closely for signs of toxicity
- When therapy is initiated, theophylline levels should be drawn frequently as the dosage is titrated.
- Signs of toxicity- serum theophylline level should be drawn
- Once stabilized, monitoring should be done every 6 to 12 months

71. Mild intermittent asthma

- Symptoms occur less often than twice a week and the patient is asymptomatic between exacerbations; nighttime symptoms occur less than twice a month; and peak expiratory flow (PEF) is greater than 80% predicted. The use of short-acting beta₂

agonists (SABA) should be less than twice a week, unless used for exercise-induced bronchospasm (EIB).

72. Mild persistent asthma

- Symptoms occur more often than twice a week but less often than once a day and exacerbations may affect activity; nighttime symptoms occur 3 to 4 times a month; and PEF is greater than 80% predicted. Patients with mild persistent asthma may use their short-acting beta₂ agonists more than twice a week but not daily, and not more than once daily.

73. Moderate persistent asthma

- The patient is having daily symptoms; requires daily use of a beta₂ agonist; exacerbations affect normal activity; nighttime symptoms occur more often than once a week; and PEF is greater than 60% to less than 80%.

74. Severe persistent asthma

- The patient has some degree of symptoms all the time; extremely limited physical activity and frequent exacerbations; frequent nighttime symptoms, often 7 days a week; and decreased lung function (PEF less than 60% predicted). Table 30-1 outlines the classifications of asthma severity in patients aged 12 years or older.

75. Risk factors for fatal asthma attacks

- Previous severe exacerbations requiring intubation or ICU.
- Two or more hospitalizations.
- More than 3 ED visits in the past year.
- Use of more than 2 SABA canisters per month.
- Difficulty perceiving airway obstruction or worsening asthma.
- Low socioeconomic status or inner-city residence.

76. Asthma step therapy

- The Expert Panel Report 3: Guidelines (NAEPP, 2007) recommends a stepwise approach to the pharmacological management of asthma. Management can begin at a higher level and gradually step down or start low and move up, depending on the patient's status when beginning treatment.
 - o Step 1: SABA PRN
 - o Step 2: Low dose ICS
 - o Step 3: Medium dose ICS
 - o Step 4: Medium dose ICS + LABA or Montelukast
 - o Step 5: High dose ICS + LABA or Montelukast
 - o Step 6: High dose ICS + LABA or Montelukast + oral corticosteroids

77. COPD therapy and goals of treatment

- Slow the disease process
- Maintain quality of life
- Medications
- Quit smoking

- Nutrition
- Infection protection
- Exercise -pulmonary rehabilitation improve function and quality of life

78. Respiratory drug interactions with digoxin

- Albuterol can lower digoxin levels in body

79. Patient education for treatment of asthma

- Basic facts about asthma.
- Medication skills.
- Self-monitoring skills.
- Specific to drug therapy.
- Reasons for the drug.
- Drugs as part of the total treatment regimen.

80. Use of oral corticosteroids in the treatment of COPD

- Corticosteroids have nonspecific anti-inflammatory activity at multiple points in the inflammatory process. Because of the cellular-level airway changes that define COPD, corticosteroids' effects are less dramatic in COPD than those seen in asthma. Yet corticosteroids are key components in the management of stable COPD and COPD exacerbations.
- The use of daily inhaled corticosteroids (ICS) in the COPD patient has mixed results in clinical studies.
- inhaled corticosteroids do not modify the long-term decline in FEV1 seen in COPD, but as both monotherapy and in combination with inhaled bronchodilators they decrease exacerbations and improve health status in patients with symptomatic COPD
- Therefore, the current ACP and GOLD guidelines recommend starting a patient on moderate- to high-dose inhaled corticosteroids
- Combination therapy of ICS and a long-acting beta agonist, such as Advair (salmeterol/fluticasone), is more effective in decreasing exacerbations than either agent alone
- combination therapy should be considered in any patient with moderate to severe COPD defined by FEV1 less than 60% of predicted.
- Oral corticosteroids are useful in the short-term treatment of acute COPD exacerbation

81. Use of oral corticosteroids in the treatment of asthma

- Inhaled corticosteroids are the preferred long-term control medications for managing the inflammatory process associated with asthma.
- Dosages for the inhaled corticosteroids vary with the specific product and the delivery method.
- The patient with persistent asthma is started on inhaled corticosteroids according to the
- All patients with mild persistent asthma are started on a low dose of inhaled corticosteroids.

