

Cardiovascular diseases

Hypertension
Heart Failure
Dysrhythmias
Angina & Myocardial Infarction
Lipids
Coagulation
Anemia

Classes of Medication for Hypertension

1. Diuretics make you pee
Diuretics Reduce blood volume through urinary excretion of water and electrolytes (Na⁺, Ca⁺⁺, Cl⁻, K⁺)
Specific mechanism of action varies within the class (thiazide, loop, potassium-sparing)
Depends on where (i.e. which part of the nephron) it works
Effective, Well tolerated
First line treatment for hypertension
Due to manipulation of electrolytes, monitoring is important!

Classes of Medication for Hypertension (cont)

a. Thiazide diuretics
Hydrochlorothiazide (HCTZ)
Largest, most commonly prescribed class of diuretics ('gentler' than loop diuretics)
Decrease the reabsorption of sodium in the early distal tubule, which increases the production and excretion of urine
More sodium (and therefore water) is excreted
Treat mild to moderate hypertension and edema that is associated with heart, hepatic, and renal failure

b. Loop diuretics
Furosemide
Are the most effective diuretics
Prevents reabsorption of sodium and chloride in the loop of Henle
Reduce edema associated with heart, hepatic, or renal failure
Cause large amounts of fluid to be quickly excreted – along with potassium (K⁺)
Used to provide short-term hypotension, not so much for blood pressure maintenance



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Classes of Medication for Hypertension (cont)

c.Pota- ssium sparing diuretics <i>Spiron- olactone</i>	Block either sodium pump (leaving more sodium in tubule) or aldosterone further along in the nephron (late distal tubule and collecting duct) Achieve diuresis without affecting blood potassium levels Preferred in patients at high risk of developing hypokalemia Sometimes combined with other diuretics (as an add-on) to minimize potassium loss
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2.Calcium channel blockers (CCB)	Muscle contraction is controlled by calcium moving in and out of channels across cell membranes (Ca++ influx causes contraction) Blocking the channels limits muscular contraction, relaxing muscle in both the periphery and heart Reduce blood pressure by lowering peripheral resistance and cardiac output
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<i>Nifedipine</i>	Mechanism of action: blocks calcium channels in myocardial and vascular smooth muscle (blood vessels > heart) – long-acting dihydropyridine (LA-DHP)
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Classes of Medication for Hypertension (cont)

Anything that causes vasodilation will also cause reflex tachycardia
Therefore, anything that causes vasodilation requires heart rate monitoring

3.Renin-angi- otension-ald- osterone system (RAAS) agents	RAAS is triggered in times of low blood pressure End result of uninterrupted RAAS is increased blood pressure drugs affecting RAAS Reduce blood pressure by: Reducing peripheral resistance Decreasing blood volume
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a.Angiotensin- converting- enzyme inhibitors (ACE Inhibitors) <i>-pril</i>	Inhibit angiotensin-converting-enzyme (ACE), resulting in less angiotensin II and aldosterone, which reduces blood pressure Prevents conversion of angiotensin I to angiotensin II, therefore: Prevents aldosterone secretion Prevents the direct vasoconstriction <i>Pregnancy category D</i>
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Classes of Medication for Hypertension (cont)

b.Angi-otensin II receptor blockers (ARBs) -sartan	In the same pathway (RAAS), ARBs block angiotensin II from causing vasoconstriction, and block the release of aldosterone at the adrenal gland Very similar uses and adverse effects as ACE-I **Also newer drugs involved in the RAAS: Aliskiren – renin inhibitor
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4. Adrenergic agents

Classes of Medication for Hypertension (cont)

a.β-Blockers -olol	β-receptors in heart (β1), lungs (β2), blood vessels (β2) + many others Cardio-selective: reduce heart rate and slow down myocardial conduction and contractility = reduce cardiac output Non-selective: also produce vasodilation = lower peripheral resistance and reduce cardiac output Because of their action in the heart, their primary use is for angina, arrhythmias, heart failure, and post-myocardial infarction Also used off-label for migraine prevention, or as a performance enhancing drug Drug dependence occurs, so upon abrupt discontinuation = reflex tachycardia (Requires tapering)
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Classes of Medication for Hypertension (cont)

b.α1-B-blockers Block α1-receptors in the periphery – relaxes smooth muscle and reduces peripheral resistance and cardiac output (indirectly)
 Vasodilation = ↓ venous return to heart = ↓ cardiac output
 Primary continuous use is for urinary incontinence and benign prostatic hyperplasia (BPH)
 Work very quickly to reduce blood pressure

Doxazosin Blocks vasoconstriction caused by stimulation of α-receptors, therefore reducing peripheral resistance
 Used to treat urinary incontinence, BPH, hypertension

Classes of Medication for Hypertension (cont)

c.α2-A-agonists Stimulate α2-receptors in the CNS, which causes the identical response as the α1-blockers in the periphery ☑
 vasodilation ☑ reduces peripheral resistance and cardiac output (indirectly)
 When α2-receptors are stimulated, the outflow of sympathetic nerve impulses from the CNS to the heart and blood vessels is inhibited
 α2-agonists and α1-blockers = same clinical result
 Rarely used for long-term (*clonidine, methyldopa*)
 Reserved for patients with hypertension which has been resistant to other therapies

d.Miscellaneous *Labetalol*: partial agonist @ β2, blocks @ α1 & β1
Carvedilol: blocks β1&2 and α1

