

# www.natures.ir

More Free USMLE , MCCEE ,MCQe and AMQ Flashcards

What does tachycardia do to coronary artery blood flow?

decreases filling time and decreases diastole

(NOTE: coronary vessels fill in diastole)

What is the most common site of coronary artery thrombosis?

LAD

(NOTE: ant portion of left ventricle and ant two thirds of the interventricular septum)

What is the most common manifestation of coronary artery disease?

angina pectoris, which is the most common type of ischemic heart disease

What is the most important risk factor for angina pectoris?

age

(more common in men and peaks in after age of 60 and peaks after 70 in women)

What is the most common type of angina?

stable angina (exercise induced substernal chest pain)

(within one year of stable angina diagnosis, 20% develop MI or unstable angina)

What is the pathogenesis of chronic stable angina and what do you see on EKG?

Subendocardial ischemia due to decreased coronary artery blood flow or thick muscle wall with ST segment depression

What is the pathogenesis of Prinzmetal's angina?	vasospasm at rest with or without coronary artery atherosclerosis with transmural ischemia and ST segment elevation
What are some characteristics of unstable angina?	angina at rest; multivessel disease; disrupted plaques  (patient will have frequent bouts of angina at rest with min exertion)
What is a treatment for Prinzmetal's angina?	calcium channel blockers that vasodilate coronary arteries
What is seen in patients with chronic ischemic heart disease?	replacement of myocardial tissue with noncontractile scar tissue
Is coronary artery thrombosis usually present in Sudden cardiac death?	no, typically cause of death is ventricular fibrillation
What is the definition of sudden cardiac death?	unexpected death within 1 hour after the onset of symptoms  (diagnosis of exclusion)
What is the most common cause of death in the US?	Acute MI  (prominent in males 40-65)

What is the typical pathogenesis of AMI?	<p>rupture of disrupted plaque -&gt; platelet thrombus -&gt; AMI</p> <p>(thromboxane A2 causes vasospasm of artery to reduce blood flow)</p>
Can cocaine use with normal coronary arteries cause AMI?	Yes
What type of MI involves Q wave formation? non Q wave?	<p>Q wave = transmural infarction</p> <p>non-Q wave = subendocardial infarction</p>
What can increase the short and long term survival of AMI?	Reperfusion injury following thrombolytic therapy
What is contraction band necrosis following reperfusion mean?	hypercontraction myofibrils due to $\text{Ca}^{2+}$

<p>What are some gross and microscopic findings of AMI within 24 hours? 1-3 days? 3-7 days? 7-10 days? 2 months?</p>	<p>24 hrs - no gross changes but there is coagulation necrosis</p> <p>1-3 days - neutrophils abundant and lyse dead myocardial cells</p> <p>3-7 days - heart is softest and danger of rupture</p> <p>7-10 days - necrotic area yellow and collagen formation present</p> <p>2 months - infarcted tissue replace with white, patchy, noncontractile scar tissue</p>
<p>What are classic clinical findings in AMI?</p>	<p>retrosternal pain, radiation to left arm/shoulder, diaphoresis</p>
<p>Is there a higher mortality rate in Q wave or non-Q wave AMI?</p>	<p>Q wave</p> <p>Non Q wave is increased risk for sudden cardiac death</p>
<p>What is the most common cause of death in AMI?</p>	<p>ventricular fibrillation</p>
<p>What is the most common time for the heart to rupture post AMI?</p>	<p>3- 7 days</p>

What are the consequences of a posteromedial papillary muscle rupture?	RCA thrombosis and mitral regurgitation and LHF
What is a danger of a mural thrombus?	embolization because its a thrombus of a large vessel
What type of fibrinous pericarditis develops 1-7 days of a Q wave AMI?	Early acute inflammation AMI due to increased vessel permeability in pericardium and exudate of acute inflammation
When does autoimmune pericarditis develop?	6-8 weeks after an AMI and autoantibodies direct against damaged pericardial antigens (late complication)
When a ventricular aneurysm forms following AMI, what is the most common cause of death?	CHF occurs due to the lack of contractile tissue  (Rupture is uncommon because of the good tensile strength of scar tissue)
What are some clinical findings in a RV AMI?	RHF, hypotension, preserved LV function  (assoc with RCA thrombosis)
What are the common cardiac lab tests to run for AMI?	creatine kinase isoenzyme (CKMB) within 4-8 hrs, peaks at 24 hrs  cardiac troponins I and T within 3-12 hrs, peaks at 24 hrs  LDH 1-2 "flip" within 10 hrs, peaks at 2-3 days

What is the gold standard for diagnosis of AMI?	cardiac troponins I and T  (NOTE: these cannot diagnose reinfarction so CKMB is used in conjunction with troponins to diagnose)
What do you see in labs if reinfarction occurs?	reappearance of CKMB after 3 days
What are classic EKG findings with AMI?	inverted T waves, elevated ST segment, Q waves
What is the primary site for O <sub>2</sub> exchange?	chorionic villus  (the chorionic villus vessels become the umbilical vein)
In fetal circulation, what has the highest PO <sub>2</sub> content?	umbilical vein
What are some unique features of fetal circulation?	foramen ovale and ductus arteriosus are patent
What is there an increased risk for with only a single umbilical artery?	congenital abnormalities

What does the ductus arteriosus become after closure within 2-8 weeks post birth?	ligamentum arteriosum
What is the most common heart disease in children?	congenital heart disease and it increases with increase maternal age
What is a danger of left to right shunts if reversal is not corrected?	reversal of shunt and cyanosis
What is the most common congenital heart disease in children?	ventricular septal defect (defect in the membranous septum)
What is the most common congenital heart disease in adults?	Atrial septal defect and the most common type is the patent foramen ovale (secundum type)
Why is there a fixed splitting of S2 in ASD?	excess blood in RA causes delay in closure of pulmonary valve
What is Patent ductus arteriosus closed with?	indomethacin

What is differential cyanosis mean and what is it seen in?	PDA and it means that you have a pink upper body and cyanotic lower body
What is the most common cyanotic congenital heart disease?	tetralogy of fallot
What is the importance with the degree of pulmonary valve stenosis?	minimal pulmonary valve stenosis is absence of cyanosis  severe PV stenosis leads to cyanosis and increased right to left shunting through VSD
What are the cardioprotective shunts with tetralogy of fallot?	ASD and PDA
What are Tet spells?	hypoxic spells caused by sudden increase in hypoxemia and cyanosis  (squatting increases systemic vascular resistance, causing temporary reversal of the shunt)
What are the heart's anatomical features in complete transposition of the great vessels?	aorta empties RV, pulmonary artery empties LV, and atria are NORMAL
What is infantile coarctation associated with?	Turner's syndrome  (constriction between subclavian artery and ductus arteriosus in infantile)  (constriction of aorta distal to ligamentum arteriosum in adult type)



What is clinically seen in adult coarctation?	<p>disparity between upper/lower extremity blood pressure &gt; 10 mmHg</p> <p>also, see a dilation of aorta and aortic valve ring</p>
Why is hypertension seen in adult coarctation?	<p>decreased blood flow to lower body cause a decrease renal blood flow and this activated the RAA system causing hypertension</p>
What are the types of collateral circulations seen in adult coarctation?	<p>Anterior intercostal arteries with post intercostal arteries</p> <p>sup epigastric artery and inf epigastric artery to external iliac artery</p>
When does acute Rheumatic fever occur?	<p>after group A streptococcal pharyngitis, usually 20 days</p>
What is the pathogenesis of acute Rheumatic fever?	<p>immune mediated type II hypersensitivity reaction because antibodies cross react with similar proteins in human tissue</p> <p>also may be cell-mediated immunity type IV</p>
What is the most common initial presentation with RF?	<p>migratory polyarthritis</p> <p>(occurs in large joints, ankles, wrists)</p>
What is the most common cause of death in RF?	<p>myocarditis</p> <p>(Aschoff bodies present)</p>

In RF, what valves are most often involved?	mitral valve followed by aortic valve
What is seen in acute and chronic attacks of RF?	acute - mitral regurg chronic - mitral stenosis
What are some of the major criteria according to the Jones criteria for RF?	carditis, arthritis, chorea, erythema marginatum, subcutaneous nodules
What is seen in lab test with RF?	increased ASO and DNase B titers (peak at 4-5 weeks after pharyngitis)
What is the most common cause of mitral valve stenosis?	recurrent RF
What is a common clinical finding in mitral stenosis?	atrial fibrillation
Describe the murmur with mitral stenosis.	opening snap followed by an early to mid-diastolic rumble

What is the most common cause of mitral regurg?	mitral valve prolapse
Describe the murmur with mitral regurg.	pansystolic murmur; S3/S4 ; no increase intensity with deep held inspiration
What is assoc with mitral valve prolapse?	Marfan and Ehlers Danlos syndromes (autosomal dominant in some cases)
What is the pathophysiology for MVP?	myxomatous degeneration of mitral valve leaflets and excess production of dermatan sulfate
Describe the murmur with MVP	systolic click followed by murmur
What do the increased and decreased preloads assoc with MVP cause?	decreased preload causes the click and murmur to move closer to S1  increased preload causes click and murmur to move closer to S2
What do you do in a symptomatic MVP patient?	Beta-blocker treatment  (decreases HR and force of contraction leading to less stretch and trauma to the prolapsed leaflets)

What is the most common cause of aortic valve stenosis in patients > 60 years old?	calcific AV stenosis
What is the most common valvular lesion causing syncope and angina with exercise?	AV stenosis
What are some clinical findings with aortic stenosis?	microangiopathic hemolytic anemia with schistocytes, hemoglobinuria
What is the most common cause of aortic regurg?	isolated aortic valve root dilation  (most common infectious disease cause of aortic regurg is infective endocarditis)
What does aortic regurg do to the pulse pressure (difference between systolic and diastolic pressures)?	increases it, produces hyperdynamic circulation
What are some clinical findings with aortic regurg?	early diastolic murmur; bounding pulses; S3, S4; no increased intensity with inspiration
What does the presence of an Austin Flint murmur indicate?	sign for AV replacement

What is the most common cause of tricuspid valve regurg?	stretching of TV ring from RHF, hypertension, dilated cardiomyopathy
What are 3 structures inside the carotid sheath? (Mnemonic)	Internal jugular Vein (lateral), common carotid Artery (medial), vagus Nerve (posterior) ("VAN")
The acute marginal artery supplies which part of the heart?	Right ventricle
The circumflex artery (CFX) supplies which part of the heart?	Posterior left ventricle
The posterior descending/interventricular artery (PD) supplies which part of the heart?	Posterior septum
The left anterior descending artery (LAD) supplies which part of the heart?	Apex and anterior interventricular septum
What artery supplies the AV and SA nodes?	Right coronary artery (RCA)

Where does the posterior descending (PD) artery arise from?	80% of the time it stems from the RCA (=right dominant). 20% of the time it stems from the circumflex artery (= left dominant)
Coronary artery occlusion most commonly occurs where?	Left anterior descending artery (LAD), which supplies the anterior interventricular septum
Do coronary arteries fill on systole or diastole?	Diastole
What is the most posterior part of the heart? What can enlargement of this part cause?	The most posterior part is the left atrium. Enlargement can cause dysphagia or hoarseness.
What are two ways to calculate cardiac output?	$CO = (\text{stroke volume}) \times (\text{heart rate})$ <p>Fick principle: <math>CO = (\text{rate of O}_2 \text{ consumption}) / [(\text{arterial O}_2 \text{ content}) - (\text{venous O}_2 \text{ content})]</math></p>
What are 2 ways to calculate mean arterial pressure (MAP)?	$MAP = \frac{2}{3}(\text{diastolic pressure}) + \frac{1}{3}(\text{systolic pressure})$ $MAP = CO \times (\text{total peripheral resistance})$
How do you calculate pulse pressure?	Pulse pressure = systolic pressure - diastolic pressure. It is proportional to stroke volume.

