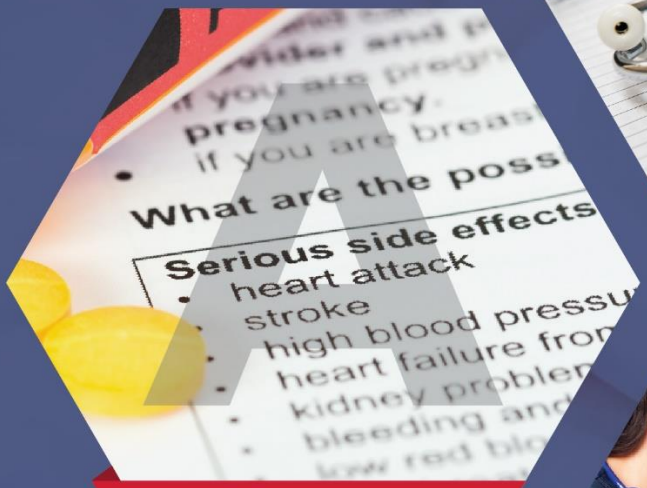
PHARMACOLOGY NOTES

NURSING IMPLICATIONS FOR CLINICAL PRACTICE

Administration

Therapeutic Effects



Adverse Effects

Teaching

GLORIA VELARDE

PHARMACOLOGY NOTES

NURSING IMPLICATIONS FOR CLINICAL PRACTICE

Overview

There are currently nine (9) units comprising this *Pharmacology Notes* resource. Units are broken down by body system and published individually for ease of retrieval:

Unit A: Autonomic Nervous System (ANS) Pharmacology

Unit B: Cardiovascular (CV) System Pharmacology

Unit C: Hematological System Pharmacology

Unit D: Central Nervous System (CNS) Pharmacology

Unit E: Skeletal System: Bone and Joint Pharmacology

Unit F: Immune System Pharmacology

Unit G: Digestive System Pharmacology

Unit H: Endocrine System Pharmacology

Unit I: Respiratory System Pharmacology

Last edited May 17, 2019

UNIT B

CARDIOVASCULAR (CV) SYSTEM PHARMACOLOGY

Table of Contents

(MC) Antidysrhythmics
- (SC) Sodium (Na^+) Channel Blockers
- (SC) Beta Adrenergic Blockers/Antagonists (BBs)
- (SC) Potassium (K^+) Channel Blockers
- (SC) Calcium Channel Blockers (CCBs)
- (SC) Other Antidysrhythmics
  - (SSC) Cardiotonics/Cardiac Glycosides

(MC) Antilipidemics
- (SC) Hydroxymethylglutaryl – Coenzyme A Reductase Inhibitors (HMG-CoA) Reductase Inhibitors
- (SC) Cholesterol Absorption Inhibitors
- (SC) Fibric Acid Agents or Fibrates
- (SC) Bile Acid Resins or Sequestrants
- (SC) Nicotinic Acid

Cardiovascular (CV) System Pharmacology

I. Anatomy and Physiology/Pathophysiology Review

A. Factors Affecting Cardiac Output (CO): CO is a product of stroke volume (SV) and heart rate (HR)

CO = SV x HR

1. Stroke Volume is affected by:
   a. Preload: The amount of tension or stretch, in the ventricles right before contraction (or end-diastolic volume).
      - Influenced by: overall blood volume, venous blood return
   b. Afterload: The force or pressure the ventricles must exert or overcome to eject the blood out.
      - Influenced by: cardiac size, peripheral vascular resistance
   c. Contractility: The force with which ventricular ejection occurs; the ability of the ventricles to squeeze those ventricles with each beat.
      - influenced by: muscle condition, strength
2. Heart Rate is affected by:
   a. Autonomic nervous system (ANS): SNS and PNS
   b. Fluid volume
   c. Activity/exertion

B. Blood Pressure (BP) Regulation: is a product of blood volume (BV) or fluid volume (FV) or cardiac output (CO) and peripheral vascular resistance (PVR)

BP = BV (or FV or CO) x PVR

1. ↑ BV or ↑ FV or ↑ CO → ↑ BP
   ↓ BV or ↓ FV or ↓ CO → ↓ BP
2. ↑ PVR (vasoconstriction) → ↑ BP
   ↓ PVR (vasodilation) → ↓ BP