5.Direct-acting vasodilators Directly relax arteriolar smooth muscle in blood vessels
 ☑ reduce peripheral resistance
 Work at cellular level – each differently
 Quickly reduce blood pressure
Hydralazine, minoxidil, nitroglycerin
 Generally reserved for acute care to dilate quickly under close monitoring



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Normal sinus rhythm



About Action Potentials

Resting State (kind of) Na⁺ and Ca⁺⁺ are outside cell, K⁺ is inside cell (+ charge higher outside, than inside – POLARIZED)

Depolarization Na⁺ and Ca⁺⁺ channels open, and both rush into the cell to try and balance out charges (it is mostly the Ca⁺⁺ increase inside the cell responsible for muscle contraction)

Repolarization In a further attempt to get back to resting state, the K⁺ channels open and K⁺ rushes out

Remember that calcium also has a role in muscle contraction!
If a patient is asymptomatic with an arrhythmia, we don't have to treat it

We only treat arrhythmias that affect cardiac output or increase risk of clots

Electric shock / defibrillation – like a reset button for the SA node – hoping to return to normal sinus rhythm in an emergency

Patients with certain types of dysrhythmias are at an increased risk of a clot – therefore, often also on anticoagulants

What are dysrhythmias/arrhythmias ?

Any abnormality of electrical conduction (in the generation or conduction) that results in a disturbance of the heart rate or rhythm

Atrial dysrhythmias are more common and less severe than ventricular dysrhythmias

Diagnose using ECG

Myocardial action potential:

Conduction is sent along the pathway using Na⁺, K⁺, and Ca⁺⁺ channels

Drugs correct dysrhythmias by either:

Manipulating these channels

Altering autonomic activity (α and β receptors)

Classes of medication for Arrhythmias

Sodium channel blockers (class I)
Propranolol
Slow down depolarization by preventing Na⁺ from rushing into the cell
Lengthen the duration of the action potential
Can also suppress ectopic activity (arrhythmias coming from an incorrect source)
Similar in structure to anesthetics, therefore potential for CNS effects

β -blockers (class II)
Slows heart rate
Decreases conduction velocity through the AV node
Altering the adrenergic nervous system
Usually used for dysrhythmias associated with heart failure (choose cardio-selective ones)
Remember: DO NOT STOP ABRUPTLY = reflex tachycardia
Contraindicated in clients with heart block, severe bradycardia, asthma, COPD, elderly, diabetics



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Classes of medication for Arrhythmias (cont)

Potassium channel blockers (class III) Delays repolarization and lengthens refractory period
Mostly used for ventricular arrhythmias (repolarization is one of the last steps – in ventricles)
Many have multiple actions at other receptors
ex. *Sotalol* – β -blocker and potassium-blocker

Amiodarone Amiodarone is a potassium channel blocker and a sodium channel blocker, among other mechanisms of action
Widely distributed and stored in tissues, so toxicity can be difficult to get rid of
Primarily used to treat resistant ventricular tachycardia and atrial dysrhythmias
LOW THERAPEUTIC range

Calcium channel blockers (class IV) Reduce automaticity, slows conduction through AV node, slows heart rate
Remember: *diltiazem* and *verapamil* were more selective for heart AND it is $Ca^{++} > Na^{+}$ that influences cardiac muscle contraction

Miscellaneous

Classes of medication for Arrhythmias (cont)

Digoxin Decreases automaticity of SA node and slows conduction through AV node – but not by blocking any ion channels
Requires therapeutic drug monitoring
Remember to teach signs of toxicity
Remember importance of potassium

Adenosine An endogenous nucleoside that reduces automaticity of SA node and slows conduction through AV node
Sometimes used in diagnosing patients who cannot complete a stress test
10 second half-life (bolus IV injection)

If it can correct a arrhythmia, it can also cause a arrhythmia
By manipulating the action potential OR the nervous system, we are also manipulating factors / variables that affect blood pressure (cardiac output and peripheral resistance)
Therefore, monitoring would include for ALL:
ECG
Blood pressure
Heart rate

About anemia

Anemia occurs when red blood cells (erythrocytes) or hemoglobin have a diminished capacity to carry oxygen Due to: blood loss, excessive destruction, or diminished synthesis

Erythropoiesis the process of making erythrocytes in bone marrow
If we are lacking a substance for erythropoiesis, we won't have as many RBCs



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About anemia (cont)

Erythr- – the hormone released by the kidneys that instructs the
opoietin bone marrow to make RBCs

Anemias are classified according to appearance of erythrocyte, which tells pathologists which ingredient is missing

General signs and symptoms of anemia

General fatigue

Weakness

Pale skin

Shortness of breath (dyspnea)