What are two ways to calculate stroke volume (SV)?	$SV = CO/HR = EDV - ESV$
What are 3 factors that affect stroke volume? (Mnemonic)	Stroke Volume is affected by Contractility, Afterload, and Preload "SV CAP") SV increases when preload increases, afterload decreases, or contractility increases
What is preload?	ventricular EDV
What is afterload?	mean arterial pressure (proportional to peripheral resistance)
What is the difference between the effects of venodilators and vasodilators on the heart?	Venodilators (e.g. nitroglycerin) decrease preload; Vasodilators (e.g. hydralazine) decrease afterload
What relationship does the Starling curve describe?	describes that the force of contraction is proportional to the initial length of cardiac muscle fibers (preload)
How do you calculate ejection fraction (EF)?	$EF = SV/EDV = (EDV-ESV)/EDV$ EF is normally greater than or equal to 55%

What equation relates resistance, pressure, and flow?	$\Delta P = Q \times R$ <p> <math>\Delta P</math> = change in pressure  <math>Q</math> = flow  <math>R</math> = resistance </p>
What's a way to measure resistance in a tube using viscosity? What does viscosity mostly depend on in blood vessels?	$\text{Resistance} = \frac{8 \times \text{viscosity} \times \text{length}}{\pi \times r^4}$ <p>viscosity depends on hematocrit</p>
What type of blood vessels account for most of the peripheral resistance?	Arterioles
Which phase of the left ventricle cardiac cycle has the highest O <sub>2</sub> consumption?	Isovolumetric contraction - period between mitral valve closure and aortic valve opening
What makes up the S1 sound?	Mitral and tricuspid valve closure. Loudest at mitral area.
What makes up the S2 sound?	Aortic and pulmonary valve closure. Loudest at left sternal border.
What makes up the S3 sound?	In early diastole rapid ventricular filling phase. Associated with increased filling pressures and more common in dilated ventricles (but normal in children)



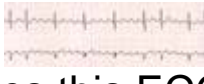
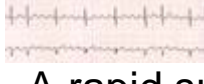
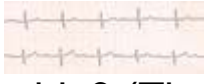
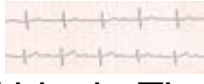


What makes up the S4 sound?	"Atrial kick" - high atrial pressure. Associated with ventricular hypertrophy.
What are the a, c, and v waves in the jugular venous pulse?	a wave = Atrial contraction c wave = RV contraction (tricuspid bulging into atrium) v wave = increased atrial pressured due to filling against closed tricuspid valve
How do the physiologic heart sounds change with inspiration and expiration?	Expiration: S1 stays the same, A2 and P2 (in S2) get closer together Inspiration: S1 stays the same, A2 and P2 (in S2) get farther apart
Wide splitting, where A2 and P2 are not as close together as is normal during expiration and farther apart than is normal during inspiration is associated with what?	Pulmonic stenosis
Fixed splitting, where A2 and P2 are always a fixed distance apart regardless of exhalation or inhalation, is associated with what?	Atrial septal defects (ASD)
Paradoxical splitting, where P2 comes before A2, and where the gap between them is greater on exhalation than inhalation, is associated with what?	Aortic stenosis
When do you get "Pulsus parvus et tardus"?	Pulses weak compared to heart sounds - aortic stenosis

What murmur is described as "Holosystolic, high-pitched blowing murmur"? Where is it loudest and where does it radiate to?	Mitral/tricuspid regurgitation. Mitral - loudest at apex and radiates toward axilla. Tricuspid - Loudest at tricuspid area and radiates to right sternal border.
What murmur is described as having a "crescendo-decrescendosystolic ejection murmur following an ejection click (EC) that radiates to carotids/apex"?	Aortic stenosis. LV >> aortic pressure during systole. Has "Pulsus parvus et tardus" - pulses weak compared to heart sounds.
What murmur is described as a "holosystolic, harsh sounding murmur that is loudest at the tricuspid area"?	VSD
What murmur is described as a "late systolic murmur with midsystolic click (MC). Loudest at S2" ?	Mitral prolapse. Enhanced by vasalva maneuver and decreased by squatting (standing and vasalva decreases the volume of the left ventricle causing prolapse to occur sooner and more severely)
What murmur is described as a "immediate high-pitched blowing diastolic murmur"?	Aortic regurgitation. Wide pulse pressure when chronic
What murmur "Follows opening snap (OS). Delayed rumbling late diastolic murmur"?	Mitral stenosis. Tricuspid stenosis gets louder with inspiration (more blood flows into RA upon inspiration)
What murmur is described as a "continuous machine-like murmur. Loudest at time of S2"?	PDA

What happens during Phase 0 of the ventricular action potential?	Rapid upstroke - voltage gated Na channels open
What happens during Phase 1 of the ventricular AP?	Initial repolarization - inactivation of voltage-gated Na channels. Voltage gated K channels begin to open.
What happens during Phase 2 of the ventricular AP?	Plateau - Ca influx through voltage-gated Ca channels balances K efflux. Ca influx triggers Ca release from sarcoplasmic reticulum and causes myocyte contraction.
What happens during Phase 3 of the ventricular AP?	Rapid repolarization - massive K efflux due to opening of voltage-gated slow K channels and closure of voltage-gated Ca channels.
What happens during Phase 4 of the ventricular AP?	Resting potential - normal K and Na permeability
What phases do pacemaker APs have, and how are they different than ventricular APs?	<p>There's only Phase 0, 3, and 4</p> <p>Phase 0: Upstroke - opening of voltage gated Ca channels.</p> <p>Phase 3 = Rapid repolarization (similar to ventricular AP) Inactivation of the Ca channels and increased activation of the K channels --&gt; K efflux</p> <p>Phase 4 = slow diastolic depolarization - membrane potential spontaneously depolarizes as Na conductance increases. Accounts for the automaticity of SA and AV nodes.</p>

On an ECG, what does the P wave indicate?	Atrial depolarization
On an ECG, what does the PR interval indicate?	Conduction delay through the AV node (normally < 200 msec)
On an ECG, what does the QRS complex indicate?	Ventricular depolarization (normally < 120 msec)
On an ECG, what does the QT interval indicate?	Mechanical contraction of the the ventricles
On an ECG, what does the T wave indicate?	Ventricular repolarization
Where is atrial repolarization on the ECG?	Masked by the QRS complex
On an ECG, what does the ST segment indicate?	Isoelectric, ventricles depolarized

On an ECG, what is a U wave?	Caused by hypokalemia or bradycardia
Associate ventricular tachycardia with shifting sinusoidal waveforms on ECG that can progress to V-fib with...	Torsades des pointes. Anything that prolongs the QT interval can predispose to torsades des pointes.
When there is an accessory conduction pathway from the atria to ventricle (bundle of Kent) that bypasses the AV node, the ventricles may begin to partially depolarize earlier. What characteristic wave is produced, and what can it cause?	Creates a delta wave. May result in reentry current leading to supraventricular tachycardia.
 <p>What does the top line of the ECG show? (the bottom line shows sinus rhythm)</p>	 <p>Atrial fibrillation - chaotic and erratic baseline with no discrete P waves in between irregularly spaced QRS complexes</p>
 <p>What does this ECG show?</p>	 <p>Atrial flutter - A rapid succession of identical, back to back atrial depolarization waves. The identical appearance accounts for the "sawtooth" appearance of the flutter waves.</p>
 <p>What finding is this? (The bottom line is clearer)</p>	 <p>1st degree AV block. The PR interval is prolonged (&gt;200 msc). Asymptomatic.</p>



What finding does this ECG show?

2nd degree AV block Mobitz I (Wenckebach). Progressive lengthening of PR interval until a beat is "dropped" (a P wave not followed by a QRS complex). Usually asymptomatic.



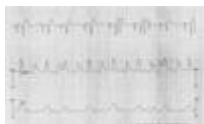
What abnormality does this ECG show?

2nd degree AV block Mobitz II. Dropped beats that are not preceded by a change in the length of the PR interval (as in type I). These abrupt, nonconducted P waves result in a pathologic condition. It is often found as 2:1 block where there are 2 P waves to 1 QRS response (the ratio of P:QRS is always constant). May progress to 3rd degree block.



What condition does this ECG show?

3rd degree (complete) AV block. The atria and ventricles beat independently of each other. Both P waves and QRS complexes are present, although the P waves bear no relation to the QRS complexes. The atrial rate is faster than the ventricular rate. Usually treat with a pacemaker.



What does this ECG show?

Ventricular fibrillation. A completely erratic rhythm with no identifiable waves. Fatal arrhythmia without immediate CPR and defibrillation.

The baro and chemoreceptors on this structure transmit via the vagus nerve to the medulla.


Aortic arch.

How does BP affect the aortic arch?	The aortic arch only responds to increased BP
The baro and chemoreceptors on this structure transmit via the glossopharyngeal nerve to the medulla.	Carotid sinus
How does BP affect the carotid sinuses?	The carotid sinuses respond to both increased and decreased BP
What is the effect of hypotension on baroreceptors?	Decreased BP --> decreased afferent baroreceptor firing --> increased efferent sympathetic firing and decreased efferent parasympathetic stimulation --> vasoconstriction, increased HR, increased contractility, increased BP
What do peripheral chemoreceptors respond to?	Respond to decreased PO <sub>2</sub> (< 600mmHg), increased PCo <sub>2</sub> , and decreased blood pH
What do central chemoreceptors respond to?	Changes in pH and PCO <sub>2</sub> of the brain interstitial fluid, which in turn are influenced by arterial CO <sub>2</sub> . Do not directly respond to PO <sub>2</sub> .

What is the Cushing reaction? What chemoreceptors are responsible for it?	Cushing reaction: increased ICP constricts arterioles --> cerebral ischemia --> hypertension (sympathetic response) and reflex bradycardia. (Cushing triad = hypertension, bradycardia, respiratory depression). The central chemoreceptors are responsible.
What organ takes the largest share of systemic cardiac output?	The liver
Which organ has the highest blood flow per gram of tissue?	Kidney
Which organ has the highest arteriovenous O <sub>2</sub> difference?	The heart
Pulmonary capillary wedge pressure is a good approximation of what? How do you measure it?	Good approximation of left atrial pressure. Measured with a Swan-Ganz catheter.
In pulmonary vasculature, hypoxia causes what response? In all other organs?	In pulmonary vasculature, hypoxia causes vasoconstriction. In all other organs hypoxia causes vasodilation.
What type of shunts cause "blue babies," right to left or left to right?	Right to left shunts



What are 5 congenital syndromes with right to left shunts? (Mnemonic)	"The 5 T's" Tetralogy of Fallot Transposition of great vessels Truncus arteriosus Tricuspid atresia Total anomalous pulmonary venous return (TAPVR)
What is the most common cause of early cyanosis in infants?	Tetralogy of fallot
In right to left shunts, what may children do to increase systemic vascular resistance?	Squat
What are causes of 3 left to right shunts that can lead to "blue kids" (late cyanosis)?	Frequency - VSD>ASD>PDA
What is Eisenmenger's syndrome?	People who have initial left to right shunts will increase pulmonary vascular resistance due to arteriolar thickening. Eventually the progressive pulmonary hypertension will cause the left to right shunt to become a right to left shunt.
What is the most common congenital cardiac anomaly?	VSD
What drug can you give to close a PDA?	Indomethacin

What 4 conditions are involved in the Tetralogy of Fallot? (Mnemonic)	Pulmonary stenosis (most important determinant for prognosis), Right ventricular hypertrophy (RVH), Overriding aorta (overrides the VSD), VSD ("PROVe")
What is the shape of the heart on an x-ray in tetralogy of Fallot?	 <p>Shaped like a boot</p>
How does tetralogy of Fallot develop?	Tetralogy of Fallot is caused by anterosuperior displacement of the infundibular septum
What is transposition of the great vessels?	The aorta leaves the RV (anterior) and the pulmonary trunk leaves the LV (posterior). Not compatible with life unless a shunt is present to allow adequate mixing of blood (e.g. VSD, PDA, or patent foramen ovale).
How does transposition of the great vessels develop?	Due to failure of the aortopulmonary septum to spiral.
What is the infantile type of coarctation of the aorta? (Mnemonic)	Aortic stenosis proximal to insertion of the ductus arteriosus (preductal) ("Infantile: IN close to the heart")
What is the adult type of coarctation of the aorta?	Aortic stenosis is distal to ductus arteriosus (postductal). Associated with notching of the ribs (due to collateral circulation), hypertension in the upper extremities, weak pulses in lower extremities. Associated with Turner's syndrome.

Is coarctation of the aorta more prevalent in males or females?	Male to female ratio 3:1
What drug can be used to close a PDA? What drug can be used to keep it open?	Close: Indomethacin Keep open: PGE
22q11 deletion syndromes are associated with what heart defects?	(persistent) truncus arteriosus, tetralogy of Fallot
Down syndrome is associated with what heart defects?	ASD, VSD, AV septal defect (endocardial cushion defect)
Congenital rubella is associated with which cardiac defects?	Septal defects, PDA, pulmonary artery stenosis
Turner's syndrome is associated with which cardiac defect?	Coarctation of the aorta
Marfan's syndrome is associated with which cardiac defect?	Aortic insufficiency/regurgitation (late complication)

What cardiac defect is associated with being born to a diabetic mother?

Transposition of great vessels

What is the definition of hypertension?

BP > 140/90

What are atheromas?

Plaques in blood vessel walls

Atheromas, xanthomas, and corneal arcus are all signs of what?

Hyperlipidemia

What is Monckeberg arteriosclerosis?

