Hemodynamic Monitoring

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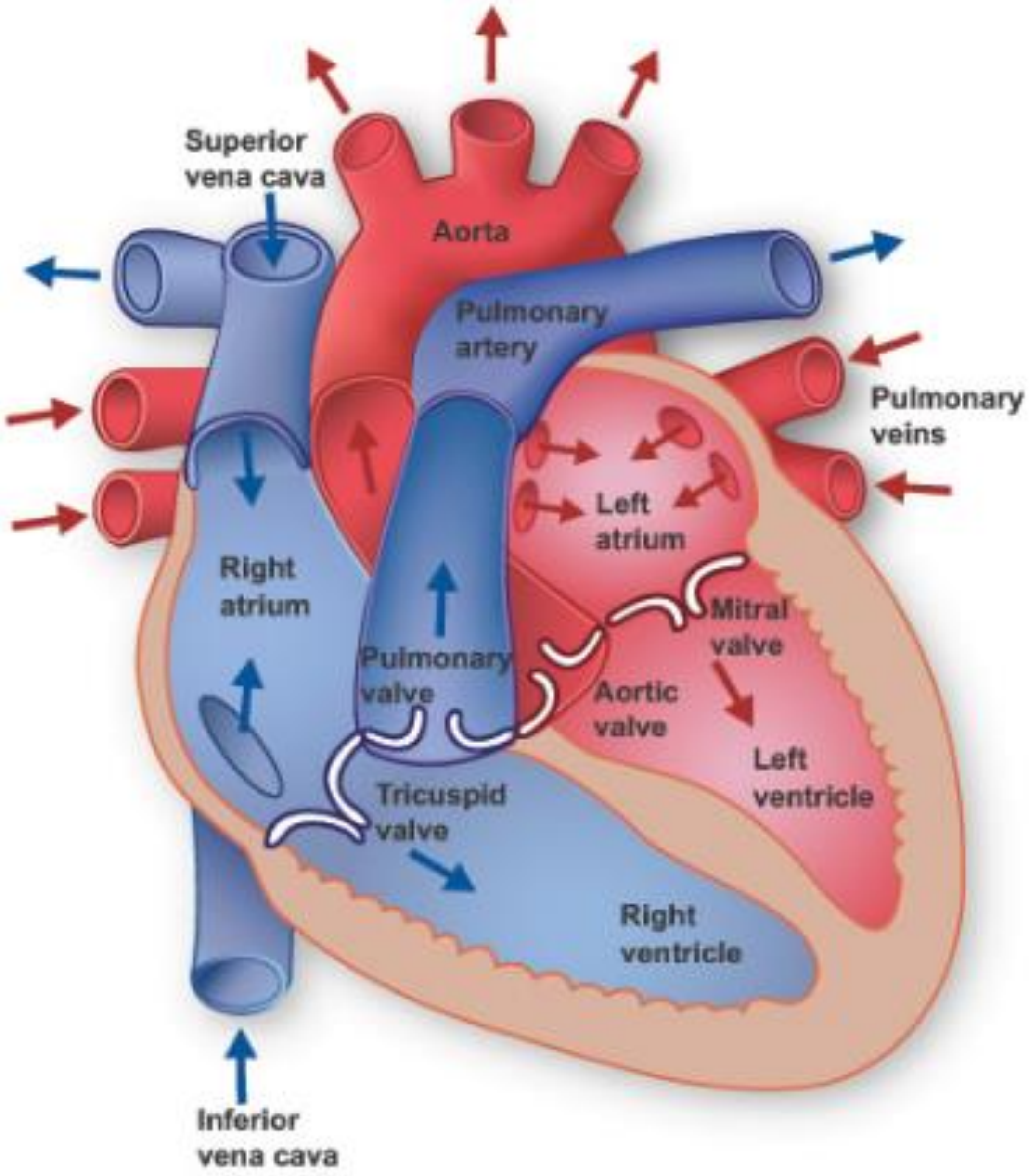
Co-Owner- A & T Lectures

Learning Objectives

- Describe the functional anatomy of the heart.
- Detail Measures of hemodynamic montoring and cardiac output,
- Review factors which can impact cardiac output and hemodynamics.
- Review interventions for hemodynamic abnormalities.
- Furnish Additional resources.

