Heart Failure and Pulmonary Over circulation

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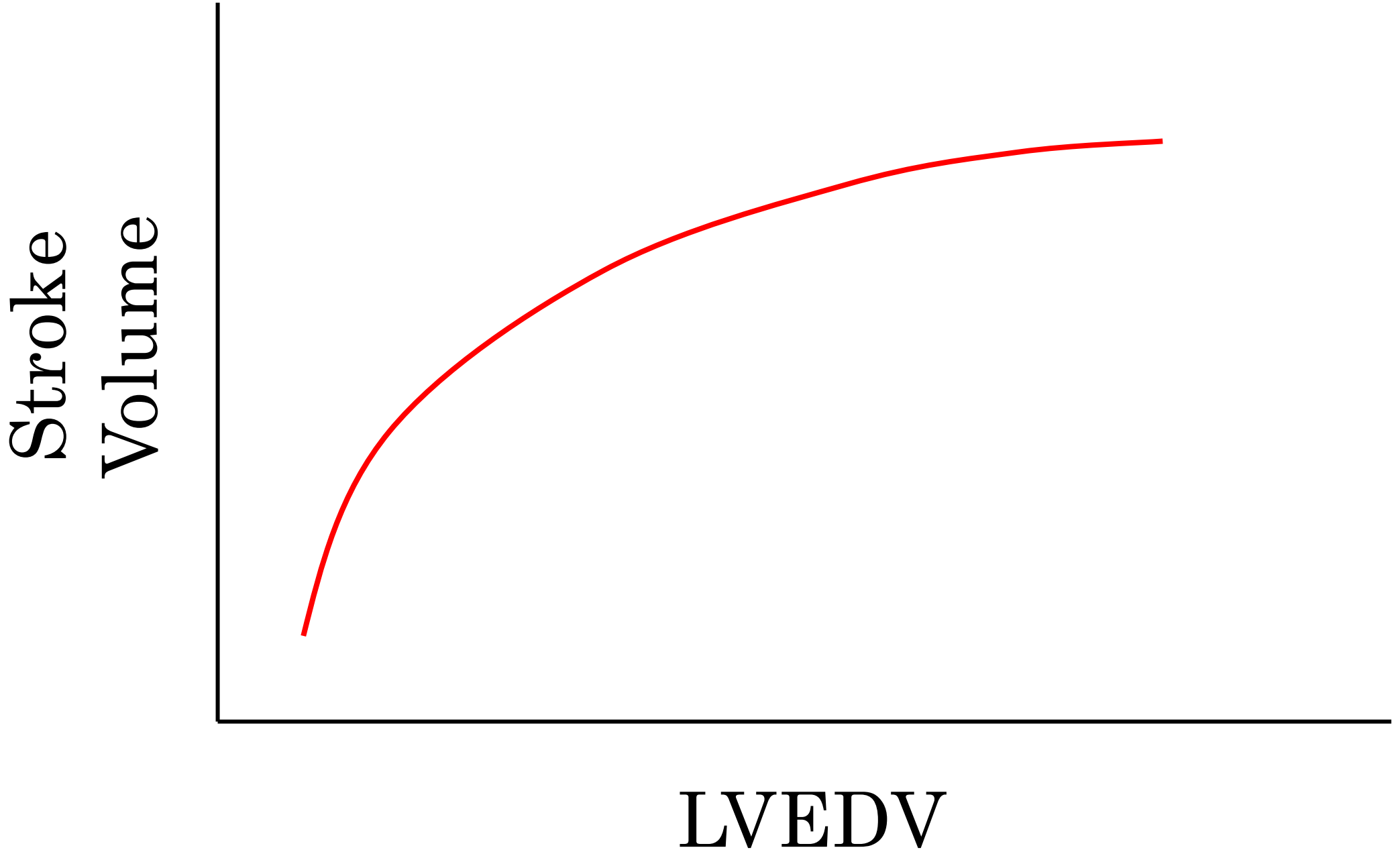
Objectives

- Understand the physiologic basis of systolic and diastolic dysfunction
- Discuss the clinical presentation and management of heart failure
- Discuss the pathophysiology and management of pulmonary overcirculation

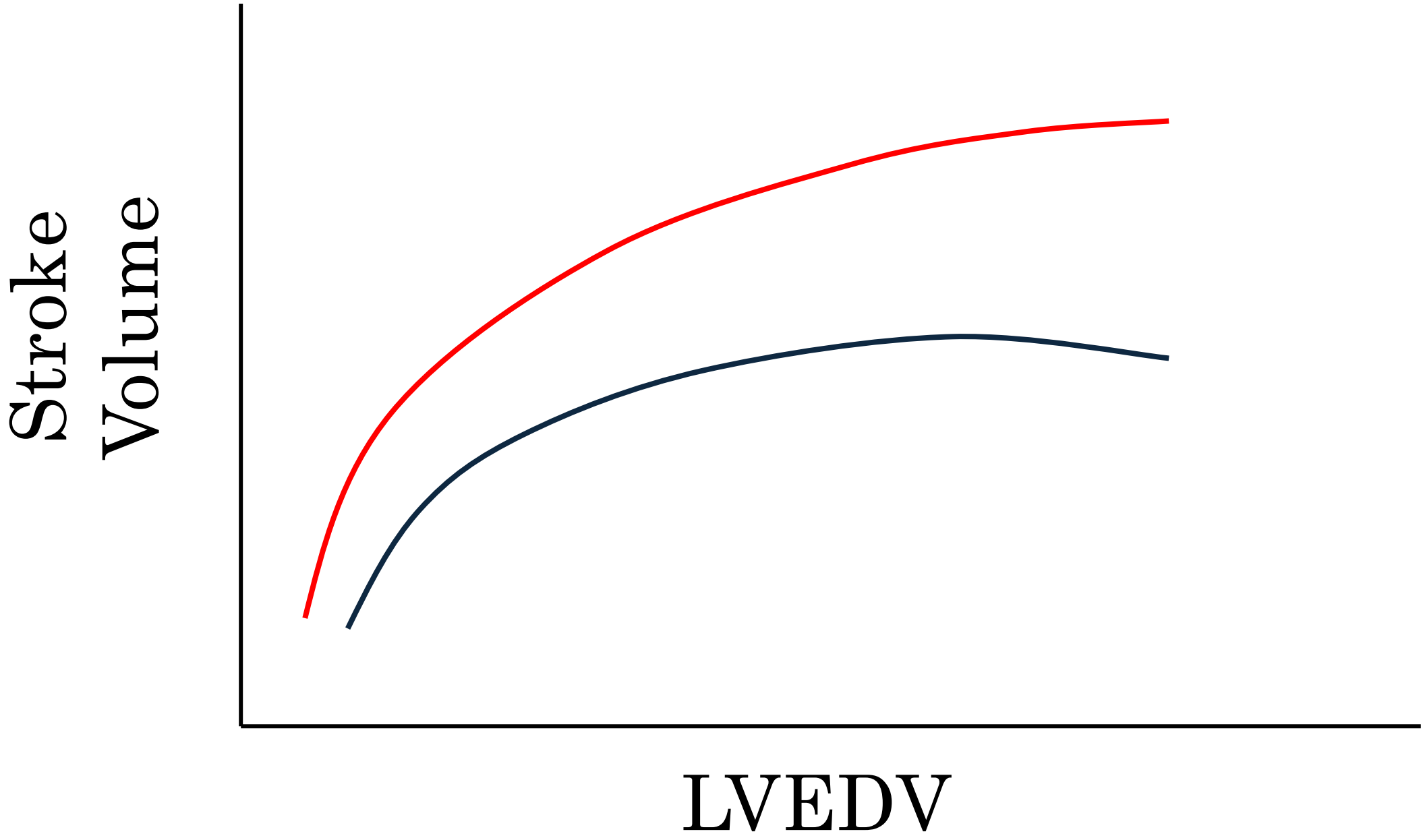
Frank – Starling Curve



Question

You are taking care of 3 week old neonate who presented with increased WOB and poor feeding. CXR showed cardiomegaly. You order an echo and it shows severely depressed function with EF of 21%.

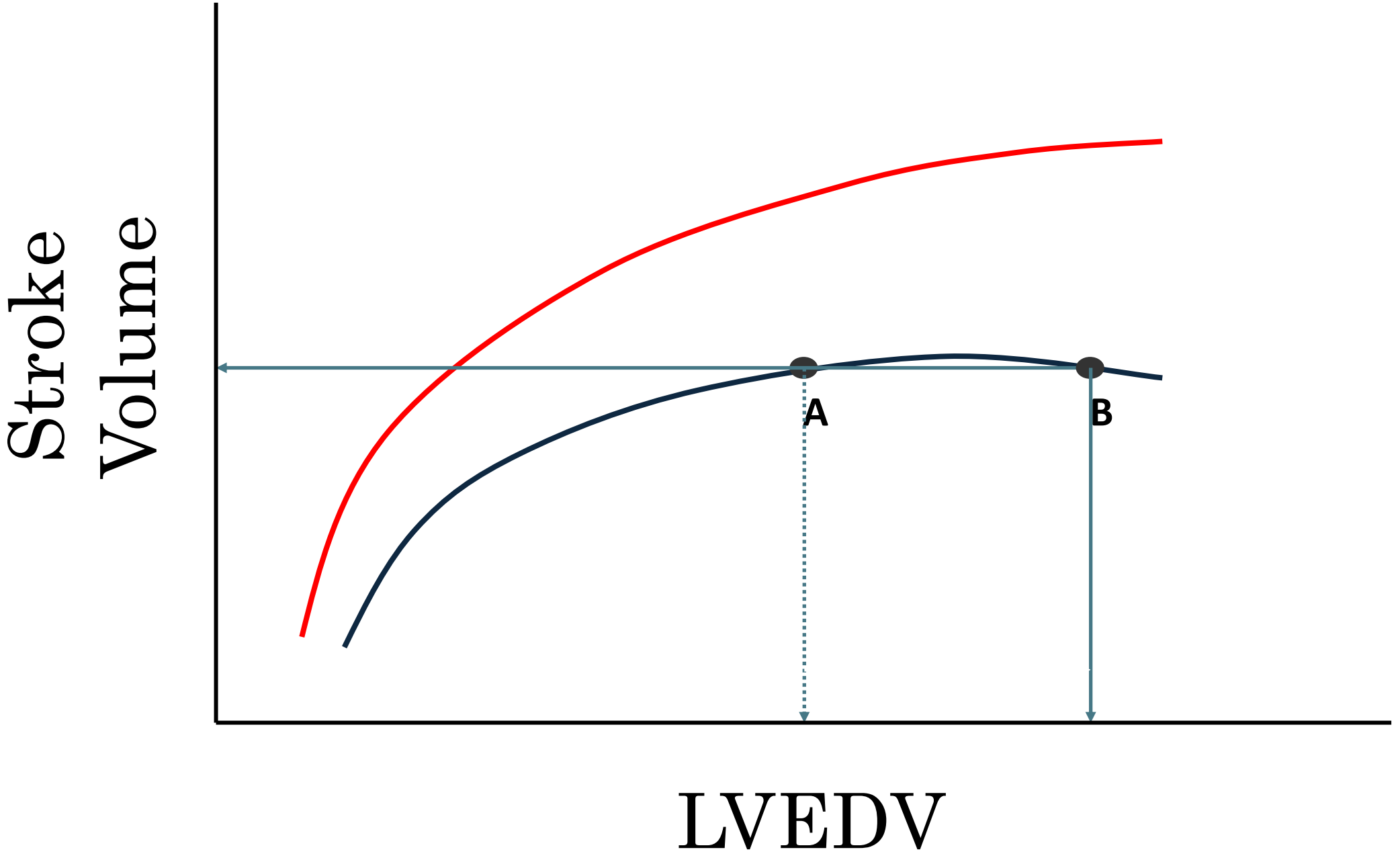
Indicate on your drawing what would happen in the setting of depressed contractility


Stroke Volume
LVEDV

Question

You are called to bedside because the patient is hypotensive. You decide to give a fluid bolus.

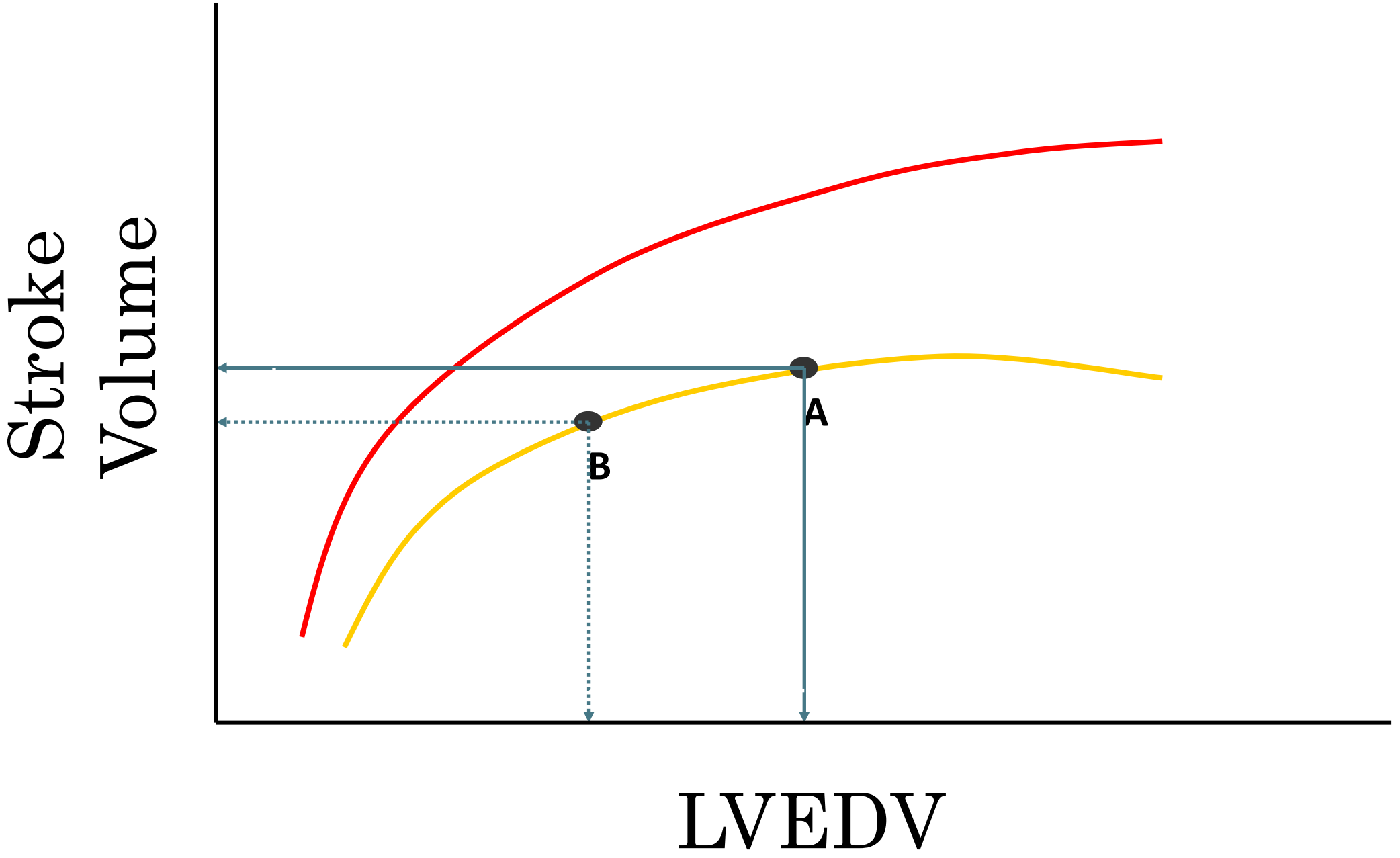
Given the two curves used on the previous question and starting at point A, what effect would a fluid bolus have?


