Domain I

I.A.1

Cardiac Physiology: Cycle, Output, Coronary Flow

YOU SHOULD BE ABLE TO

- ✓ Walk the cardiac cycle and draw it as a pressure-volume loop
- ✓ Separate preload, afterload, contractility and compliance, and predict what each one does to stroke volume
- ✓ Balance myocardial oxygen supply against demand, including coronary perfusion pressure and diastolic time

THE LESSON ON ONE PAGE

The cycle

- Kick, IVC, eject, IVR, fill
- Both valves shut in IVC and IVR
- Atrial kick 20 to 30 percent
- S1 mitral, S2 aortic

The numbers

- EDV 120, ESV 50, SV 70 mL
- EF 55 to 70 percent
- CO 4 to 8 L/min, CI 2.5 to 4.0
- CO = HR times PAC

The loop

- Width is SV, area is stroke work
- ESPVR slope is contractility
- EDPVR is diastolic stiffness
- Preload widens, afterload heightens

Coronary balance

- Supply DOC, demand HAC
- CPP = aortic diastolic minus LVEDP
- Left in diastole, right throughout
- Subendocardium suffers first

KEY TABLES

Lesions on the loop

Intervention	Main effect	How the loop moves
Fluid bolus	Preload up	Bottom right corner slides right; loop widens
Phenylephrine	Afterload up	Loop gets taller and narrower; ESV rises
Nitroglycerin	Preload down (venodilation)	Loop shifts left and narrows; wall stress falls
Sodium nitroprusside	Afterload down	Loop gets shorter and wider; ESV falls
Epinephrine or dobutamine	Contractility up	ESPVR rotates up and left; ESV falls, loop widens
Esmolol	Rate and contractility down	ESPVR flattens; longer diastole fills the loop wider

BOARD TRAPS

"Ejection fraction is a measure of contractility."

Not by itself. Ejection fraction is **load dependent**. Raise afterload and it falls with no change in the muscle. In mitral regurgitation the ventricle empties into a low-pressure atrium, so an ejection fraction of 55 percent actually signals **impaired** contractility. The load-independent index is the slope of the ESPVR.

"Coronary flow stops during systole, so the whole heart is starved then."

Only the left side. The **right** ventricle is perfused in systole and diastole because its wall pressure stays well below aortic pressure. Add severe pulmonary hypertension or RV hypertrophy and the right ventricle starts behaving like the left: diastole dependent, and easy to make ischemic.

"A high CVP means the patient has enough volume."

False. CVP is a **pressure**, and it reflects right-sided compliance, intrathoracic pressure and tricuspid function as much as volume. A stiff ventricle, high PEEP or tamponade all raise it without filling anybody. Static pressures predict fluid responsiveness poorly.

"Thermodilution overestimates output in severe tricuspid regurgitation."

Backwards. Regurgitant flow recirculates the cold indicator, which spreads the curve out and usually makes the reported output **falsely low**. The things that read falsely **high** are injectate that is too warm, too small a volume, and a left-to-right shunt.

"Nitroglycerin helps angina by dilating the coronary arteries."

Mostly not. Its main benefit is **venodilation**: less preload, a smaller radius, less wall stress and a lower LVEDP, which also improves the subendocardial perfusion gradient. It does dilate epicardial conduit vessels and relieve spasm, but coronary steal is the risk with pure arteriolar dilators.

MEMORY DEVICES

Everything that sets cardiac output

HR × PAC

- **HR** Heart rate: the only factor outside the ventricle
- **P** Preload: how full it is when systole starts
- **A** Afterload: what it has to push against
- **C** Contractility: how hard it squeezes at that load