Functional Anatomy of the Heart



Cardiac Output

- The amount of blood pumped out of the ventricles in 1 min.
- Stroke Volume x Heart Rate = CO
- 60-130 ml/beat x 60-100bpm = 4-8 lpm at rest

Cardiac Index

- Determined by dividing the CO by body surface area
- Normal CI is 2.5 to 4.0 L/min/m^2
- CI measurement allows a standardized interpretation of the cardiac function

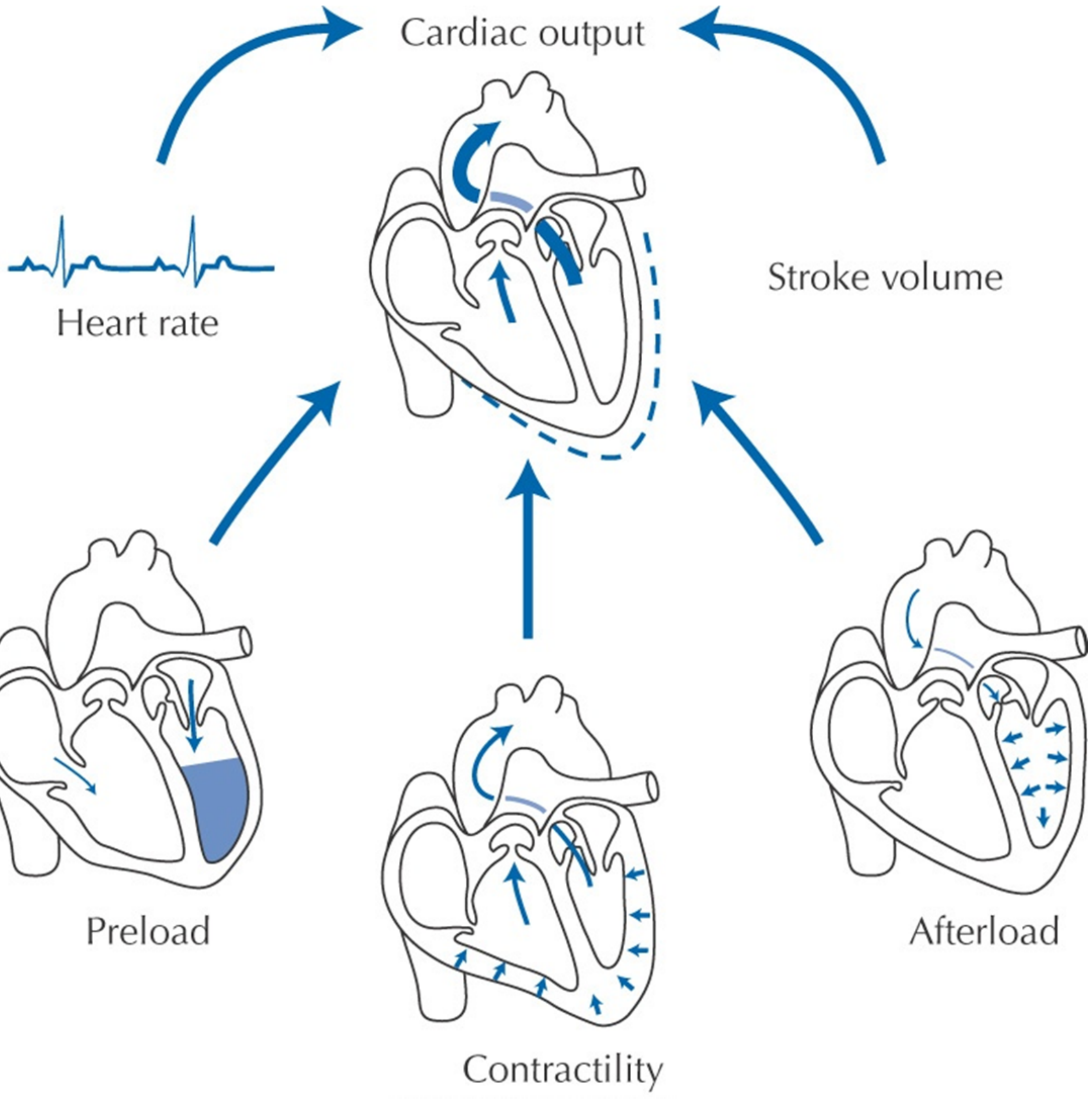
Ejection Fraction

- The fraction of end-diastolic volume ejected with each systole
