Cardio in review [dh]

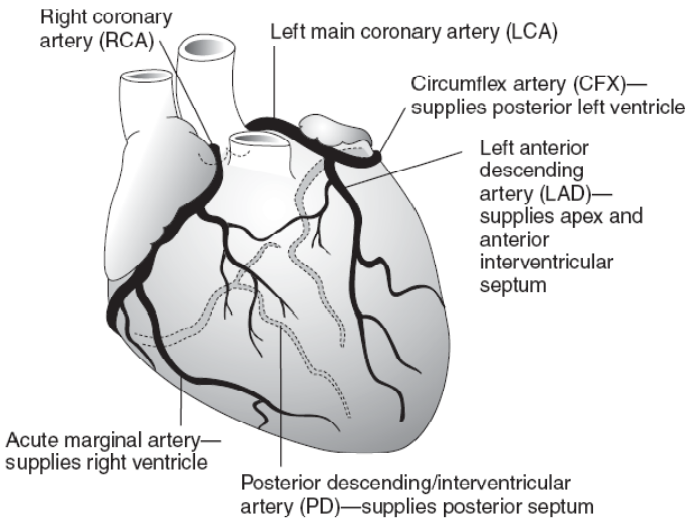
Carotid sheath

3 structures inside:

1. Internal jugular Vein (lateral)
2. Common carotid Artery (medial)
3. Vagus Nerve (posterior)

VAN.

Coronary artery anatomy



(Adapted, with permission, from Ganong WF. *Review of Medical Physiology,* 19th ed. Stamford, CT: Appleton & Lange, 1999: 592.)

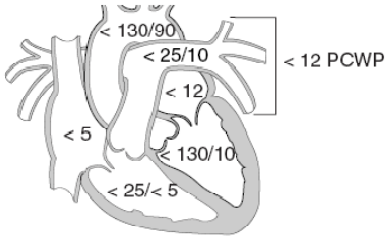
In the majority of cases, the SA and AV nodes are supplied by the RCA. 80% of the time, the RCA supplies the inferior portion of the left ventricle via the PD artery (= right dominant). 20% of the time, the PD arises from the CFX.

Coronary artery occlusion most commonly occurs in the LAD, which supplies the anterior interventricular septum.

Coronary arteries fill during diastole.

The most posterior part of the heart is the left atrium; enlargement can cause dysphagia or hoarseness.

Normal pressures



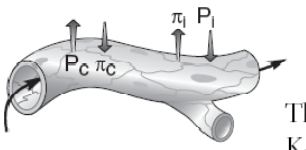
PCWP—pulmonary capillary wedge pressure (in mmHg) is a good approximation of left atrial pressure.
Measured with Swan-Ganz catheter.

Autoregulation

Organ	Factors determining autoregulation
Heart	Local metabolites—O_2, adenosine, NO
Brain	Local metabolites—CO_2 (pH)
Kidneys	Myogenic and tubuloglomerular feedback
Lungs	Hypoxia causes vasoconstriction
Skeletal muscle	Local metabolites—lactate, adenosine, K^+
Skin	Sympathetic stimulation most important mechanism—temperature control

Note: the pulmonary vasculature is unique in that hypoxia causes vasoconstriction. In other organs, hypoxia causes vasodilation.

