Cardiovascular diseases

Hypertension
Heart Failure
Dysrhythmias
Angina & Myocardial Infarction
Lipids
Coagulation
Anemia

Classes of Medication for Hypertension

1. Diuretics	Diuretics make you pee 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

Classes of Medication for Hypertension (cont)	
c.Potassium sparing diuretics *Spironolactone*	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
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
Nifedipine	Mechanism of action: blocks calcium channels in myocardial and vascular smooth muscle (blood vessels > heart) – long-acting dihydropyridine (LA-DHP)

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-angio-tension-aldo-sterone 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
a.Angiotensin-converting-enzyme inhibitors (ACE Inhibitors) *-pril*	Inhibit angiotensin-converting-enzyme (ACE), resulting in less angiotensin II and aldosterone, which reduces blood pressure Prevents conversion of angiotensin I to angiot-ensin II, therefore: Prevents aldosterone secretion Prevents the direct vasoconstriction *Pregnancy category D*

Classes of Medication for Hypertension (cont)	
b.Angiotensin 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
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)

Classes of Medication for Hypertension (cont)	
b.α1-B-lockers	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-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 *Hydralazine, minoxidil, nitroprusside*	Directly relax arteriolar smooth muscle in blood vessels reduce peripheral resistance Work at cellular level – each differently Quickly reduce blood pressure Generally reserved for acute care to dilate quickly under close monitoring

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) *Procainamide*	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

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
Amiod-arone	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
Adeno-sine	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
Erythr-opo-iesis	the process of making erythrocytes in bone marrow If we are lacking a substance for erythropoiesis, we won't have as many RBCs

About anemia (cont)

Erythr-opoietin	– the hormone released by the kidneys that instructs the 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

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

Classes of Medication for Anemia (cont)

Folic Acid	Required for erythropoiesis
Folate	Does not require intrinsic factor to absorb from GI (more readily absorbed) 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 (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 *Atorvastatin (Lipitor®)*	Inhibit HMG-CoA-reductase, which is involved in the synthesis of cholesterol in the liver Reduces the amount of cholesterol made by our body 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.

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

Classes of medication for lipids (cont)

PCSK9 Inhibitors *Alirocumab, evoloc-umab*	Class of biologics for very high-risk patients (of a cardiovascular event) who have not reached targets with statins 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 *Hydrochlorothiazide (HCTZ)*	Electrolyte imbalances (especially loss of potassium - hypokalemia) – monitor all electrolytes 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 *Nifedipine*	Dizziness, hypotension, headache, flushing, reflex tachycardia*, constipation, peripheral edema

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 *Doxazosin*	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 *Hydralazine, minoxidil, nitroprusside*	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

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 *Digoxin*	dysrhythmias, nausea, vomiting, anorexia, visual disturbances 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 *Furosemide*	hypokalemia, dysrhythmias (related to K+), dehydration, hypotension
Sodium Channel Blockers *Procainamide*	nausea, anorexia, diarrhea, vomiting, abdominal pain, headache, dysrhythmias, hypotension High doses may result in confusion or psychosis Lupus effect – agranulocytosis, bone marrow depression, anemias – 30-50% of patients using > 1 year

Adverse effects of Cardiovascular medication (cont)	
Potassium Channel Blockers *Amiodarone*	pneumonia-like syndrome, blurred vision, photosensitivity, nausea, vomiting, anorexia, fatigue, dizziness, and hypotension 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) *Verapamil or diltiazem* (cardioselective)	headache, constipation, hypotension, peripheral edema, dizziness 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 *ASA (acetylsalicylic acid)*	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 *Atorvastatin (Lipitor®)*	intestinal cramping, diarrhea, constipation and rarely liver damage, rhabdomyolysis All adverse effects (even nausea and vomiting) are rare
Fibrates *Fenofibrate*	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 *Cholestyramine*	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 *Alirocumab, evolocumab*	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.

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.

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 × 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

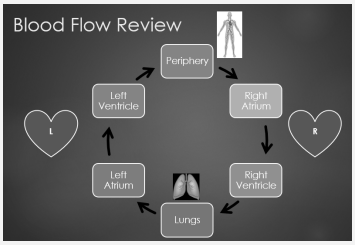
2.Renin-angiotensin-aldosterone system (RAAS)

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 ǂ 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

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 glycos-ides *Digoxin*	Slows heart rate by acting on SA and AV nodes = improves cardiac output 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

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
Vasodil-ators	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) --> thromb-in--> 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

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.Unfract-ionated 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) *Tinzaparin, enoxap-arin, dalteparin -parin*	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 SC injection or directly in hemodialysis catheter; no IM Still use protamine as antidote, but not as effective

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 *ASA, dipyridamole, clopidogrel, ticlopidine*	Can be given along with anticoagulants, because affect different places in clotting cascade +++ 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)

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

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, transderm-ally, 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 Enviro-nmental (pets, foods, pollens), NSAIDs, cold weather

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)

Classes of Medication for Respiratory diseases (cont)	
1. β-Agonists	Open up the airway very quickly Relax bronchial smooth muscle – selective for β2 (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)	
Ipratropium (Atrovent®)	Used mostly in COPD Must be dosed quite often due to short duration of action (~q4h – approximately every 4 hours)
3. Methylxanthines *Theophylline, aminophylline, oxtriphylline*	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 *dextromethorphan (DM), codeine*	suppress cough by stimulating opioid (sigma) receptors
Decongestants *pseudoephedrine, phenylephrine*	stimulants that cause vasoconstriction and shrinks swollen mucous membranes
Expectorants *guafenesin*	increases mucous flow/movement so it can be expelled by coughing
Anti-histamines *diphenhydramine, chlorpheniramine*	antagonize histamine receptors (involved in allergic response); better for allergy symptoms than common cold; may help sneezing

Classes of Medication for Respiratory diseases (cont)	
Acetaminophen	fever or aches/pains, included if product says "…& Flu"; an extra ingredient in most combo products

Adverse effects of Respiratory Medication	
β-Agonists *Salbutamol (Ventolin®)*	tachycardia, anxiety, arrhythmias, nervousness, restlessness, tremor, vertigo, headache, hypokalemia Typical dose: 1-2 puffs up to QID PRN Caution if arrhythmias or on β-blockers
Anticholinergics *Ipratropium (Atrovent®)*	hoarseness, dry mouth, cough, bitter taste (rinse mouth after use) 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 *Theophylline, aminophylline, oxtriphylline*	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

Adverse effects of Respiratory Medication (cont)	
Corticosteroids *Fluticasone (Flovent®)*	hoarseness, change in voice, thrush, watch for systemic steroid effects (hypertension, hyperglycemia, osteoporosis) MUST RINSE MOUTH AFTER USE TO PREVENT THRUSH (ORAL CANDIDIASIS – FUNGAL INFECTION) DUE TO IMMUNO-SUPPRESSIVE QUALITIES
Leukotriene Receptor Antagonists	Few adverse effects/well tolerated: headache, cough, GI upset