FIRST AID FOR THE®

USMLE STEP 1 2023

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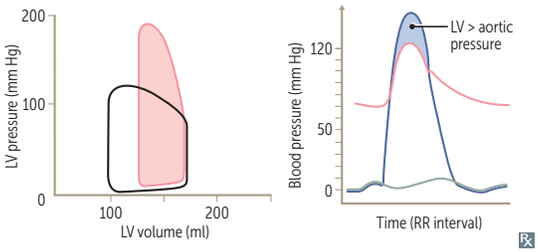
Pressure-volume loops and valvular disease

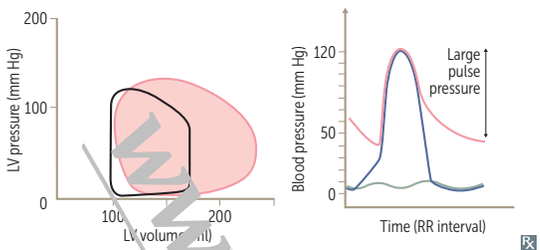
Aortic stenosis



↑ LV pressure
↑ ESV
No change in EDV (if mild)
↓ SV

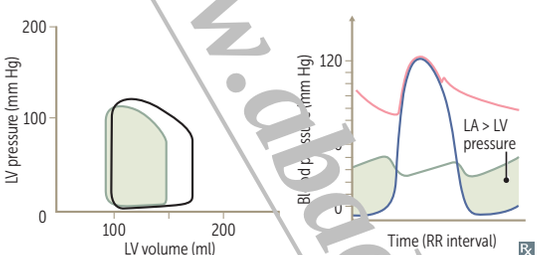
Ventricular hypertrophy → ↓ ventricular compliance → ↑ EDP for given EDV

Aortic regurgitation



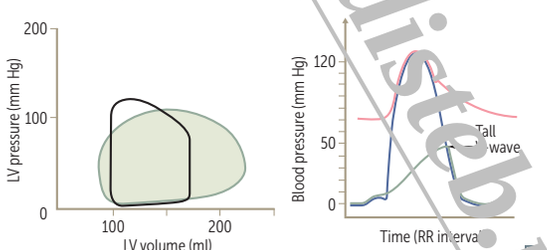
No true isovolumetric phase
↑ EDV
↑ SV
Loss of dicrotic notch

Mitral stenosis



↑ LA pressure
↓ EDV because of impaired ventricular filling
↓ ESV
↓ SV

Mitral regurgitation

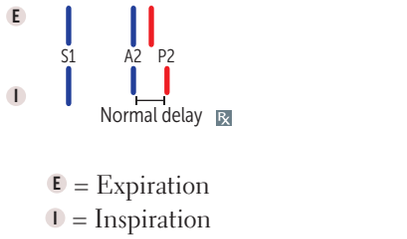


No true isovolumetric phase
↓ ESV due to ↓ resistance and ↑ regurgitation into LA during systole
↑ EDV due to ↑ LA volume/pressure from regurgitation → ↑ ventricular filling
↑ SV (forward flow into systemic circulation plus backflow into LA)

Splitting of S2

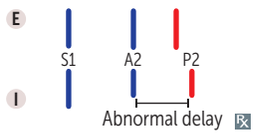
Physiologic splitting

Inspiration → drop in intrathoracic pressure → ↑ venous return → ↑ RV filling → ↑ RV stroke volume → ↑ RV ejection time → delayed closure of pulmonic valve. ↓ pulmonary impedance (↑ capacity of the pulmonary circulation) also occurs during inspiration, which contributes to delayed closure of pulmonic valve.



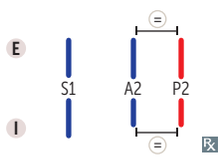
Wide splitting

Seen in conditions that delay RV emptying (eg, pulmonic stenosis, right bundle branch block). Causes delayed pulmonic sound (especially on inspiration). An exaggeration of normal splitting.



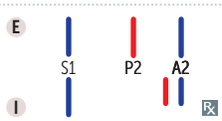
Fixed splitting

Heard in ASD. ASD → left-to-right shunt → ↑ RA and RV volumes → ↑ flow through pulmonic valve → delayed pulmonic valve closure (independent of respiration).

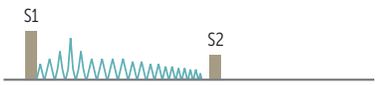
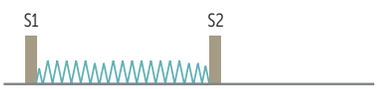
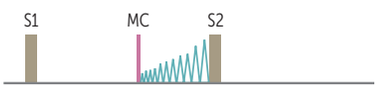
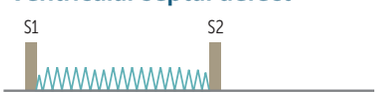
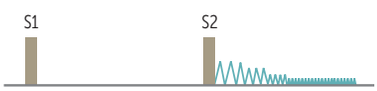
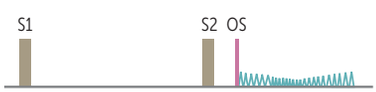
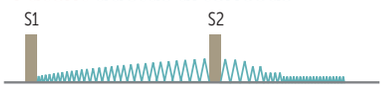


Paradoxical splitting

Heard in conditions that delay aortic valve closure (eg, aortic stenosis, left bundle branch block). Normal order of semilunar valve closure is reversed: in paradoxical splitting P2 occurs before A2. On inspiration, P2 closes later and moves closer to A2, "paradoxically" eliminating the split. On expiration, the split can be heard (opposite to physiologic splitting).