Dizziness

Strange cravings to eat items that aren't food, such as dirt, ice, or clay

Tingling or crawling feeling in the legs

Tongue swelling or soreness

Classes of Medication for Anemia (cont)

Folic Required for erythropoiesis
Acid Does not require intrinsic factor to absorb from GI (more readily absorbed)
Folate Deficiency results in anemia, but no neurological symptoms
Require folic acid during neural tube formation in pregnancy – suggest supplements in any woman of child-bearing age
Green, leafy vegetables – or supplements (1-5mg)
Corrected in 2 weeks ➡ 1 month

Classes of Medication for Anemia

Vitamin Required for erythropoiesis
B12 Does not absorb very well from GI tract – must have
Cyana intrinsic factor present to absorb (genetic differences)
coba- Often given by IM injection (monthly maintenance)
lamin B12 deficiency presents as memory loss, confusion, unsteadiness, tingling in limbs, delusions, mood disturbances (more CNS effects)



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Classes of Medication for Anemia (cont)

Iron Involved in the oxygen carrying capacity of the erythrocytes
 Different formulations (or salts) have different absorptions and bioavailability
 Ferrous sulfate (red), ferrous gluconate (green), ferrous fumarate
 Iron interferes with absorption of many other drugs (antibiotics, thyroid meds)
 It is an ion that binds to some medications, forming a complex too large to absorb
 It is better absorbed (↑ to 10%) in presence of vitamin C
 Antacids decrease absorption of iron (by changing the pH of the gastric contents) and need to be separated by ~ 2 hours
 General recommendation: separate iron supplements from other meds by 2 hours if possible

Classes of Medication for Anemia (cont)

Growth Factors When anemia is a result of a lack of growth factor, we can replace the growth factor with biologics
 Erythropoietin alfa or darbepoietin alfa = to replace erythropoietin (EPO)
 Hormone secreted by kidneys – low in kidney failure and cancers
 EPO = “blood doping” ☑ oxygen carrying capacity is increased, boosting endurance

Monitoring for Anemia:
 B12, folate levels
 CBC (RBC, hemoglobin, hematocrit)
 Iron, ferritin
 Potassium
 Neuro status (confusion, etc.)
 Arrhythmias
 Resolving of symptoms (fatigue, pale colour)
 GI adverse effects with iron

Classes of medication for lipids

Statins Inhibit HMG-CoA-reductase, which is involved in the synthesis of cholesterol in the liver
Atorva-statin Reduces the amount of cholesterol made by our body
(Lipitor®) Also increases the amount of LDL removed from the blood
 First drug of choice; therapy continues for life
 Very well tolerated
 DO NOT USE IN PREGNANCY
 Choice of statin is dependent on lipid profile Some are good at lowering LDL, some better at raising HDL, etc.



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Classes of medication for lipids (cont)

Fibrates

Fenofibrate

"Lipid metabolism regulator" – changes production levels of lipoproteins, but different pathway than statins
Lower triglyceride levels and raise HDL levels
Some also lower LDL
More gastrointestinal adverse effects than statins
May be used with a statin in some cases

Niacin

Nicotinic acid / nicotinamide / niacinamide / vitamin B3

Available without a prescription (OTC)
Exact mechanism is unknown, but reduces synthesis of LDL, VLDL, and increases HDL
Also causes peripheral vasodilation ☑
flushing
More gastrointestinal effects than statins

Classes of medication for lipids (cont)

Bile Acid

Resins

Bind to bile acid made by the liver to enhance excretion of cholesterol
Bile acid then does not absorb through intestinal wall (forms a complex, too big to pass through plasma membrane), so once bile acid is bound, it is excreted with feces
The liver responds by getting rid of even more cholesterol
Drug of choice in pregnancy (no absorption occurs!)

Miscellaneous

Ezetimibe

inhibits intestinal cholesterol absorption – used along with a statin

Orlistat

doesn't allow fats to be absorbed from intestine – *anal discharge* (anti-obesity drug)

Omega-3

insufficient evidence for cholesterol, but likely no harm

Psyllium (Metamucil®)

similar mechanism to bile acid resin



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Classes of medication for lipids (cont)

PCSK9 Inhibitors	Class of biologics for very high-risk patients (of a cardiovascular event) who have not reached targets with statins
<i>Alirocumab, evolocumab</i>	Monoclonal antibodies for PCSK9, which promotes LDL degradation – reduce LDL substantially
	Administered SC every 2 weeks (q2w), monitor within 4-8 weeks

Adverse effects of Cardiovascular medication

Thiazide Diuretics	Electrolyte imbalances (especially loss of potassium - hypokalemia) – monitor all electrolytes
<i>Hydrochlorothiazide (HCTZ)</i>	Hyperglycemia – monitor blood glucose Dizziness – monitor upon standing Hypotension – monitor blood pressure/vitals Some drug interactions – most mild and require ↑ monitoring; sulfa drug Important to warn patient about ↑ peeing! AM dosing!
Calcium Channel Blockers	Dizziness, hypotension, headache, flushing, reflex tachycardia*, constipation, peripheral edema
<i>Nifedipine</i>	