Stroke Volume
A
B
LVEDV

Question

Your senior colleague walks into the room and astutely points out that you should give a small dose of furosemide instead.

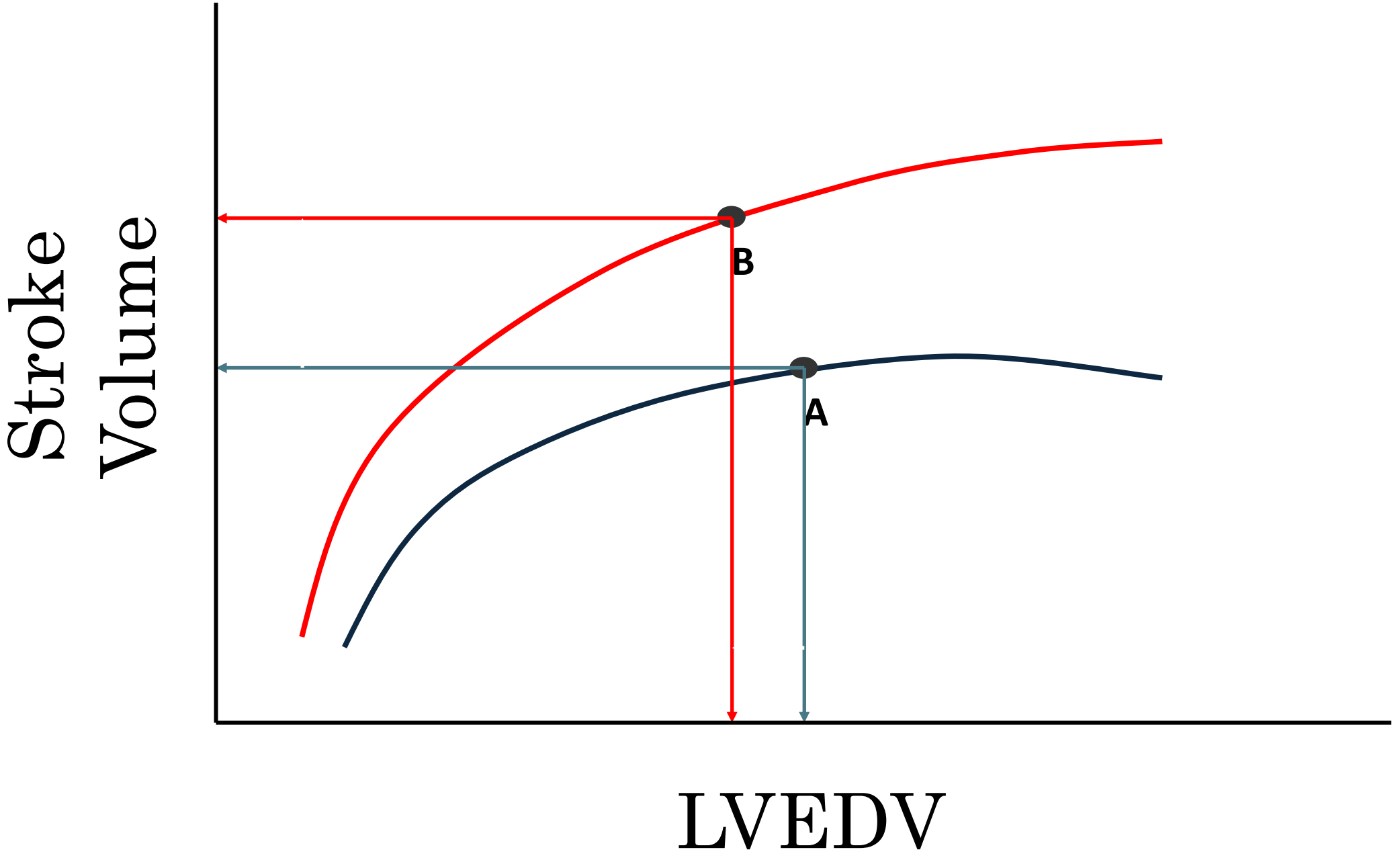
Again, starting with a normal Starling curve and a curve demonstrating decreased contractility, what would be the effect of a diuretic regimen if a patient started at point A?


Stroke Volume
A
B
LVEDV

Question

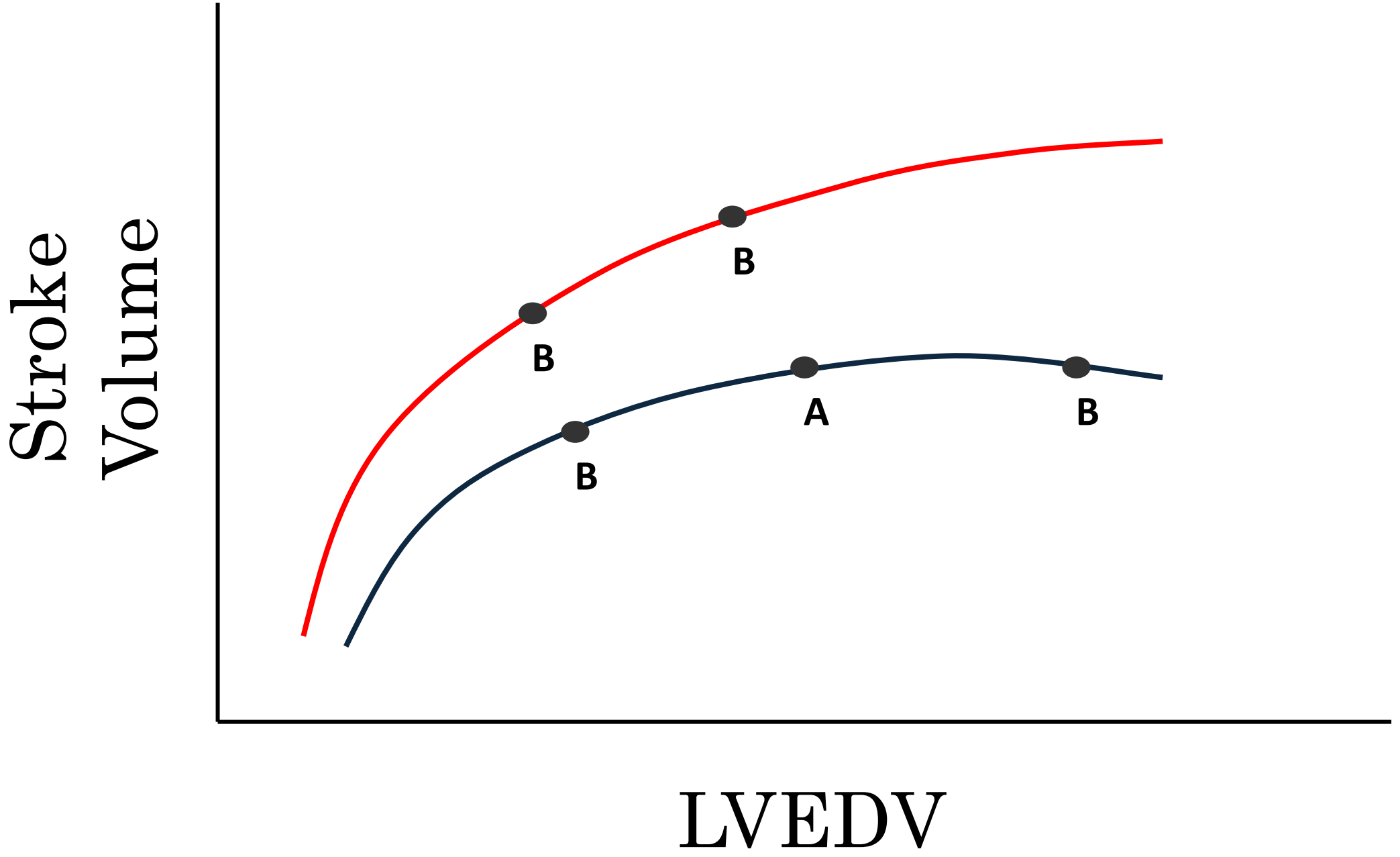
The patient remains hypotensive with minimal response to furosemide. NIRS are dropping. So, you decide to initiate epinephrine infusion.

Again, starting with the normal and decreased contractility curves and starting at point A, what would the effect be of starting an inotropic agent?

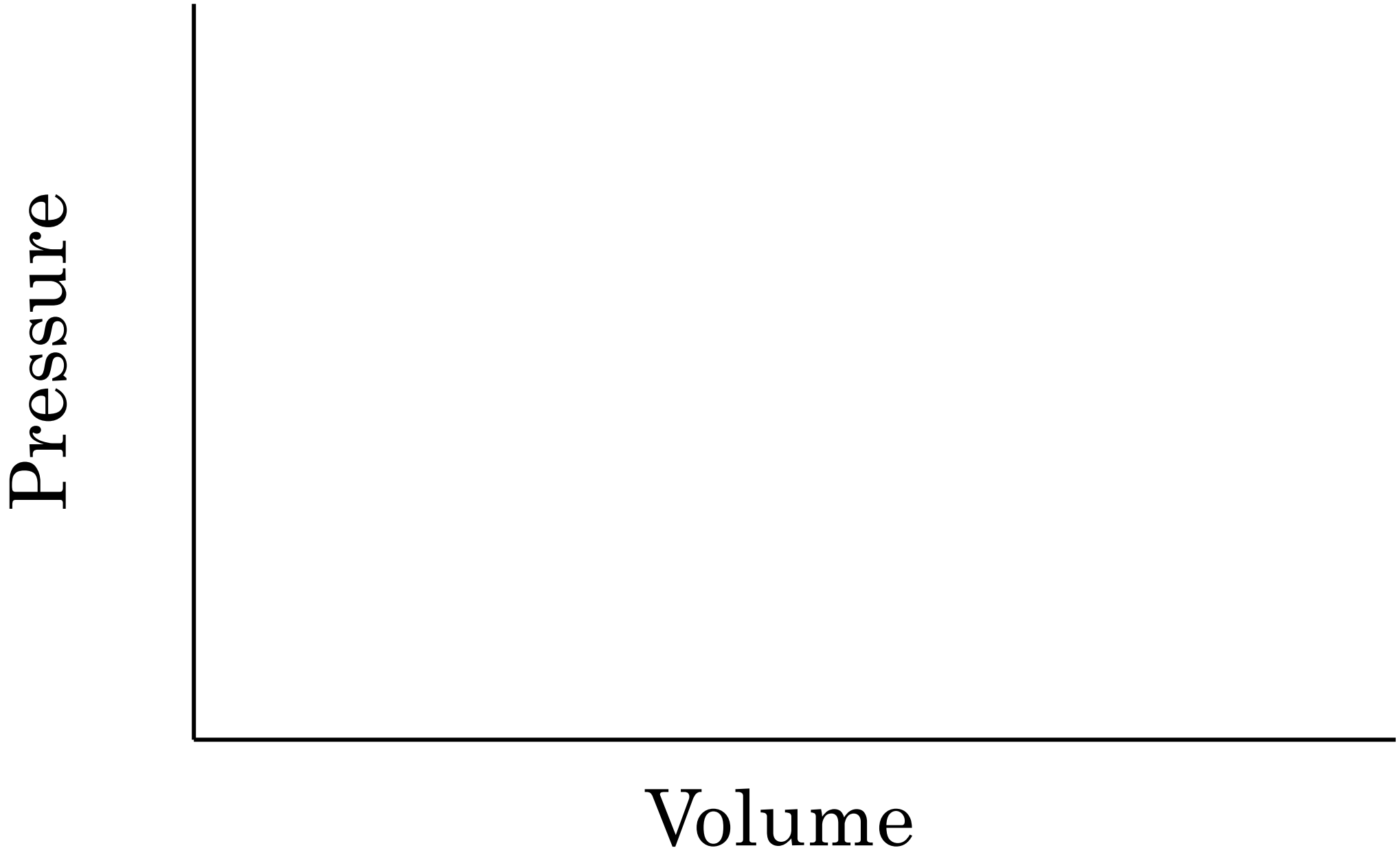

Stroke Volume
B
A
LVEDV

KEY CONCEPTS: The Starling Curve

- Altering the intravascular volume results in a move ALONG THE SAME CURVE
- A change in the contractility of the myocardium, for whatever reason, results in a CURVE SHIFT