Calcification in the media of the arteries, especially the radial or ulnar arteries. Usually benign; "Pipestem" arteries

What is the name for hyaline thickening of small arterioles in essential hypertension and diabetes mellitus?

Arteriosclerosis



Associate hyperplastic "onion skinning" of arterioles with...



Malignant hypertension

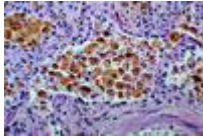
Associate fibrous plaques and atheromas forming in intima of arteries with...	Atherosclerosis
Associate tearing chest pain radiating to the back with...	Aortic dissection
How does aortic dissection appear on x-ray?	Mediastinal widening
What are the 4 most common sites of atherosclerosis?	Abdominal aorta > Coronary artery > Popliteal artery > Carotid artery
What percent of a coronary artery must be occluded to produce angina symptoms?	> 75%
Associate coronary artery spasm with...	Prinzmetal's angina
What is the definition of sudden cardiac death?	Death from cardiac causes within 1 hour of onset of symptoms, most commonly due to a lethal arrhythmia

<p>This type of infarct occurs in loose tissues with collaterals, such as the liver, lungs, and intestine</p>	<p>Red (hemorrhagic) infarcts</p>
<p>This type of infarct occurs in solid tissues with single blood supply, such as heart, kidney, and spleen.</p>	<p>Pale</p>
<p>An infarct that is reperfused is this color</p>	<p>Red ("REd = REperfusion")</p>
<p>What are the 3 most commonly blocked coronary artery branches?</p>	<p>LAD &gt; RCA &gt; Circumflex</p>
<p>How long after a MI do you first start to see contraction bands?</p>	<p>Within 4 hours</p>
<p>When do macrophages first start to replace neutrophils in a MI?</p>	<p>By day 5-10</p>
<p>In the first 2-4 days after a MI, what is there a risk for?</p>	<p>Arrhythmia</p>

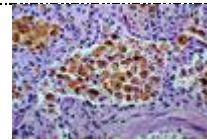
In days 5-10 after a MI, what is there a risk for?	Free wall rupture
Several weeks after a MI, what is there a risk for?	Ventricular aneurysm
What is the gold standard for detecting a MI within the first 6 hours?	ECG
How long after a MI does troponin elevate and for how long?	Cardiac troponin (most specific protein marker) rises after 3 to 6 hours, peaks at 24 hours, and disappears after 7-10 days
How long does it take for CK-MB to rise after a MI?	Appears within 4 to 8 hours, peaks at 24 hours, and disappears within 1.5 - 3 days
A transmural infarct will have what appearance on a ECG?	ST elevation, pathological Q wave
A subendocardial infarct will have what appearance on a ECG?	ST depression

What is Dressler's syndrome?	Autoimmune phenomenon resulting in fibrinous pericarditis (several weeks post-MI)
What type of cardiomyopathy causes systolic dysfunction?	Dilated (congestive) cardiomyopathy
What type of cardiomyopathy causes diastolic dysfunction?	Hypertrophic and restrictive/obliterative cardiomyopathies
What are 6 causes of dilated cardiomyopathy? (Mnemonic)	("ABCCCD") Alcohol abuse, Beriberi, Coxsackie B virus myocarditis, Cocaine use, Chaga's disease, Doxorubicin
Associate sudden death in young athletes with...	Hypertrophic cardiomyopathy
How do you treat hypertrophic cardiomyopathy?	beta blockers, or non-dihydropyridine calcium channel blockers (e.g. verapamil)
What does hypertrophic cardiomyopathy sound like on auscultation?	Loud S4, systolic murmur





The brown cells in this lung tissue are a sign of what?



Heart failure. They are hemosiderin laden macrophages (blood backs up into the lungs due to left ventricle failure and macrophages phagocytose them), and are known as "heart failure cells"

What is the appearance of the liver in CHF?



Nutmeg liver

Right heart failure is most often due to what?

Left heart failure

Isolated right heart failure is usually due to what?

Cor pulmonale

What are 6 types of emboli?  
(Mnemonic)




Fat, Air, Thrombus, Bacteria, Amniotic fluid, Tumor  
("an embolus moves like a FAT BAT")

Fat emboli are associated with what?

Long bone fractures and liposuction

DIC can result from what type of emboli?

Amniotic fluid

What is Virchow's triad, and what can it predispose to?	Stasis, hypercoagulability, endothelial damage. Can lead to deep vein thromboses --> pulmonary embolism.
What is a Roth's spot?	 <p>A round white spot on the retina surrounded by hemorrhage - an indication of bacterial endocarditis</p>
What are Janeway lesions?	 <p>Small erythematous lesions on palm or sole. A sign of bacterial endocarditis.</p>
What is a splinter hemorrhage?	 <p>Hemorrhage on nailbed. A sign of bacterial endocarditis.</p>
What are 8 signs of bacterial endocarditis? (Mnemonic)	Fever, Roth's spots, Osler's nodes, Murmur, Janeway lesions, Anemia, Nail-bed hemorrhage, Emboli ("bacteria FROM JANE")
Associate large vegetations on previously normal valve and rapid onset with...	<i>S. aureus</i>
Associate smaller vegetations on congenitally abnormal or diseased valves and an insidious onset with..	<i>Strep viridans</i> (and dental procedures)

What is the most common valve involved in bacterial endocarditis?

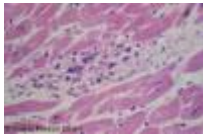
Mitral

What is tricuspid valve endocarditis associated with?


Tricuspid - IV drug abuse ("Don't TRI drugs")

What is a disease where you get Libman-Sacks endocarditis (non bacterial endocarditis)? (Mnemonic)

SLE ("SLE causes LSE")




What is this lesion called and what is it an indication of?




It is an Aschoff body, and it is associated with rheumatic endocarditis. Aschoff bodies are granulomatous structures consisting of fibrinoid change, lymphocytic infiltration, occasional plasma cells, and characteristically abnormal macrophages surrounding necrotic centres. Some of these macrophages may fuse to form multinucleated giant cells.



What type of cells are these, and what do they indicate?



They are Anitschkow cells, and they are pathognomonic for rheumatic endocarditis. The cells are also called caterpillar cells, as they have a large amount of clear cytoplasm surrounding a rod-shaped nucleus that to some resembles a caterpillar.

What are 7 symptoms of rheumatic fever? (Mnemonic)	Fever, Erythema marginatum, Valvular damage, ESR increase, Red-hot joints (polyarthritis), Subcutaneous nodules (Aschoff bodies), St. Vitus' dance (chorea) ("FEVERSS")
Associate electrical alternans (beat-to-beat alternations of QRS complex height), pulsus paradoxus (exaggerated decrease in amplitude of pulse during inspiration), and distant heart sounds with...	Cardiac tamponade
What type of pericarditis is caused by SLE, rheumatoid arthritis, viral infection, and uremia?	Serous pericarditis
What type of pericarditis is caused by uremia, MI, and rheumatic fever?	Fibrinous pericarditis
What type of pericarditis can be caused by TB and malignancies?	Hemorrhagic pericarditis
Syphilis causes dilation of the aorta and valve ring via what mechanism?	Tertiary syphilis disrupts the vasa vasorum leading to dilation of the aorta and valve ring. Can result in aneurysm of the ascending aorta or aortic arch and aortic valve incompetence.
What is the most common primary cardiac tumor in adults?	 <p>Myxomas. They are a benign tumor usually described as a "ball valve" obstruction in the atria (mostly LA).</p>

What is the most common primary cardiac tumor in children?	Rhabdomyomas - associated with tuberous sclerosis.
What are the most common heart tumors overall?	Metastases (melanoma, lymphoma)
Associate arteriovenous malformation in small vessels that look like dilated capillaries with	Telangiectasia. Affects small vessels.
Associate decreased blood flow to the skin due to arteriolar vasospasm in response to cold temperature or emotional stress with...	Raynaud's disease. Affects small vessels.
Associate focal necrotizing vasculitis, necrotizing granulomas in the lung and upper airway, and necrotizing glomerulonephritis with...	Wegner's granulomatosis
What is a strong marker for Wegner's granulomatosis?	c-ANCA
How do you treat Wegner's granulomatosis?	Cyclophosphamide and corticosteroids

What are 3 pANCA positive vasculitides?

Microscopic polyangitis, primary pauci immune crescentic glomerulonephritis (pauci immune = paucity of antibodies), Churg-Strauss syndrome.

Associate granulomatous vasculitis with eosinophilia, involving lung, heart, skin, kidneys, and nerves, and often seen in atopic patients with...

Churg-Strauss syndrome



Associate port-wine stain on face and leptomeningeal angiomatosis (intracerebral AVM) with...

Sturge-Weber disease



Associate palpable purpura after a URI with...

Henoch-Schonlein purpura. Common triad: skin, joints, GI

What is the most common form of childhood systemic vasculitis?

Henoch-Schonlein purpura

Associate idiopathic, segmental, thrombosing vasculitis of small and medium peripheral arteries and veins, often seen in heavy smokers with...

Buerger's disease (thromboangiitis obliterans) - affects small and medium sized vessels

What are 3 symptoms of Buerger's disease?  
What is the treatment?

Intermittent claudication, superficial nodular phlebitis, cold sensitivity (Raynaud's phenomenon)  
Treatment: Smoking cessation

Associate acute, self-limiting necrotizing vasculitis in infants and kids that presents with fever, congested conjunctiva, changes in lips/oral mucosa ("strawberry tongue") with...	Kawasaki disease
Children with Kawasaki disease are at risk for what?	Coronary aneurysms
What size arteries does Kawasaki disease affect?	Small and medium vessels
Associate necrotizing immune complex inflammation of medium-sized muscular arteries associated with Hepatitis B seropositivity in 30% of patients with...	Polyarteritis nodosa
How do the ages of the lesions appear in Polyarteritis nodosa? What vessels are typically affected?	Lesions are of different ages. Typically involves renal and visceral vessels
How do you treat polyarteritis nodosa?	Corticosteroids, cyclophosphamide
Associate granulomatous thickening of aortic arch and/or proximal great vessels with...	Takayasu's arteritis

What are 7 symptoms of Takayasu's arteritis? (Mnemonic)	Fever, Arthritis, Night sweats, MYalgia, SKIN nodules, Ocular disturbances, Weak pulses in upper extremities ("FAN MY SKIN On Wednesday")
What's the most common vasculitis that affects medium and large arteries, usually branches of the carotid artery?	Temporal arteritis (giant cell arteritis)
What are common symptoms of temporal arteritis (giant cell arteritis)?	Unilateral headache, jaw claudication, impaired vision (occlusion of ophthalmic artery) ("TEMPoral arteritis has signs near TEMples")
How do you treat temporal arteritis?	High-dose steroids
What is stressed volume?	The volume of blood in the arteries (because it is under high pressure)
What adrenergic receptors are found in arterioles of the skin, splanchnic, and renal circulations?	alpha-1 adrenergic receptors: causes constriction
What adrenergic receptors are found on the arterioles of skeletal muscle?	Beta-2 adrenergic receptors: causes relaxation



The highest proportion of blood in the CV system is found in what type of vessels?	Veins (called "unstressed volume")
What is an equation for blood flow velocity in the blood vessels?	$v = Q/A$ where: $v$ = velocity (cm/sec) $Q$ = blood flow (mL/min) $A$ = cross sectional area (cm <sup>2</sup> )
What is the formula for parallel resistance?	Illustrated by systemic circulation: $1/R_{total} = 1/R_a + 1/R_b + \dots 1/R_n$ where: $R_a$ , $R_b$ , and $R_n$ are the resistances of the renal, hepatic, and other arteries respectively
What is the formula for series resistance?	Illustrated by arrangement of blood vessels within a given organ: $R_{total} = R_{artery} + R_{arterioles} + R_{capillaries}$
In each parallel artery, _____ is the same	Pressure
In each series artery, _____ is the same	Flow

What is Reynold's number and what affects it?	<p>Reynold's number predicts whether blood flow will be laminar or turbulent. When Reynold's number is increased, there is greater tendency for turbulence.</p> <p>Reynold's number (and therefore turbulence) is increased by:</p> <p>Decreased blood viscosity (e.g. decreased hematocrit, anemi), and increased blood velocity (e.g. narrowing of a vessel)</p>
What is capacitance? Capacitance is inversely related to what?	<p>Capacitance describes the distensability of blood vessels. Capacitance is inversely related to elastance, or stiffness.</p>
What is the formula for Capacitance?	$C = V/P$ <p>where:</p> <p>C= capacitance (mL/mm Hg)  V = volume (mL)  P = pressure (mm Hg)</p>
Is capacitance greater for arteries or veins? How does capacitance change with age?	<p>Much greater for veins.</p> <p>Capacitance of blood vessels decrease with age, leading to an increase in blood pressure</p>
What are the mean pressures in the aorta, arterioles, capillaries, and vena cava?	<p>Aorta, 100 mm Hg  Arterioles, 50 mm Hg  Capillaries, 20 mm Hg,  Vena cava, 4 mm Hg</p>

<p>What is the difference between absolute refractory period (ARP), Effective refractory period (ERP), and Relative refractory period (RRP)?</p>	<p>ARP - Reflects the time during which no action potential can be initiated, regardless of how much inward current is supplied.</p> <p>ERP - Is the period during which a conducted action potential cannot be elicited.</p> <p>RRP - Is the period during which an action potential can be elicited, but more than the usual inward current is required.</p>
<p>What are chronotropic effects and how are they produced?</p>	<p>Chronotropic effects produce changes in heart rate. A positive chronotropic effect increases heart rate by increasing the firing of the SA node.</p>
<p>What are dromotropic effect?</p>	<p>Dromotropic effects produce changes in the conduction velocity, primarily in the AV node. (Note: negative dromotropic effects increase the PR interval)</p>
<p>Which parts of the heart have parasympathetic vagal innervation? Which part doesn't?</p>	<p>SA node, atria, and AV node have parasympathetic vagal innervation. The ventricles do not. The neurotransmitter is ACh which acts at muscarinic receptors.</p>
<p>What part of the SA node action potential is altered in producing chronotropic effects?</p>	<p>Phase 4 depolarization (decreased heart rate by decreasing the Na influx)</p>
<p>Which part of the AV node action potential is affected to produce dromotropic effects?</p>	<p>Decreased inward Ca current and in phase 0</p>

What accounts for the observation that the heart behaves as an electrical syncytium?