C. Cholesterol Synthesis

1. Blood lipids/cholesterol is regulated by:
   a. Endogenous factors – cholesterol synthesis in the body – primary site: LIVER
   b. Exogenous factors – diet:
      1) ↑ saturated fats/cholesterol →↑ blood lipid levels
      2) ↑ polyunsaturated fats →↓ blood lipids levels
      3) ↑ dietary fiber → binds with bile acids and prevents reabsorption →↓ blood lipid levels

Blood cholesterol = Rate of synthesis – rate of destruction (utilization and/or excretion/elimination)

2. There are different types of lipoproteins, which contain varying amounts of cholesterol, triglycerides, and phospholipids and a protein carrier:
   a. **L**ow-density lipoproteins (LDL) and very **l**ow-density lipoproteins (VLDL) = "lousey" or "bad"
   b. **H**igh-density lipoproteins (HDL) = "**h**appy" or "good"

D. Common CV Disorders

1. Hypertension (HTN):
   a. Description: high blood pressure; persistent elevation ≥ 130 systolic and/or ≥ 80 diastolic
   b. Causes/Risk factors (primary or essential HTN):
      1) modifiable: smoking, obesity, diet, inactivity, dyslipidemia, DM, stress
      2) non-modifiable: family hx, race (African-American), age, gender
      3) s/sx: often asymptomatic
      4) consequences/complications: cardiac disease, stroke, renal disease, peripheral artery disease (PAD), blindness

2. Coronary Artery Disease (CAD):
   a. Description: impedance or blockage of one or more arteries that reduces blood supply to heart
   b. Causes/Risk factors:
      1) dyslipidemia → atherosclerosis*
      2) HTN + others listed for HTN
   c. Outcome:
      1) imbalance between oxygen supply and oxygen demand

         O_2 supply ≠ O_2 demand

      2) blood flow/supply is unable to meet cardiac workload

         BF/supply ≠ cardiac workload

   d. Key s/sx:
      1) angina/chest pain*
      2) shortness of breath (SOB)
      3) GI sx
      4) myocardial infarction

3. Cardiac Dysrhythmias/Arrhythmias:
   a. Description: abnormal heart rate and/or rhythm
   b. Causes: ectopic foci or "extra" beat(s) due to ↑ irritability or ↑ excitability of the heart
   c. Contributing/Risk factors: heart disease, electrolyte imbalances, conditions causing hypoxia (e.g. respiratory conditions); endocrine disorders (i.e. thyroid)
   d. Outcome: ↓ CO due to ineffective myocardial contractility ⇒ ↑ cardiac workload or ↑ O_2 demand
   e. S/sx: ↓ CO or HF

4. Heart Failure = Sequelae or Consequences of ↓ CO:
    a. Description: when heart (pump) no longer has the ability to provide adequate blood to the body
    b. Causes: any CV condition or disease that ↓ CO (can be acute or chronic)
    c. Outcome: ↓ forward blood flow and ↑ blood back up
    d. S/sx:
        1) L-sided – ↓ blood delivered to periphery, kidneys, GI, heart, and ↑ blood back-up into lungs:
            a) ↓ peripheral circulation, ↓ renal function, ↓ GI function, chest pain, fatigue
            b) lung congestion
        2) R-sided – ↓ blood delivery to lungs; and ↑ blood back-up into venous system:
            a) hypoxia
            b) peripheral edema, + JVD, hepatic congestion, hepatomegaly, ascites

II. PHARMACOLOGY

A. Pharmacologic Connections for CV Agents

1. CV agents will act on one or more factors that affect <u>cardiac output</u> (CO = SV x HR)
   ⇒ ***mechanism of action (MOA)*** or pharmacodynamics

2. The goals of CV agents are to:
    a. ***Maintain*** or attain adequate CO
    b. ***Restore*** balance between O_2 supply and oxygen demand by:

        O_2 Supply = O_2 Demand

        1) ↑ blood flow or supply by promoting vasodilation
        2) ↓ cardiac workload by ↓ HR and/or cardiac irritability or excitability
        3) when you think of pre**load** and after**load**, think cardiac work**load**
    c. ***Manage*** BP by:
        1) ↓ blood volume **or** ↓ fluid volume **or** ↓ cardiac
        2) ↓ PVR
    d. ***Regulate*** HR and rhythm by:
        1) ↓ ectopic beats →
        2) ↓ cardiac irritability/excitability →
        3) ↓ cardiac workload
    e. In the presence of CV disease, the **goal** of several CV drugs is to **reduce** cardiac work**load**