Capillary fluid exchange



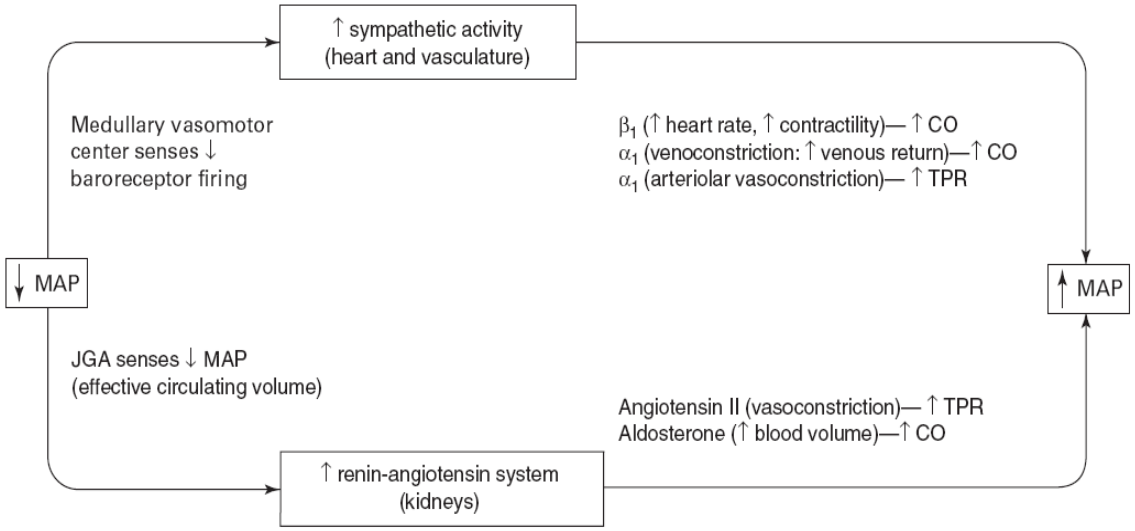
Starling forces determine fluid movement through capillary membranes:

1. P_c = capillary pressure—pushes fluid out of capillary
2. P_i = interstitial fluid pressure—pushes fluid into capillary
3. π_c = plasma colloid osmotic pressure—pulls fluid into capillary
4. π_i = interstitial fluid colloid osmotic pressure—pulls fluid out of capillary

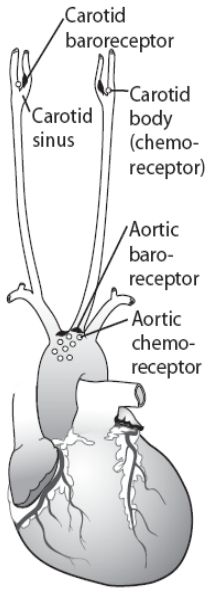
Thus, net filtration pressure = $P_{net} = [(P_c - P_i) - (\pi_c - \pi_i)]$.
K_f = filtration constant (capillary permeability).
Net fluid flow = $(P_{net})(K_f)$.
Edema—excess fluid outflow into interstitium commonly caused by:

1. ↑ capillary pressure (↑ P_c; heart failure)
2. ↓ plasma proteins (↓ π_c; nephrotic syndrome, liver failure)
3. ↑ capillary permeability (↑ K_f; toxins, infections, burns)
4. ↑ interstitial fluid colloid osmotic pressure (↑ π_i; lymphatic blockage)

Control of mean arterial pressure



Baroreceptors and chemoreceptors



Receptors:

1. Aortic arch transmits via vagus nerve to medulla (responds only to ↑ BP)
2. Carotid sinus transmits via glossopharyngeal nerve to medulla (responds to ↓ and ↑ in BP).

Baroreceptors:

1. Hypotension—↓ arterial pressure → ↓ stretch → ↓ afferent baroreceptor firing → ↑ efferent sympathetic firing and ↓ efferent parasympathetic stimulation → vasoconstriction, ↑ HR, ↑ contractility, ↑ BP. Important in the response to severe hemorrhage.
2. Carotid massage—↑ pressure on carotid artery → ↑ stretch → ↑ afferent baroreceptor firing → ↓ HR.