The presence of gap junctions that act as low-resistance paths between cells that allow for rapid electrical spread of APs

What structures in heart sarcomeres invaginate the cells at the Z lines and carry APs into the cell interior?

T tubules

What structure is the site of storage and release of Ca for excitation-contraction coupling?

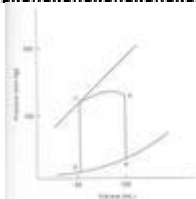
Sarcoplasmic reticulum

How do positive inotropic agents affect the heart?

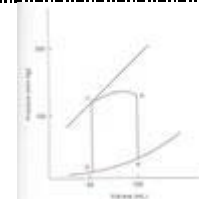
They increase contractility

How do cardiac glycosides increase contractility of the heart?

They inhibit the Na,K-ATPase in the myocardial cell membrane --> increased intracellular [Na], diminishing the Na gradient across the cell membrane. Na<sup>+</sup>-Ca<sup>+</sup> exchange (the mechanism that extrudes Ca from the cell), depends on the size of the Na gradient and thus is diminished, producing increased intracellular [Ca]



This is a LV pressure-volume loop. Where does the mitral valve open and close? Where does the aortic valve open and close?



Mitral valve opens at "d."  
Mitral valve closes at "a."  
Aortic valve opens at "b."  
Aortic valve closes at "c."

What does the vasalva maneuver detect?	<p>Detects the integrity of the baroreceptor mechanism. Vasalva maneuver --&gt; increased intrathoracic pressure --&gt; decreased venous return - -&gt; decreased cardiac output and arterial pressure</p> <p>If the baroreceptor mechanism is intact, the decrease in arterial pressure is sensed by the baroreceptors, leading to an increase in sympathetic outflow to the heart and blood vessels, causing an increase in heart rate.</p>
What are 3 actions of atrial natriuretic peptide?	<p>Is released from the atria in response to an increase in blood volume and atrial pressure:</p> <p>causes relaxation of vascular smooth muscle</p> <p>causes increased excretion of Na and water by the kidney</p> <p>Inhibits renin secretion</p>
What does endothelium-derived relaxing factor (EDRF) do?	<p>Causes local relaxation of vascular smooth muscle. One form of EDRF is NO.</p>
If perfusion pressure to an organ is suddenly decreased, compensatory vasodilation of the arterioles will occur to maintain a constant flow. This is called....	Autoregulation
If metabolic activity in an organ increases, blood flow will increase to that organ proportionately to meet metabolic demands. This is called...	Active hyperemia

An increase in blood flow to an organ after a period of occlusion of flow is called...	Reactive hyperemia
What are 2 vasoactive metabolites in the coronary arteries?	Hypoxia, adenosine
What are 2 vasoactive metabolites in the cerebral circulation?	CO <sub>2</sub> , H <sup>+</sup>
What are 3 vasoactive metabolites in skeletal muscle?	Lactate, K <sup>+</sup> , adenosine
What is the most important method of controlling blood flow to skeletal muscle at rest? During exercise?	Rest: sympathetic control Exercise: Local metabolic control (lactate, K <sup>+</sup> , adenosine)
What is the myogenic hypothesis for local control of blood flow?	Is based on the observation that vascular smooth muscle contracts when it is stretched. Explains autoregulation, but not active or reactive hyperemia.
What is the metabolic hypothesis for local control of blood flow?	Tissue supply of O <sub>2</sub> is matched to the tissue demand for O <sub>2</sub> . Vasodilator metabolites, produced when there is metabolic activity, mediates increased O <sub>2</sub> supply during increased O <sub>2</sub> demand.

What is the effect of histamine on blood vessels?	Causes arteriolar dilation and venous constriction --> increased capillary pressure --> local edema
What is the effect of bradykinin on blood vessels?	Causes arteriolar dilation and venous constriction --> increased capillary pressure --> local edema (similar to histamine)
What is the effect of serotonin on blood vessels?	Causes arteriolar constriction and is released in response to blood vessel damage to help prevent blood loss. Has been implicated in the vascular spasms of migraine headaches.
What is the effect of the following prostaglandins on blood vessels: Prostacyclin E-series prostaglandins F-series prostaglandins Thromboxane A2	Prostacyclin: Vasodilator E-series prostaglandins: Vasodilator F-series prostaglandins: Vasoconstrictor Thromboxane A2: Vasoconstrictor
What is coronary circulation controlled by?	Almost exclusively by local metabolic factors (hypoxia, adenosine)
What is the main method of blood vessel regulation in skin?	Sympathetic control (cold temp --> sympathetic activation --> vasoconstriction)
How do you calculate LDL in the blood? How does the presence of chylomicrons affect this calculation?	Calculated LDL = cholesterol - HDL - triglyceride/5  Chylomicrons falsely lower calculated LDL by increasing diet-derived triglycerid



What molecule can help remove cholesterol from atherosclerotic plaques for disposal in the liver?	HDL
What lipoprotein is required for chylomicron assembly?	B-48
What lipoprotein is required for VLDL assembly and secretion?	B-100
How does hypertriglyceridemia affect the appearance of plasma?	Causes turbidity
Diet derived triglycerides are found in...	Chylomicrons
Liver-derived triglycerides are found in...	VLDL
What is Type II hyperlipoproteinemia? What is an example?	Increased LDL due to decreased LDL receptors (E.g. familial hypercholesterolemia - Autosomal Dominant)



What is type III hyperlipoproteinemia and what is the problem? What's an example	Familial dysbetalipoproteinemia Accumulation of "remnants" (chylomicron and IDL) due to deficiency of apolipoprotein E (apoE)
What is Type IV hyperlipoproteinemia?	Accumulation of VLDL due to increased in synthesis or decrease in catabolism. Type IV is the most common lipid disorder.
What is the most common cause of Type IV hyperlipoproteinemia?	Most common cause is alcohol excess
What are signs of apolipoprotein B deficiency?	Deficiency of chylomicrons, VLDL, and LDL (decreased cholesterol and triglycerides). Leads to malabsorption, because chylomicrons accumulate in villi and prevent reabsorption of micelles. Marked decrease in vitamin E. Ataxia, hemolytic anemia, and thorny RBCs (acanthocytes).
What is the first step in atherosclerosis?	Endothelial cell injury (e.g. due to smoking, hypertension, homocysteine, LDL)
What's the pathognomonic lesion of atherosclerosis?	Fibrous cap (plaque) composed of smooth muscle, foam cells, inflammatory cells, and extracellular matrix
What is an excellent marker of disrupted atherosclerotic plaques?	C-reactive protein

What is the most common site for atherosclerosis?	Abdominal aorta
What are two diseases associated with hyaline arteriolosclerosis?	Diabetes mellitus, hypertension
What's the most common aneurysm in men older than 55 years of age?	Abdominal aortic aneurysm
How does vessel wall diameter affect vessel wall stress?	Increased diameter causes increased vessel wall stress
What is the triad of a ruptured abdominal aortic aneurysm?	Left flank pain, hypotension, pulsatile mass
What is a mycotic aneurysm?	Vessel wall weakening due to an infection
Where are berry aneurysms found?	At the junction of communicating branches with main cerebral vessels

Where does the blood go in a ruptured berry aneurysm?	Subarachnoid space
Aortic arch aneurysm is associated with which organism?	Syphilis (tertiary) - caused by vasculitis of the vasa vasorum
How does aortic regurgitation create such a large pulse pressure?	There is increased stroke volume because the back flow of blood increases LVEDV. After the heart beats, there is a drop in diastolic pressure due to blood rapidly draining back into the left ventricle.
What is the most common cause of death in Marfan syndrome and Ehlers-Danlos Syndrome?	Aortic dissection
What is the most common group to have aortic dissections?	Men with a mean age of 40 to 60 years with antecedent hypertension (also common during pregnancy due to 2X higher plasma volume)
What is the histologic hallmark change in patients with Marfan syndrome who have aortic dissections?	Cystic medial degeneration/necrosis. Elastic tissue fragmentation leads to matrix material collecting in areas of fragmentation in the tunica media
What is the most common cause of death in aortic dissection?	Cardiac tamponade

Associate severe retrosternal chest pain radiating to the back ('tearing pain') and loss of upper extremity pulse (or pulse differences between left and right arms) with...	Aortic dissection
What type of valve dysfunction can you get in aortic dissection?	Aortic valve regurgitation due to aortic valve ring dilation
What is the most common site of varicose veins?	Superficial saphenous veins
What is phlebothrombosis?	Thrombosis of a vein without inflammation
What is the most common cause of phlebothrombosis? What is the most common site of phlebothrombosis.	Stasis of blood flow. Most common in the deep vein of the calf.
 <p>Stasis dermatitis, an orange discoloration (hemosiderin) around the ankles caused by rupture of the penetrating branches is a sign of what condition?</p>	 <p>Deep vein thrombosis</p>
Superficial migratory thrombophlebitis is associated with what?	Carcinoma of the pancreatic head - due to release of thrombogenic substances by cancer



Superior vena cava syndrome (pic on the left), which includes puffiness and blue to purple discoloration of the face, arms, and shoulders due to extrinsic compression of the superior vena cava, is most often caused by what?



Primary lung cancer (90% of cases) usually a small cell carcinoma of the lung

Compression of the neurovascular compartment of the neck that leads to vascular signs (arms falling asleep while person is sleeping), nerve root signs (e.g. numbness, parasthesias) sometimes due to a cervical rib is called...

Thoracic outlet syndrome

What is a test for thoracic outlet syndrome?

Positive Adson test - pulse disappears when the arm is outstretched and the patient looks to the side of the outstretched arm



Lymphangitis, inflammation of the lymphatics that often appears as a "red streak" (in this picture it is going to the patient's axilla) is most often caused by what?



Cellulitis caused by Strep. pyogenes



Lymphedema of the hands and feet in newborns is associated with which syndrome?





Turner's. Also associated with webbed neck due to a lymphatic abnormality.



How do you treat capillary hemangiomas in newborns?



No need to. They normally regress with age.

c-ANCA antibodies are against what structure?	Proteinase 3 (e.g. found in Wegener's granulomatosis)
What are p-ANCA antibodies against?	myeloperoxidase (e.g. found in Churg-Strauss syndrome, microscopic polyangitis)
Fibrosis is usually associated with what size vessel disease?	Medium-sized vessel vasculitis
Large vessel vasculitis is usually associated with what histological structures?	Granulomas
Polyarteritis nodosa always spares what arteries?	Pulmonary arteries
 <p>Strawberry tongue and MIs in children are associated with which vasculitis?</p>	 <p>Kawasaki disease</p>
Can you give corticosteroids in Kawasaki disease?	No, contraindicated because of danger of vessel rupture

What's the difference between Raynaud's disease and Raynaud's phenomenon?	Raynaud's phenomenon is secondary to other diseases (e.g. systemic sclerosis, CREST syndrome) whereas Raynaud's disease is a primary disease
Systolic blood pressure correlates with...	Stroke volume
Diastolic blood pressure correlates with...	tonicity of total peripheral resistance arterioles
What is the effect of excess sodium on TPR arterioles?	Causes them to vasoconstrict. Sodium enters the arteriole smooth muscle cells and opens calcium channels, causing vasoconstriction. Increases diastolic blood pressure
What is the most common type of hypertension?	Essential hypertension (95% of HTN) - genetic factors reduce renal sodium excretion
What is renovascular hypertension?	Hypertension due to activation of renin-angiotensin-aldosterone system. E.g. Atherosclerotic plaque (in elderly men) or fibromuscular hyperplasia (in middle aged women) partially blocks blood flow in the renal artery, leading to activation of the renin-angiotensin-aldosterone system in the affected kidney.