Adverse effects of Cardiovascular medication (cont)

ACE Inhibitors	electrolyte imbalances (esp. potassium) first-dose syncope orthostatic hypotension unexplained persistent dry cough Theory: due to high levels of bradykinin usually broken down by ACE angioedema (rare)
Angiotensin II Receptor Blockers (ARBs)	Same adverse effects as ACE-I
α1-Blockers <i>Doxazosin</i>	orthostatic hypotension (first-dose syncope), dizziness, headache
α2-Agonists	more CNS adverse effects than α1-blockers: Sedation, depression, fatigue, + orthostatic hypotension, dizziness, headache, etc
Direct Vasodilators <i>Hydralazine, minoxidil, nitroprusside</i>	Multiple dangerous side effects limit use to emergencies and acute care: Reflex tachycardia, lupus-like syndrome (hydralazine), pericardial effusions (minoxidil), sodium and fluid retention * arthralgia, arthritis, fever, myalgia, pleural effusions; resolves upon discontinuation



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Adverse effects of Cardiovascular medication (cont)

β-Blockers IF a β-blocker is stopped abruptly = REBOUND
TACHYCARDIA + Tachycardia, headache, tremor, chest pain, arrhythmia or myocardial infarction
IF a β-blocker needs to be discontinued, it should be tapered slowly over 1-2 weeks
Hypotension, Bradycardia, Hyper/hypoglycemia (depends on individual agent), Hyperlipidemia, Nausea, Shortness of breath, fatigue, diminished libido, Dizziness
Depending on the selectivity of the individual agent, β-blockers can cause both hypoglycemia AND hyperglycemia
In addition, they can also MASK symptoms of hypoglycemia (things like tachycardia, tremor, and anxiety) (see Module 6) The only symptom that remains unopposed is SWEATING

Cardiac Glycosides dysrhythmias, nausea, vomiting, anorexia, visual disturbances
Digoxin Narrow therapeutic range = toxicity

Adverse effects of Cardiovascular medication (cont)

Digoxin Toxicity Acute Toxicity: anorexia, nausea, vomiting, lethargy, confusion, weakness, hyperkalemia, dysrhythmias
Chronic Toxicity: abdominal pain, anorexia, dysrhythmias, confusion, delirium, disorientation, headache, hypokalemia, hypomagnesemia, nausea, vomiting, ocular disturbances

Loop Diuretics hypokalemia, dysrhythmias (related to K+), dehydration, hypotension

Furosemide

Sodium Channel Blockers nausea, anorexia, diarrhea, vomiting, abdominal pain, headache, dysrhythmias, hypotension
Propranolol High doses may result in confusion or psychosis
Propranolol Lupus effect – agranulocytosis, bone marrow depression, anemias – 30-50% of patients using > 1 year



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Adverse effects of Cardiovascular medication (cont)

Potassium Channel Blockers	pneumonia-like syndrome, blurred vision, photosensitivity, nausea, vomiting, anorexia, fatigue, dizziness, and hypotension
<i>Amiodarone</i>	Corneal microdeposits = blurred vision = permanent blindness Neurological abnormalities in 20-40% patients (Delirium, confusion, tremors, sleep disturbances) Pulmonary abnormalities in 10-15% GI – 25% Further dysrhythmias Elevated liver enzymes = Cirrhosis Blue/grey skin abnormality; photosensitivity; alopecia (hair loss) Hypo- or hyperthyroidism
Calcium Channel Blockers (CCBs)	headache, constipation, hypotension, peripheral edema, dizziness
<i>Verapamil or diltiazem</i> (cardioselective)	Less peripheral effects than nifedipine (vessels > heart) Avoid grapefruit juice (possible toxicity due to CYP3A4 inhibition)

Adverse effects of Cardiovascular medication (cont)

Warfarin	Hemorrhage – of any type Upper and lower GI tract (gums --> rectum) Respiratory Genitourinary tract Skin All other adverse effects are rare
Antiplatelets <i>ASA (acetylsalicylic acid)</i>	Can cause GI upset because they also inhibit prostaglandin synthesis in the stomach, which ↓ mucosal lining nausea, dyspepsia, increased risk of bleeding 81mg = "Baby Aspirin"- often recommended to prevent cardiac event in high risk patients We do not give Aspirin to babies
Thrombolytics	high bleeding risk, watch for cognitive change which could be a sign of cerebral hemorrhage
Anti-fibrinolytics	Most common adverse effect = infusion site reactions They also slow down blood flow = bradycardia, hypotension
Vitamin B12	Rare adverse effect: low potassium
Iron	All oral supplements can cause nausea, dyspepsia, GI bleeding, constipation, black stool (Take with food)



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Adverse effects of Cardiovascular medication (cont)