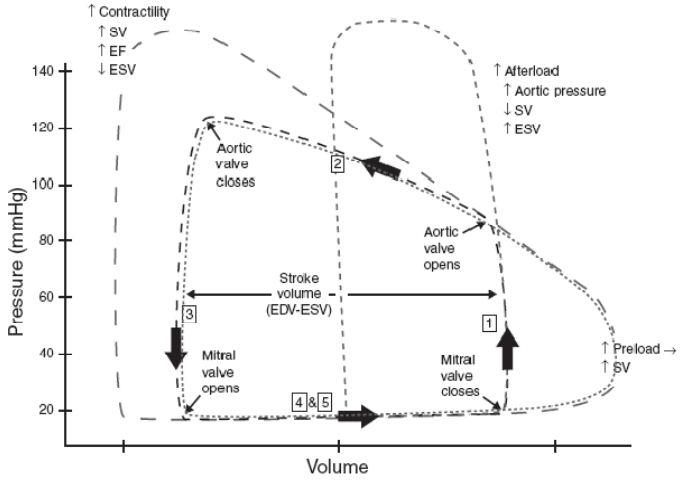
Chemoreceptors:

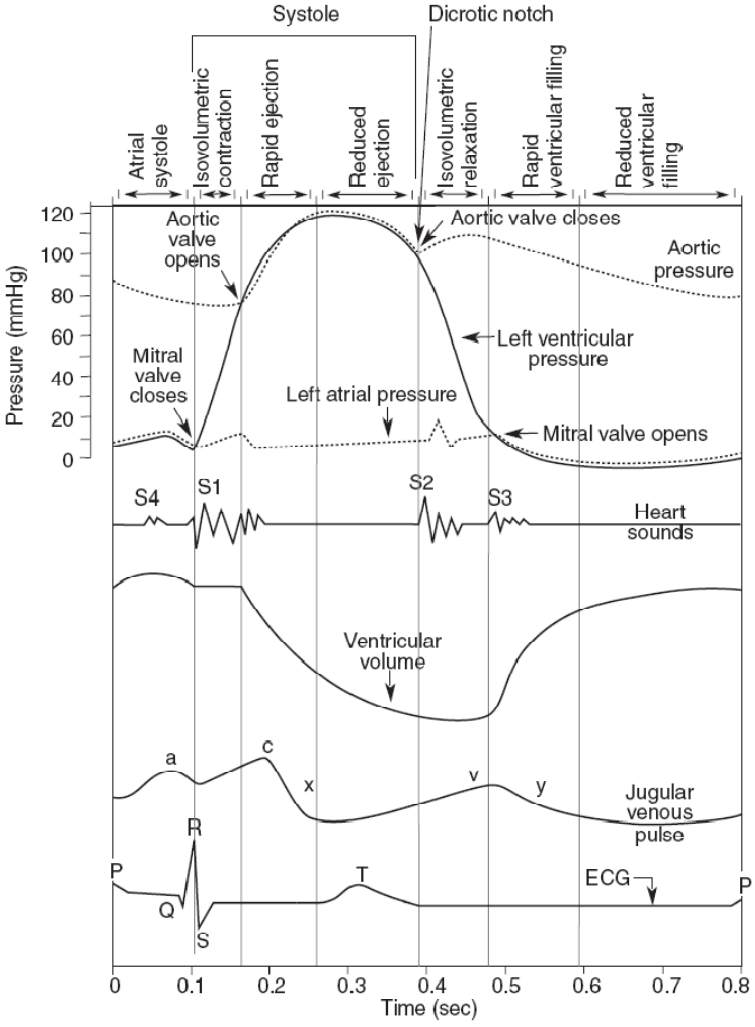
1. Peripheral—carotid and aortic bodies respond to ↓ P_{O_2} (< 60 mmHg), ↑ P_{CO_2}, and ↓ pH of blood.
2. Central—respond to changes in pH and P_{CO_2} of brain interstitial fluid, which in turn are influenced by arterial CO_2. Do not directly respond to P_{O_2}. Responsible for Cushing reaction— ↑ intracranial pressure constricts arterioles → cerebral ischemia → hypertension (sympathetic response) and reflex bradycardia. Note: Cushing triad = hypertension, bradycardia, respiratory depression.