What is the most common cause of hypertension in young women?	Oral contraceptives: increased synthesis of angiotensinogen
What is the most common overall complication of hypertension?	Left ventricular hypertrophy
What is the most common cause of death in hypertensive patients?	Acute MI
How does heart wall stress affect the muscle?	Causes sarcomere duplication. Duplication in parallel thickens muscle. Duplication in series lengthens muscle.
What is the most common cause of left ventricular hypertrophy?	Essential hypertension
Increased afterload causes what type of hypertrophy?	Concentric hypertrophy
Increased preload causes what type of hypertrophy?	Eccentric (dilation)



What sound is produced in ventricular hypertrophy?	S4 heart sound - correlateds with atrial contraction in late diastole. Caused by blood entering a noncompliant ventricle.
What is the most common type of CHF?	Left sided heart failure
What is the general cause of heart failure?	The heart fails when it is unable to eject blood delivered to it by the venous system
"Forward failure" is another name for what? What does it cause?	Left-sided heart failure --> pulmonary edema.
What is the difference between systolic and diastolic dysfunction?	Systolic dysfunction is decreased ventricular contraction (decreased ejection fraction). Diastolic dysfunction is increased resistance to filling of the ventricle (EF is normal).
What is the first cardiac sign of LHF?	S3 heart sound. Occurs in early diastole. Caused by blood entering a volume-overloaded left ventricle. The intensity of the heart sound increases with expiration.
Expiration increases the intensity of what sort of heart murmurs and abnormal heart sounds?	Left sided ones

What causes paroxysmal nocturnal dyspnea?	Lying down increases venous return to the right side of the heart. If there is LHF, blood backs up into the lungs producing pulmonary edema and the person cannot breathe.
Backward failure is also known as what? What does it cause?	Also known as right sided heart failure. -->increase in venous hydrostatic pressure
Inspiration increases the intensity of what sorts of murmurs and abnormal heart sounds?	Right sided ones
What is high-output heart failure?	Form of heart failure in which cardiac output is increased compared with values for the normal state (e.g. due to increased stroke volume, decreased blood viscosity, vasodilation of peripheral resistance arterioles, or arteriovenous fistula)
What is the most common cause of arteriovenous fistula?	Trauma from a knife wound
What effect does tachycardia have on the coronary arteries?	Decreases filling time, leading to ischemia
What is the most common manifestation of coronary artery disease?	Angina pectoris

What does stable angina look like on a ECG?	Shows subendocardial ischemia with ST-segment depression
What does Prinzmetal's angina look like on ECG?	Transmural ischemia with ST-segment elevation
Describe the general sequence of pathology in a MI	Rupture of disrupted plaque --> Platelet thrombus --> Acute MI
A Q-wave infarction is a...	Transmural infarction
A non-Q wave infarction is a ...	Subendocardial infarction
In what population do "silent MI's" occur?	In elderly or in individuals with diabetes mellitus due to high pain threshold or problems with the nervous system
What is the most common cause of death in acute MI?	Ventricular fibrillation

What is the most common arrhythmias post MI?	Ventricular premature contractions
What is the most common cause of death after ventricular aneurysms?	CHF due to lack of contractile tissue
Wall rupture most commonly occurs how long after a MI?	3 to 7 days
Fibrinous pericarditis with or without effusion commonly occurs how long after a MI?	1 to 7 days
When do ventricular aneurysms commonly occur after a MI?	Clinically recognized within 4 to 8 weeks
What is the (LDH)1-2 "flip" that occurs during an acute MI?	Normally, LDH2 is higher than LDH1. In acute MI, LDH1 in cardiac muscle is release causing the "flip". LDH1-2 appears within 10 hours, peaks at 2-3 days, and disappers within 7 days
How do ECG changes correlate with microscopic changes after a MI?	Inverted T waves - correlate with areas of ischemia at periphery of infarct. Elevated ST segment - correlates with injured myocardial cells surrounding the area of necrosis. New Q waves - correlate with the area of coagulation necrosis.

In fetal circulation, blood that comes from the SVC and IVC go where?	SVC: Right atrium to right ventricle IVC: Right atrium to Left atrium via the foramen ovale
In fetal circulation, where does most of the pulmonary artery blood go?	Shunted through the patent ductus arteriosus into the aorta. Kept open by prostaglandin E2.
What vessels in fetal circulation have the highest and lowest oxygen concentrations?	Highest: Umbilical vein Lowest: Umbilical arteries (two of them)
What is the most common congenital heart disease in children?	Ventricular septal defect (VSD)
What is the most common congenital disease in adults?	Atrial septal defects (ASD)
Associate a pink upper body and a cyanotic lower body in an infant with...	patent ductus arteriosis
In Tetralogy of Fallot, what correlates with the presence or absence of cyanosis?	The degree of pulmonary stenosis

What is total anomalous pulmonary venous return?	A type of cyanotic congenital heart disease: Pulmonary vein empties oxygenated blood into the right atrium.
A disparity between upper and lower extremity blood pressure > 10 mm Hg is a sign of what?	Adult coarctation of the aorta
What type of valve lesions are typical of Rheumatic fever?	Mitral regurgitation in acute attack, mitral stenosis in chronic attack
Opening snap followed by a mid-diastolic rumble is associated with...	Mitral stenosis
What is the most common cause of mitral regurgitation?	Mitral valve prolapse
How does mitral regurgitation affect the left ventricle?	Leads to left ventricular hypertrophy, because mitral regurgitation leads to increased atrial volume, which leads to volume overload of the left ventricle --> LHF
Mitral valve prolapse is associated with what two diseases?	Marfan and Ehlers-Danlos syndrome

What is the pathophysiology of mitral valve prolapse?	It is due to myxomatous degeneration (pathologic weakness of CT) due to excess production of dermatan sulfate
What sound is produced in mitral prolapse?	Mid-systolic click due to sudden restrain by the chordae of the prolapsed valve, followed by mid to late systolic regurgitant murmur
What is the most common cause of aortic stenosis?	Calcification of normal or bicuspid valve
What is the most common valvular lesion causing syncope and angina with exercise?	Aortic stenosis. Decreased blood flow through the stenotic valve leads to decreased blood flow to the brain.
What is the most common cause of aortic regurgitation?	Isolated aortic valve root dilation.
What is the most common cause of acute aortic regurgitation?	Infective endocarditis.
Early diastolic murmur associated with bounding pulses is indicative of...	Aortic regurgitation

What is an Austin Flint murmur and what does it indicate?	Regurgitant stream from incompetent aortic valve hits the anterior mitral valve leaflet producing a diastolic murmur
Carcinoid tumor is associated with what type of valve disease?	Tricuspid valve regurgitation and pulmonary valve stenosis. Liver metastasis from a carcinoid tumor of the small intestine secretes serotonin, which causes fibrosis of the tricuspid valve and pulmonary valve.
What is the most common cause of infective endocarditis?	Strep. viridans
What is the most common cause of infective endocarditis in IV drug users?	Staph aureus
What is the most common cause of infective endocarditis due to prosthetic devices?	Staph epidermidis
What is the most common cause of infective endocarditis in ulcerative colitis or colorectal cancer?	Strep bovis
What valve is most often affected in infective endocarditis in IV drug users?	Tricuspid



Nonbacterial thrombotic endocarditis, where circulating mucin from mucin-producing tumors create sterile, nondestructive mitral valve vegetations is associated with which cancers?	Colon, pancreas
What is the most common cause of myocarditis and pericarditis?	Coxsackie
A young woman with pericarditis and effusion most likely has...	SLE
What is the most common cause worldwide of constrictive pericarditis?	TB
What is the most common cardiomyopathy?	Dilated
What causes the familial form of hypertrophic cardiomyopathy?	Mutations in the heavy chain of beta-myosin and in the troponins. Autosomal dominant.
What is the most common cause of sudden death in young individuals?	Hypertrophic cardiomyopathy

What is the most common part of the heart for metastasis?	Pericardium
What are the main primary cardiac tumors in adults and children?	Adults: Myxomas Children: Rhabdomyomas
What are 4 class IA antiarrhythmics? (Mnemonic)	Quinidine, Amiodarone, Procainamide, Disopyramide ("Queen Amy Proclaims Diso's Pyramid")
What are 3 class IB antiarrhythmics? (Mnemonic)	Lidocaine, Mexiletine, Tocainide "I'd Buy Lidy's Mexican Taco's"
How are cardiac output (CO), left ventricular end diastolic pressure (LVEDP) and pulmonary vascular resistance (PVR) affected in hypovolemic shock?	Decreased CO (due to decreased blood volume) Decreased LVEDP Increased PVR (due to vasoconstriction from catecholamines and angiotensin II, released due to decreased CO)
How are cardiac output (CO), left ventricular end diastolic pressure (LVEDP) and pulmonary vascular resistance (PVR) affected in cardiogenic shock?	Decreased CO (due to decreased force of contraction from left ventricle - e.g. MI) Increased LVEDP (blood accumulates in LV) Increased PVR (due to vasoconstriction from catecholamines and angiotensin II, released due to decreased CO)

How are cardiac output (CO), left ventricular end diastolic pressure (LVEDP) and pulmonary vascular resistance (PVR) affected in septic shock (initial phase)?	<p>Increased CO (due to rapid blood flow through dilated arterioles caused by endotoxins, and anaphylatoxins like C3a and C5a)</p> <p>Decreased LVEDP (due to neutrophil transmigration through pulmonary capillaries into alveoli producing noncardiogenic pulmonary edema)</p> <p>Decreased PVR (due to vasodilation of peripheral resistance arterioles)</p>
What's the most common cause of death in shock?	Multiorgan dysfunction
what is the mechanism of small vessel injury in diabetes?	non-enzymatic glycosylation renders vessels permeable to probein --> gives hyalinization and narrows lumen
what is gross appearance of kidney with HTN?	shrunk cobblestones appearance due to lacunar strokes and hyalinization of arterioles and capillaries
onion-skinned appearance of small vessels in kidney	malignant hypertension
why is the abdominal aorta the most common site of aneurysm?	absence of vaso vasorum... has no blood supply below renal arteries

sudden onset of severe left flank pain, hypotension, pulsatile mass on PE means what?	ruptured abdominal aortic aneurysm
what is underlying pathology of syphilitic lesions?	vasculitis (all of syphilis is vasculitis) treponeme infects vasa vasorum
key factors for causing dissecting aortic aneurysm	hypertension
what is pathogenesis of dissecting aortic aneurysm	elastic tissue fragmentation, cystic medial necrosis (GAGs)
most common type of aortic dissection?	proximal dissection --> occludes upper arteries, causes absent pulse on left
what conditions predispose to aortic dissection?	marfan syndrome, ehlers danlos syndrome, pregnancy
sturge weber...what is it?	vascular malformation in trigeminal region of face. also has AV malformation in brain on same side. predisposes to bleeding. people are retarded

how do you treat capillary hemangiomas?

leave it alone

cause of kaposi's sarcoma

herpes virus 8

lesion only seen in AIDS patients that looks like kaposi's sarcoma but isn't. what is it?

vacillary angiomatosis caused by bartonella henselae

angiosarcoma of liver is caused by what?

arsenic and polyvinyl chloride

small vessel vasculitis is caused by what?

type III hypersensitivity --> immune complex deposition --> palpable purpura

most common cause of coronary artery disease in children

kawasaki's

what lesion do you see in muscular arteries?

infarction

what kind of vessels are affected in people with no pulse?	elastic arteries (takayasu's)
temporal arteritis caused by what?	granulomatous inflammation (multinucleated giant cells present) of temporal artery
digital necrosis of fingers and toes in heavy smoker	buerger's disease
14 y/o boy with URI one week ago presents with polyarthrits, joint pains, hematuria with RBC casts and palpable purpura of buttocks and lower leg. what is the cause?	henoch-schonlein purpura --> IgA Abs in small vessels
most common vasculitis in children?	henoch-schonlein purpura
saddle nose deformity, chronic sinus infections, nodular masses in lungs, glomerular disease. what test will diagnose?	wegener's granulomatosis --> C-ANCA --> treat with cyclophosphamide
polyarteritis nodosa. what antibody is associated with this?	P-ANCA --> associated with HBV surface antigen