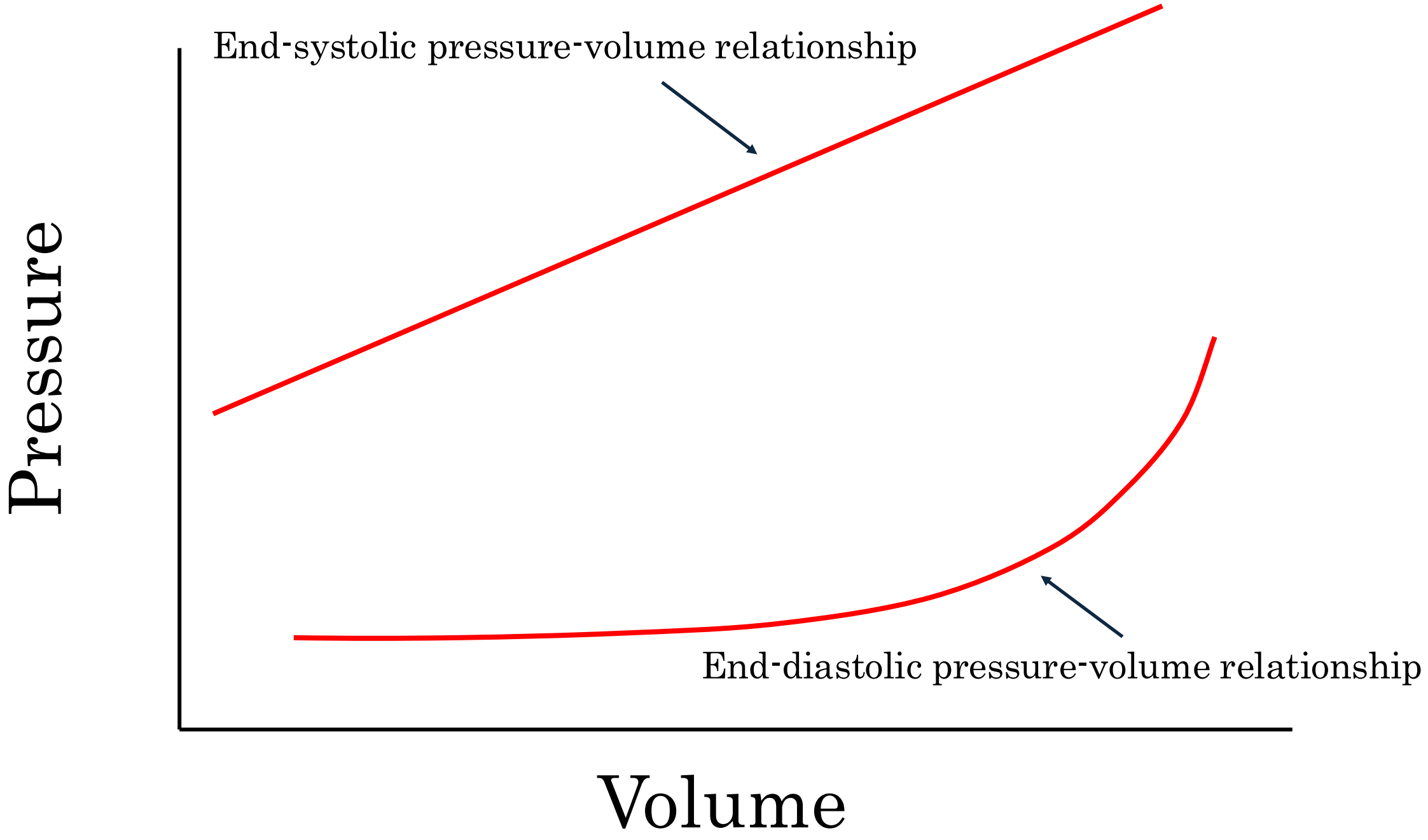

Stroke Volume
B
B
A
B
B
LVEDV

Draw and correctly label the axes for a cardiac output pressure volume loop


Pressure
Volume

Question

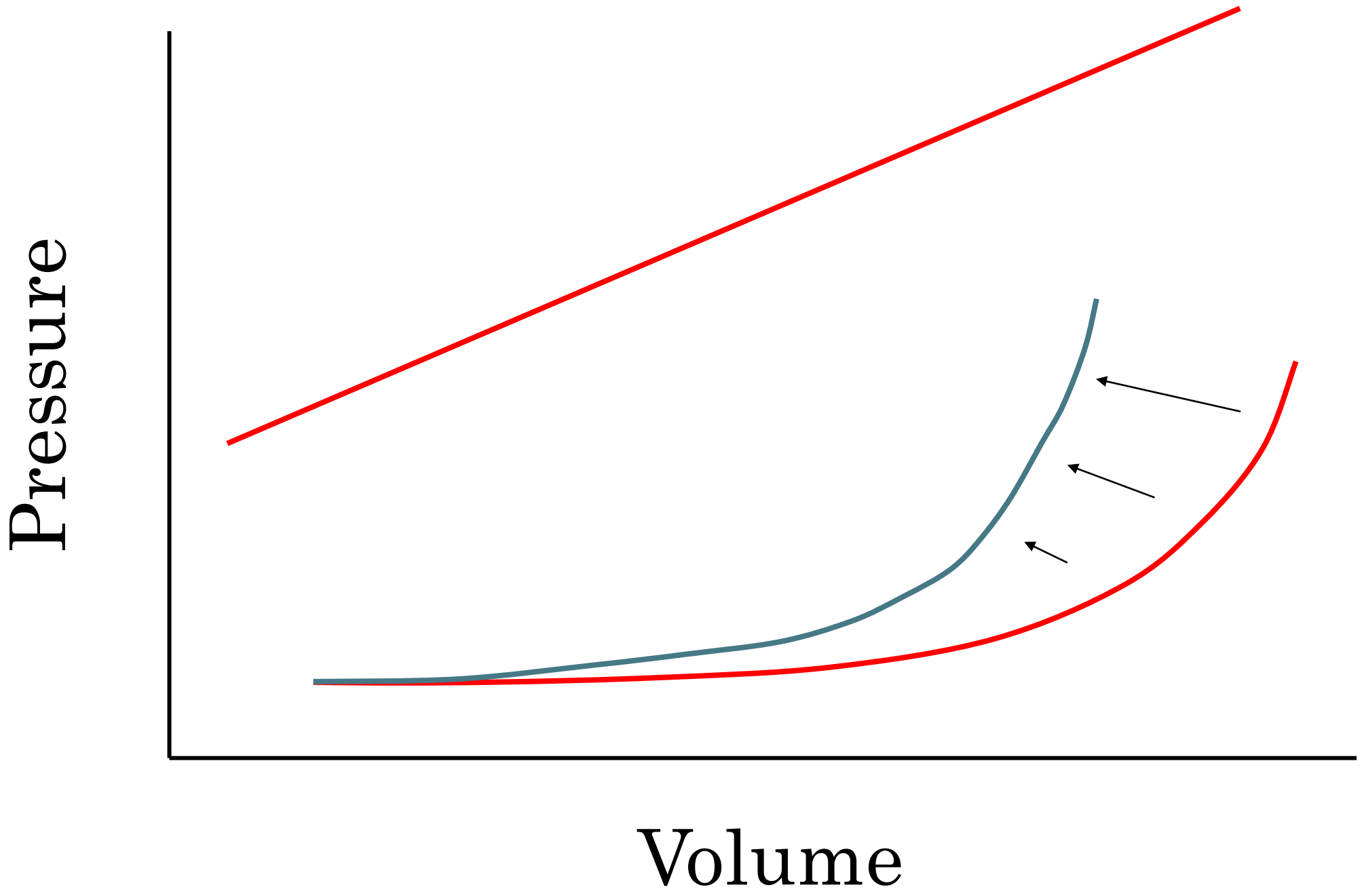
Now add the two lines that represent the end-systolic and end-diastolic pressure-volume relationships


End-systolic pressure-volume relationship
Pressure
End-diastolic pressure-volume relationship
Volume

Question

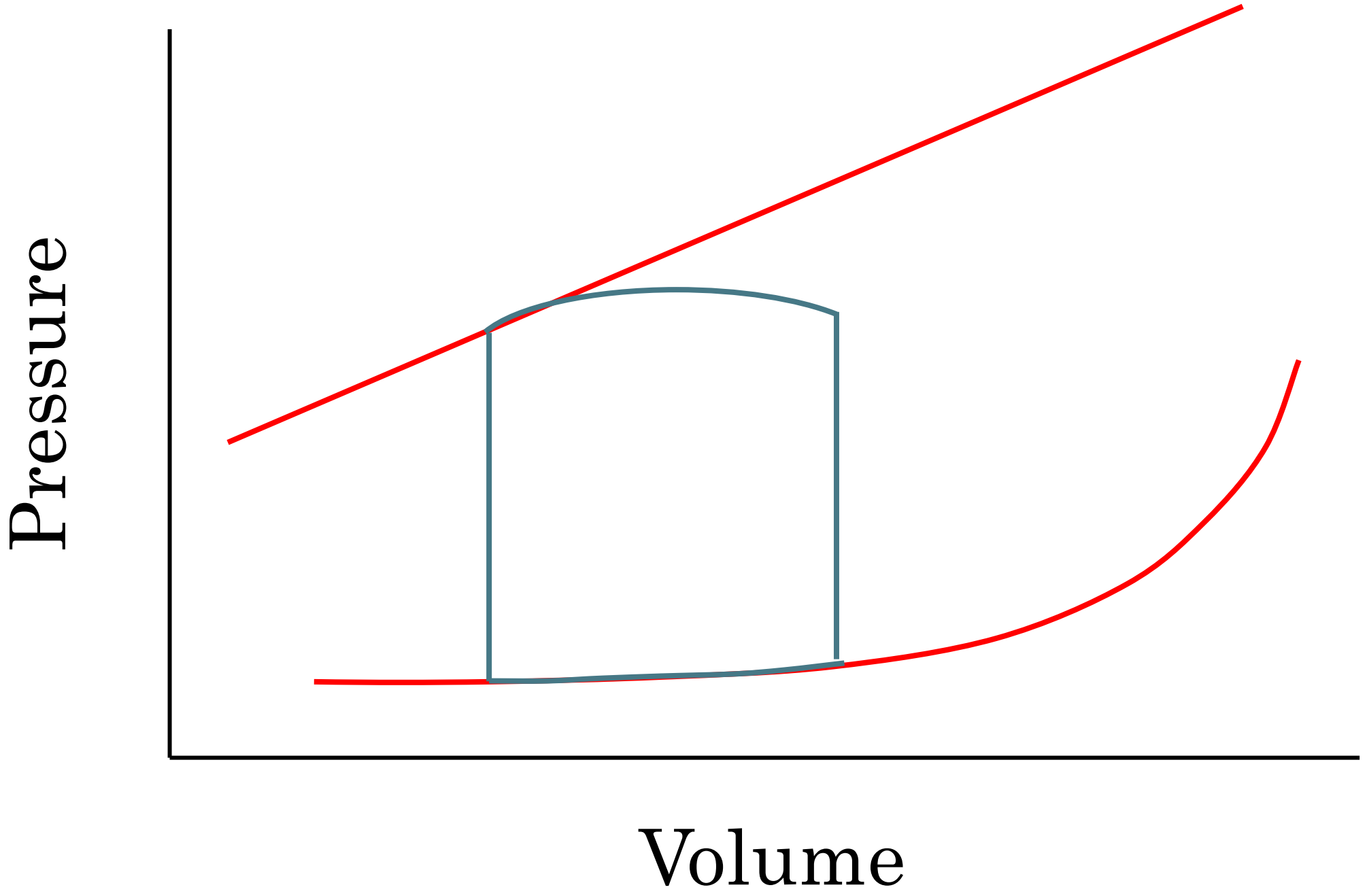
You are taking care of IDM with LV hypertrophy. You have a medical student rounding with you, who curiously asks you about P-V relationship for this patient.

Edit the drawing to exhibit what happens in the setting of diastolic dysfunction


Pressure
Volume

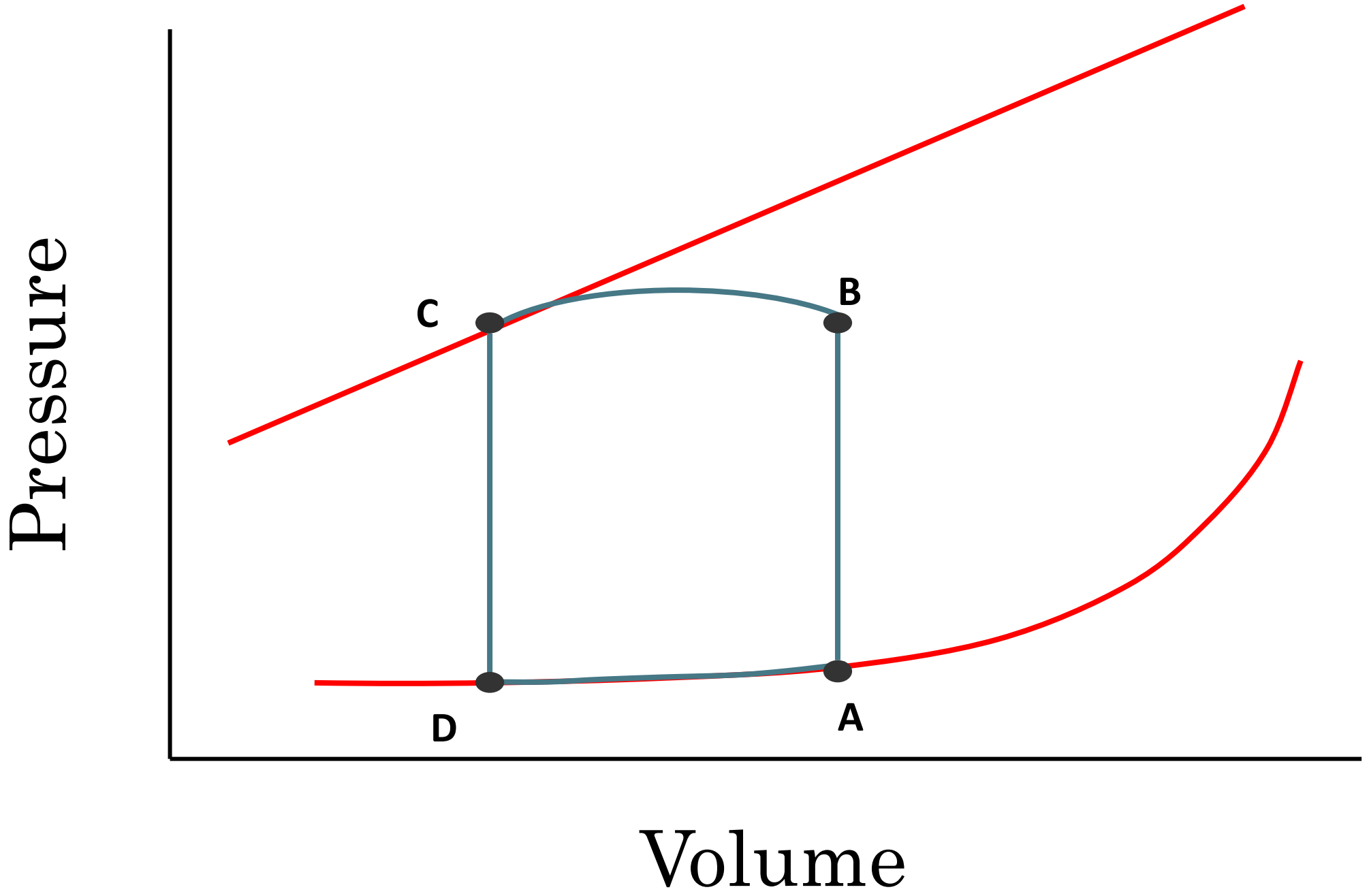
Question

Starting with the normal pressure volume relationship lines drawn in for diastole and systole, construct a normal pressure-volume loop