Circulation through organs

Liver	Largest share of systemic cardiac output.
Kidney	Highest blood flow per gram of tissue.
Heart	Large arteriovenous O_2 difference. ↑ O_2 demand is met by ↑ coronary blood flow, not by ↑ extraction of O_2.

Cardiac cycle





(Adapted, with permission, from Ganong WF. *Review of Medical Physiology,* 22nd ed. New York: McGraw-Hill, 2005.)

Phases—left ventricle:

1. Isovolumetric contraction—period between mitral valve closure and aortic valve opening; period of highest O_2 consumption
2. Systolic ejection—period between aortic valve opening and closing
3. Isovolumetric relaxation—period between aortic valve closing and mitral valve opening
4. Rapid filling—period just after mitral valve opening
5. Reduced filling—period just before mitral valve closure

Sounds:

S1—mitral and tricuspid valve closure. Loudest at mitral area.

S2—aortic and pulmonary valve closure. Loudest at left sternal border.

S3—in early diastole during rapid ventricular filling phase. Associated with ↑ filling pressures and more common in dilated ventricles (but normal in children).

S4 ("atrial kick")—high atrial pressure. Associated with ventricular hypertrophy.

a wave—atrial contraction.

c wave—RV contraction (tricuspid valve bulging into atrium).

v wave—↑ atrial pressure due to filling against closed tricuspid valve.

S2 splitting: aortic valve closes before pulmonic; inspiration ↑ this difference.

Normal:

Expiration	|	||	
	S_1	A_2	P_2
Inspiration	|	| |	

Wide splitting (associated with pulmonic stenosis):

Expiration	|	| |	
	S_1	A_2	P_2
Inspiration	|	|	|

Fixed splitting (associated with ASD):

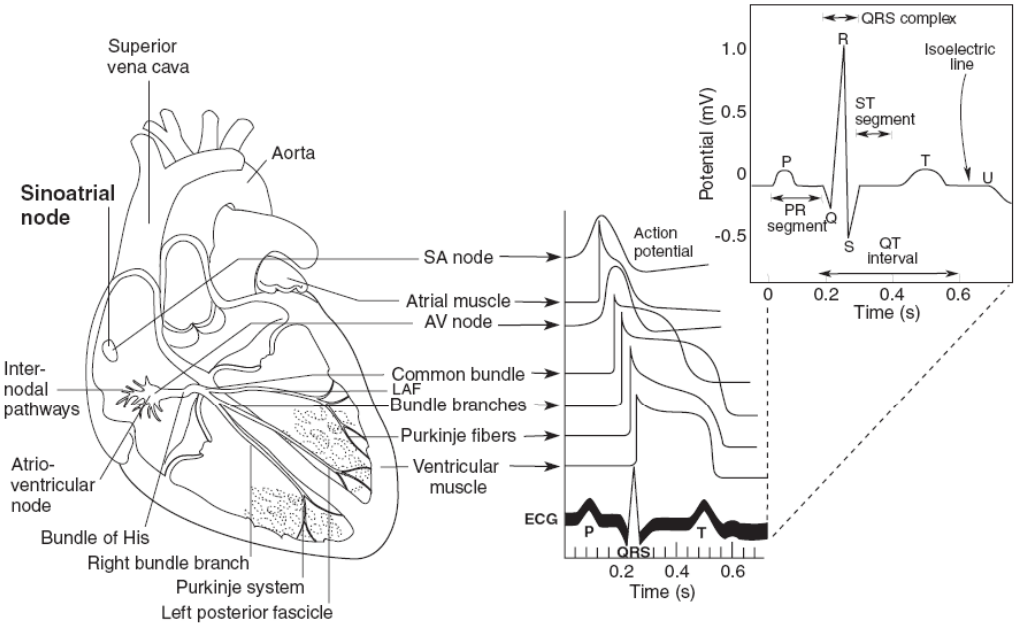
Expiration	|	|	|
	S_1	A_2	P_2
Inspiration	|	|	|