- Children older than age 12 and adults may be treated with cromolyn, nedocromil, leukotriene modifiers, or theophylline as alternative therapy.

82. **Angina**

- Pain in the heart region caused by lack of oxygen. Ischemia caused by the imbalance between myocardial oxygen supply (MOS) and myocardial oxygen demand (MOD) produces pain referred to as _____.

83. **Angina risk factors**

- smoking, hypertension, hypercholesterolemia, low high-density lipoprotein (HDL) cholesterol, diabetes mellitus.

84. **Class I Angina**

- Proven coronary artery disease without symptoms
- Ordinary physical activity, such as walking or climbing stairs, does not cause angina. Angina occurs with strenuous, rapid, or prolonged exertion at work or recreation.

85. **Class II Angina**

- Angina only with unusually strenuous physical exertion
- Slight limitation of ordinary activity. Angina occurs on walking or climbing stairs rapidly; walking uphill; walking or stair climbing after meals; in cold wind; under emotional stress; or only during the few hours after awakening.
- Walking more than two blocks on the level and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions does not cause angina.

86. **Class III Angina**

- Angina during routine physical activity
- Marked limitations of ordinary activity. Angina occurs on walking one to two blocks on the level and climbing one flight of stairs in normal conditions and at a normal pace.

87. **Class IV Angina**

- Angina during minimal activity or rest
- Inability to carry on any physical activity without discomfort.
- Angina may occur at rest.

88. **Short-acting Nitrates**

- can be used in patients with mild, stable CAD for immediate relief on an as needed basis
- Short-acting forms are also less expensive but must be taken several times each day.

89. **Long-acting nitrates**

- can be used for treatment of angina in patients when beta blockers and ACE inhibitors are ineffective or contraindicated
- For patients who respond well to sublingual or translingual nitroglycerin and who experience angina episodes more than "rarely," and who are intolerant of beta blockers, these oral or transdermal nitrates are generally indicated.

90. Angina Treatment

- All appropriate patients with angina should be on aspirin 81 to 162 mg/d
- If aspirin is contraindicated, clopidogrel (Plavix) 75 mg daily may be an effective substitute
- Patients with angina only on exertion, a normal resting ECG, and symptoms that can be controlled by rest and intermittent nitroglycerin
- ACE inhibitors and beta-adrenergic blockers are the mainstays of initial drug therapy for patients with angina
- The second-generation dihydropyridine CCBs (amlodipine and felodipine) and long-acting nitrates can be used for treatment of angina in patients when beta blockers and ACE inhibitors are ineffective or contraindicated
- Short-acting, sublingual nitrates can be used in patients with mild, stable CAD for immediate relief on an as needed basis

91. Drugs which increase myocardial oxygen supply

- One mechanism available to increase oxygen supply is to dilate the coronary arteries and bring more blood flow to the myocardium. Nitrates can do this in patients with normal hearts.
- ACE inhibitors also affect both the MOS and the MOD sides of the equation.
- statins should be included in the treatment regimen for angina. Their role is on the MOS side of the equation; reduction in LDL cholesterol levels plays a significant role in decreasing the formation of atherosclerotic plaque. This plaque is central to the narrowing of the arterial lumen.

92. Long-acting nitrate effects

- They widen your blood vessels to increase blood flow to the heart.

93. Nitroglycerine rationale for route of administration chosen

- For patients who respond well to sublingual or translingual nitroglycerin and who experience angina episodes more than "rarely," and who are intolerant of beta blockers, long-acting oral or transdermal nitrates are generally indicated
- Nitroglycerin 0.3 to 0.4 mg sublingual tablets or translingual spray is used for immediate symptom relief.