3. The desired effects of CV agents are to:
    a. ↓ s/sx of CV condition or disease
    b. ↓ s/sx or prevent HF

4. Specific to lipid-lowering agents:
    a. Blood cholesterol is managed by:
        1) ↓ cholesterol synthesis
        2) ↓ cholesterol absorption
        3) ↑ cholesterol excretion/elimination
    b. Management is directed at:
        1) ↓ "bad" lipoproteins
        2) ↑ "good" lipoproteins

Nursing Implications: CV Pharmacology: Antihypertensives BP = BV (or FV or CO) x PVR

Subclass	MOA	Prototype – generic	Prototype – trade	A – Admin	T – √ Therapeutic Effects – General (MC)	A – √ Adverse Effects – Common	A – √ Adverse Effects – Specific (SC)	T – Teaching – General
Diuretics	Blocks absorption of water → ↑ UO →↓ FV → ↓ BP			Route: PO/IV; IV for rapid action Timing: early in day	Treatment of underlying causes of HTN (e.g. excess FV: • ↓wgt, ↓edema, ↓lung congestion) Treatment of hypertension: • ↓BP ↓s/sx of HTN (HA, blurry vision) Ø signs of HTN-related complications: • CAD, CVD (neuro), PAD, renal, eyes	• CV: hypotension – dizziness, light-headedness, syncope, + orthostasis • Other: arrhythmias, angina, s/sx HF • GI: N/V/D/C • Sexual dysfunction	• F&E: dehydration, electrolyte loss/ imbalances, ↑ BG	***Selection based on HTN class:*** • Timing: same time; stagger if >1 • Ø abruptly stop • Safety precautions • Ø excess alcohol • Ø hot weather • Lifestyle changes • ✓ BP & HR record
	• thiazide	hydrochloro-thiazide	HydroDiuril, Microzide				• Hypokalemia	**SPECIFIC** to diuretics: • Timing: Ø evening • ✓ wgt • Diet restrictions to ↓ excess FV (↓ Na^+) • Report: o Sudden wgt △'s o △'s HR or rhythm o ↑ breathing sx • ✓ electrolyte labs, CBC
	• loop	furosemide	Lasix				• ↑hypokalemia; • ENT: otoxicity, esp when given IV	
	• K+ sparing/ aldosterone antagonist	spironolactone	Aldactone				• Hyperkalemia	
	• osmotic	mannitol	Osmitrol				• Dry mouth, excessive thirst, HA • Pulmonary congestion	
	• carbonic anhydrase inhibitors	acetazolamide	Diamox	For acetazola-mide: • Take 1-2 days before climbing (altitude sickness)	Other uses for acetazolamide: • Ø s/sx altitude sickness • ↓ IOP for glaucoma • ↓ or Ø seizure activity		• Neuro: paresthesias, tinnitus • Hypersensitivity • Blood dyscrasias	

Subclass	MOA	Prototype – generic	Prototype – trade	A – Admin	T – √ Therapeutic Effects – General (MC)	A – √ Adverse Effects – Common	A – √ Adverse Effects – Specific (SC)	T – Teaching – General
Angiotensin converting enzyme inhibitors (ACEIs)	Blocks enzyme that converts angiotensin I → angiotensin II → vasodilation → ↓ PVR → ↓ BP Blocks aldosterone secretion → ↑ UO → ↓ FV → ↓ BP	lisino***pril*** fosinopril captopril	Prinivil, Zestil Monopril Capoten	Route: PO for most; IV for rapid action	**(See previous page)**	**(See previous page)**	• Hyperkalemia • Hypersensitivity (rash) • Angioedema • Cough • Teratogenicity • GU: ↑ creatinine	**(See previous page)** **SPECIFIC** to A2 subclasses: • Monitor electro-lytes, esp. K^+ • Contraception • Monitor renal function: BUN, Cr, EGFR • Teach re: use of salt substitutes
Angiotensin II receptor antagonists/ blockers (A2RBs)	Blocks action of angiotensin II after it's formed (outcome same as ACEIs)	losartan valsartan	Cozaar Diovan					
Renin inhibitors	Binds w/ renin → ↓ angiotensin II and aldosterone → ↓ PVR and ↓ FV →↓ BP	aliskiren	Tekturna					
Calcium channel blockers (CCBs)	Blocks Ca^{++} channels in vascular smooth muscle walls → vasodilation → ↓ PVR →↓ BP	nifedipine diltiazem verapamil amlodipine	Adalat, Procardia Cardizem Calan, Covera-HS, Isoptin SR Norvasc				• Bradycardia or reflex tachycardia • Constipation (primarily verapamil)	**SPECIFIC** to CCBs: • Monitor bowel function – add fiber to diet