Heart murmurs

	AUSCULTATION	CLINICAL ASSOCIATIONS	NOTES
Systolic			
Aortic stenosis 	Crescendo-decrescendo ejection murmur, loudest at heart base, radiates to carotids Soft S2 +/– ejection click "Pulsus parvus et tardus"—weak pulses with delayed peak	In older (>60 years old) patients, most commonly due to age-related calcification In younger patients, most commonly due to early-onset calcification of bicuspid aortic valve	Can lead to **S**yncope, **A**ngina, **D**yspnea on exertion (**SAD**) LV pressure > aortic pressure during systole
Mitral/tricuspid regurgitation 	Holosystolic, high-pitched "blowing" murmur MR: loudest at apex, radiates toward axilla TR: loudest at tricuspid area	MR: often due to ischemic heart disease (post-MI), MVP, LV dilatation, rheumatic fever TR: often due to RV dilatation Either MR or TR: infective endocarditis	
Mitral valve prolapse 	Late crescendo murmur with midsystolic click (MC) that occurs after carotid pulse Best heard over apex Loudest just before S2	Usually benign, but can predispose to infective endocarditis Can be caused by rheumatic fever, chordae rupture, or myxomatous degeneration (1° or 2° to connective tissue disease)	MC due to sudden tensing of chordae tendineae as mitral leaflets prolapse into LA (**c**hordae cause **c**rescendo with **c**lick)
Ventricular septal defect 	Holosystolic, harsh-sounding murmur Loudest at tricuspid area	Congenital	Larger VSDs have lower intensity murmur than smaller VSDs
Diastolic			
Aortic regurgitation 	Early diastolic, decrescendo, high-pitched "blowing" murmur best heard at base (aortic root dilation) or left sternal border (valvular disease)	Causes include **BEAR**: ▪ **B**icuspid aortic valve ▪ **E**ndocarditis ▪ **A**ortic root dilation ▪ **R**heumatic fever Wide pulse pressure, pistol shot femoral pulse, pulsing nail bed (Quincke pulse)	Hyperdynamic pulse and head bobbing when severe and chronic Can progress to left HF
Mitral stenosis 	Follows opening snap (OS) Delayed rumbling mid-to-late murmur (↓ interval between S2 and OS correlates with ↑ severity)	Late and highly specific sequelae of rheumatic fever Chronic MS can result in LA dilation and pulmonary congestion, atrial fibrillation, Ortner syndrome, hemoptysis, right HF	OS due to abrupt halt in leaflet motion in diastole after rapid opening due to fusion at leaflet tips LA >> LV pressure during diastole
Continuous			
Patent ductus arteriosus 	Continuous **machine**like murmur, best heard at left infraclavicular area Loudest at S2	Often due to congenital rubella or prematurity	You need a **patent** for that **machine**.

Myocardial action potential

Phase 0 = rapid upstroke and depolarization—voltage-gated Na^+ channels open.

Phase 1 = initial repolarization—inactivation of voltage-gated Na^+ channels. Voltage-gated K^+ channels begin to open.

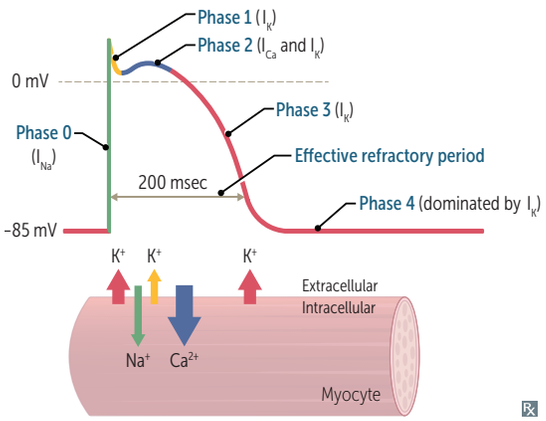
Phase 2 = plateau ("plat**two**")—Ca^{2+} influx through voltage-gated Ca^{2+} channels balances K^+ efflux. Ca^{2+} influx triggers Ca^{2+} release from sarcoplasmic reticulum and myocyte contraction (excitation-contraction coupling).

Phase 3 = rapid repolarization—massive K^+ efflux due to opening of voltage-gated slow delayed-rectifier K^+ channels and closure of voltage-gated Ca^{2+} channels.

Phase 4 = resting potential—high K^+ permeability through K^+ channels.

In contrast to skeletal muscle:

- Cardiac muscle action potential has a plateau due to Ca^{2+} influx and K^+ efflux.
- Cardiac muscle contraction requires Ca^{2+} influx from ECF to induce Ca^{2+} release from sarcoplasmic reticulum (Ca^{2+}-induced Ca^{2+} release).
- Cardiac myocytes are electrically coupled to each other by gap junctions.



Occurs in all cardiac myocytes except for those in the SA and AV nodes.

Pacemaker action potential

Occurs in the SA and AV nodes. Key differences from the ventricular action potential include:

Phase 0 = upstroke—opening of voltage-gated Ca^{2+} channels. Fast voltage-gated Na^+ channels are permanently inactivated because of the less negative resting potential of these cells. Results in a slow conduction velocity that is used by the AV node to prolong transmission from the atria to ventricles.

Phases 1 and 2 are absent.

Phase 3 = repolarization—inactivation of the Ca^{2+} channels and ↑ activation of K^+ channels → ↑ K^+ efflux.

Phase 4 = slow spontaneous diastolic depolarization due to I_f ("funny current"). I_f channels responsible for a slow, mixed Na^+ inward/K^+ outward current; different from I_{Na} in phase 0 of ventricular action potential. Accounts for automaticity of SA and AV nodes. The slope of phase 4 in the SA node determines HR. ACh/adenosine ↓ the rate of diastolic depolarization and ↓ HR, while catecholamines ↑ depolarization and ↑ HR. Sympathetic stimulation ↑ the chance that I_f channels are open and thus ↑ HR.