IV drug abuser with chronic hepatitis B has nodular inflamed mass on lower extremity and hematuria. what does he have?	polyarteritis nodosa
diabetic ketoacidosis with frontal lobe cerebral abscess related to what fungus?	mucormycosis
what are the spirochetes?	leptospira, borrellia, treponeme
cryoglobulinemia associated with what hepatitis?	HCV
digital vasculitis leads to fibrosis. manifests as Raynaud's syndrome. what is it?	scleroderma
CREST syndrome associated with what?	Calcinosis/Centromere Ab Raynaud's Esophageal dysmotility Sclerodactyly Telangiectasia
three causes of raynaud's?	cold-reacting antibodies, vasculitis, vasoconstriction

essential hypertension has what renin level in blood?	low
where do most hypertensive ruptures occur in brain?	in branches from middle cerebral artery
concentric hypertrophy is caused by what?	increased after-load
dilated hypertrophy is caused by what?	increased preload (volume overload)
what causes and S3 heart sound?	occurs in early diastole, so it must be caused by volume overload
S4 heart sound is caused by what?	occurs in late diastole --> problem with compliance (atrium encountering a problem filling the ventricle)
what abnormal heart sound is found in hypertension?	S4



all right sided heart murmurs and abnormal heart sounds increase in intensity during what?	inspiration (due to increased filling)
aortic valve is heard where?	right second intercostal space
pulmonic valve heard best where?	left second intercostal space
mitral valve heard best where?	apex
tricuspid valve is heard best where?	right sternal border
left sided heart murmurs and abnormal heart sounds are increased in intensity during what?	expiration (increased intrapleural pressure)
IV drug abuser with fever, pansystolic murmur along parasternal border that increases on inspiration, distended neck veins. what is most likely diagnosis?	infective endocarditis of tricuspid valve

signs of AV fistula

bruit over area, pulsatile, pressing on it causes decrease in HR

O2 sat on right side is what?

75%

O2 sat on left side of heart is what?

95%

eisenmenger's syndrome is what?

tardive cyanosis. --> reversal of L-to-R shunt

what is most common teratogen with ASD associated?

Fetal alcohol syndrome

murmur heard btwn shoulder blades caused by what?

patent ductus arteriosus

teratogen associated with patent ductus

congenital rubella

4 things in tetralogy of Fallot	aorta straddles septum, VSD, pulmonic stenosis (below valve), pulmonic hypertrophy
what in tetralogy of Fallot causes cyanosis?	degree of pulmonic stenosis
which shunts are protective in tetralogy of fallot?	ASD and patent ductus
what abnormalities do you get with right-to-left shunt?	polycythemia and high risk for infective endocarditis
Clinical features of stable angina	Chest pain induced by exercise or emotions. ST segment depression (subendocardial ischemia) Relieved by rest or nitroglycerin
Clinical features of Prinzmetal angina	Chest pain caused by coronary artery vasospasm. ST segment elevation. Relieved by nitroglycerin
Clinical features of unstable angina	Non-occlusive thrombus triggers release of TXA2 (vasoconstrictor). Occurs at rest. Risk of MI

Coronary irrigation of the heart	<p>Left anterior descending artery supplies anterior portion of left ventricle and anterior 2/3 of interventricular septum (produces heart blocks) (45% of MI). Circumflex artery, branch of left coronary artery (15% of MI). Right coronary artery supplies posterior and inferior left ventricle, right ventricle, SA node (sinus bradycardia), papillary muscle (mitral insuficiency) (35% of MI)</p>
Risk factors for coronary artery disease	<p>Age, family history, cigarette smoke, hipertension, low HDL, high LDL, diabetes</p>
Clinical presentation of AMI	<p>Sudden onset of acute substernal chest pain radiated to left arm, jaw and neck. Shortness of breath, diapgoresis, nausea, vomiting and anxiety</p>
Serum markers of miocardial infarction	<p>CK-MB elevated by 8h, peaks 18h, normal in 3 days. Troponin elevated by 6h, peaks 16h, normal in 10 days. LDH elevted by 24h, peaks 6 days, normal in 14 days.</p>
Gross changes in miocardial infarction	<p>18h, no change. 24h vague pallor. 1-7d yellow pallor. 7-28d central pallor with red border. Months - white firm scar</p>
Microscopic changes in miocardial infarction	<p>4-24h coagulative necrosis. 1-3d neutrophilic infiltrate. 3-7d macrophages. 7-28d granulation tissue. Months - fibrotic scar</p>

Complications of MI	<p>Arrhythmias (MC COD), CHF, pericarditis, rupture (4-7 days post-infarct). Ventricular free wall (LAD) --&gt; cardiac tamponade. Interventricular septum (LAD) --&gt; left to right shunt. Papillary muscle (RCA) mitral insufficiency</p>
Features of sudden cardiac death	<p>Death within 1 hour of onset of symptoms by fatal arrhythmia. CAD (80%), hypertrophic cardiomyopathy, mitral valve prolapse, aortic stenosis</p>
Pathophysiology of heart failure	<p>Left ventricle fails --&gt; decreased cardiac output --&gt; RAA system and retention of Na and H<sub>2</sub>O --&gt; increased venous return causes edema and partial compensation of CO. There's backward pulmonary congestion that causes dyspnea and pulmonary edema with decreased RV output that adds up to systemic edema. Increased sympathetic tone and volume retention are compensation mechanisms</p>
Signs and symptoms of left heart failure	<p>Dyspnea (due to increased pulmonary hydrostatic pressure), pillow orthopnea (no gravity increases venous return with pulmonary congestion), rales, S3 gallop (volume overloaded ventricle)</p>
Complications of left heart failure	<p>Pulmonary edema, excessive RAA leads to secondary hyperaldosteronism, cardiogenic shock</p>
Features and treatment of systolic left heart failure	<p>Due to decreased contractility after infarction. EF&lt;0.4. Rx. Inotropics (digitalis), decrease afterload with vasodilators (ACE inhibitor)</p>

Features and treatment of diastolic left heart failure	<p>Due to decreased compliance of left ventricle (increases left atrial pressure and pulmonary congestion). <math>EF &gt; 0.4</math>.</p> <p>Due to left ventricular hypertrophy, restrictive cardiomyopathy. Rx.: increase preload by decreasing heart rate (calcium channel blockers and <math>\beta</math>-blockers)</p>
Causes of right heart failure	Left heart failure (MCC), cor pulmonale (primary pulmonary hypertension)
Clinical features of right heart failure	Jugular venous distension, nutmeg liver hepatomegaly, dependant pitting edema, ascites, pleural effusions, tricuspid insufficiency
Causes and features of mitral stenosis	Chronic rheumatic fever is MCC. Mid-diastolic murmur. Dyspnea and hemoptysis (pulmonary congestion), atrial fibrillation (left atrial dilation), dysphagia for solids (enlarged left atrium compresses esophagus), hoarseness (irritation of recurrent laryngeal nerve)
Causes and features of mitral prolapse	Valve leaflets undergo myxomatous degeneration. Associated with lethal ventricular arrhythmias in Marfan. Mid-systolic click. Infectious endocarditis and rupture of chordae tendinae are complications Rx.: CCA, $\beta$ -blockers and negative inotropic agents
Causes and features of mitral insufficiency	Caused by mitral prolapse, left heart failure, infective endocarditis, RCA thrombosis (papillary muscle). Systolic murmur, S3 heart sound.

Causes and features of aortic stenosis	MCC is calcified congenital bicuspid valve, rheumatic fever, old age. Decreased stroke volume and cardiac output, increased afterload. Left ventricular hypertrophy. Systolic murmur. Associated with angina (less coronary filling), syncope (reduced cardiac output) and microangiopathic hemolytic anemia with schistocytes
Causes and features of aortic insufficiency	MCC is essential hypertension, infective endocarditis, syphilitic and aortic aneurysms. Left ventricular hypertrophy, increased preload. Diastolic murmur, bounding pulse.
Pathophysiology of rheumatic fever	Antibodies against streptococcal M protein cross react with heart valves producing fibrosis/stenosis, as well as systemic features
Jones major criteria of rheumatic fever	Migratory polyarthritits, pancarditis, subcutaneous nodules, erythema marginatum, sydenhan chorea
Pathognomonic lesion of rheumatic heart fever	Aschoff body. Fibrinoid necrosis surrounded by macrophages (Anitschkow cells), lymphocytes and plasma cells
Clinical features of subacute endocarditis	Strep viridans colonizes damaged valves. "FROM JANE". Fever, Roth spots on retina, Osler nodes (painful subcutaneous nodules on fingers and toes), murmur, Janeway lesions (painless red lesions on palms and soles), anemia, nailbed hemorrhage, septic emboli

Preductal coarctation of the aorta	Associated with Turner syndrome. Narrowing of aorta proximal to ductus arteriosus. Usually associated with PDA that supplies oxygenated blood to distal aorta. Presents in newborn with CHF, weak pulses and cyanosis of lower extremities.
Postductal coarctation of the aorta	Narrowing of aorta distal to ductus arteriosus. Hypertension in upper extremities and hypotension in lower extremities. Can produce aortic insufficiency, berry aneurysms and secondary hypertension due to increased RAA (low renal flow)
Right to left shunts	Early cyanosis due to blood shunt past the lungs. Tetralogy of Fallot, transposition of great vessels, truncus arteriosus, tricuspid atresia.
Left to right shunts	Late cyanosis due to Eisenmenger syndrome. VSD, ASD, PDA
Eisenmenger syndrome	Right side of the heart hypertrophies due to a septal defect or PDA and shunt reverses from left-right to right-left producing cyanosis
Tetralogy of Fallot	Overriding aorta, pulmonic stenosis, right ventricular hypertrophy, VSD. Cyanosis depends on degree of pulmonic stenosis. PDA or ASD are cardioprotective.
Transposition of the great vessels	Inversion of aorta and pulmonary arteries. Infants of diabetic mothers. Must have ASD, VSD or PDA to survive.



Truncus arteriosus	Common pulmonary artery and aortic trunk. Massive blood flow to the lungs causes pulmonary hypertension. Early cyanosis and CHF.
VSD	Communication between ventricles. Large defect leads to pulmonary hypertension and Eisenmenger syndrome. Systolic murmur.
ASD	Communication between atriums. Associated with fetal alcohol syndrome.
PDA	Communication between aorta and pulmonary artery. Associated with congenital rubella. During pregnancy PDA is kept by PGE2. Close with indomethacin. Machinery murmur. Eisenmenger syndrome.
Dilated cardiomyopathy	Idiopathic, postpartum, alcohol, Coxackie B infections, doxorubicin and cocaine. Presents as CHF with decreased ejection fraction
Hypertrophic cardiomyopathy	Cause of death in young athletes. Autosomal dominant. Asymetrical hypertrophy in ventricular septum. Decreased compliance and stroke volume. Rx.: increase preload with beta blockers (decrease HR)
Carcinoid heart disease	Right sided endocardial and valvular fibrosis secondary to serotonin in patients with carcinoid metastasis to liver. Skin flushing, diarrhea, cramping, bronchospasm, wheezing, telangiectasia