Statins <i>Atorvastatin</i> (<i>Lipitor®</i>)	intestinal cramping, diarrhea, constipation and rarely liver damage, rhabdomyolysis All adverse effects (even nausea and vomiting) are rare
Fibrates <i>Fenofibrate</i>	heartburn, abdominal pain, diarrhea, nausea, flatulence, skin reactions (itchiness, redness, rash), rhabdomyolysis, liver damage Not as well tolerated as statins
Niacin	: flushing, nausea, abdominal pain, hyperglycemia, gout, flatulence, rhabdomyolysis
Bile Acid Resins <i>Cholestyramine</i>	Adverse effects limited to gastrointestinal reactions: constipation (ensure sufficient water intake), bloating, gas, nausea, steatorrhea Drug interactions: May potentially alter absorption of any drug, vitamin, or mineral Separate by 2 hours (you will see variations of this)
PCSK9 Inhibitors <i>Alirocumab</i> , <i>evolocumab</i>	local injection site reactions, upper respiratory tract infections, itch
Nitroglycerin	headache, reflex tachycardia, flushing, hypotension ANYTHING THAT CAUSES VASODILATION WILL CAUSE REFLEX TACHYCARDIA

Adverse effects of Cardiovascular medication (cont)

β-Blockers	hypotension, bradycardia, hypoglycemia, hyperglycemia, etc.
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The 3 Variables of Blood Pressure

Blood Volume	Blood volume is regulated by the kidneys. Blood volume measurement may be used in people with congestive heart failure, chronic hypertension, kidney failure and critical care.
Peripheral resistances	the resistance of the arteries to blood flow. As the arteries constrict, the resistance increases and as they dilate, resistance decreases. Peripheral resistance is determined by three factors: 1. Autonomic activity: sympathetic activity constricts peripheral arteries. 2. Pharmacologic agents: vasoconstrictor drugs increase resistance while vasodilator drugs decrease it. 3. Blood viscosity: increased viscosity increases resistance.



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The 3 Variables of Blood Pressure (cont)

Cardiac output the amount of blood pumped by each ventricle per minute.
To calculate this value, multiply stroke volume (SV), the amount of blood pumped by each ventricle, by the heart rate (HR) in beats per minute.
Use following equation: $CO = HR \times SV$.

These are the different things that we can manipulate (with drugs), in order to affect blood pressure

Hormones and Neurotransmitter involved in BP

Antidiuretic hormone (ADH) released by hypothalamus and pituitary that: Keeps fluid in the body Constricts blood vessels

Epinephrine and norepinephrine both constrict blood vessels via adrenergic receptors

Aldosterone released by adrenal glands that tells kidney to keep sodium (and therefore water) in the body

Remember Wherever sodium goes, water follows

Homeostasis

Detected by:

1. chemoreceptors measure CHEMICALS levels like pH, levels of oxygen, carbon dioxide
2. baroreceptors measure PRESSURE levels

Controlled by:

1. Autonomic nervous system
2. Renin-angiotensin-aldosterone system (RAAS)

Kidneys and Diuretics

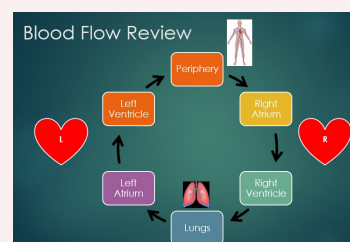
Filtration when urine is first created, substances are filtered from blood --> urine

Reabsorption substances move back from urine --> blood through tubules

Secretion – substances move from blood --> urine through tubules

Urine = Filtration- Reabsorption + Secretions

What is heart Failure ?



The inability of the heart to pump enough blood to meet the body's metabolic demands

A weakened heart

Pre-load ≠ Afterload

Classic Presentation = FED

Fatigue, Edema, Dyspnea

If Heart Failure is in the left, it will back up into the lungs (congestion and pulmonary edema)

If Heart Failure is in the right, it will back up into periphery (peripheral edema, leg edema)

Classes of medication for Heart Failure

ACE inhibitors (ACE-I) Reduce afterload = improve cardiac output
Dilate vessels = decreasing preload
Interrupts the RAAS, which enhances excretion of sodium and water
Lowers peripheral resistance and reduces blood volume
Drug of choice for heart failure because it interrupts both compensatory mechanisms



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Classes of medication for Heart Failure (cont)

Angiotensin II receptor blockers (ARBs)	Reduce afterload = improve cardiac output Indirectly dilate vessels = decreasing preload Block angiotensin II from causing vasoconstriction and block adrenal glands from releasing aldosterone Same pathway as ACE-I, different place in the pathway Like ACE-Is, interrupt both compensatory mechanisms used in clients who have not responded to ACE-I
β-blockers	Slow heart rate and reduce blood pressure = reduce cardiac workload and provides rest Negative inotropic effect Decreased heart contractility Blocks the over-stimulation of sympathetic nervous system (fight-or-flight) that occurs in patients with heart failure Must be introduced slowly and NEVER abruptly stopped Generally, we avoid β-blockers in patients with Diabetes (Type 1 & 2) and patients who are at a high risk of hypoglycemia (ex. elderly)

Classes of medication for Heart Failure (cont)