Pressure
Volume

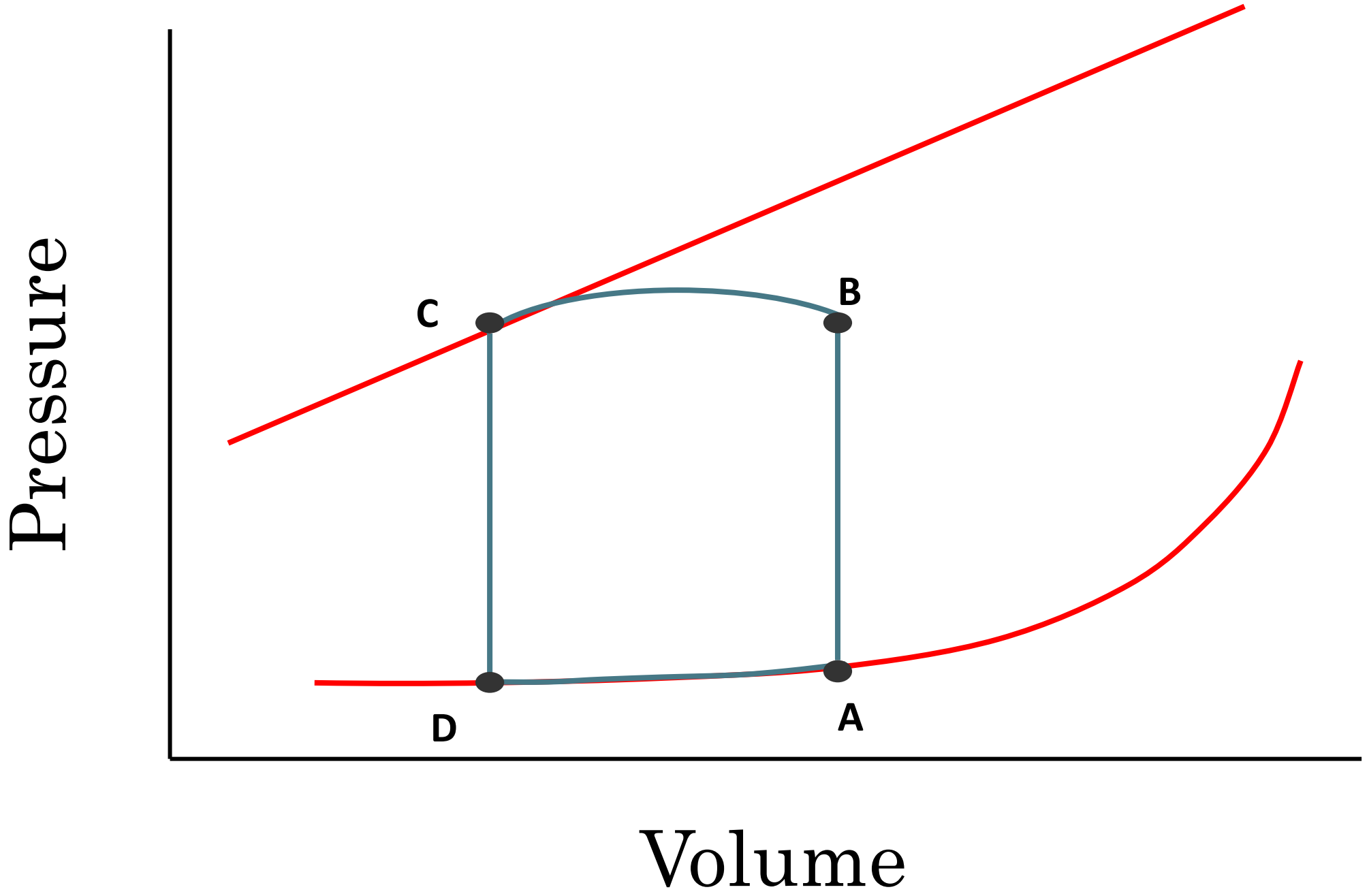
Question

On the following pressure-volume loop, which letter represents end-systole?


Pressure
C
B
D
A
Volume

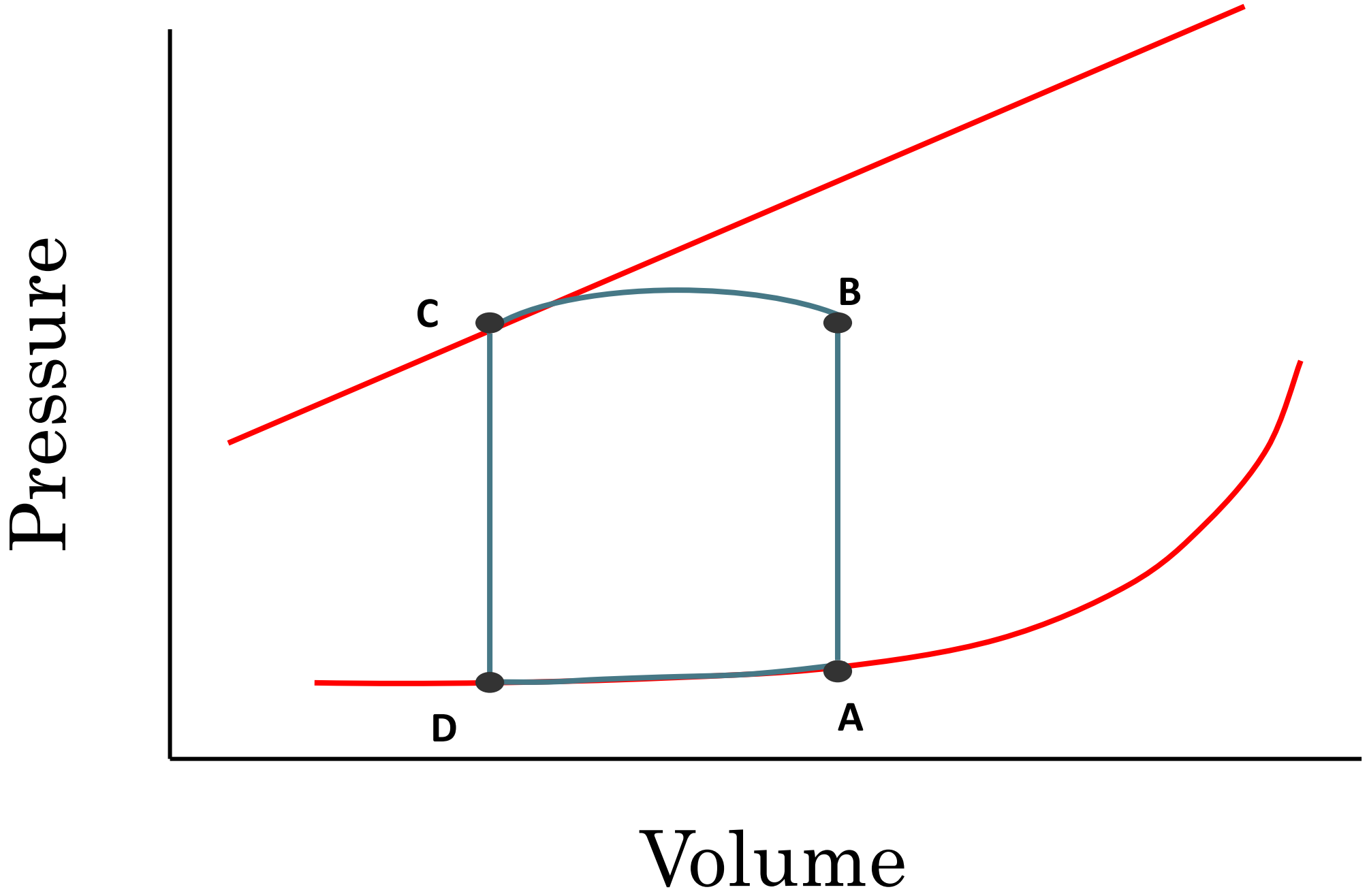
Question

On the following pressure-volume loop, which letter represents end-diastole?


Pressure
C
B
D
A
Volume

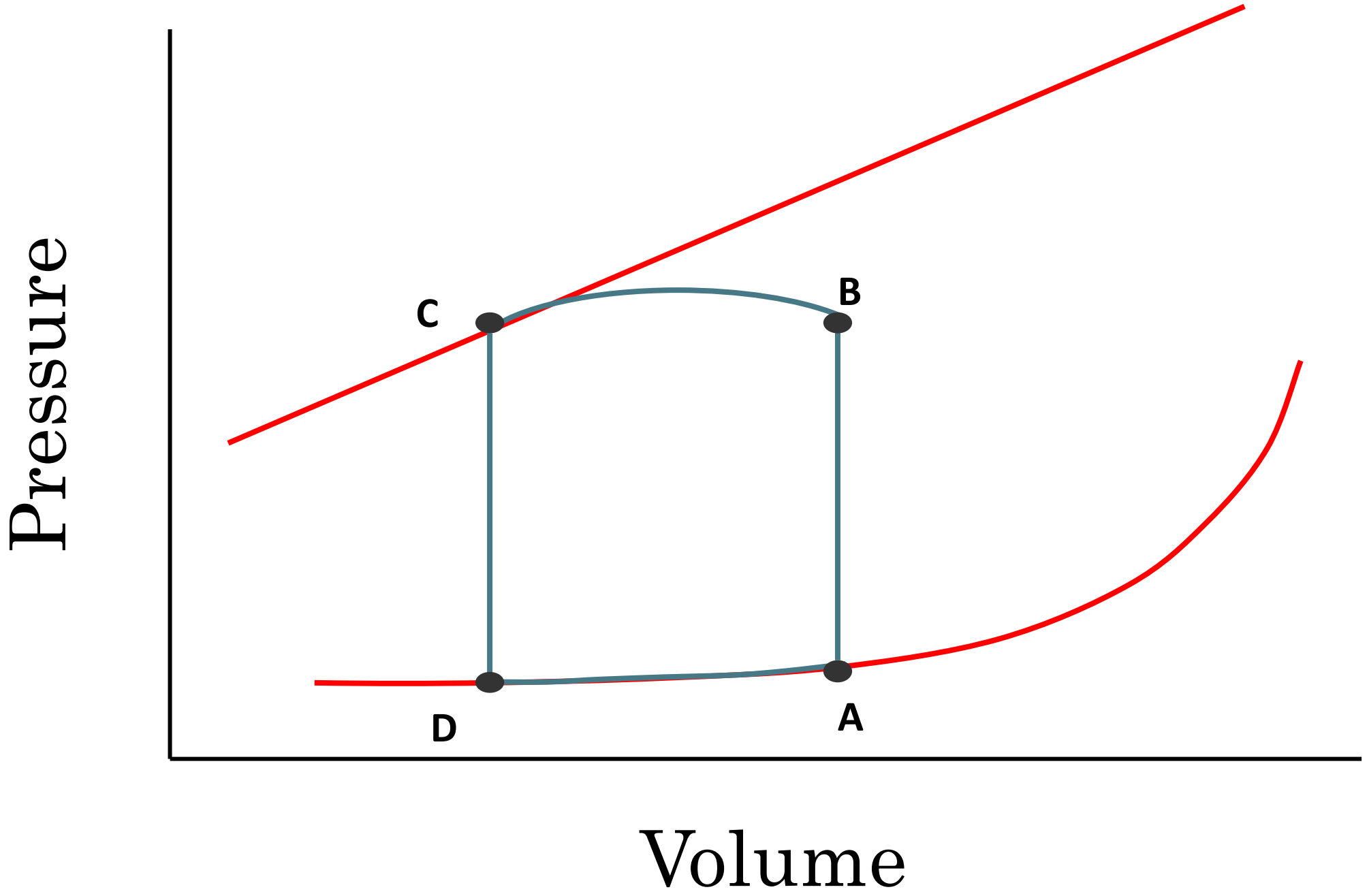
Question

On the following pressure volume loop, isovolumic contraction is represented by what segment?


Pressure
C
B
D
A
Volume

Question

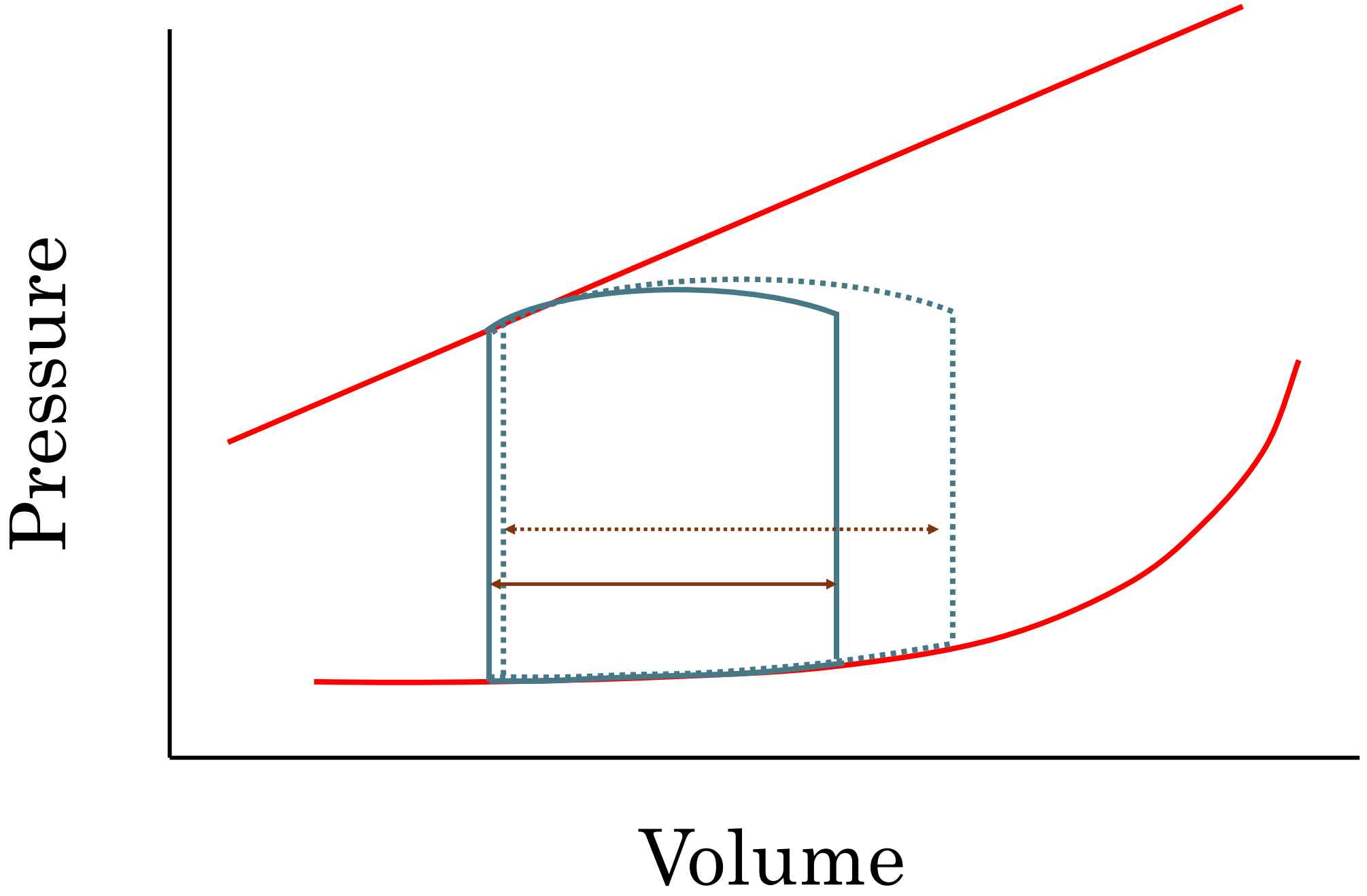
On the following pressure-volume loop, isovolumic relaxation is represented by what segment?


Pressure
C
B
D
A
Volume

Question

You are taking care of neonate with VSD. The medical student is highly impressed with your knowledge and asks you to draw the PV loop for this patient.