List 3 causes of Right and Left ventricular hypertrophy	<ol style="list-style-type: none"> <li>1. sustained pressure increases wall stress</li> <li>2. contraction against an increased resistance (afterload)</li> <li>3. volume overload (increased preload)</li> </ol>
Sustained pressure can increase the wall stress of the right and left ventricles causing hypertrophy. What effects does this have on the sarcomeres	causes duplication of the sarcomeres, duplicates causes parallel thick muscles and duplication in series lengthens muscle
T/F contraction against an increased resistance (after-load) produces concentric thickening of the ventricular wall	true- new sarcomeres duplicate in parallel to the long axes of the cells
list the 2 common causes of left ventricular hypertrophy	<ol style="list-style-type: none"> <li>1. essential hypertension (most common)</li> <li>2. aortic stenosis</li> </ol>
list the 2 common causes of right ventricular hypertrophy	<ol style="list-style-type: none"> <li>1. pulmonary hypertension</li> <li>2. pulmonary artery stenosis</li> </ol>
T/ F volume overload (increased preload) causes constriction and hypotrophy of the ventricular wall	False- volume overload (increased preload) causes DILATION and HYPERTrophy of the ventricular wall
list 2 causes of eccentric hypertrophy of left ventricles	<ol style="list-style-type: none"> <li>1. mitral valve or aortic valve regurgitation</li> <li>2. left to right shunting of blood (ventricular septal defect) which causes more blood to return to the left side of the heart</li> </ol>

what causes eccentric hypertrophy of the right ventricle	tricuspid valve of pulmonary valve regurgitation
list 3 consequences of ventricular hypertrophy	<ol style="list-style-type: none"> <li>1. left or right sided heart failure</li> <li>2. angina (mostly LVH)</li> <li>3. S4 heart sound ( correlated with the atrial contraction in late diastole and by blood entering a noncompliant ventricle)</li> </ol>
<p>Definition:</p> <p>the heart fails when it is unable to eject blood delivered to it by the venous system</p>	congestive heart failure
list the types of CHF	<ol style="list-style-type: none"> <li>1. left sided heart failure- most common</li> <li>2. right sided heart failure</li> <li>3. biventricular heart failure</li> <li>4. high output heart failure</li> </ol>
why is it said that left sided heart failure is a forward failure	<ol style="list-style-type: none"> <li>1. left side of the heart can not eject blood into the aorta</li> <li>2. increase in left ventricular end-diastolic volume and pressure</li> <li>3. backup of blood into the lungs causes pulmonary edema</li> </ol>

in LHF, the decrease in ventricular contraction, noncompliance of the ventricles and increased workload causes what effects	<ol style="list-style-type: none"> <li>1. decreased ventricular contraction (systolic dysfunction) causes ischemia and myocardial fibrosis, myocarditis, cardiomyopathy</li> <li>2. noncompliant ventricles (diastolic dysfunction) restricts filling of the ventricle and causes concentric LVH and infiltration of muscle with amyloid, iron or glycogen</li> </ol>
what is the difference between systolic dysfunction and diastolic dysfunction	<ol style="list-style-type: none"> <li>1. systolic dysfunction- low ejection fraction (&lt;40%)</li> <li>2. diastolic dysfunction-high EF and an S4 gallop due to increased resistance to filling in late diastole so there is an increase in left atrial pressure (there could be a normal EF)</li> </ol> <p>EF=stroke volume/ LV end-diastolic volume , normal 55%-80%</p>
what is the gross and microscopic findings of LHF	<ol style="list-style-type: none"> <li>1. lungs are congested and exude a frothy pink transudate (edema)</li> <li>2. alveolar macrophages contain hemosiderin ( heart failure cells)</li> </ol>
what are the differences on clinical exam of pt with LHF vs RHF	<p>left sided heart failure</p> <ol style="list-style-type: none"> <li>1. dyspnea- can not inspire</li> <li>2. pulmonary edema-bibasilar inspiratory crackles</li> <li>3.*** Left side S3 heart sound- first finding in LHF</li> <li>4. mitral valve regurgitation</li> <li>5. paroxysmal nocturnal dyspnea</li> </ol> <p>right sided heart failure</p> <ol style="list-style-type: none"> <li>1. prominent jugular veins</li> <li>2. right sides S3 sound due to volume overload</li> <li>3. tricuspid valve regurgitation</li> <li>4. painful hepatomegaly</li> <li>5. dependent pitting edema and ascites</li> </ol>

the pulmonary edema in LHF will show what finding on chest radiograph	congestion in upper lobes and alveolar infiltrates
why is a left sided S3 heart sound heard in LHF and when is this sound heard on exam	<p>occurs in early diastole- intensity of sound increases with expiration</p> <p>caused by blood entering a volume overloaded left ventricle</p> <p>*** this S3 heart sound is the first cardiac finding in LHF</p>
in LHF, the mitral valve regurgitates blood, why and where is the murmur best heard	<p>mitral valve regurgitates blood bc it is caused by stretching of the valve ring which causes a problem closing the mitral valve during systole.</p> <p>blood entering the Left Atrium during systole produces a pansystolic murmur that increases in intensity on expiration</p> <p>murmur best heard at the apex</p>
what is paroxysmal nocturnal dyspnea and why does it occur in LHF	<p>choking sensation at night due to increased venous return to the failed left side of the heart</p> <p>blood backs up in the lungs producing pulmonary edema</p> <p>relieved by standing or placing pillow under head (pillow orthopnea) to increase the effects of gravity on reducing venous return to the heart</p>
why is right sided heart failure considered a backward failure	<p>right side of the heart cannot pump blood from the venous system to the lungs</p> <p>blood accumulated in the venous system which leads to increase in venous hydrostatic pressure</p>

RHF can be caused by what 4 pathogenesis	<p>1. decreased contraction- right ventricular infarction</p> <p>2. noncompliant right ventricle - RVH</p> <p>3. increase afterload- LHF</p> <p>4. increase preload- tricuspid valve regurgitation</p>
in Right sided heart failure, what causes the prominence of the jugular veins	increase in the venous hydrostatic pressure
in right sided heart failure what causes the Right S3 heart sound	due to volume overload in the ventricle which increases in intensity with inspiration
In right sided heart failure what causes the tricuspid valve regurgitation and where is it best heard	<p>caused by stretching of the valve ring which causes problem closing the tricuspid valve during systole.</p> <p>blood entering the right atrium during systole produces a pansystolic murmur that increases with inspiration</p> <p>murmurs best heard on left parasternal border</p>
why is there painful hepatomegaly in RHF	passive liver congestion due to back up of venous blood in the central veins
what kind of edema is seen in RHF and why	dependent pitting edema and ascites caused by the increase in venous hydrostatic pressure

<p>Defintion:</p> <p>Form of heart failure in which cardiac output is increased compared with values for the normal resting state</p>	<p>high output heart failure</p>
<p>high output heart failure can be caused by what 4 condition</p>	<ol style="list-style-type: none"> <li>1. increase in stroke volume- hyperthyroidism</li> <li>2. decrease blood viscosity- severe anemia</li> <li>3. vasodilation of peripheral resistance arterioles- thiamine deficiency, early phase of endotoxic shock</li> <li>4. arteriovenous fistulas-bc AV communications bypass the mircocirculation which increases venous return to the heart- trauma from knife wound or surgical shunt from hemodialysis</li> </ol>
<p>Definition:</p> <p>Imbalance between myocardial O2 demand and supply from the coronary arteries</p>	<p>ischemic heart disease</p>
<p>when doe coronary vessels fill</p>	<p>diastole</p>
<p>what is the effect of tachycardia on the coronary artery blood flow</p>	<ol style="list-style-type: none"> <li>1. Tachycardia (&gt;180bpm) decreases filling time, leading to ischemia.</li> <li>2. decreases diastole which decreases filling of coronary arteries</li> </ol>

which coronary artery accounts for about 50% of the coronary artery thromboses	left anterior descending coronary artery
the LAD supplies which portions of the heart	<ol style="list-style-type: none"> <li>1. Anterior portion of the left ventricle</li> <li>2. Anterior two thirds of the interventricular septum</li> </ol>
the right coronary artery supplies which portions of the heart	<ol style="list-style-type: none"> <li>1. Posteroinferior part of the left ventricle</li> <li>2. Posterior one third of the interventricular septum</li> <li>3. Right ventricle</li> <li>4. Posteromedial papillary muscle in left ventricle</li> <li>5. Both atrioventricular and sinoatrial nodes</li> </ol>
which coronary artery Supplies the lateral wall of the left ventricle and Accounts for 15% to 20% of coronary artery thromboses	left circumflex coronary artery
what are the 4 types of ischemic heart disease	<ol style="list-style-type: none"> <li>1. Angina pectoris (most common type)</li> <li>2. Chronic ischemic heart disease</li> <li>3. Sudden cardiac death</li> <li>4. Myocardial infarction</li> </ol>



<p>what risk factors are associated with ischemic heart disease</p>	<ol style="list-style-type: none"> <li>1. Age- Men 45 years old and up, women 55 years old and up</li> <li>2. Family history of premature coronary artery disease or stroke</li> <li>3. Lipid abnormalities- Low-density lipoprotein above 160 mg/dL or High-density lipoprotein below 35 mg/dL</li> <li>4 Smoking tobacco,</li> <li>5. hypertension</li> <li>6. diabetes mellitus</li> </ol>
<p>what is angina pectoris-</p>	<p>chest pain or discomfort due to coronary heart disease.</p> <p>Angina is a symptom myocardial ischemia. It occurs when the heart muscle (myocardium) doesn't get as much blood (hence as much oxygen) as it needs. This usually happens because one or more of the heart's arteries (coronary blood vessels that supply blood to the heart muscle) is narrowed or blocked.</p>
<p>Clinical findings:</p> <p>Exercise-induced substernal chest pain lasting 30 seconds to 30 minutes</p> <p>Relieved by resting or nitroglycerin</p> <p>Stress test shows ST-segment depression.</p> <ol style="list-style-type: none"> <li>a. stable angina</li> <li>b. Prinzmetal's angina</li> <li>c. unstable angina</li> <li>d. myocardial infarction</li> </ol>	<p>stable angina</p>

list 3 causes of stable angina	1. Atherosclerotic coronary artery disease (most common)  2. Aortic stenosis with concentric LVH  3. Hypertrophic cardiomyopathy
Pathogenesis Subendocardial ischemia due to decreased coronary artery blood flow  a. stable angina b. Prinzmetal's angina c. unstable angina d. myocardial infarction	a. stable angina
Pathogenesis:  Intermittent coronary artery vasospasm at rest  Vasoconstriction due to platelet thromboxane A2 or decrease in endothelin  a. stable angina b. Prinzmetal's angina c. unstable angina d. myocardial infarction	b. Prinzmetal's angina
Clinical findings:  Stress test shows ST-segment elevation (transmural ischemia).  Responds to nitroglycerin and calcium-channel blocker (vasodilator)  a. stable angina b. Prinzmetal's angina c. unstable angina d. myocardial infarction	b. Prinzmetal's angina

<p>Pathogenesis: Severe, fixed, multivessel atherosclerotic disease and Disrupted plaques with or without platelet nonocclusive thrombi</p> <p>Clinical findings Frequent bouts of chest pain at rest or with minimal exertion and May progress to acute myocardial infarction</p> <p>a. stable angina b. Prinzmetal's angina c. unstable angina d. myocardial infarction</p>	<p>c. unstable angina</p>
<p>list 3 revascularization procedures for angina pectoris</p>	<ol style="list-style-type: none"> <li>1. percutaneous transluminal coronary angioplasty (PTCA) and stenting- Balloon angioplasty dilates and ruptures the atheromatous plaque (Problem with restenosis)</li> <li>2. Intracoronary stents- Decrease the rate of restenosis. Most common early complication is a localized dissection with thrombosis</li> <li>3. Coronary artery bypass graft (CABG)- Used for multivessel coronary artery atherosclerosis <ol style="list-style-type: none"> <li>a. Internal mammary artery graft- Best graft patency after 10 years</li> <li>b. Saphenous veins- "Arterialization" of the vessels, fibrosis, and occlusion common after 10 years</li> </ol> </li> </ol>
<p>Definition:</p> <p>Progressive CHF resulting from long-term ischemic damage to myocardial tissue</p> <p>Replacement of myocardial tissue with noncontractile scar tissue</p>	<p>Chronic ischemic heart disease</p>

what is the cause of death in a sudden cardiac death	<p>Unexpected death within 1 hour after onset of symptoms</p> <p>Pathogenesis: Severe atherosclerotic coronary artery disease Disrupted fibrous plaques Absence of occlusive vessel thrombus (&gt;80% of cases)</p> <p>Cause of death is ventricular fibrillation.</p>
describe the common pathogenesis of a myocardial infarction	<p>Sequence:</p> <ol style="list-style-type: none"> <li>1. Sudden disruption of an atheromatous plaque</li> <li>2. Exposed subendothelial collagen or thrombogenic necrotic material</li> <li>3. Platelet adhesion and eventual formation of a platelet thrombus</li> </ol>
list 4 less common causes of an MI	<ol style="list-style-type: none"> <li>1. Vasculitis (e.g., polyarteritis nodosa, Kawasaki disease)</li> <li>2. Cocaine use</li> <li>3. Embolization of plaque material</li> <li>4. Thrombosis syndromes (e.g., antithrombin III deficiency, polycythemia)</li> </ol>
How does acute RF develop?	after group A streptococcal pharyngitis
how do immunitis develop against acute RF?	<p>immunemeditated</p> <p>type II hypersensitivity reaction</p> <p>cell-meditated immunity type IV</p>

What is the mos common initial presentation of acute RF?	migratory polyarthritis most common initial presentation
what is the most common cause of death death in acute RF?	myocarditis is the most common type of death
What happens with RF in an acute attack? in chronic disease?	mitral regurgitation in acute attack mitral stenosis in chronic disease
How do you diagnose acute RF?	with Jones criteria
What are the major criteria in diagnosing RF?	1) Carditis 2) Arthritis 3) Chorea 4) Erythema marginatum subcutaneous nodules
in acute RF what happens to ASO and DNase B titers?	↑ ASO ↑ DNase B titers
Which valve causes acute RF?	Acute RF: MV most often involved followed by AV

What is the most common cause of MV stenosis?	recurrent RF
Mitral valve stenosis effects?	Atrial fibrillation Pulmonary venous hypertension RHF
What is the sound of MV stenosis?	opening snap followed by an early to mid-diastolic rumble
What is mitral regurgitation most commonly caused by?	mitral valve prolapse
what is the sound of MV regurgitation?	pansystolic murmur, S3/S4 heart sounds no ↑ intensity with deep held inspiration
MV prolapse is associated with which syndromes?	Marfan and Ehlers-Danlos syndromes
how does MVP occur?	-myxomatous degeneration -excess dermatan sulfate

heart sound of MVP?

systolic click followed by murmur

What happens to the MVP sound if ↑ preload?

preload alters click and murmur relationship to S1/S2

Treatment of Symptomatic MVP?