94. Patient education for nitroglycerine

- All patients with angina should carry some form of rapid-acting nitrate with them at all times. They should be instructed to use this medication at the first sign of angina, even if they are uncertain if the symptoms are angina. If symptoms have not improved after 5 minutes of taking one dose of this drug, the patient should call 9-1-1 for medical attention. This recommendation has been updated from the previous recommendation of taking up to three doses before calling EMS.
- The timing of the nitrate-free interval should coincide with the time of fewest episodes of angina, which is typically at night. The administration schedule that seems most effective is 7 a.m. and 2 p.m. daily.
- Headache is the most common adverse reaction but resolves over time. Starting with low doses and slowly increasing the dose reduces the incidence of headache.

95. Reducing tolerance to nitrate therapy

- Among the available drugs, the most cost-effective are isosorbide mononitrate (Imdur) given daily or nitroglycerin transdermal patches applied daily with a 10 to 12 hour nitrate-free interval to prevent nitrate tolerance.
- The timing of the nitrate-free interval should coincide with the time of fewest episodes of angina, which is typically at night.

96. Initial drug therapy for patients with angina and alternative agents

- All appropriate patients with angina should be on aspirin 81 to 162 mg/d
- If aspirin is contraindicated, clopidogrel (Plavix) 75 mg daily may be an effective substitute
- Patients with angina only on exertion, a normal resting ECG, and symptoms that can be controlled by rest and intermittent nitroglycerin
- ACE inhibitors and beta-adrenergic blockers are the mainstays of initial drug therapy for patients with angina
- The second-generation dihydropyridine CCBs (amlodipine and felodipine) and long-acting nitrates can be used for treatment of angina in patients when beta blockers and ACE inhibitors are ineffective or contraindicated
- Short-acting, sublingual nitrates can be used in patients with mild, stable CAD for immediate relief on an as needed basis

97. Drugs contraindicated in patients with angina

- Beta blockers are contraindicated for patients with severe, uncontrolled reactive airway diseases and vasospastic angina

98. Patho of heart failure

- Heart tries to compensate for not pumping an adequate amt of blood
- Increased heart rate
- Blood vessels dilate
- Heart hypertrophy
- Right side triggered by MI or lung dx
- Vascular resistance
- Greater O₂ demand
- Cells become hypoxic

99. HF Stage A

- High risk for development of HF; no underlying structural cardiac disease (HTN, DM, hyperlipidemia, etc)
- ACE inhibitors
- Treat HTN + lipids
- Lifestyle changes

100. HF Stage B

- Structural heart disease but asymptomatic.
- ACE inhibitors

- Beta-blockers

101. HF Stage C

- Structural heart disease with past or current symptoms of HF
- ACE inhibitors and beta-blockers
- Diuretics
- Digitalis
- Dietary salt restriction

102. HF Stage D

- End-stage disease. Requires specialized treatment strategies such as mechanical circ support, continuous inotropic infusions, cardiac transplantation, or hospice care.

103. Systolic Dysfunction

- Left ventricular dysfunction (systolic heart failure) begins with injury to the myocardium and is usually a progressive process, even in the absence of additional myocardial insults. The principal mechanism relates to remodeling, which occurs as a homeostatic mechanism to decrease wall stress through increases in wall thickness.

104. Diastolic Dysfunction

- Diastolic dysfunction, also known as heart failure with preserved ejection fraction (HF-pEF), results from inadequate relaxation and loss of muscle fiber elasticity, resulting in a slower filling rate and elevated diastolic pressures. Although cardiac output is reduced, ejection fractions remain within normal limits

105. Diastolic Dysfunction Causes

- Valvular dysfunction, hypertrophic and ischemic cardiomyopathy, uncontrolled HTN, hypothyroidism.

106. Systolic Dysfunction Causes

- Injury to the myocardium

107. Diagnostic testing for HF

- 2D ECG with Doppler flow studies - most useful!
- Chest xray, CBC, urinalysis, serum electrolytes, BUN, creatinine, HbA2c, liver fxn studies, fasting lipid profiles, and thyroid-stim hormone.
- Brain natriuretic peptide (BNP) to id patient with elevated left-ventricular filling pressures.