Subclass	MOA	Prototype – generic	Prototype – trade	A – Admin	T – √ Therapeutic Effects – General (MC)	A – √ Adverse Effects – Common	A – √ Adverse Effects – Specific (SC)	T – Teaching – General
Alpha$_1$ (α_1) adrenergic antagonists/ blockers	Blocks sympathetic receptors in arterioles → vasodilation → ↓ PVR →↓ BP	prazosin doxazosin	Minipress Cardura	**(See previous page)**	**(See previous page)**	**(See previous page)**	• 1st dose syncope • CNS: nervousness, fatigue	**(See previous page)**
Alpha$_2$ (α_2) adrenergic agonists	Centrally-acting → ↓ SNS activity to heart & arterioles → vasodilation → ↓ PVR →↓ BP	clonidine methyldopa	Catapres Aldomet				• Drowsiness, sedation, • Dry mouth • Rebound hypertension if abruptly stopped • Blood dyscrasias	
Beta adrenergic antagonists/ blockers (BBs)	Blocks beta receptors in heart → ↓ CO →↓ BP	metopro***lol*** propranolol carvedilol	Lopressor, Toprol Inderal Coreg				• Bradycardia • Bronchospasms – use w/ caution w/ pts having COPD, asthma • Rebound tachycardia if abruptly stopped	
Miscellaneous vasodilators	Relaxes smooth muscles in blood vessels → vasodilation → ↓ PVR → ↓ BP	nitroprusside sodium	Nitropress	Route: IV Used for: hypertensive crisis			• Fast, profound hypotension	

Nursing Implications: CV Pharmacology: Antianginals (CAD/HF management) – Restore balance: O_2 supply = O_2 demand by: ↑ O_2 supply (blood flow) and/or ↓ O_2 demand (cardiac workload)

Subclass	MOA	Prototype – generic	Prototype – trade	A – Admin	T – √ Therapeutic Effects – General (MC)	A – √ Adverse Effects – Common	A – √ Adverse Effects – Specific (SC)	T – Teaching – General
Beta adrenergic antagonists/ blockers (BBs)	Blocks beta receptors in heart →↓ CO →↓ cardiac workload →↓ O_2 demand	metopro***lol*** propranolol carvedilol	Lopressor, Toprol Inderal Coreg	***Subclasses given depends on goal of tx: short- or long-term mgmt. of chest pain*** Route: short-term: • SL, TL spray, ointment, IV Route: long-term: • PO, transdermal patch/disc Timing: same time; stagger w/ other anti-HTN meds	• Manage acute episode of chest pain • Manage long-term myocardial ischemia →↓ episodes of acute chest pain • ↑ cardiac output: ○ improve activity level & tolerance	• CV: hypotension – dizziness, light-headedness, syncope, + orthostasis • Other: arrhythmias, angina, s/sx HF • GI: N/V/D/C	• Bradycardia • Bronchospasms – use w/ caution w/ pts having COPD, asthma • Rebound tachycardia if abruptly stopped • Bradycardia or tachycardia reflex • Constipation (primarily verapamil)	• Timing: same time; evenly spaced for long-acting • Ø abruptly stop • Safety precautions • Ø excess alcohol • Lifestyle changes – reinforce activity restrictions • ✓ BP & HR record • ✓ cardiac enzymes: troponin, CK, LDH, myoglobin
Calcium channel blockers (CCBs)	Blocks Ca^{++} channels in vascular smooth muscle walls → • vasodilation → ↑blood flow → ↑O_2 supply • ↓ BP →↓ afterload →↓ O_2 demand Blocks Ca^{++} influx into myocardial cells →↓ cardiac excitability →↓ cardiac workload →↓O_2 demand	nifedipine verapamil amlodipine diltiazem	Adalat, Procardia Calan, Covera-HS, Isoptin SR Norvasc Cardizem					