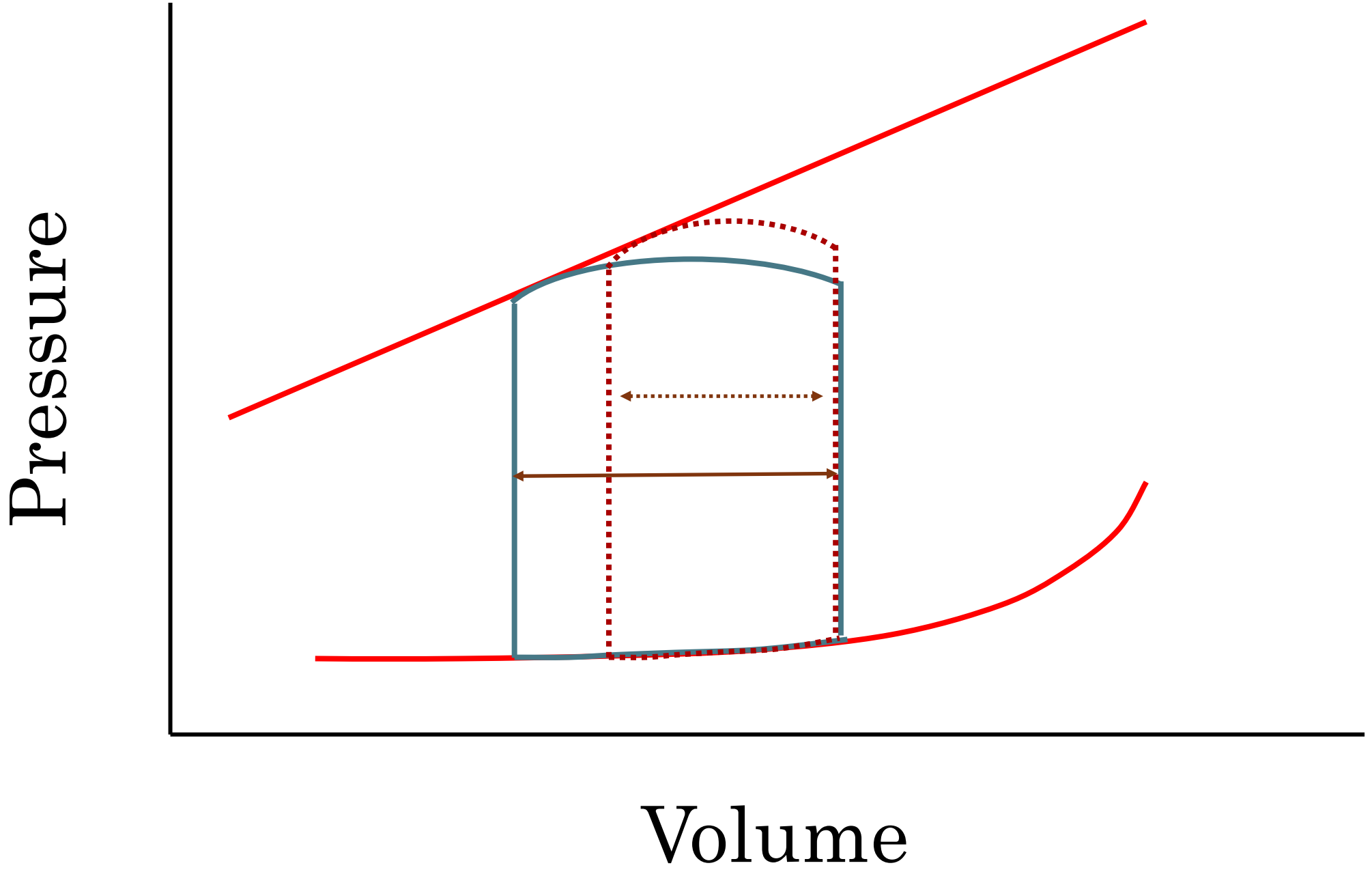
Starting with a normal pressure volume loop, how does the curve shift in the setting of increased preload?


Pressure
Volume

Question

You are taking care of neonate with Aortic Stenosis. The medical student is highly impressed with your knowledge and asks you to draw the PV loop for this patient.

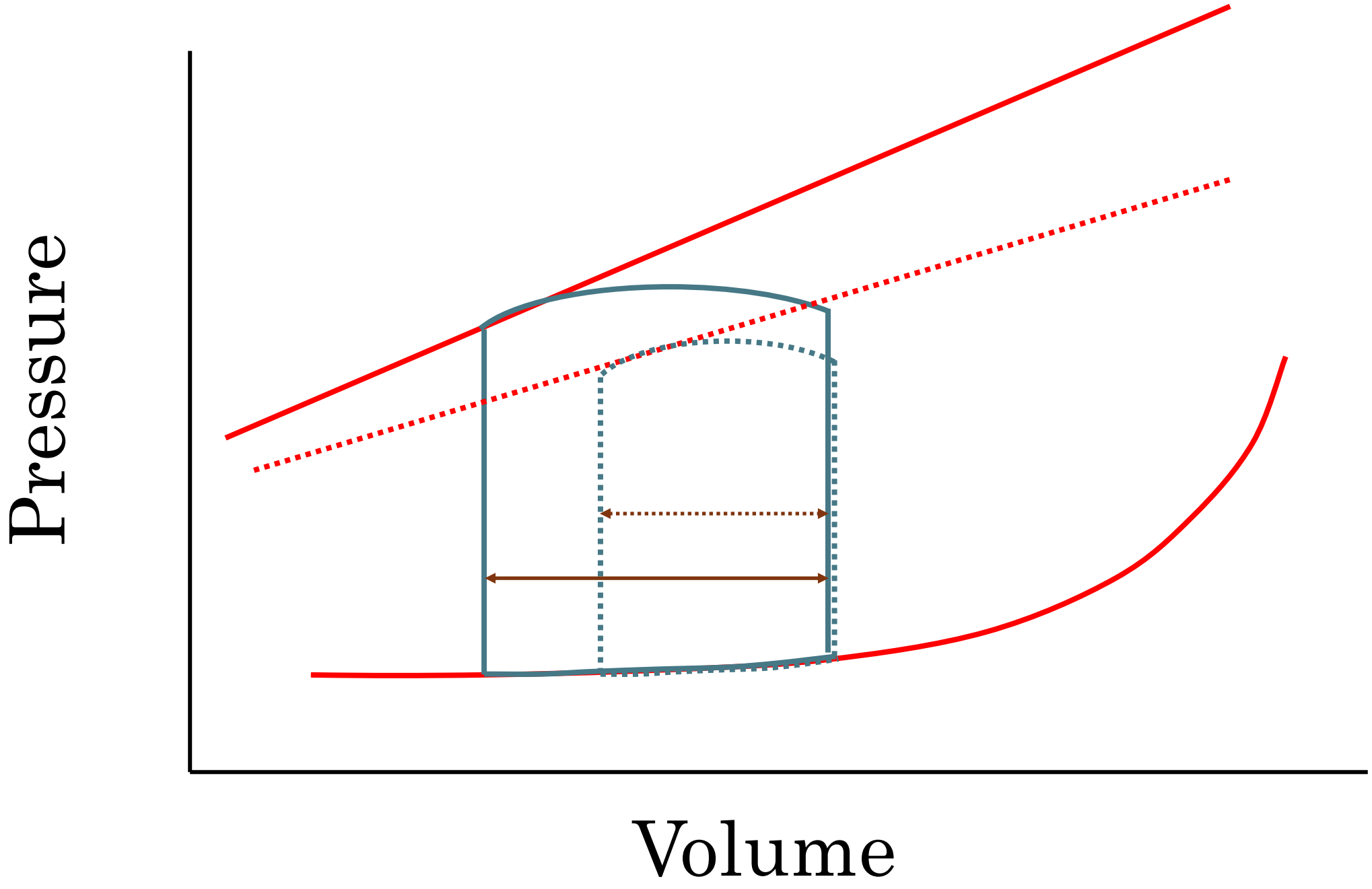
Starting with a normal pressure volume loop, how does the curve shift in the setting of increased afterload?


Pressure
Volume

Question

You correctly draw the loop for previous patient, increasing the excitement of your medical student. He then asks you to draw the loop for your myocarditis patient.

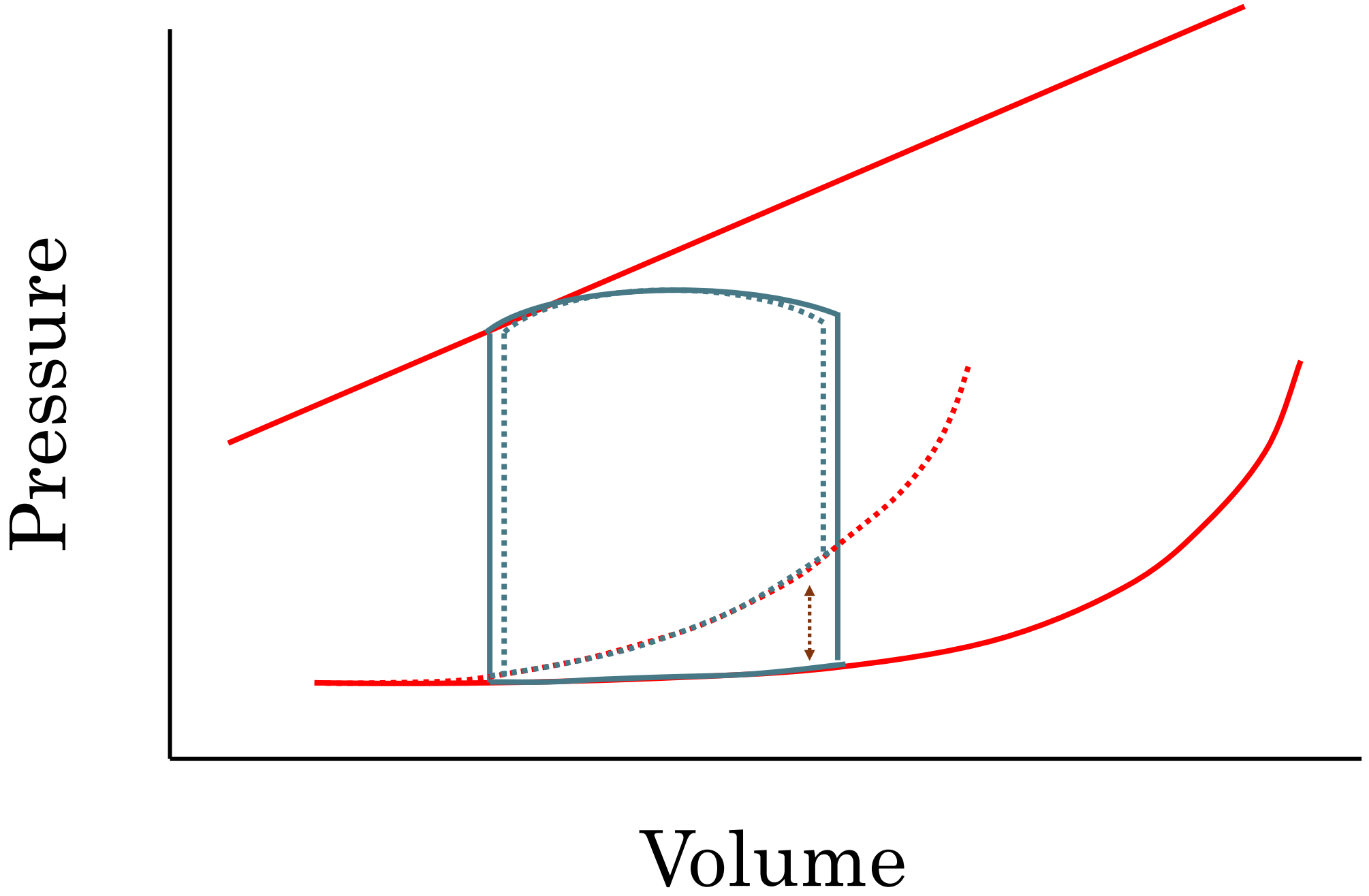
Starting with a normal pressure-volume loop, how does the curve shift in the setting of decreased myocardial contractility?


Pressure
Volume

Question

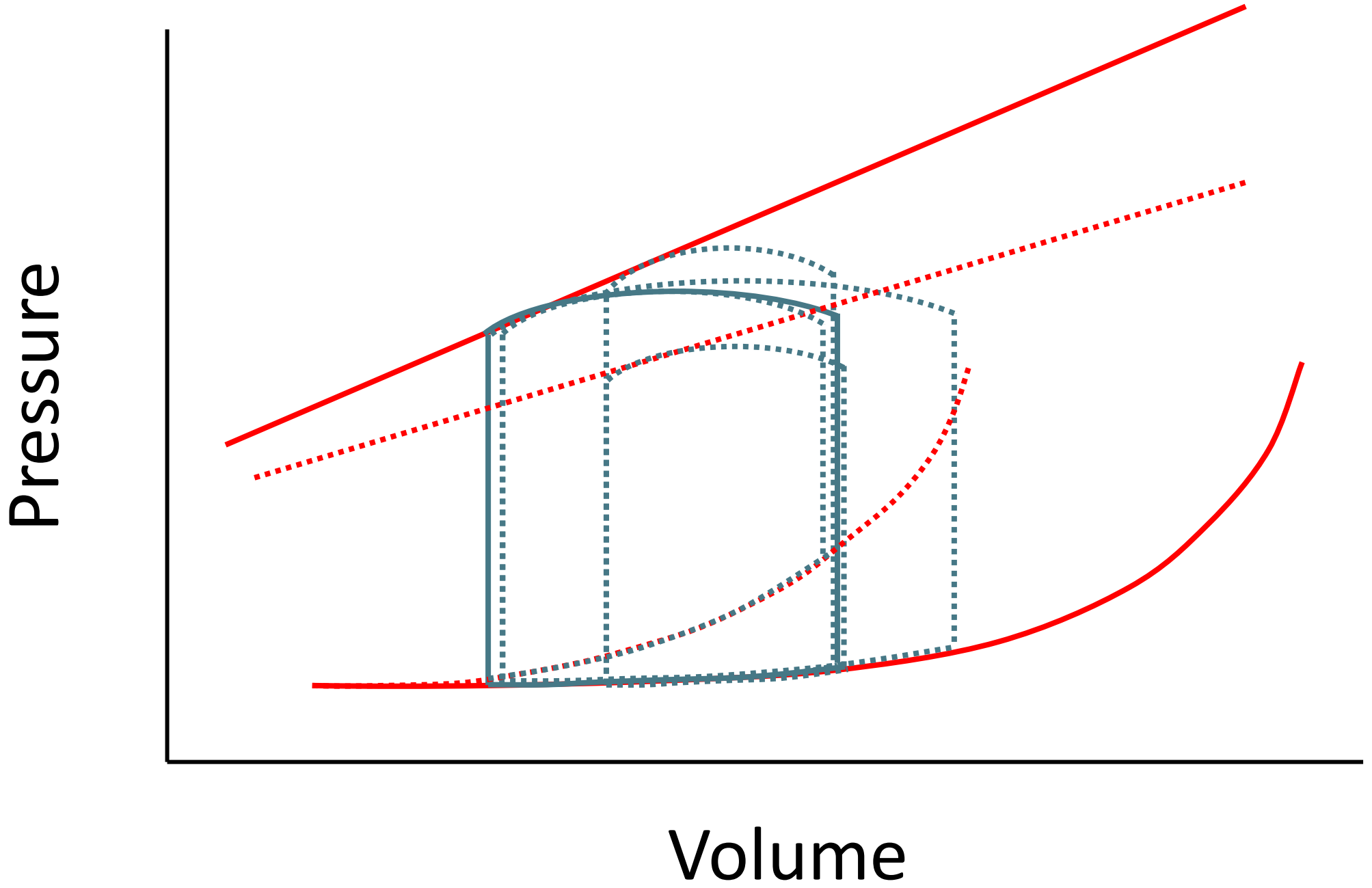
Continuing with his inquisitiveness, he asks you to draw the PV loop for IDM with LV hypertrophy.