Paradoxical splitting (associated with aortic stenosis):

Expiration	|	|	|
	S_1	P_2	A_2
Inspiration	|	|	|

Electrocardiogram

P wave—atrial depolarization.
PR interval—conduction delay through AV node (normally < 200 msec).
QRS complex—ventricular depolarization (normally < 120 msec).
QT interval—mechanical contraction of the ventricles.
T wave—ventricular repolarization.
Atrial repolarization is masked by QRS complex.
ST segment—isoelectric, ventricles depolarized.
U wave—caused by hypokalemia, bradycardia.



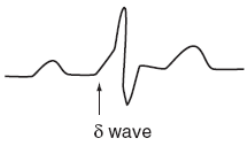
SA node "pacemaker" inherent dominance with slow phase of upstroke
AV node - 100-msec delay - atrioventricular delay

(Adapted, with permission, from Ganong WF. *Review of Medical Physiology,* 22nd ed. New York: McGraw-Hill, 2005: 548, 550.)

Torsades des pointes

Ventricular tachycardia characterized by shifting sinusoidal waveforms on ECG. Can progress to V-fib. Anything that prolongs the QT interval can predispose to torsades des pointes.

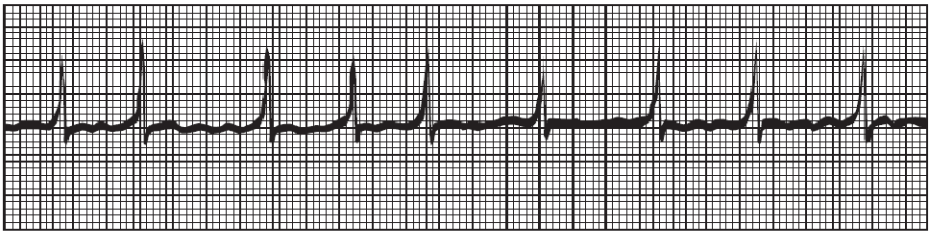
Wolff-Parkinson-White syndrome



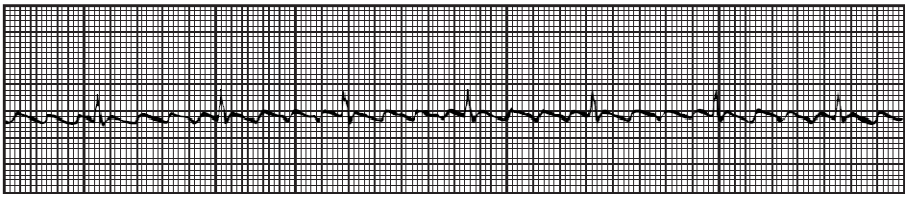
Accessory conduction pathway from atria to ventricle (bundle of Kent), bypassing AV node. As a result, ventricles begin to partially depolarize earlier, giving rise to characteristic delta wave on ECG. May result in reentry current leading to supraventricular tachycardia.

ECG tracings

Atrial fibrillation — Chaotic and erratic baseline (irregularly irregular) with no discrete P waves in between irregularly spaced QRS complexes.

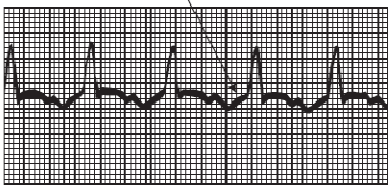


Atrial flutter — A rapid succession of identical, back-to-back atrial depolarization waves. The identical appearance accounts for the "sawtooth" appearance of the flutter waves.