Subclass	MOA	Prototype – generic	Prototype – trade	A – Admin	T – √ Therapeutic Effects – General (MC)	A – √ Adverse Effects – Common	A – √ Adverse Effects – Specific (SC)	T – Teaching – General
Nitrates	Relaxes both arterial & venous smooth muscles: • arterial dilation → ↑blood flow → ↑O_2 supply • venous dilation →↑blood pools →↓blood return →↓preload →↓cardiac workload →↓O_2 demand	nitroglycerin (NTG) isosorbide	Nitrostat, Nitrobid, Nitro-Dur Isordil	**(See previous page)**	**(See previous page)**	**(See previous page)**	• CV: tachycardia, headache, peripheral edema	**SPECIFIC** to short-acting NTG: • Have pt. stop activity • ✓ vs, pain level: **NTG 1 tab SL every 5 min x 3 for chest pain PRN** • Storage: cool, out of light, replace q 24 months • Avoid skin contact w/ NTG ointment/ patches → headache • Patches/ointment: apply to clean, dry, hairless areas; rotate sites, avoid distal portions • Remove (12 hrs on/ 12 hrs off – ↓tolerance
Angiotensin converting enzyme inhibitors (ACEIs)	Blocks enzyme that converts angiotensin I → angiotensin II → vasodilation → ↑ blood flow → ↑ O_2 supply Blocks aldosterone secretion →↑ UO →↓ FV → →↓CO →↓ cardiac workload →↓ O_2 demand	lisino***pril*** fosinopril captopril	Prinivil, Zestil Monopril Capoten				• Hyperkalemia • Hypersensitivity (rash) • Angioedema • Cough • Teratogenicity • GU: ↑ Cr	**SPECIFIC** to A2 subclasses: • Monitor electrolytes, esp. K^+ • Monitor renal function: BUN, Cr, EGFR • Teach re: use of salt substitutes • Contraception

Subclass	MOA	Prototype – generic	Prototype – trade	A – Admin	T – √ Therapeutic Effects – General (MC)	A – √ Adverse Effects – Common	A – √ Adverse Effects – Specific (SC)	T – Teaching – General
Angiotensin II receptor antagonists/ blockers (A2RBs)	Blocks action of angiotensin II after it's formed *(outcome same as ACEIs)*	losartan valsartan	Cozaar Diovan	**(See previous page)**	**(See previous page)**	**(See previous page)**	• Hyperkalemia • Hypersensitivity (rash) • Angioedema • Cough • Teratogenicity • GU: ↑ Cr	**SPECIFIC** to A2 subclasses: • Monitor electrolytes, esp. K^+ • Monitor renal function: BUN, Cr, EGFR • Teach re: use of salt substitutes • Contraception
Renin inhibitors	Binds w/ renin →↓ angiotensin II & aldosterone *(outcome same as ACEIs)*	aliskiren	Tekturna					
Angiotensin receptor – neprilysin inhibitors (ARNIs)	Blocks action of angiotensin II after formed & inhibits neprilysin (enzyme that metabolizes peptides) →↑ blood levels of natriuretic peptides: • promotes renal excretion of sodium & water • promotes myocardial relaxation & inhibits hypertrophy & fibrosis • suppress SNS outflow in brain • stimulates vasodilation	valsartan/ sacubitril	Entresto		• ↓ s/sx heart failure • improved ejection fraction			

Nursing Implications: CV Pharmacology: Anti-Dysrhythmics –

↓ ectopic foci →↓ cardiac irritability/excitability (↓ O_2 demand/cardiac workload)