108. Limited role of digoxin in treatment of HF

- Although digoxin increases the force of contraction and modulates the RAAS, thereby improving functioning and symptoms and reducing hospitalizations, it has little if any effect on mortality. Published data suggest that digoxin does not improve quality of life, symptoms, or mortality rates

- The development of ACE inhibitors, combined with the risks of toxicity and multiple drug interactions associated with the cardiac glycosides (CGs), has moved digoxin to a third-line drug except for selected cases.

109. Symptoms of digoxin toxicity

- Confusion.
- Irregular pulse.
- Loss of appetite.
- Nausea, vomiting, diarrhea.
- Fast heartbeat.
- Vision changes (unusual), including blind spots, blurred vision, changes in how colors look, or seeing spots.

110. Drugs contraindicated in patients with HF

- a triple combination of ACE inhibitor, ARB, and beta blocker should be avoided because it may lead to additional undesired side effects.
- Non-steroidal anti-inflammatory drugs (NSAIDS). These include: ibuprofen, Advil, Motrin, Aleve, Toradol, Celebrex. These medicines hold fluid and cause swelling. They also can harm your kidneys.
- Cold and cough medicines with pseudoephedrine or phenylephrine. Check with your doctor before using a cold medicine.
- Alka-Seltzer® - this has too much sodium (salt).
- Calcium channel blockers such as diltiazem (Cardizem) or verapamil (Calan, Verelan).
- These lessen the heart's ability to pump if you have systolic heart failure. They may be used if you have diastolic heart failure or hypertrophic cardiomyopathy.

111. Hyperlipidemia Diagnosis

- Guidelines for identifying risk for CAD and CVD have traditionally focused on serum cholesterol levels above 200 mg/dL, fasting triglyceride TG levels above 150 mg/dL, and LDL levels above 100 mg/dL. The newest guidelines place a stronger emphasis upon individual risk factors with lifestyle and pharmacological therapies individualized to reduce that risk.

112. Hyperlipidemia Treatment Goals

- The therapeutic goal for the management of hyperlipidemia is to reduce morbidity and mortality from CVD by reducing atherosclerosis. Research suggests that at least a 30% to 40% decrease in LDL levels needs to be achieved to reach this goal

113. Hyperlipidemia Risk Factors

- Age
- Male: ≥ 45
- Female: ≥ 55
- Family history
- Premature CHD (MI or sudden death before 55 yr in father or other male first-degree relative or before 65 yr in mother or female first-degree relative)
- Cigarette smoking

- BP \geq 140/90 mm Hg or on antihypertensive medication
- HDL \leq 40 mg/dL
- Diabetes mellitus
- Presence, especially if poorly controlled

114. Hyperlipidemia testing and monitoring

- Monitoring for effectiveness of dietary therapy involves weight loss, BMI reduction, and lipid lowering.
- Drug therapy is not usually initiated until a 3-month trial of dietary therapy has been completed; the exception is concurrent drug therapy for those patients with DM or metabolic syndrome.
- Baseline lab data
- Lipids
- Cholesterol
- (liver function, ALT or AST, and CK) should be gathered before drug treatment begins. Serial monitoring of liver labs are no longer indicated for most patients. Specific diagnostic tests for monitoring each drug class are discussed in Chapter 16.
- Intermittent monitoring of HDL and non-HDL levels is a practice that provides a clearer picture of the actual CVD risk status for the patient but may not be covered by insurance.
- Attention to reductions in the C-reactive protein levels is more critical if measuring treatment effect.