- normally 65% to 70%; **drops with cardiac failure**

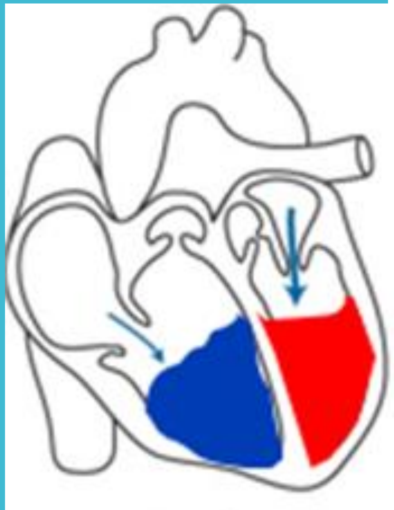
Heart Rate

- Compensatory mechanism: ⇓ HR ➔ ⇑ SV
- Ventricular filling and tachycardia
- Dysrhythmias
- HR is primarily determined by CNS
- CO is directly related to HR
  - HR > 160–180 is exception yielding…
    - Decreased EDV, EF, SV, & CO

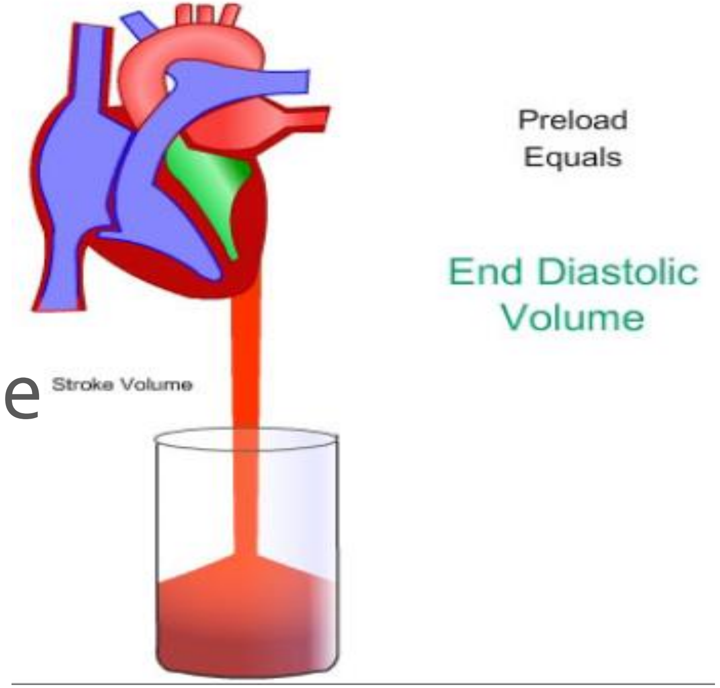
Cardiac Output Cycle



Preload



- Stretch on Ventricle Before Contraction
- Filling
- Venous Return
- Compliance
- End Diastolic Volume
  - Blood Left in Atria