Subclass	MOA	Prototype – generic	Prototype – trade	A – Admin	T –√ Therapeutic Effects – General (MC)	A – √ Adverse Effects – Common	A – √ Adverse Effects – Specific (SC)	T – Teaching – General
Na^+ channel blockers (Class I A-C)	Blocks the movement of Na^+ (fast channels) ***into*** myocardial cells → delays depolarization → delays action potential →↓ excitability	lidocaine procainamide propafanone	Xylocaine Pronestyl, Procanbid Rythmol	• ✓ AP for one full minute ○ HOLD if < 60 • ✓ BP • ✓ electrolyte levels Timing: evenly spaced intervals; same time daily Contraindications: pregnancy, heart block	• HR returned to baseline (60-100 beats per minute) • Rhythm = regular • No s/sx HF	• Dysrhythmias – most common = **bradycardia** • △'s BP = hypotension • S/sx of ↓ CO or HF (CO = SV x **HR**)	• CV: cardiotoxicity ○ ↑ PR & QT intervals ○ widening QRS complex • MS: painful inflamed joints • Heme: blood dyscrasias	• How to take pulse – one full minute • Have BP monitored regularly • Timing of medications esp. if taking more than one • Safety re: position changes • Follow-up labs ○ electrolyte levels • Report any worsening sx: SOB, CP, palpitations • Use OTC cold remedies, appetite suppressants, & anti-sleep preparations w/ caution
Beta adrenergic antagonists/ blockers (BBs) (Class II)	Blocks beta receptors in heart →↓ CO →↓ excitability	metopro***lol*** propranolol carvedilol	Lopressor, Toprol Inderal Coreg				• Bradycardia • Bronchospasms – use w/ caution w/ pts having COPD, asthma • Rebound tachycardia if abruptly stopped	
K^+ channel blockers (Class III)	Blocks movement of K^+ out of myocardial cells →prolongs repolarization →↑ refractory period →↓ cardiac excitability	amiodarone sotalol	Cordarone Betapace				• Pulmonary toxicity • Visual disturbances • Liver/thyroid dysfunction • GI sx • CNS	
Calcium channel blockers (CCBs) (Class IV)	Blocks Ca^{++} influx into myocardial cells → delays myocardial contraction that follows depolarization ↓cardiac excitability	nifedipine verapamil diltiazem amlodipine	Adalat, Procardia Calan, Covera-HS, Isoptin SR Cardizem Norvasc				• Bradycardia or reflex tachycardia • Constipation (primarily verapamil)	

Subclass	MOA	Prototype – generic	Prototype – trade	A – Admin	T –√ Therapeutic Effects – General (MC)	A – √ Adverse Effects – Common	A – √ Adverse Effects – Specific (SC)	T – Teaching – General
Other: Anti-dysrhythmics (Class V)	Cardiotonics/ Cardiac glycosides →↑ strength of myocardial contrx (↑ contractility → + inotropic effect) Suppresses SA node →↓ conduction thru AV node	digoxin	Lanoxin	• ✓ AP for one full minute ○ HOLD if <60 • ✓ BP • ✓ **electrolyte levels** Timing: evenly spaced and/or same time daily Contraindications: pregnancy, heart block liver or kidney disorders	• HR returned to baseline (60-100 beats per minute) • Rhythm=regular • ↑ CO → ↓ s/sx HF • Serum blood levels normal *(i.e. digoxin) – 0.5–2 ng/mL (narrow therapeutic range)*	• Dysrhythmias – most common = bradycardia • △'s BP = hypotension • S/sx ↓ CO or HF (CO = SV x **HR**)	**Long half-life** → due to high protein-binding: • **given 1x/day** • **loading dose required** • GI: N/V/D/A – anorexia *is most common early sign of digoxin toxicity* • Visual △'s –yellow halos around objects, blurring • Electrolyte imbalances that ↑ risk for toxicity: ○ hypokalemia ○ hypomagnesemia ○ hypercalcemia • Antidote: digoxin immune fab (Digibind)	• How to take pulse – one full minute • Have BP monitored regularly • Timing of medications esp. if taking more than one • Safety re: position changes • Follow-up labs – electrolyte levels – (**esp. K^+, Mg^{++}, Ca^{++} for digoxin)** • Report any worsening sx: SOB, CP, palpitations • Do not use OTC cold remedies, appetite suppressants, & anti-sleep preparations w/ caution • Use caution w/ **diet supplements**
	Cardiotonics/ phosphodiester-ase inhibitors PDE3I) →↓ cAMP phosphodiesteras e activity in heart →+ inotropic effect & vasodilation	milrinone	Primacor	Route: IV			• Neuro: HA, tremors • Heme: easy bruising or bleeding	

