

VERSION A

How do positional maneuvers affect blood flow and murmurs?

a) Standing/Valsalva

b) Squatting/Lying down

c) Sustained handgrip - ANSWER- **-standing/valsalva - decreased cardiac filling, decreases most murmurs except MVP and HOCM**

-squatting/ lying down - increase cardiac volume, increased murmurs except MVP, HOCM

-sustained handgrip - increases systemic resistance, decreases murmur in HOCM, AS

learnexams

What are the stages of the Valsalva maneuver? - ANSWER- **-Phase one is the onset of straining with increased intrathoracic pressure. The heart rate does not change but blood pressure rises.**

-Phase two is marked by the decreased venous return and consequent reduction of stroke volume and pulse pressure as straining continues. The heart rate increases and blood pressure drops.

-Phase three is the release of straining with decreased intrathoracic pressure and normalization of pulmonary blood flow.

-Phase four marks the blood pressure overshoot (in the normal heart) with return of the heart rate to baseline.

What causes a physiologic split S2? - ANSWER- Increased blood volume in the RV prolongs systole and delays pulmonary valve closure

What causes a fixed split S2? - ANSWER- Pulmonary stenosis, PE, LV pacer, RBBB, MR (early AV closure), ASD, RV failure

What causes a paradoxical split S2 - ANSWER- LBBB, RV pacing, HOCM

What causes an S3? - ANSWER- Rapid LV filling - acute ventricular decompensation, severe AR or MR

What causes a S4? - ANSWER- Decreased ventricular compliance during atrial contraction - ischemic heart dz, AS, MR, HOCM, hypertrophic or diabetic cardiomyopathy, HTN heart dz, concentric LVH

Can you have a S4 with atrial fibrillation? - ANSWER- No - no atrial contraction

What are the parts of the venous waveform? - ANSWER- A wave - atrial contraction

X descent - atria relax, RV fills rapidly; Bottom/middle of x descent is TC valve closure (c wave)

V wave - ventricle contacting against closed TC valve

Y descent - TC valve opens, passive emptying into ventricle

What gives elevated a and v waves - ANSWER- **Pulmonary HTN, RV infarction**

What leads to Large r side v waves - ANSWER- **Septal rupture**

What diseases lead to Large v waves - ANSWER- **TR (right), MR (left)**

Rapid x and y descent - ANSWER- **Constrictive pericarditis, restrictive cardiomyopathy, tamponade (x descent only, loss of y descent)**

Large a waves - ANSWER- **TS, severe RVH (on right), MS**

Cannon a waves - ANSWER- **AV disassociation - complete heart block, ventricular pacing**

Slow Y descent - ANSWER- **Delayed atrial emptying - TS**

Most important prognostic factor with CAD - ANSWER- **Degree of LV dysfunction**

Causes of resting ST elevation - ANSWER- **MI, pericarditis, LV aneurysm, LBBB, ventricular pacing, LVH, early repolarization**

Giving nitrates causes severe decompensation in a IWMI pt. What happened? - ANSWER- Pt had R side infarction as well, the preload reduction from the nitrate now meant little flow getting to the L side of the heart

MR due to papillary muscle rupture is most common with MI in this region - ANSWER- Inferior; posteromedial papillary muscle has only single vessel supply (RCA) while the anterolateral has two vessels

VSD is more likely with MIs here - ANSWER- Anterior, inferior

Contraindications for B-blockers - ANSWER- Bradycardia, hypotension, 2nd or 3rd degree AVB, pulmonary edema, asthma. NOT DM

When to use non-dihydropyridine CCBs in ACS - ANSWER- Contraindications to B blockers, continued ischemia, but NO LV dysfunction

What anticoagulant to use with ACS - ANSWER- Enoxaparin good, but have to stop 12-24 hrs before CABG

Fondaparinux is increased risk of bleeding, do not use if going to do PCI - increased risk of catheter thrombosis and coronary complications

If using Fondaparinux and decide to do PCI, change to heparin or bivalirudin