Starting with a normal pressure-volume loop, how does the loop shift in the setting of diastolic dysfunction?


Pressure
Volume

KEY CONCEPTS: Cardiac Pressure-Volume Loops

- Changes in the functional performance of the myocardium for any reason result in a SHIFT IN EITHER THE SYSTOLIC OR DIASTOLIC FUNCTION CURVES, with a corresponding alteration in the pressure volume loop
- Changes in conditions outside of the myocardium, such as alterations in volume status or afterload, result in no change in the function curves but instead lead to alterations in the pressure volume loop along the SAME FUNCTION CURVES


Pressure
Volume

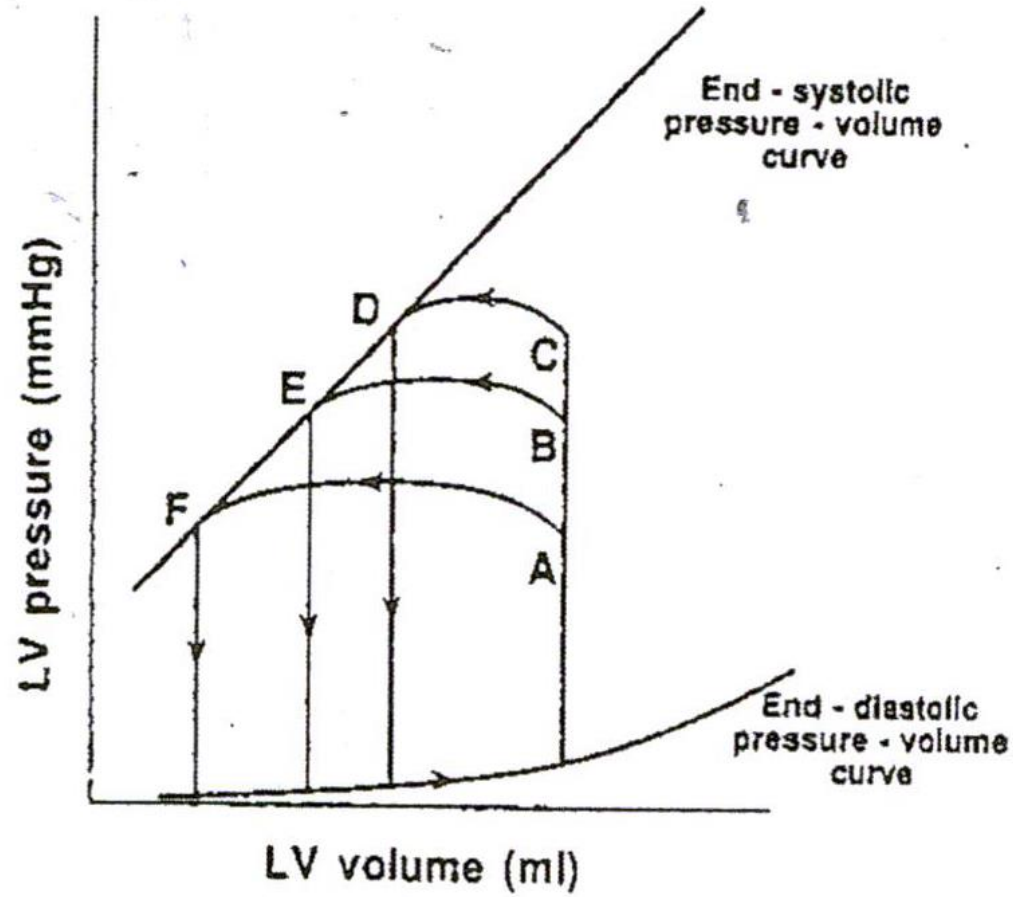


FIGURE 10.11. Schematic pressure-volume loops demonstrating the effect of increasing afterload on stroke volume during normal contraction when preload and contractility are held constant. As afterload increases, stroke volume diminishes. The points D, E, and F at end ejection describe a line known as the end-systolic pressure-volume relationship, the slope of which is used as a load-independent index of contractility.

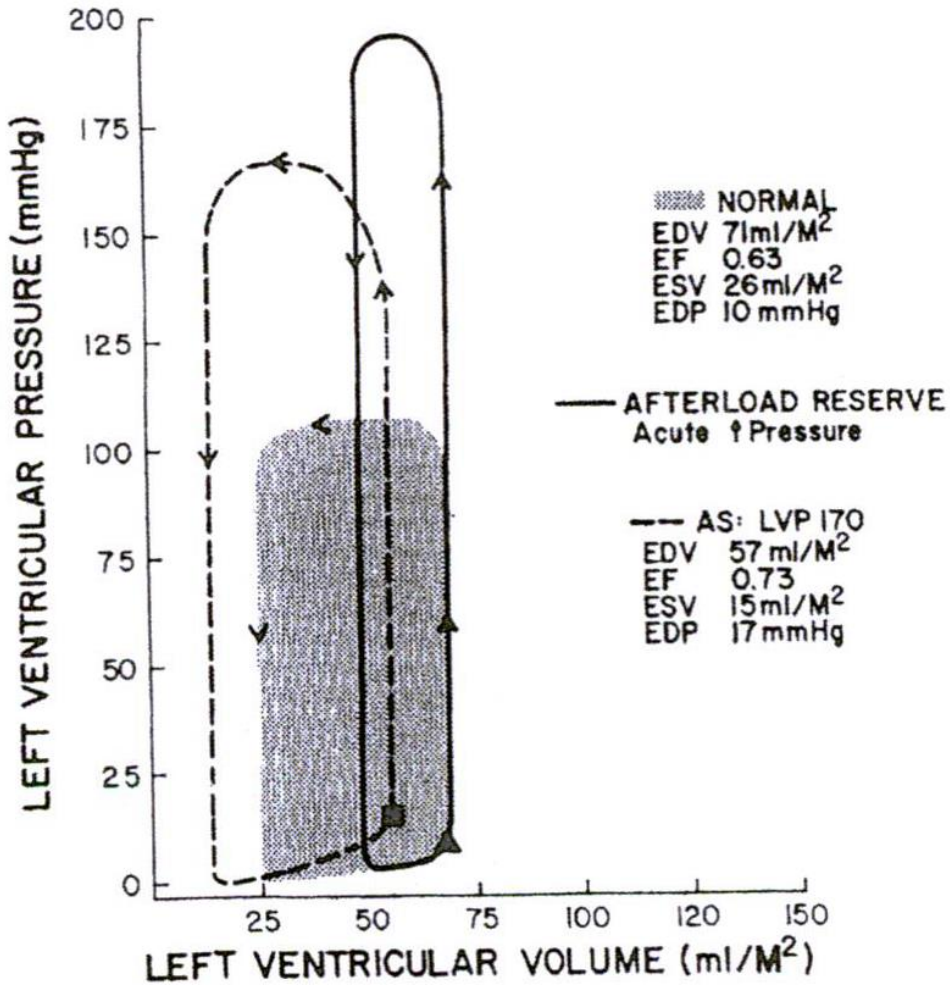


FIGURE 9. *Schematic pressure–volume loops for a normal 10-year-old, illustrating concepts of acute and chronic pressure overload. AS, aortic stenosis; EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; EDP, end-diastolic pressure; LVP, left ventricular pressure.*

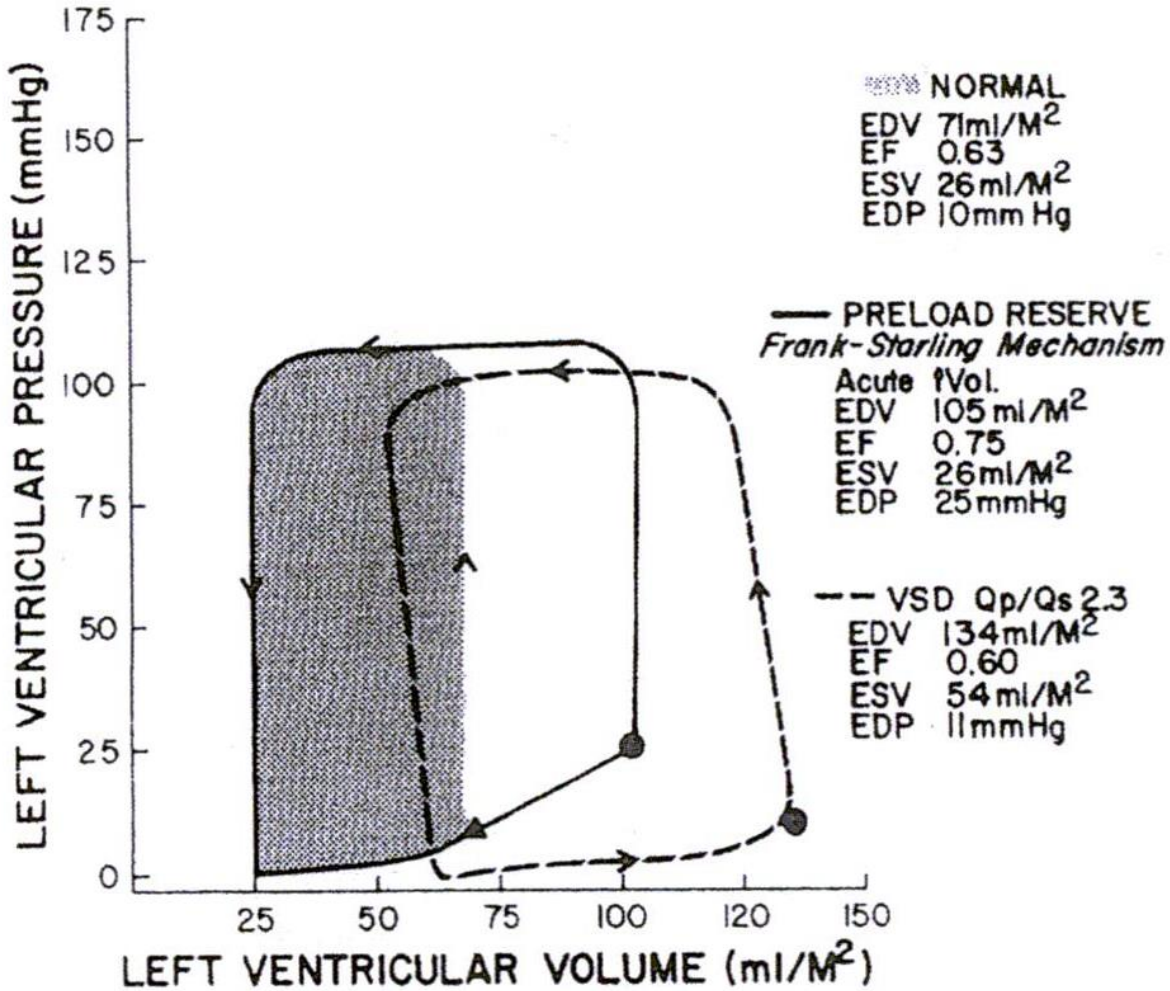


FIGURE 1. *Normal pressure–volume relations contrasted with preload reserve and chronic volume overload secondary to large ventricular septal defect (VSD). EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; EDP, end-diastolic pressure.*