Common Drugs that Effect Preload

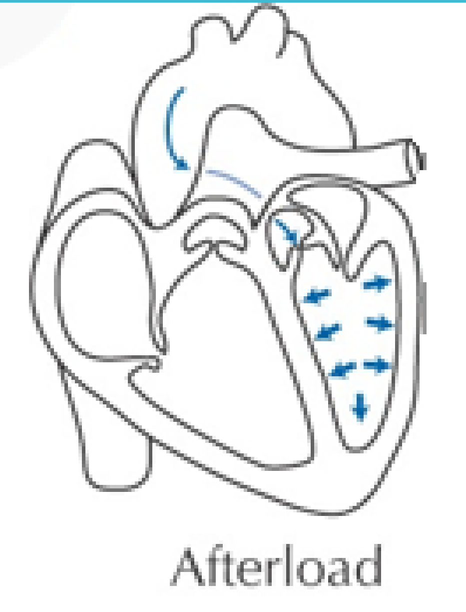
Reduces Preload

- Nitroglycerin
- Diuretics
  - Furosemide (Lasix)
- Morphine (Astramorph)

Increases Preload

- Fluids
- Blood Products
- Volume Expanders
  - Colloids
  - Crystalloids
    - D5W
    - Normal Saline
    - Ringers Lactate

Afterload



- Resistance to Ventricular Emptying
- Two Components:
  - Peripheral Vascular Resistance
  - Ventricular Wall Tension
- Increases with Vasoconstriction
- Increase in the oxygen demand
- Decreases with Vasodilation
  - Improves SV
  - Issues with BP if low volume
- Blood Viscosity
- Negative Intrathoracic Pressure

Common Drugs that Effect Afterload

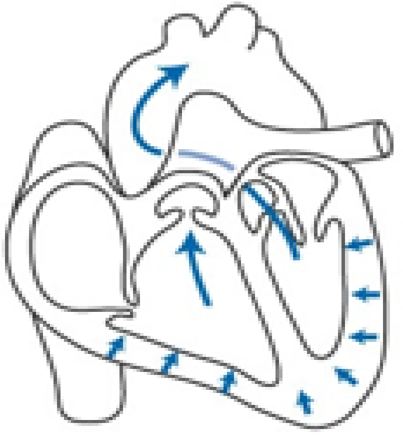
Reduce Afterload

- Nitroprusside (Nitropress)
- Enalapril (Vasotec)
- Captopril (Capoten)

Increase Afterload

- Epinephrine
- Norepinephrine
- Dopamine

Contractility



- Strength of Ventricular Contraction
- Sympathetic Stimulation
- Inotropes
- Physiologic Depressants
  - Hypoxia
  - Hypercapnia
  - Acidosis
- Coronary Flow
- Heart Muscle Damage

Drugs that Effect Contractility

Positive Inotropes

- Calcium
- Digitalis
- Epinephrine
- Norepinephrine
- Dopamine
- Dobutamine
- Amrinone,
- Isoproterenol
- Caffeine

Negative Inotropes

- Beta Blockers
  - Propranolol
- Barbiturates
- Procainamide
- Quinidine

Stroke Volume

Effected Only By:

* Preload
* Contractility
* Afterload

- Volume ejected by the ventricle with each contraction.
  - Contraction/Beat = Systole
  - SV = EDV-ESV

Pulse Pressure

- The difference between Systolic and Diastolic
- Normal Pulse Pressure 30 – 40 mmHg
- **A pulse pressure <30 mm Hg indicates a low stroke volume (LVSV) by the left ventricle.**
- If the blood pressure increases with fluid therapy, the patient was probably hypovolemic

Factors that effect Venous Return, Cardiac Output, and Preload

- Changes in circulating blood volume:
  - Hemorrhage
  - Loss of volume (dehydration, diuresis)
  - Gain on volume: IVs, Colloids
- Changes in distribution of blood volume:
  - Third Spacing: burns, sepsis, shock
  - Changes in body position, venous tone and Intrathoracic pressures
- Atrial contraction:
  - Atrial kick contributes up to 30% of the total CO
- Positive Pressure Ventilation