Cardiac glycosides	Slows heart rate by acting on SA and AV nodes = improves cardiac output
<i>Digoxin</i>	Requires steady levels of potassium for action Positive inotropic effect Increases heart contractility Second-line treatment for heart failure (primary treatment for arrhythmias) NARROW THERAPEUTIC RANGE DRUG Requires drug monitoring to ensure proper loading dose, digitalization, and doses to maintain steady state Mechanism of action: increases the contractility of myocardial contraction (+ inotropic) – requires steady levels of potassium for action Used for dysrhythmias and heart failure IF other drugs fail



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Classes of medication for Heart Failure (cont)

Diuretics Work in different places in the nephron of kidney
Reduce blood volume and cardiac workload
ALSO reduce edema and pulmonary congestion Mostly for symptom relief of excess fluid
Used in addition to other heart failure drugs
As heart failure progresses, we see the stronger loop diuretics (furosemide) used more often, at higher doses
Mechanism of action: prevents reabsorption of sodium and chloride, primarily in the Loop of Henle to increase urine flow, reduce blood volume and cardiac workload
For symptomatic relief of excess fluid

Vasodi-lators Relax blood vessels = lowers blood pressure = reduces afterload and preload
Minor role in heart-failure treatment
Hydralazine: arteries > veins (afterload)
Isosorbide: veins > arteries (preload) For heart failure, sometimes are used together for highest effect

Bleeding disorders

Can be due to disease of bone marrow (where we make blood cells), or genetics

Hemophilia's there are lots of types, depending on which factor they lack

Von Willebrand's Disease lack von Willebrand factor

We focus treatment on trying to get the blood to clot, or stopping bleeding

VIP clotting factors

Factors involved in forming a blood clot:

Platelets

Prothrombin --> (prothrombin activator) --> thrombin --> fibrinogen --> fibrin strands

Vitamin K

Factors involved in dissolving a blood clot (fibrinolysis): Plasminogen --> (tissue plasminogen activator) --> plasmin

Thrombus = a stationary clot

Embolus = a travelling clot

Deep Vein Thrombosis (DVT) = clot in veins of leg (calf)

Pulmonary Embolism (PE) = clot that has travelled to the lung

Cerebrovascular Accident (CVA) (Stroke) = clot that has travelled to the brain a stroke can also be caused by a bleed in the brain

Clinical presentation of Clot

Swift neurological status change

Swollen, red, sore calf (DVT)

Signs of myocardial infarction (chest pain)

Signs of stroke (one-sided weakness or numbness, sudden confusion, trouble speaking, difficulty understanding speech, vision loss, loss of balance and coordination)

Dyspnea, chest pain, coughing up blood (pulmonary embolism)

Colour changes in skin



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Classes of medication for bleeding disorders

Anticoagulants	Prevent a clot from forming, either by inhibiting a specific clotting factor or by inhibiting platelet action NOT = BLOOD THINNERS
a. Unfractionated heparin	Does not dissolve a clot, but prevents them from getting bigger and new ones from forming Binds to multiple clotting factors SC or IV only - no oral or IM Do not massage injection site (bleeding & bruising) Short half-life (1.5h) – used in situations where we need it to work quickly, or have the ability to stop it quickly (like pre-surgery) Antidote = protamine – works within 5 minutes Dose is dependent on condition

Classes of medication for bleeding disorders (cont)

b. Low molecular weight heparins (LMWH)	Longer duration of action and more predictable response, so often a choice for discharge (can teach patient to do SC injection) Doses are decided according to patient weight and what we're treating (post-surgery, treat DVT, prevent clot for dialysis) – so, DOUBLE OR TRIPLE CHECK CORRECT DOSAGE
<i>Tinzaparin, enoxaparin, dalteparin</i>	SC injection or directly in hemodialysis catheter; no IM Still use protamine as antidote, but not as effective



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Classes of medication for bleeding disorders (cont)

c. Warfarin Inhibits the synthesis of multiple clotting factors
 Oral therapy for people with a long-term need for anticoagulation (atrial fibrillation, valve replacement, treatment of DVT or PE)
 Warfarin takes ~ 3 days to reach a therapeutic level, so when transitioning from heparin/LMWH to warfarin, there must be an overlap of therapies
 Even higher risk of bleeding during overlap
 Antidote = vitamin K – works in a few hours
 This is why we caution foods high in vitamin K, because we want stability of anticoagulation
 Important to take at same time each day (most institutions will give all warfarin at the same time – like supper)
MUST GIVE CORRECT DOSE
 Patient must be consistent with checking for drug interactions and signs of bleeding

Classes of medication for bleeding disorders (cont)

d. New Oral anticoagulants (NOACs) Inhibit more specific clotting factors *Rivaroxaban (Xarelto®)*, *apixaban (Eliquis®)* = inhibit Factor Xa
Dabigatran (Pradaxa®) = thrombin inhibitor
 Pros: No INRs, predictable response, one dose ✓ less chance of error
 Cons: No antidote, need dosage adjustment in kidney failure, \$\$, more dyspepsia than warfarin, more difficult to individualize therapy with restricted doses
 All still cause bleeding, many drug interactions