115. Statin Strengths

- High-Intensity Rx Daily
- Est. 50% reduction in LDL
- Atorvastatin (40-80)mg daily
- Rosuvastatin (20-40)mg daily
- Moderate-Intensity Rx Daily
- Est. 30%-49% reduction LDL
- Atorvastatin (10-20) mg
- Fluvastatin 20-40 mg
- Fluvastatin XL 80 mg
- Lovastatin 20 m
- Pitavastatin (2-4) mg
- Pravastatin (40-80) mg
- Rosuvastatin (5-10) mg
- Pravastatin 10-20 mg
- Low-Intensity Rx Daily
- Less than 30% reduction
- Fluvastatin 20-40 mg
- Lovastatin 40 mg
- Pitavastatin 1 mg
- Simvastatin (20-40) mg
- Simvastatin 10 mg

116. Hyperlipidemia Treatment and Drug Selection

- Central aspects of treatment are lifestyle modifications, especially dietary, which include the reduction of elements that are often perceived as "making food taste good."
- The factors to consider before developing a treatment plan are the presence or the absence of CVD, any associated risk factors, specific patient variables and desires, patient interest, plus a realistic consideration of a cost-benefit ratio.
- The level of individual risk has become a cornerstone of the new guidelines. The following sections blend the new guidelines with the decades of evidence of all risk factors for the prescriber to consider when selecting medications.
- The higher the CV risk, the more aggressive the statin treatment recommendations in the new guidelines.
- Any risk prediction over 7.5% is now earmarked as needing a discussion about whether to start statin therapy
- Four statin benefit groups
- Evidence of clinical ASCVD
- Primary LDL >190 mg/dL
- DM patients with LDL 70-189 mg/dL without clinical ASCVD (age 40-75)
- Patients age 40-75 without DM or ADCVD with a 10 yr risk of 7.5% or higher and LDL of 70-189 mg/dL.
- Statins allow most high-risk patients to attain lowered serum LDL levels. Patients treated with a statin may also see a modest decrease in serum TG and an increase in serum HDL.
- Nicotinic acid is best for treating patients who have elevated total cholesterol and TG and low HDL levels who cannot tolerate statins.
- Bile acid-binding resins have a strong record of efficacy and safety and are most useful for patients with moderately elevated LDL levels and a low CVD risk profile who are unable to reduce their LDL by TLC alone.
- Fibric-acid derivatives are effective triglyceride-lowering drugs that may modestly lower LDL and raise HDL for some patients. Because these drugs usually do not produce substantial reductions in LDL cholesterol, they are not appropriate for effective lowering of LDL levels as a primary LDL-lowering agent. They can be valuable in combination with a statin for patients with very high triglyceride levels, for diabetic patients with elevated TG and good renal function, and for patients with familial dysbetalipoproteinemia.
- For patients with elevated LDL and TG below 200 mg/dL, the main goal of treatment is to lower LDL levels first as hypertriglyceridemia does not carry the same risk for CVD as elevated LDL cholesterol levels. Combination therapy can be achieved by ordering two separate agents or by ordering more convenient, but more expensive, single-dosage formulations.

117. Alternative Hyperlipidemia Therapies

- Omega 3 Fatty Acids
- Omacor is a prescription formulation that is a more concentrated (840 mg) and quality-controlled source of both the eicosapentanoic acid (EPA) and docosahexaenoic acids (DHA) than those found in OTC supplements (avg 200 to 500 mg). Omacor has the same "fish burp" side effect as its OTC counterparts but may be more tolerable to patients because of its twice-a-day dosing schedule.

118. Benefits and mechanism of action for non-statin hyperlipidemia medications

- Nicotinic acid- lowers total cholesterol and triglycerides and raises HDLs
- Bile-acid-binding resins- useful in pts with moderately elevated LDLs and low CVD risk profile, who are unable to reduce LDL by TLC alone
- Fibric-acid derivatives- effective trigly lowering drugs that may modestly lower LDL and raise HDL for some patients

119. Prehypertension

- 120-139/80-89

120. Hypertension stage 1

- 140-159/90-99

121. Hypertension stage 2

- >160/>100

122. HTN risk factors

- obesity, high sodium intake, sedentary lifestyle, excessive use of alcohol, smoking

123. HTN Treatment

- Without compelling indications and SBP 159 or DBP 90-99: Thiazide-type diuretics for most; may consider ACEI, ARB, CCB or combination.
- Without compelling indications and SBP >160 or DBP >100: 2-drug combination - thiazide-type diuretics and ACEI, ARB, BB or CCB.
- WITH compelling indications: other hypertensive drugs (other diuretics, ACEI, ARB, alpha blockers, CCB as needed)

124. Antihypertensives contraindicated in pregnancy

- ACE Inhibitors, Angiotensin II receptor blockers

125. Preferred antihypertensives for blacks

- Calcium Channel Blockers

126. Contraindications for beta blockers

- contraindicated in patients with symptomatic bradycardia, AV block, decompensated heart failure, and asthma.