Changes in Preload

Increased Preload

- Leaky Valves due to Stenosis
  - Mitral
  - Tricuspid
- Decreased Heart Rate
- Increased Stroke Volume

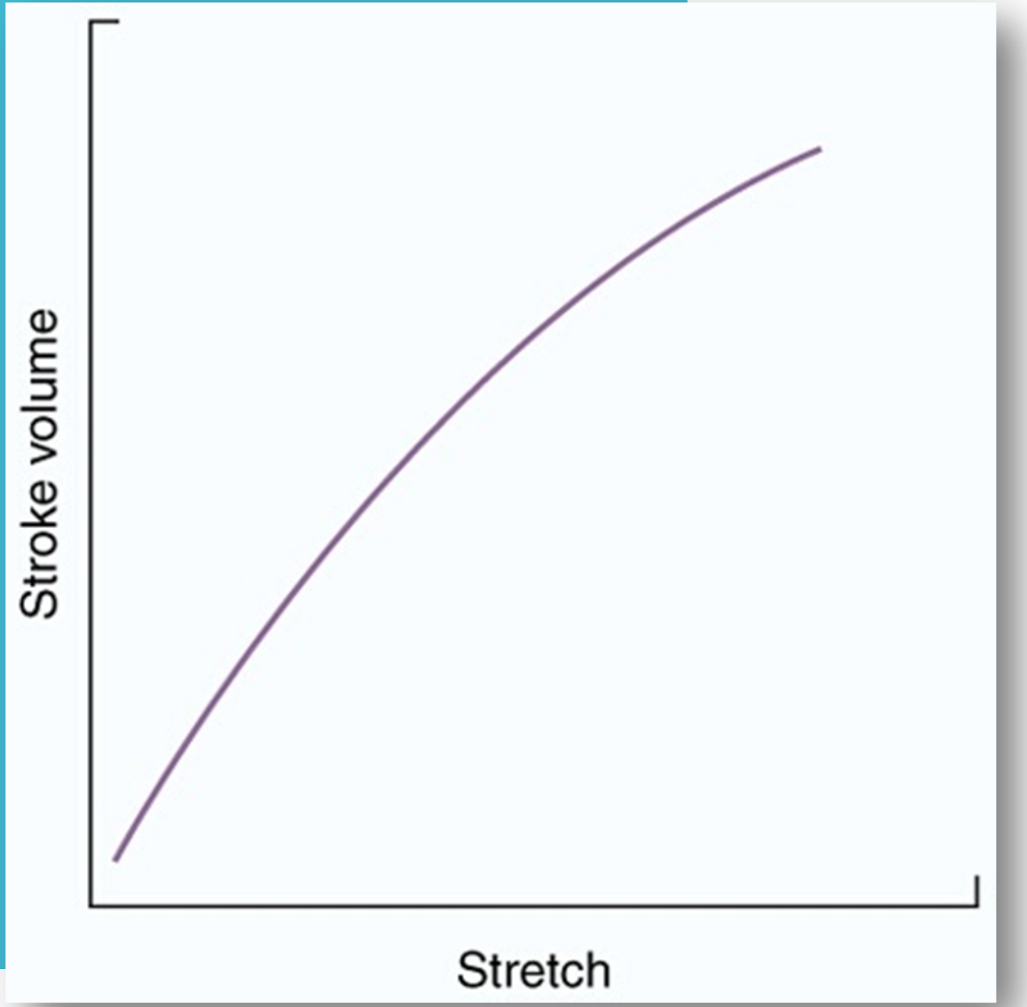
Decreased Preload

- Hypovolemia
- Increased Heart Rate
- Decreased Stroke Volume
- Increase in Intrathoracic Pressures

Positive Pressure Ventilation

- Normal Spontaneous Inspiration:
  - Augments preload
  - Augments CO
- Effects of lung compliance
  - The effects on venous return depend on how much pressure is transmitted to *the pleura space*
- PEEP/CPAP ➔ ⇓ venous return
- ICP