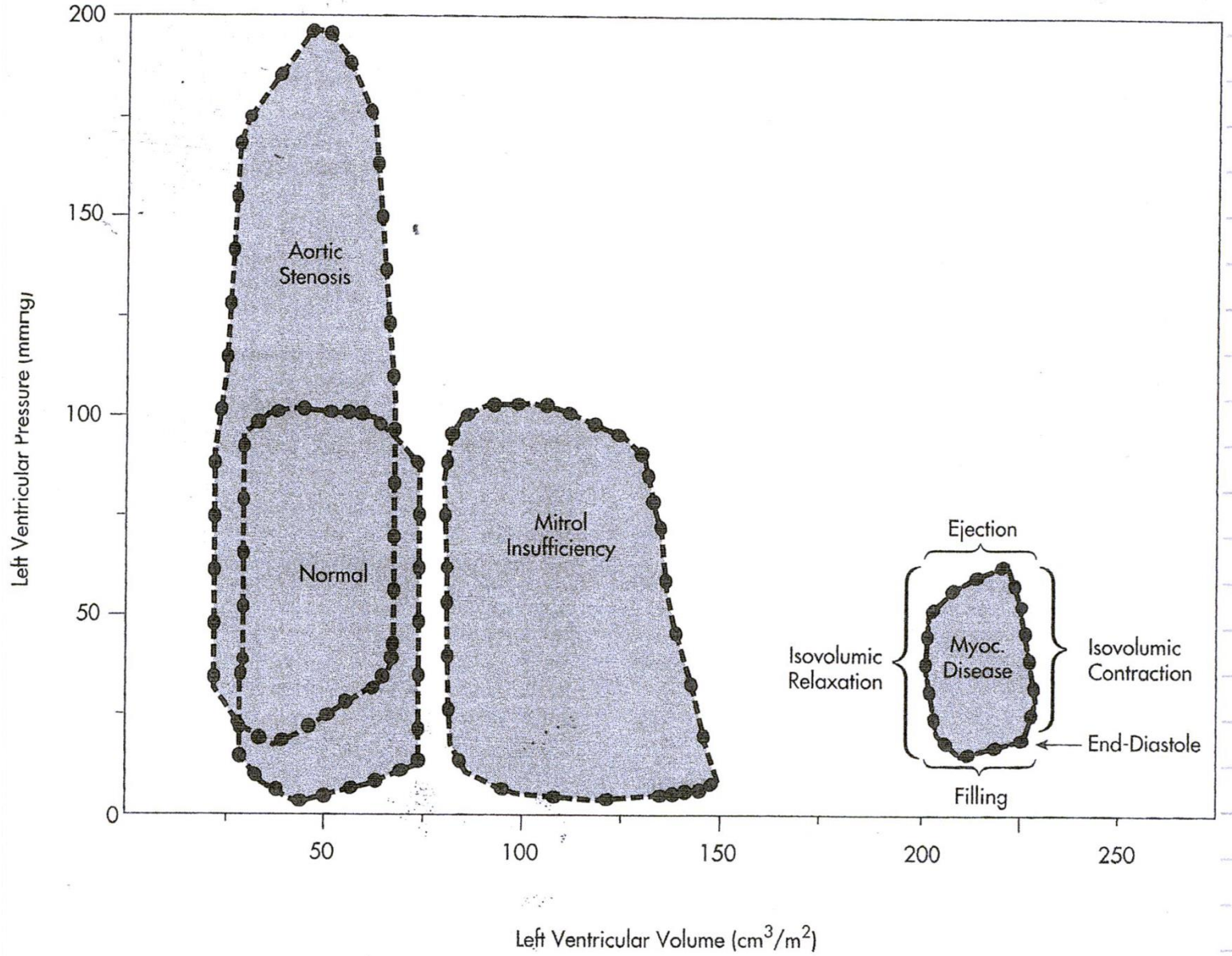
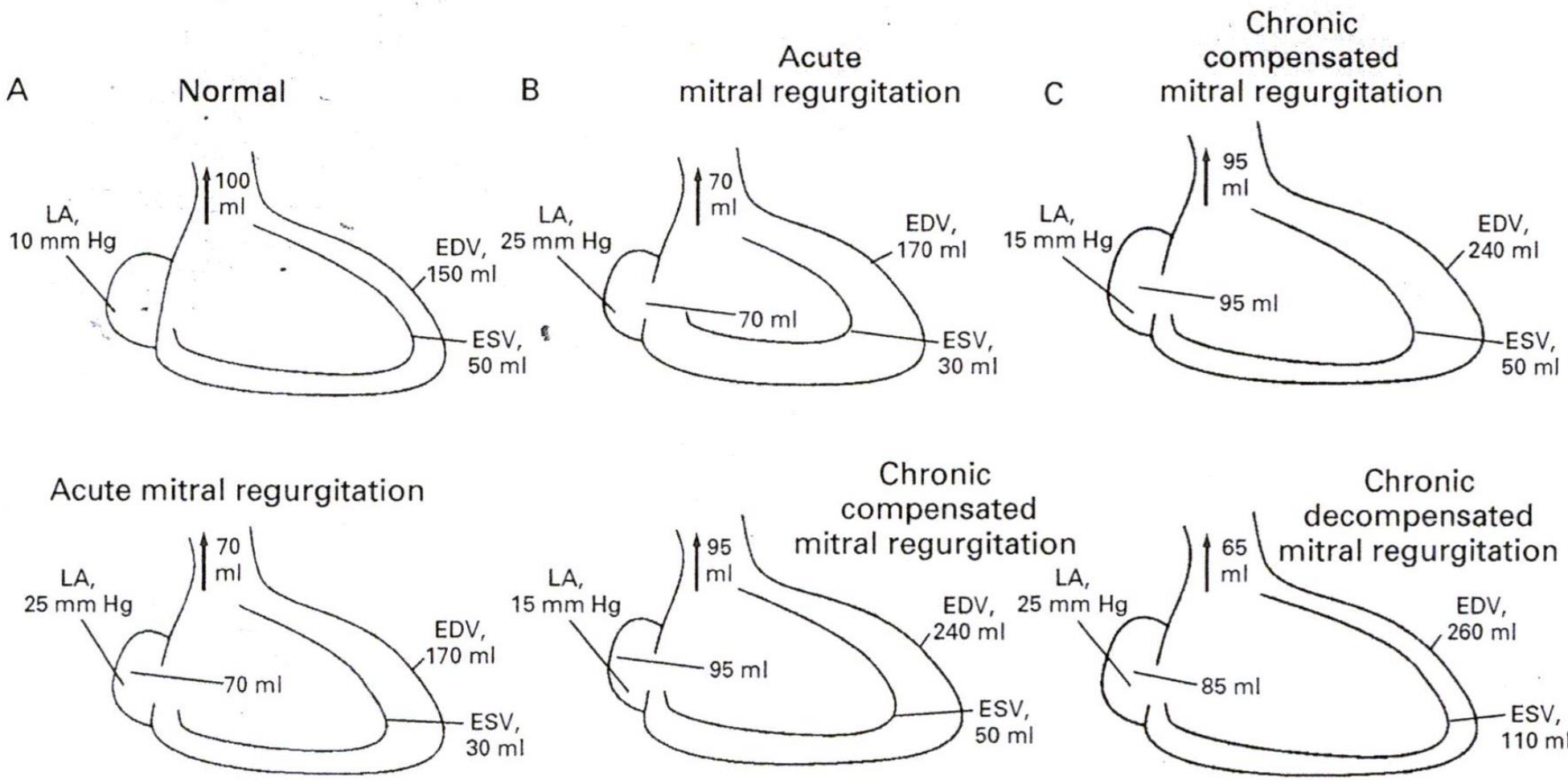


Fig. 2-9. Pressure-volume loops for children with various congenital heart diseases. Myocardial disease, volume overload (mitral insufficiency), and pressure overload (aortic stenosis) are demonstrated. (From Graham TP Jr, Jarmakani MM: Evaluation of ventricular function in infants and children, *Pediatr Clin North Am* 18:1109-1132, 1971; with permission.)



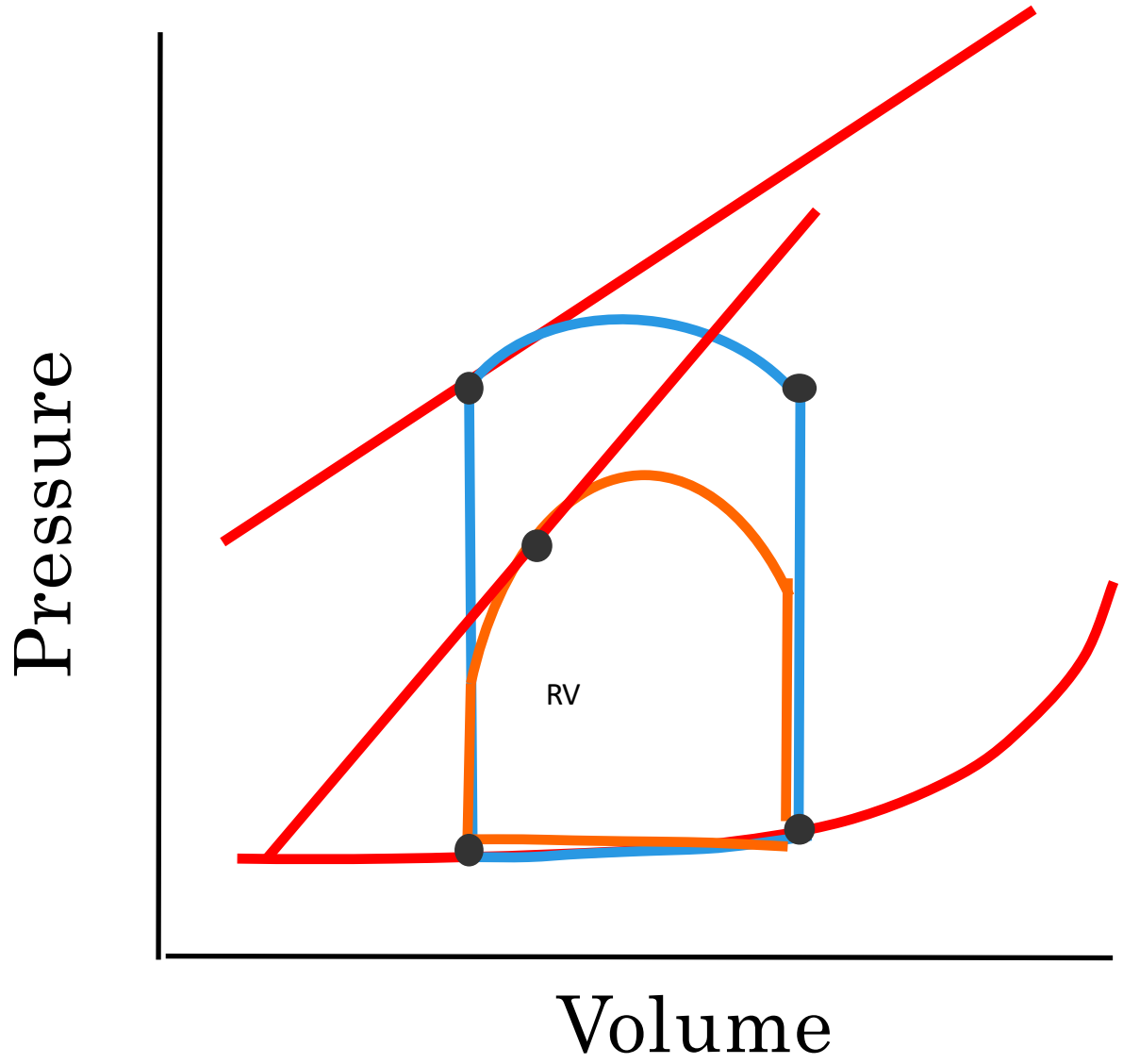
STAGE	PRE-LOAD SL μm	AFTER-LOAD ESS kdyn/cm²	CF	EF	RF	FSV ml
Normal	2.07	90	N	0.67	0.00	100
AMR	2.25	60	N	0.82	0.50	70

STAGE	PRE-LOAD SL μm	AFTER-LOAD ESS kdyn/cm²	CF	EF	RF	FSV ml
AMR	2.25	60	N	0.82	0.50	70
CCMR	2.19	90	N	0.79	0.50	95

STAGE	PRE-LOAD SL μm	AFTER-LOAD ESS kdyn/cm²	CF	EF	RF	FSV ml
CCMR	2.19	90	N	0.79	0.50	95
CDMR	2.19	120	↓	0.58	0.57	65

Figure 3. Pathophysiologic Stages of Mitral Regurgitation.

- # EXPLAIN THE DIFFERENCES BETWEEN THE LV AND RV PRESSURE VOLUME LOOPS



The LV is a square loop with ejection ending at the onset of isovolumetric relaxation (D)

The RV is significantly smaller because of low PVR, increased compliance and lower work output. It is triangular shaped with less concrete isovolumetric relaxation periods with ejection continuing into diastole

Heart Failure

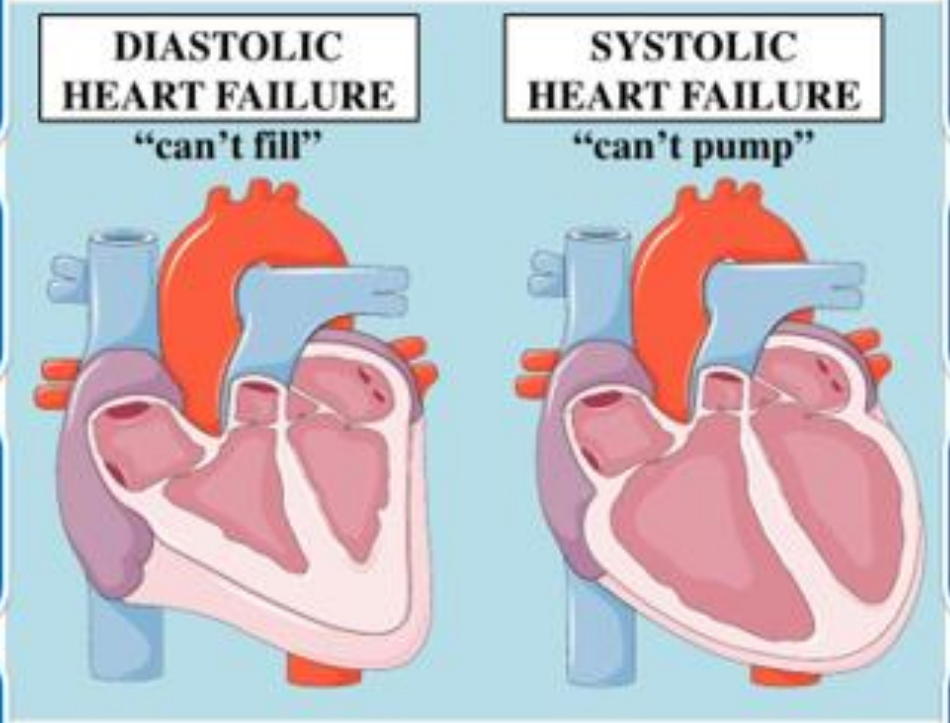
Systolic vs. Diastolic

ventricle unable to relax normally

heart can't properly fill with blood during diastole

leads to fluid backing up into lungs

preserved ejection fraction



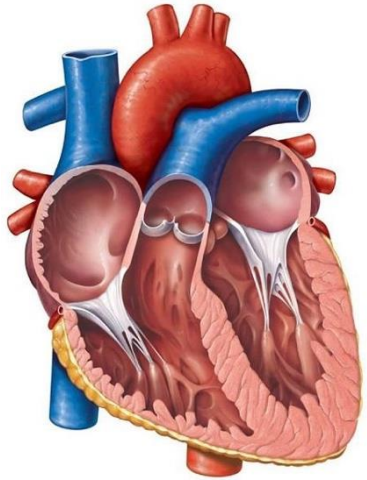
ventricle unable to contract normally

can't pump with enough force to provide adequate blood into circulation

eventually causes low cardiac output

ejection fraction decreased

Heart Failure
Right vs. Left



Right heart failure

Systemic congestion

- hepatomegaly
- peripheral edema
- weight gain
- jugular venous distension
- pleural effusion

Left heart failure

Decreased cardiac output

- tachycardia
- gallop
- diaphoresis
- decreased perfusion
- fatigue
- poor feeding

Pulmonary congestion

- tachypnea
- cyanosis
- respiratory distress
- pulmonary edema
- wheezing
- cough

Heart Failure: Management

Improve Cardiac Function	Fluid Management	Decrease Metabolic Demands	Improve Tissue Oxygenation
Oral medications • ACE inhibitors • Beta blockers • Digoxin • Iron supplementation **Vasoactive infusions** • Milrinone • Epinephrine • Dopamine Manage HTN Reduce afterload	• Diuretics • Strict I&O • Monitor electrolytes closely • Fluid and sodium restriction • Max concentrate calories	• Conservation of energy • Normothermia • NG feeds	• Reduce work of breathing • Treat anemia • Possibly O2 or other respiratory support • Mechanical ventilation

GOALS OF THERAPY

- Relieve symptoms
- Restore cardiac performance
- Improve quality of life
- Reduce cardiovascular (CV) hospitalizations
- Slow/reverse heart failure progression
- Improve/prolong patient survival

Inotropic drugs: digoxin, milrinone
Diuretics: chlorothiazide, furosemide
Vasodilators: hydralazine, nitrate
Ivabradine