Antiplatelets Can be given along with anticoagulants, because affect different places in clotting cascade
ASA, *dipyridamole*, *clopidogrel*, *ticlopidine* +++ bleeding risk if combined
 Can cause GI upset because they also inhibit prostaglandin synthesis in the stomach, which ↓ mucosal lining

ASA Irreversibly binds to cyclo-oxygenase in platelets, which prevents it from aggregating
 Effects of one dose lasts 7-10 days (irreversible binding)



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Classes of medication for bleeding disorders (cont)

Thrombolytics TPA = tissue plasminogen activator OR other drugs that do same thing (alteplase)
Convert plasminogen --> plasmin, which breaks down many clotting factors
Destroy a clot that's already formed --> used in emergency situations (like stroke, MI, DVT, PE)
If the patient is actively bleeding DO NOT GIVE
Dosed according to weight
Only administered by RN with special training and in facility with appropriate equipment to monitor for hemorrhage

Classes of medication for bleeding disorders (cont)

Antifibrinolytics Promote clotting, to prevent bleeding during surgery or emergency
They also slow down blood flow --> bradycardia, hypotension
Tranexamic acid most common (can give orally)
All are rarely prescribed compared to anticoagulants
Many biologics developed for genetic conditions that lack a clotting factor (products very specific to type of hemophilia)
Used to both prevent and treat bleeding – treatment would continue for life (most intervals every 3-4 days, longer intervals with newer products) but dosages change
Most developed using recombinant DNA to replace missing factor

About coronary artery disease

Atherosclerosis = narrowing or occlusion of an artery due to plaque



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About coronary artery disease (cont)

Plaque = a fatty, fibrous material that accumulates gradually due to high cholesterol – attracts WBCs, platelets, remnants of dead cells, fibrin that narrows and then eventually occludes the artery
Also makes the vasculature less elastic, which means it can't respond to dilation

Coronary Artery Disease (CAD) = narrowing or occlusion of the coronary arteries

Angina Pectoris = chest pain caused by insufficient oxygen to a portion of the myocardium

Types of Angina

1. Stable Angina – when symptoms are predictable as to frequency, intensity and duration
2. Variant Angina – when the chest pain is caused by spasms of the smooth muscle of coronary arteries rather than atherosclerosis
3. Unstable Angina – when symptoms are more intense and occur during periods of rest; unpredictable

Classes of Medication for Angina

Nitrates Potent vasodilator
Nitroglycerin Relaxes arterial and venous smooth muscle – opens up everything
Decreases workload of the heart and myocardial oxygen demand --> chest pain alleviated
Short acting formulations: nitroglycerin sublingual spray or tablets – for emergencies
Long acting formulations: isosorbide – for prevention of frequent angina episodes; nitroglycerin patch
Can be given sublingual (SL), orally, IV, transdermally, topically; SL = relief in 4 minutes

Classes of Medication for Angina (cont)

β-Blockers Reduces cardiac workload
Slows heart rate and reduces contractility
Used for prevention of chronic angina (if occurring often or unstable)
Cardio-selective preferred

Calcium Channel Blockers (CCB) Reduce cardiac workload and dilate coronary arteries, and reduce peripheral resistance (depends on selectivity)
Bring more oxygen to myocardium
Both types (cardio-selective and non) work
First choice for prevention of variant angina because they help prevent the cardiac muscle spasm
For those intolerant/contraindicated for β-blockers (elderly, diabetic, asthma/COPD)

Respiratory diseases

Asthma Chronic inflammatory disease of the airway with 2 components
Inflammation treat w/ anti-inflammatories
Bronchoconstriction treat w/ bronchodilators
Often have triggers that cause exacerbations Environmental (pets, foods, pollens), NSAIDs, cold weather



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Respiratory diseases (cont)

Chronic Obstructive Pulmonary Disease COPD	Lung disease that includes chronic bronchitis and emphysema Chronic bronchitis: airways are swollen and filled with mucous Emphysema: air sacs are damaged, leaving less surface area for oxygen to enter blood stream COPD patients have frequent lung infections and exacerbations – frequent hospitalizations
Common Cold	Viral infection of upper respiratory tract (URTI) Antibiotics not indicated or appropriate Treat symptoms only – resolves by itself Cough Congestion Fever Body aches, mild headache

Inhalers

Advantages	Large surface area for absorption, Direct to site of action, resulting in fast onset, Reduces systemic side effects (does not eliminate)
Disadvantages	Precise doses dependent on patient condition/abilities, Correct use of devices critical, Some oral absorption due to inadvertent swallowing

Types of Inhalation Devices

Inhalers (cont)

1. Metered Dose Inhaler (MDI)	Deliver drugs via a propellant (drug is in a solution) Requires hand-eye co-ordination Spacers and aerochambers improve distribution
2. Dry Powder Inhalers (DPI)	Delivers medication in a powder form, using patient's own inhalation (no propellant) Requires ability to inhale quickly and deeply Leaves slight residue in mouth Cannot use spacers with these devices
3. Nebulizers	Vaporize a liquid into a fine mist Requires a machine Takes a long time to deliver one dose (time-consuming) Inconvenience of being near machine for every dose