β-blockers

what age group is Calcific Aortic Valve (AV) stenosis common in?

patients >60 years

what sound does Aortic Valve (AV) stenosis produce?  
what happens with ↓preload?  
↑ preload?

ejection murmur  
↓intensity with ↓ preload  
↑ intensity with ↑ preload

Aortic Valve stenosis is the most common valvular lesion  
what 2 things happen with exercise?

syncope  
angina

what kind of anemia is seen with AV (Aortic Valve) stenosis?

AV stenosis: microangiopathic hemolytic anemia with schistocytes, hemoglobinuria

what is the most common cause of aortic regurgitation	isolated AV root dilation
What happens to pulse pressure in Aortic valve regurgitation?	↑ pulse pressure
What sound does Aortic valve regurgitation make?	early diastolic murmur; bounding pulses; S3, S4 no ↑ intensity of inspiration
AV regurgitation causes ____ because of a widened pulse pressure?	hyperdynamic circulation
an Austin Flint murmur in AV regurgitation indicates what should be done?	sign for AV replacement
Tricuspid Valve (TV) regurgitation happens in which age group?	adults
TV (Tricuspid Valve) regurgitation can be caused by what?	infective endocarditis carcinoid heart disease



what sound does Tricuspid Valve regurgitation make?	panystolic murmur; S3/S4; ↑ intensity with deep held inspiration
Pulmonary valve (PV) regurgitation is caused by what?	pulmonary hypertension
What 2 conditions does Carcinoid heart disease cause?	PV (Pulmonary valve) Stenosis TV (Tricuspid Valve) regurgitation
Infective endocarditis (IE) is most commonly caused by which microbial pathogen?	streptococcus viridans
IE (Infective endocarditis ) is most commonly caused by which microbial pathogen in cases of IVDA (intravenous drug abuse)?	staphylococcus aureus
What is the most common pathogen that produces nosocomial and prosthetic valve infective endocarditis?	staphylococcus epidermidis
What is the most common pathogen that produces IE in ulcerative colitis/colorectal cancer?	streptococcus bovis

What causes Tricuspid Valve regurgitation in IVDA (intravenous drug abuse)?	Tricuspid Valve regurgitation in IVDA is due to infective endocarditis
Most consistent sign of Infective endocarditis (IE) ?	Fever
signs of IE?	microembolization immunocomplex vasculitis
in the majority of cases of Infective Endocarditis what do the blood cultures test?	positive in 80% of IE cases
Libman-Sacks endocarditis is associated with what conditions? (2)	systemic lupus erythematosus(SLE) MV(mitral valve) regurgitation involved
marantic endocarditis is associated with what type of vegetation? and which syndrome?	sterile vegetation paraneoplastic syndrome
most common cause of myocarditis and pericarditis?	Coxsackievirus

what disease is the most common cause of myocarditis leading to CHF in Central/South America?	Chagas' disease
What drugs can cause myocarditis?	doxorubicin daunorubicin cocaine
in myocarditis what happens to the CK-MB? troponins I and T?	↑ CK-MB ↑ troponins I and T
coxsackievirus is the most common cause of what disease?	Pericarditis
what relieves pain in pericarditis? (2)	pain is relieved by -precordial rub -lean forward
young woman with pericarditis and effusion most likely has SLE	---
pericardial effusion on inspiration the neck vein has distention what happens to systolic blood pressure?	↓ systolic blood pressure

constrictive pericarditis = ?

incomplete filling of chambers  
pericardial knock

what are the 3 types of  
cardiomyopathy?

1. dilated
2. hypertrophic
3. restrictive

What is the most common type of  
cardiomyopathy?

dilated cardiomyopathy

cause of dilated cardiomyopathy?

myocarditis is the most common cause

what drugs cause dilated  
cardiomyopathy?

doxorubicin  
daunorubicin

what happens to the heart in dilated  
cardiomyopathy?

global enlargement of the heart

hypertrophic cardiomyopathy (HCM) is  
the most common cause of sudden  
death in which age group of people?

HCM (hypertrophic cardiomyopathy)  
most common cause of death in young  
individuals

where is the obstruction located in hypertrophic cardiomyopathy (HCM)?	below the aortic valve
What is the sound of HCM (hypertrophic cardiomyopathy)?	preload changes on murmur intensity opposite of those for AV stenosis
what is the cause of sudden death in hypertrophic cardiomyopathy?	sudden death due to ventricular tachycardia/ fibrillation
What is the treatment of HCM (hypertrophic cardiomyopathy)?	$\beta$ -blockers
restrictive cardiomyopathy which is the least common cardiomyopathy has what type of voltatge ECG	low voltatge ECG
what happens to ventricular compliance in Restrictive cardiomyopathy ?	↓ ventricular compliance
which is more common in heart tumors metastasis or primary tumors?	metastasis > primary tumors

cardiac myxoma most common in which atrium?	in left atrium
myxomas occur which age group? rhabdomyomas occur at what age?	myxomas occur in adults rhabdomyomas occur in children
what effect does wall stress have on sarcomere duplication?"	wall stress increases gene-controlled sarcomere duplication
define afterload = ?	resistance ventricle contracts against to eject blood in systole
what is the ventricular hypertrophy in relation to afterload?	increased afterload causes concentric hypertrophy
preload = ?	equivalent to LVEDV (left ventricular end diastolic volume)
what is ventricular hypertrophy in relation to preload?	increased preload causes eccentric hypertrophy

what are the consequences of hypertrophy? (3)	heart failure S <sub>4</sub> angina (LVH)
what is S <sub>4</sub> ?	blood entering noncompliant ventricle
what does left side failure lead to?	left side failure= forward failure -> pulmonary endema
what is the most common type of LHF (Left Heart Failure)?	systolic dysfunction
what is the most common type of hypertension?	Diastolic dysfunction
Systolic dysfunction = ?	↓ ventricular contraction ↓ (Ejection faction) EF
Diastolic dysfunction - what happens?	↑ resistance to filling the ventricle normal EF (ejection faction)

What happens with dyspnea?	cannot take full inspiration
pulmonary edema = ?	hydrostatic pressure > oncotic pressure
Kerley's lines indicates what?	septal edema
Heart failure cells = ?	alveolar macrophages with hemosiderin
S <sub>3</sub> heart sound = ?	first cardiac sign of LHF
PND (Paroxysmal nocturnal dyspnea)/ orthopnea = ?	↑ venous return to right side of the heart at night
What is brain natriuretic peptide (BNP) useful for?	BNP (brain natriuretic peptide) is useful for confirming/ excluding LHF



RHF = ?	RHF = backward failure -> increase in venous hydrostatic pressure
what is the most common cause of LHF?	RHF
what happens to venous hydrostatic pressure in RHF?	↑ venous hydrostatic pressure
Clinical findings of RHF?	neck vein distention hepatomegaly dependent pitting edema ascites
ACE (angiotensin-converting enzyme) inhibitors causes?	↓ afterload ↓ preload
β-Blockers cause?	↓ myocardial O <sub>2</sub> consumption ↓ heart rate
High output failure causes?	↑ SV (stroke volume) ↓ TPR (total peripheral resistance) arteriovenous fistula

Tachycardia does what?

decreases diastole and filling of  
coronary arteries

What is the most common site of  
coronary artery thrombosis?

LAD (Left anterior descending)  
coronary artery

how is Angina pectoris related to  
coronary artery disease?

Angina pectoris is the most common  
manifestation of coronary artery  
disease?

what is the most important risk factor in  
Angina pectoris

age is the most important risk factor in  
Angina pectoris

which sex Angina pectoris most  
prevalent in?

males > females

what is the most common type of  
angina

stable angina

Define stable angina:

it is exercise induced substernal chest  
pain

Stable angina is caused by what?	subendocardial ischemia with ST-segment depression
Prinzmetal's angina = ?	vasospasm with transmural ischemia and ST-segment elevation
Unstable angina = ?	angina at rest; multivessel disease, disrupted plaques
What causes Prinzmetal's angina?	calcium channel blockers vasodilate coronary arteries
What is chronic ischemic heart disease	replacement of muscle by fibrous tissue
Define sudden cardiac death	unexpected death within 1 hour after symptoms
what is usually not present at sudden cardiac arrest?	coronary artery thrombosis

MVP (mitral valve prolapse) sudden death is caused by?	arrhythmias from mitral regurgitation or CHF (congestive heart failure)
What is the most common cause of death in the US?	AMI (Acute myocardial infarction)
what is the sequence of AMI (Acute myocardial infarction)?	Rupture of disrupted plaque -> platelet -> AMI (Acute myocardial infarction)
What does AMI (Acute myocardial infarction) by cocaine look like?	AMI with normal coronary arteries
what is the significance of Q wave in AMI	Q wave type transmural non-Q wave type subendocardial
what is the significance of reperfusion in AMI	↑ short/long term survival
Contraction band necrosis = ?	reperfusion histologically alters the damaged cells hypercontraction myofibrils due to $\text{Ca}^{2+}$

How long does the AMI coagulation take	AMI : coagulation necrosis within 24 hours
during AMI when is the heart softest?	3-7 days, at which time there is danger of rupture
Clinical findings of AMI?	retrosternal pain, radiation to left arm/shoulder, diaphoresis
if Q wave AMI is present?	↑ early mortality rate
Non-Q wave AMI is present?	↑ risk for SCD (sudden cardiac death)
Ventricular fibrillation relates to AMI how?	Ventricular fibrillation is the most common cause of death in acute MI
When is Myocardial rupture common?	3-7 days

What happens when posteromdial papillary muscle ruptures?	RCA thrombosis; mitral regurgitation
Mural thrombus is not a danger to embolization T/F	F, it is a danger to embolization
Fibrinous pericarditis?	early (acute inflammation) and late complication (autoimmune)
CHF most common cause of death?	ventricular aneurysm
RV AMI Clinical findings?	hypotension RHF preserved LV function
Reinfarction?	reappearance of CK-MB (creatin kinase isoenzyme) after 3 days
cTnI and cTnT in relation to reinfarction	cTnI and cTnT cannot diagnose reinfarction

cTnl and cTnT used for AMI	cTnl and cTnT is the gold standard for AMI
What are the ECG findings in AMI?	inverted T waves elevated ST segment Q waves
Chorionic villus is the primary site for?	chorionic villus is the primary site for O2 exchange
Umbilical vein relating to CHD (congenital heart disease)?	is the highest Po2 in fetal circulation
Fetal circulation = ?	foramen ovale and ductus arteriosus are patent
Single umbilical artery = ?	↑ risk congenital abnormalities
ASD (atrial septal defect) heart sound = ?	ASD (atrial septal defect) is fixed splitting of S2; most common adult CHD

PDA (patent ductus arteriosus) treatment and sound?	closed with indomethacin or surgery machinery murmur
What is the most common cyanotic CHD?	Tetralogy of Fallot
In Tetralogy of Fallot what does the degree of PV stenosis correlate to?	degree of PV stenosis correlates to absence of cyanosis
Which cardioprotective shunts help CHD?	ASD (atrial septal defect) - steps up Sao2 in right atrium PDA (patent ductus arteriosus) - shunts blood from aorta -> pulmonary artery
what can Tet spells do in CHD?	squatting ↑ systemic vascular resistance; ↑ PaO2
transposition = ?	aorta empties RV, pulmonary artery empties LV, atria normal
What is infantile coarctation associated with?	Turner's syndrome



What happens in adult coarctation?	disparity between upper/lower extremity blood pressure > 10 mm Hg
How does hypertension develop in adult coarctation?	due to activation RAA (renin-angiotensin-aldosterone) system
Coarctation collaterals: where do they develop?	<p>AIA(anterior intercostal arteries)-PIA(posterior intercostal arteries) to aorta</p> <p>SEA(superior epigastric artery)-IEA(inferior epigastric artery) to external iliac artery</p>