Chapter 11

- Know the difference between hypertensive urgency and hypertensive emergency
- Be aware of compensatory responses to antihypertensive drugs (Table 11-1)
- Know sympathoplegics that act in the CNS – (Clonidine, Methyldopa)

_ Mechanisms of action
_ Effects
_ Clinical uses
_ Toxicity

- Be aware that ganglion blocking drugs and that they are very potent
- Recollect connection of MAO inhibitors with hypertension
- Know adrenergic blockers

_ Mechanisms of action
_ Effects
_ Clinical uses
_ Toxicity

- Vasodilators

__Names
_ Mechanisms of action
_ Effects
_ Clinical uses
_ Toxicity

- ACE and Renin inhibitors

_Names
_ Mechanisms of action
_ Effects
_ Clinical uses
_ Toxicity

- Treatment approaches for Hypertensive emergency
- Drug of choices to treat hypertension in different clinical scenarios

Chapter 17

- Physiologic effects of Angiotensin
- Angiotensin antagonists

_Names
_ Mechanisms of action
_ Effects
_ Clinical uses
_ Toxicity
_ Effects on cardiac remodeling

- Know vasopeptidase inhibitors

_Names

_ Mechanisms of action
_ Effects
_ Clinical uses
_ Toxicity
- Physiologic effects of Bradykinin and drugs that interfere with it (Ecallantide, Icatibant)

_Names
_ Mechanisms of action
_ Effects
_ Clinical uses
_ Toxicity
- Know names of natriuretic peptides (ANP,BNP)

_ Clinical implications
_ Effects
_ Know what Nesiritide is
- Know effects of endothelins
- ET A antagonists – Bosentan, Ambrisentan, Macicentan
  - Their mechanism of action
  - clinical application
  - be aware of what Riociguat is (lower yield)
- Know what VIP, Substance P, CGRP and NPY are, what are the effects that they cause
  _ drugs associated with them – Capsaicin, Aprepitant, Fosamprepitant
- Know what is the difference between effects caused by V1 and V1 receptor activation
- Know what Tolvaptan and Conivaptan are

Chapter 12

- Be able to list and describe different types of angina
- Be able to list the determinants of cardiac oxygen requirement and differentiate them as diastolic factors and systolic factors
- Be able to define preload and afterload and know what they are determined by
- Be able to define double product
- Know the major therapeutic strategies for angina
- Describe nitroglycerin

_ Which form is the most rapid one
_ Mechanism of action
_ Effects
_ Clinical uses
_ Toxicity of nitrates and nitrites

_ Mechanism of nitrites for treating Cyanide poisoning
_ Drug-to-drug interactions in case of taking nitrates together with PDE5 inhibitors

- Describe calcium channel blockers

_ Classification
_ Mechanism of action
_ Effects
_ Clinical use
_ Toxicity

- Describe beta-blockers

_ Classification
_ Mechanism of action
_ Effects
_ Clinical use
_ Toxicity

- Describe Ranolazine _ the mechanism of action, effects and clinical use.
- Describe ivabradine _ the mechanism of action, effects and clinical use.
- List nonpharmacologic therapeutic options of angina

Chapter 19

- Know what and how endogenous and exogenous NO is produced
- Effects of NO on smooth muscle (cGMP), cell adhesion, inflammation and other effects

Chapter 13

- Describe pathophysiology behind heart failure
- Know what Figure 13-1 and Figure 13-2 show
- Know and describe
  - Therapeutic strategies for treatment of HF
  - Know names and mechanisms of action of drugs that have long- term beneficial effects.
  - Table 13-1 (focus more on drug targets and mechanisms of action, still read through which types of HF they are used)
- Cardiac glycosides – Digoxin
  - Mechanism of action (figure 13-1)
  - Cardiac Effects
  - Clinical uses
  - Interactions
  - Toxicity and Approach to treatment
- Describe the use of following drugs in Heart Failure – mechanisms, effects, toxicities, indications, contraindications (under name "Other drugs used in congestive heart failure)
  - Diuretics

- Angiotensin antagonists
- Beta-1-adrenoreceptor agonists
- Beta-adrenoreceptor antagonists
- Phosphodiesterase inhibitors
- Vasodilators

Chapter 14

- Describe mechanism of action and classes of antiarrhythmic medications
- Explain according to what are they classified
- Describe mechanism of action, side effects, possible drug drug interactions if such exist and clinical applications of the following drugs:
    - Quinidine
    - Procainamide
    - Disopyramide
    - Lidocaine
    - Mexiletine
    - Flecanide
    - Propafenone
    - Beta blockers
    - Amiodarone
    - Ibutilide
    - Dofetilide
    - Sotalol
    - Diltiazem
    - Verapamil
    - Adenosine
    - Magnesium
- Identify Procainamide as one of the first choice medications for WPW syndrome
- Classify aforementioned drugs into appropriate classes