Classes of Medication for Respiratory diseases

Bronchodilators	Target the bronchoconstriction component Used in both asthma and COPD (or any time bronchodilation is needed) Literally open up the airway to let air in (make airways bigger)
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Classes of Medication for Respiratory diseases (cont)

1. β -Agonists
 - Open up the airway very quickly
 - Relax bronchial smooth muscle – selective for β_2 (stimulating sympathetic)
 - We don't give orally because 1) would not act as fast, and 2) tachycardia
 - Short acting are "rescue" agents – *salbutamol*
 - Long acting are used more as disease progresses for maintenance therapy – *salmeterol*, *formoterol*, *indacaterol*, *vilanterol*
2. Anticholinergics
 - Bronchoconstriction that occurs in both asthma and COPD is largely caused by stimulation of muscarinic receptors – so blocking this pathway makes sense
 - Don't work as fast as β -agonists
 - Does NOT make any clinical difference in secretions
 - Could either provide a benefit OR an adverse effect
 - Acute and maintenance therapy – Newer agents better for long term

Classes of Medication for Respiratory diseases (cont)

- lpratr-
opium
(Atrov-
ent@)*
 - Used mostly in COPD
 - Must be dosed quite often due to short duration of action (~q4h – approximately every 4 hours)
- 3. Methylxanthines
 - Induces Fight-or-flight response
 - Stimulants, similar in structure to caffeine ☑ stimulate the CNS ☑ relax bronchial smooth muscle
 - Narrow therapeutic range (requires monitoring), adverse effects (stimulant!), and numerous drug interactions limit its use to severe asthma that has not responded to other treatments
 - Oral or IV route

Anti-inflammatories



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Classes of Medication for Respiratory diseases (cont)

1. Corticosteroids
Anti-inflammatory and immuno-suppressive
Used to prevent exacerbations and progression of disease
Suppress airway inflammation and secretions
Must be used daily to work; won't provide "rescue" if used as needed (PRN) by patient
Dose is increased OR switched to oral during exacerbation
Inhaled route minimizes numerous systemic steroid side effects

Fluticasone (Flovent®)
Produces anti-inflammatory and immunosuppressive effects ☑ reduces inflammation and secretion
Used in both asthma and COPD

2. Leukotriene Receptor Antagonists
Reduce inflammation by blocking leukotrienes in inflammation cascade; also useful in allergies
Preventative – not "rescue"
Not as effective as corticosteroids
Must be taken daily to work
Oral

Miscellaneous

Classes of Medication for Respiratory diseases (cont)

Omalizumab a monoclonal antibody (biologic) that attaches to IgE to prevent inflammation from triggers

Roflumilast oral phosphodiesterase-4 inhibitor (PDE4); taken daily to prevent inflammation associated with COPD

Acetylcysteine a mucolytic: dissolves or breaks up mucous in lungs, making easier to get out (less viscous)

Pulmonary vasodilators specific for receptors in lungs; use potent vasodilators such as nitric oxide; will still have systemic effects (hypotension --> reflex tachycardia)

Cold symptom relief Medication

Antitussives suppress cough by stimulating opioid (sigma) receptors
dextromethorphan (DM), codeine

Decongestants stimulants that cause vasoconstriction and shrink swollen mucous membranes
pseudoephedrine, phenylephrine

Expectorants increases mucous flow/movement so it can be expelled by coughing
guaifenesin

Anti-histamines antagonize histamine receptors (involved in allergic response); better for allergy symptoms than common cold; may help sneezing
diphenhydramine, chlorpheniramine



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Classes of Medication for Respiratory diseases (cont)

Acetam fever or aches/pains, included if product says "...& Flu";
inophen an extra ingredient in most combo products

Adverse effects of Respiratory Medication

β-Agonists tachycardia, anxiety, arrhythmias, nervousness,
Salbutamol restlessness, tremor, vertigo, headache,
(Ventolin®) hypokalemia
 Typical dose: 1-2 puffs up to QID PRN
 Caution if arrhythmias or on β-blockers

Anticholinergics hoarseness, dry mouth, cough, bitter taste (rinse
lpratropium mouth after use)
(Atrovent®) Caution in conditions contraindicated to anticholinergic use (elderly, incontinence, glaucoma, kidney disease) – may still be used due to little systemic absorption but will still monitor

Methylxanthines Narrow therapeutic range (requires monitoring),
Theophylline, adverse effects (stimulant!),
aminophylline, and numerous drug interactions limit its use to
oxtriphylline severe asthma that has not responded to other treatments

Adverse effects of Respiratory Medication (cont)

Corticosteroids hoarseness, change in voice, thrush, watch for
Fluticasone systemic steroid effects (hypertension, hyperglycemia, osteoporosis)
(Flovent®) MUST RINSE MOUTH AFTER USE TO PREVENT THRUSH (ORAL CANDIDIASIS – FUNGAL INFECTION) DUE TO IMMUNOSUPPRESSIVE QUALITIES

Leukotriene Receptor Antagonists Few adverse effects/well tolerated: headache, cough, GI upset



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