Starlings Law



Frank-Starling mechanism states that the energy liberated with each cardiac contraction is a function of the length of the muscle fibers in the ventricular wall; as preload ↑, so does end-diastolic pressure, which ↑ force of ventricular contraction

Translation…Starling's Law

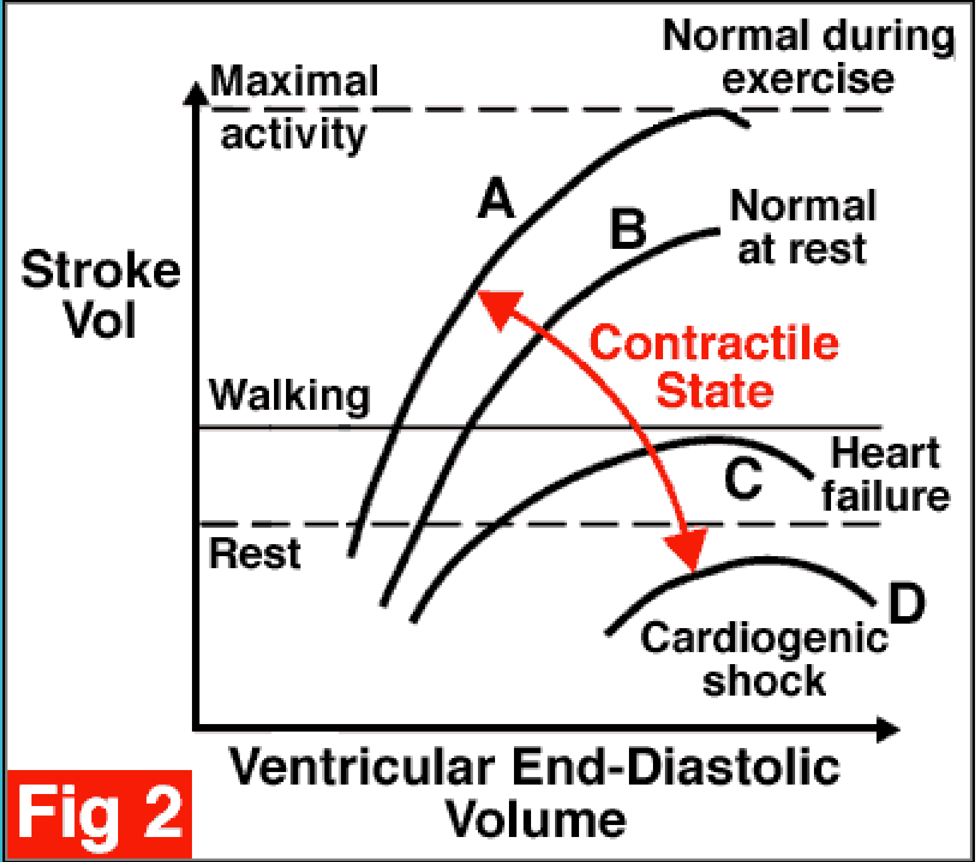


Fig 2

More volume in the ventricles….more stretch!

More stretch causes ventricles to contract more forcefully.

Heart's way of protecting it's with an increase in venous return.

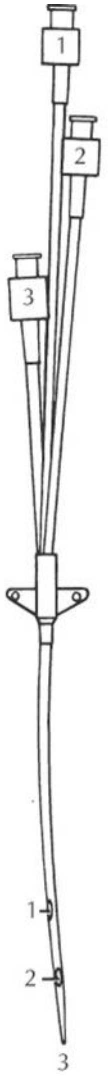
Why do we use Hemodynamic Monitoring?