127. Precautions for beta blockers

- Beta blockers should be used with caution in patients with poorly controlled diabetes mellitus because beta blockers can prolong or enhance hypoglycemia by interfering with glycogenolysis. Beta blockers may also mask the signs and symptoms of acute hypoglycemia. Because they may mask the clinical signs of hypothyroidism, beta blockers should be used with caution in patients with hyperthyroidism.

128. Education for beta blockers

- For patients with diabetes who must take a beta blocker, the diaphoresis associated with hypoglycemia is not masked by these drugs. Patients should be taught to recognize this indication of possible hypoglycemia and test their blood glucose levels whenever unexplained diaphoresis occurs.

129. Antihypertensive step therapy recommendations

- As a general rule, the following suggestions can lead to progress in achieving the BP goal in a primary care population:
 - Set an appropriate therapeutic BP goal based on individual patients and their compelling indications.
 - Be patient and work on attaining the BP goal over many weeks to months. Moving to lower BP quickly is more likely to produce side effects to the drugs that lead to nonadherence. There is no evidence that faster is better except in cases of extreme values.
 - Titrate BP medications no more often than every 4 to 6 weeks. The body needs time to demonstrate full response to the drug.
 - Do not automatically assume symptoms reported by patients are caused by the drug. What may appear to be adverse responses may have other reasons for occurrence. Assigning all symptoms to drug effects may result in changing a drug that is actually working well. Antihypertensive drugs alleviate more adverse responses than they cause.
 - Plan at the beginning of therapy for the use of more than one drug. A single drug is not likely to provide BP control to goal level if the patient is more than 15/10 mm Hg higher than the goal. Explaining to the patient early in treatment the likelihood of more than one drug decreases the risk for nonadherence.
 - Do not ignore ISH in the elderly. Treat to goal SBP in older adults even if DBP is normal, but go more slowly. Titrate to the target of 150/90.
 - Extracellular fluid volume may need to be controlled in order to achieve BP goals. Include a diuretic in any treatment regimen that includes more than one agent.

130. Preferred class for initial drug therapy in HTN treatments

- thiazide-type diuretics

131. Drug of choice for gout

- Allopurinol

132. Allopurinol adverse effects

- poor urate clearance with renal impairment, hepatotoxicity, weight loss, pruritis, rash--> more severe, fever, chills, arthralgia, cholestatic jaundice, eosinophilia, leukocytosis, leukopenia.

133. Allopurinol monitoring

- Check liver and renal function

134. Role of NSAID use in treatment of gout

- It is recommended that NSAIDs or colchicine be administered prophylactically for the first 6 months of febuxostat therapy to prevent acute gout flare.

135. Gout medications that require renal or hepatic dose adjustments

- Allopurinol, Colchicine, Salicylates

136. Allopurinol drug interactions

- ACEI - hypersensitivity.
- Aluminum salts - decreased effects.
- Ampicillin - rate of amp rash higher.
- Anticoagulants- enhanced anticoag (use)
- Cyclophosphamide - increased risk for bleeding.
- Theophylline - toxicity risk. Warfarin
- Thiazide diuretics - increased incidence of hypersensitivity reactions.
- Thiopurines - clinically significant increases in pharm and toxic effect of thio.
- Uricosuric agents - increase excretion of oxypurinol.

137. Signs of aspirin toxicity

- Signs of salicylate poisoning appear at serum levels of 30 to 60 mg/dL.
- Respiratory alkalosis is seen initially.
- Hyperpnea and tachypnea occur as a result of increased CO₂ production and a direct stimulatory effect of the salicylate on the respiratory center in the brain.
- Other symptoms include nausea, vomiting, hypokalemia, tinnitus, disorientation, irritability, seizures, dehydration, hyperthermia, thrombocytopenia, and other hematological disorders.