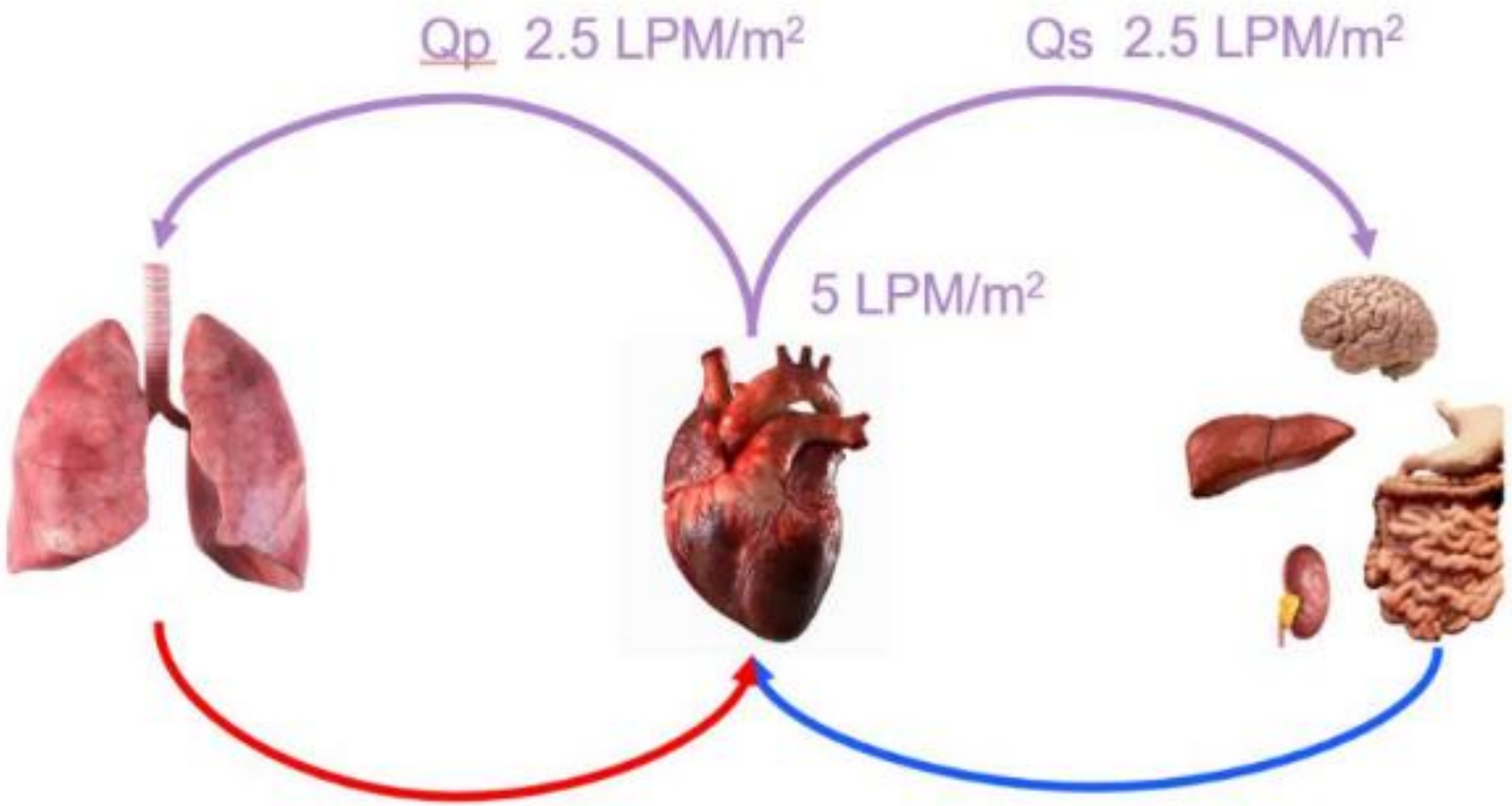
ACE-I/ARB/ARNI

Beta Blockers

Mineralocorticoid receptor antagonist (MRA)

SGLT-2 inhibitors

Pulmonary over circulation



Pulmonary Over circulation- Physiology

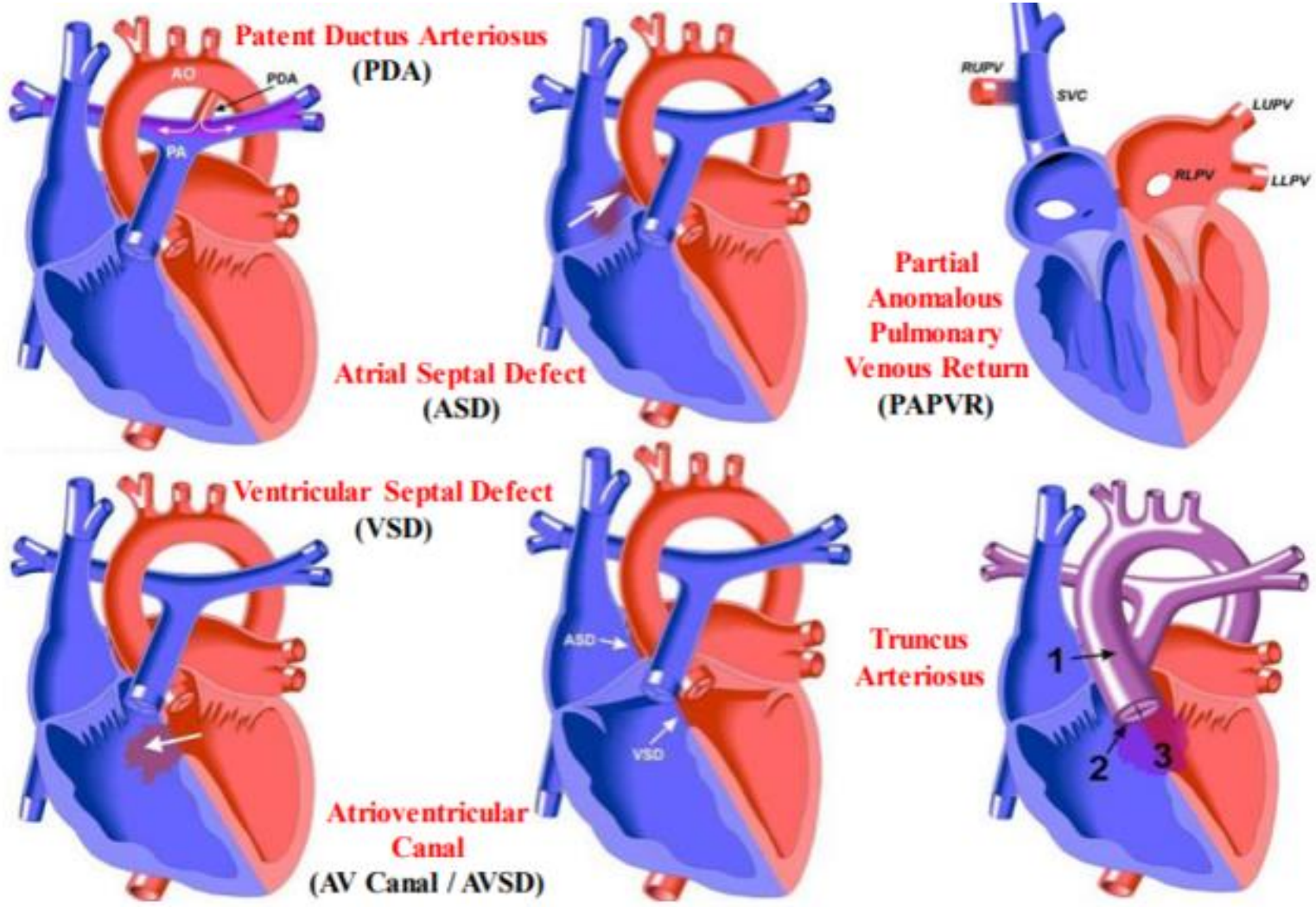
Determinants of shunt flow:

- Shunt size (defect diameter, duct size)
- Pressure gradient between chambers or vessels
- Resistance
- PVR
    - Falls after birth (key physiologic trigger)
    - Rises with hypoxia/acidosis

Too much blood flow to the lungs (Qp↑)

- Left-to-right shunt
- Lungs get flooded with flow → pulmonary edema/congestion
- Heart works harder → tachycardia, cardiomegaly, heart failure
- Systemic output can drop → poor perfusion, poor growth

Pulmonary Over circulation: Lesions



Pulmonary Over circulation: Clinical Presentation

- Tachypnea at rest
- Subcostal retractions, nasal flaring
- Diaphoresis, poor effort, fatigue, or vomiting with feeding
- Frequent “chest infections” history

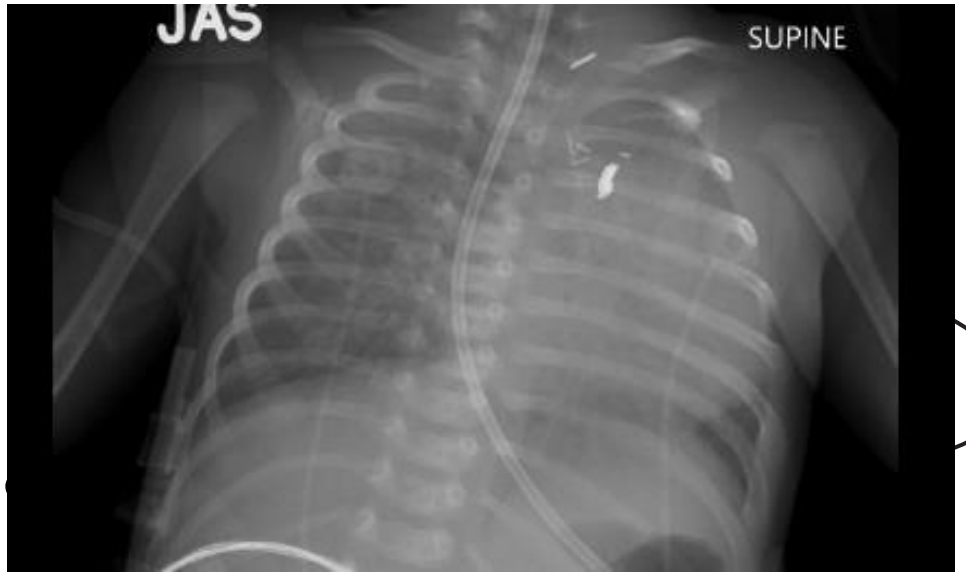
CHD > Primary lung disease

- Failure to thrive
- Hepatomegaly
- Hyperdynamic precordium
- Murmur (maybe)
- Wide pulse pressure (PDA)
- Single loud S2 (Pulmonary hypertension)

Pulmonary Over circulation: Diagnostics

- At minimum:
  - Careful exam and feeding history
  - Oxygen saturation
  - Weight/growth

- If available
  - CXR: Cardiomegaly, prominent vascular markin
  - ECG: Enlarged chambers (Not definitive)
  - Echo: Confirm anatomy, estimates pressures
  - BNP



Isn’t oxygen a good thing?!

Oxygen:

- ↓ PVR via pulmonary vasodilation
- ↓ Pulmonary arteriolar tone
- ↑ Pressure gradient from systemic → pulmonary circuit
- ↑ Qp → ↑ pulmonary edema

- Give oxygen only if hypoxemic
- If sats are normal and patient is tachypneic oxygen won’t fix it (heart failure)

Pulmonary Over circulation: Management

- Reduce pulmonary congestion
  - Diuretics
  - Careful O2 use
- Support systemic output (Qs)
- Improve nutrition/growth
- Plan definitive repair/closure

When is pulmonary over circulation dangerous?

- When it's at the expense of systemic perfusion
    - Poor perfusion, cool extremities, rising lactate, metabolic acidosis

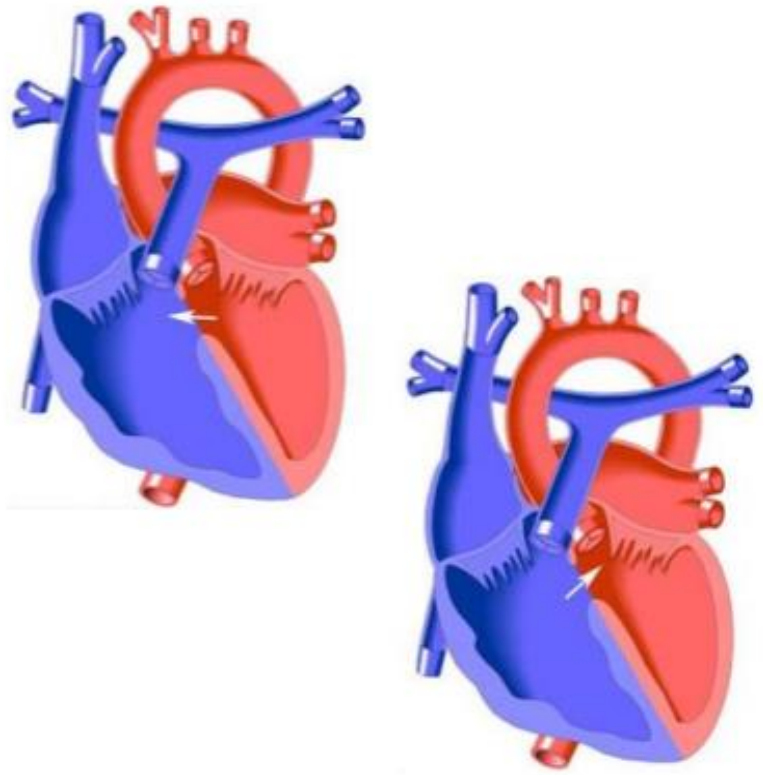
- Apnea
- Altered mental status
- Severe malnutrition
- Recurrent admissions “pneumonia”
- Decreased murmur sound + louder S2

Eisenmenger's Syndrome

- 1897- 32yr w/ exercise intolerance, cyanosis, hemoptysis/death
  - Autopsy revealed Lg VSD with overriding Aorta
- Unrepaired/untreated L-R shunts
  - Lg VSD/PDA ~50% develop PAH in childhood
  - Lg ASD- ~10% progress to PAH after 3rd decade of life
  - Truncus, AVSD- ~100% develop severe PHTN by age 2
- Survival depends on:
  - Preserved LV/RV function, valve function
  - End organ perfusion
  - Cyanosis associated with pulmonary vascular obstructive disease

Eisenmenger's Syndrome

- Increased PVR--> Persistent R-->L shunt= Eisenmenger's Syndrome
- Increased cyanosis and erythrocytosis/polycythemia
- Pathologic damage: Inflammation, cell proliferation, vasoconstriction, and fibrosis
- Irreversible remodeling of pulmonary microvasculature--> Obstruction to pulmonary blood flow (PVOD)



KEY CONCEPTS

- Blood flow is determined by pressure gradients
- A decrease in PVR will **increase** your PBF and **decrease** your SBF
    - Pulmonary overcirculation
- An increase in PVR will **decrease** your PBF and **increase** your SBF
    - Atelectasis, pulmonary infections, PAH
- Symptom recognition: Tachypnea + feeding intolerance+ poor growth+ hepatomegaly
- Management: diuretics + nutrition support + cautious Oxygen use
- Timely referral to prevent pulmonary vascular disease
    - Don't wait until a patient is cyanotic!

Questions???

THANK YOU!

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