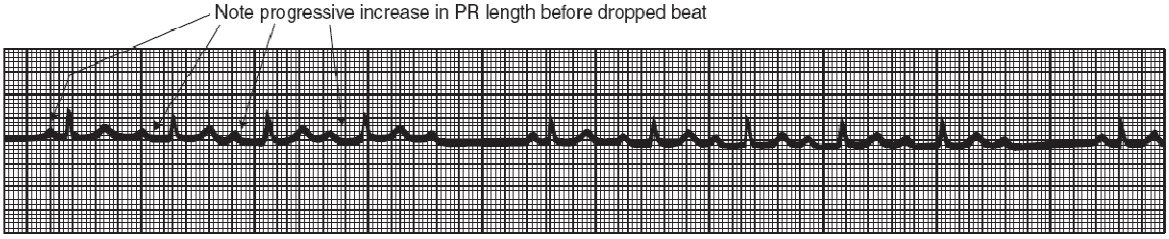
AV block

1st degree — The PR interval is prolonged (> 200 msec). Asymptomatic.

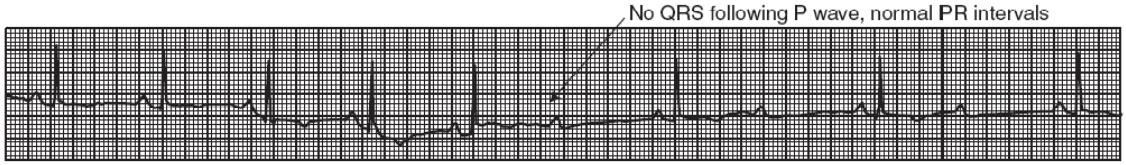


2nd degree

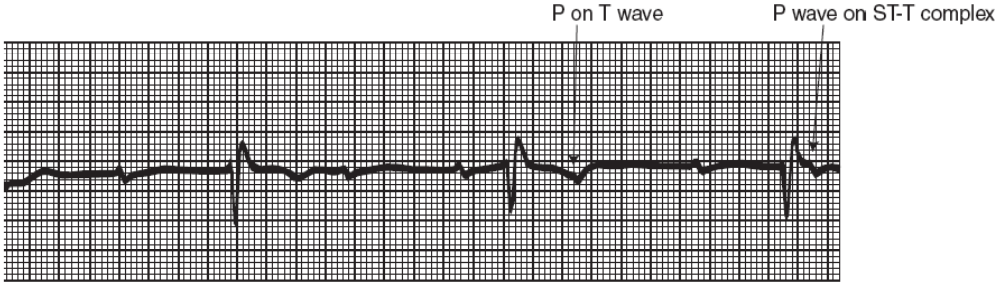
Mobitz type I (Wenckebach) — Progressive lengthening of the PR interval until a beat is "dropped" (a P wave not followed by a QRS complex). Usually asymptomatic.



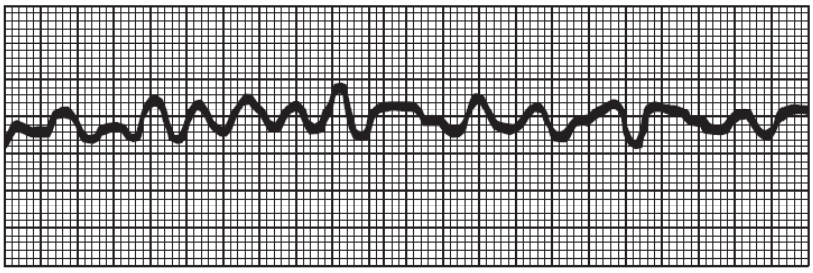
Mobitz type II — Dropped beats that are not preceded by a change in the length of the PR interval (as in type I). These abrupt, nonconducted P waves result in a pathologic condition. It is often found as 2:1 block, where there are 2 P waves to 1 QRS response. May progress to 3rd-degree block.



3rd degree (complete) — 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 pacemaker.



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



(Adapted, with permission, from Hurst JW. *Introduction to Electrocardiography.* New York: McGraw-Hill, 2001.)