Chapter 15

- Be able to describe and identify tubule transport systems in the kidney and sites of action of diuretics (Figure 15-1)
- Know the physiology of proximal convoluted tubule;
- Be able to describe carbonic anhydrase inhibitors

_ Names
_ Mechanism of action
_ Effects
_ Clinical uses
_ Toxicity

- Know the physiology of thick ascending limb of the loop of Henle
- Know the mechanism of calcium and magnesium reabsorption in the ascending limb of the loop of Henle
- Be able to describe loop diuretics

_Names
_ Mechanism of action
_ Effects
_ Clinical uses
_ Toxicity

- Know the physiology of distal convoluted tubule
- Be able to describe thiazide diuretics

_Names
_ Mechanism of action
_ Effects
_ Clinical uses
_ Toxicity

- Know the physiology of cortical collecting tubule
- Be able to describe potassium-sparing diuretics

_Names
_ Mechanisms of action
_ Effects
_ Clinical uses
_ Toxicity

- Be able to describe osmotic diuretics

_Names
_ Mechanisms of action
_ Effects
_ Clinical uses
_ Toxicity

- Be able to describe SGLT2 antagonists

_Names
_ Mechanisms of action
_ Effects
_ Clinical uses
_ Toxicity

- Be able to describe ADH agonists and antagonists

_Names
_ Mechanisms of action
_ Effects
_ Clinical uses
_ Toxicity

Chapter 34

1. Major classes of anticlotting drugs and compare their usefulness in venous and arterial thromboses.
2. Main classes of anticoagulants and their mechanisms of action
3. Explain why the onset of warfarin's action is relatively slow.
4. Compare the oral anticoagulants, standard heparin, and LMW heparins with respect to pharmacokinetics, mechanisms, and toxicity.
5. Give several examples of warfarin's role in pharmacokinetic and pharmacodynamic drug interactions.
6. Role of activated platelets at the site of a damaged blood vessel wall and sites where the major classes of antiplatelet drugs act.
7. Pharmacokinetics, clinical uses, and toxicities of the major antiplatelet drugs.
8. Mechanism of action, clinical uses, and toxicities of the oral anticoagulants (warfarin, rivaroxaban, and dabigatran).
9. Drugs used to treat disorders of excessive bleeding

Chapter 18

1. Major effects of PGE1, PGE2, PGF2α, PGI2, LTB4, LTC4, and LTD4.
2. Cellular sites of synthesis and the effects of thromboxane and prostacyclin in the cardiovascular system.
3. Types of currently available agonists and antagonists of leukotrienes and prostaglandins and their targets (receptors or enzymes).
4. Different effects of aspirin on prostaglandin, thromboxane, and leukotriene synthesis.

Chapter 36

1. Effects of NSAIDs on prostaglandin synthesis.
2. Functions of COX-1 and COX-2.
3. Actions and toxicity of aspirin, the older nonselective NSAIDs, and the COX-2-selective drugs.
4. Why several of the highly selective COX-2 inhibitors have been withdrawn from the market.
5. Toxic effects of aspirin.
6. Effects and the major toxicity of acetaminophen.
7. Disease-modifying antirheumatic drugs (DMARDs) and their toxicity.
8. Explain why patients need to be screened for tuberculosis prior to initiating anti-TNFalpha- therapy.
9. Pharmacologic treatment of acute and chronic gout.
10. Mechanisms of action and toxicity of different drug groups used in gout.

Chapter 20

1. Pathophysiology of asthma and COPD

2. Strategies of asthma therapy
    A. Treatment strategy for acute bronchospasm
    B. Long-term preventive treatment of Asthma
3. Beta adrenoreceptor agonists used for the treatment of asthma and COPD (short acting vs long acting, mechanism of action, route of administration, clinical use, toxicity)
4. Methylxanthines (mechanism of action, clinical use, toxicity)
5. Muscarinic antagonists used for asthma and COPD (mechanism of action, clinical use, toxicity)
6. Corticosteroids (aerosol vs systemic, mechanism of action, clinical use, toxicity)
7. Leukotriene antagonists (mechanism of action, clinical use, toxicity)
8. Cromolyn and nedocromil (mechanism of action, clinical use)
9. Anti-IgE antibodies (omalizumab)
10. Strategies of COPD treatment