Subclass	MOA	Prototype – generic	Prototype – trade	A – Admin	T –√ Therapeutic Effects – General (MC)	A – √ Adverse Effects – Common	A – √ Adverse Effects – Specific (SC)	T – Teaching – General
	Blocks calcium channels Suppresses SA node → ↓ AV conduction	magnesium sulfate	MgSO4	Route: IV bolus	**(See previous page)**	**(See previous page)**	• Skin: facial flushing • Resp: hypoventila-tion • CNS: sedation, confusion, muscle weakness	**(See previous page)**
	Suppresses AV conduction	adenosine	Adenocard	Route: IV			• Resp: dyspnea • Skin: facial flushing	

Nursing Implications: CV Pharmacology: Antilipidemics

Major Class	MOA	Prototype – generic	Prototype – trade	A – Admin	T – √ Therapeutic Effects – General (MC)	A – √ Adverse Effects – Common	A – √ Adverse Effects – Specific (SC)	T – Teaching – General
Hydroxymethyl-glutaryl – coenzyme A reductase inhibitors (HMG-CoA reductase inhibitors)	Inhibits/**stops** enzyme required for hepatic **synthesis** of cholesterol **"statins"**	atorvastatin lovastatin rosuvastatin simvastatin	Lipitor Mevacor Crestor Zocor	Route: Oral Timing: take w/ or w/o food/meals; preferred – eve/HS	Treatment of dyslipidemia: • ↓ LDL • ↓ VLDL or triglycerides • ↓ total cholesterol • ↑ HDL Slows progression of vessel disease: CAD, CVD, PAD	• GI: N/VC/D, flatulence. abdominal discomfort • Hypersensitivity – skin rash, pruritus • Hepatotoxicity – ↑ liver enzymes – SGOT/SGPT or AST/ALT, ↑ jaundice	• MS: myopathy, rhabdomyolysis • Skin: photo-sensitivity	• Timing • Diet + low-fat, low-cholesterol • Other life style △'s • Lab ✓'s: ○ lipid profile ○ liver enzymes ○ CK levels • Ø alcohol **SPECIFIC** to Statins: • Report muscle pain • Avoid prolonged sun exposure
cholesterol absorption inhibitors	Blocks absorption of cholesterol from small intestine→↓ cholesterol to hepatic cells →↓ blood cholesterol	ezetimibe "**easy** exit" → faster "**time**"	Zetia	Route: Oral Timing: take w/ food/meals; preferred – eve/HS Contraindications: Preg. risk cat.=X			• GI: hepatitis • MS: myopathy	**SPECIFIC** to Cholesterol-absorption inhibitors: • Contraception
fibric acid agents or Fibrates	↑ oxidation of fatty acids →↓ need for triglycerides →↓ triglyceride prod. in liver	gem***fibr***ozil	Lopid "***lo***wer li***pid***s"	Route: Oral Timing: take on empty stomach; stagger w/ other antilipidemics			• GI: gallstones • MS: myopathy	**SPECIFIC** to Fibrates: • Timing: take 30 minutes before breakfast & dinner

Major Class	MOA	Prototype – generic	Prototype – trade	A – Admin	T – √ Therapeutic Effects – General (MC)	A – √ Adverse Effects – Common	A – √ Adverse Effects – Specific (SC)	T – Teaching – General
bile acid resins or sequestrants	Binds w/ bile acids in intestinal lumen → excreted in feces; triggers more cholesterol to be broken down to bile acids → moves cholesterol from blood to liver	cholestyramine colesevelam	Questran Welchol	Route: Oral Timing: take with/ before food & 8 oz. of water	**(See pervious page)**		• GI: N/C, bloating, gas, aggravate pre-existing biliary disorders	**SPECIFIC** to Bile Acid Resins: • Take other meds. 4 hrs before
nicotinic acid	↓ VLDL → ↓ triglyceride production in liver	niacin	Niaspan	Route: Oral: standard or time-released Contraindication: gout, pregnancy			• Skin flushing w/ high doses • F/E: hyperuricemia, hyperglycemia	**SPECIFIC** to Niacin: • ✓ renal function • Take ASA/ acetaminophen before