3 Reasons to Monitor:

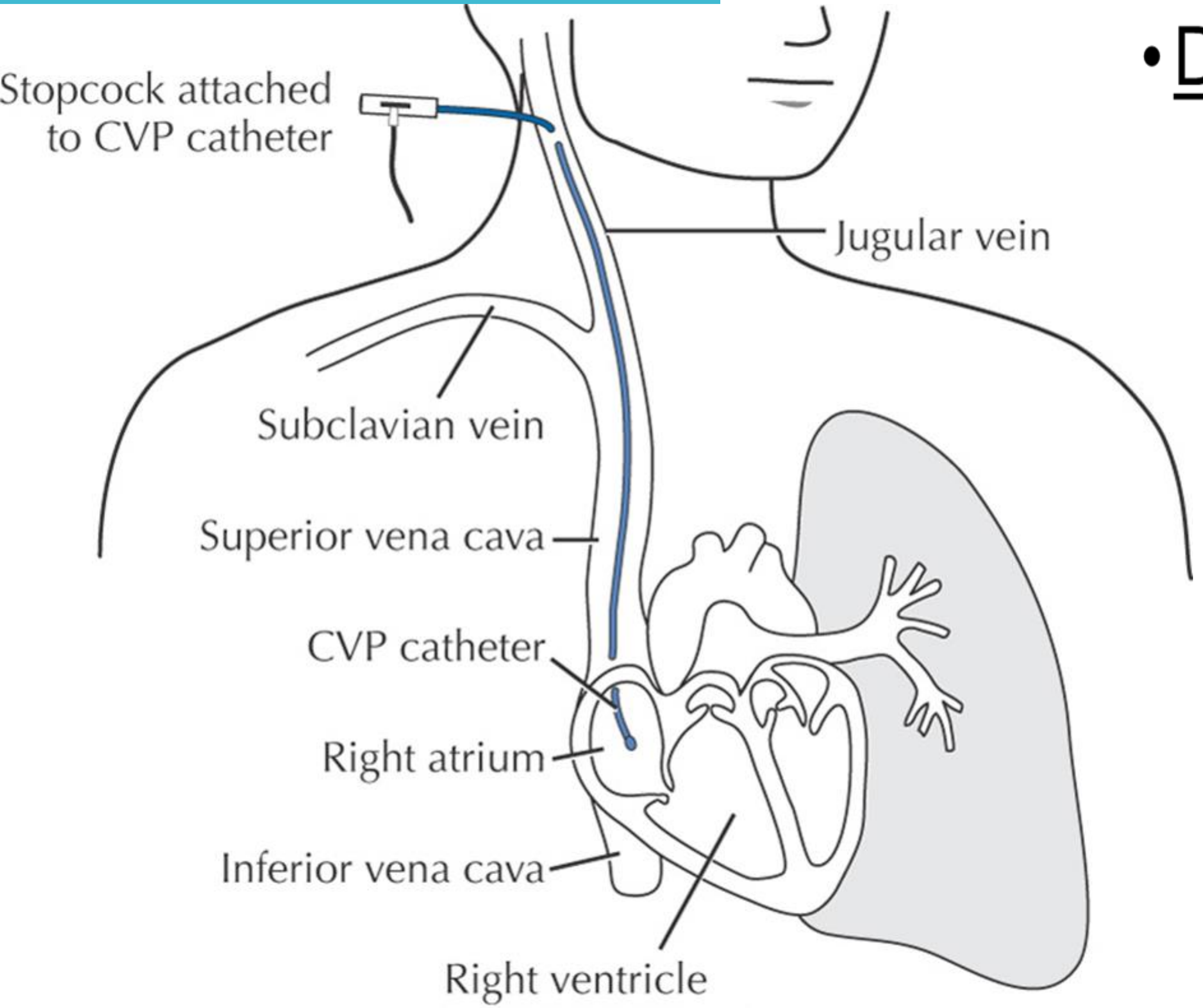
1. Intravascular fluid volume
   - CVP
   - PAWP
2. Cardiac function
   - CO
   - CI
3. Vascular function
   - PVR
   - SVR

Central Venous Pressure

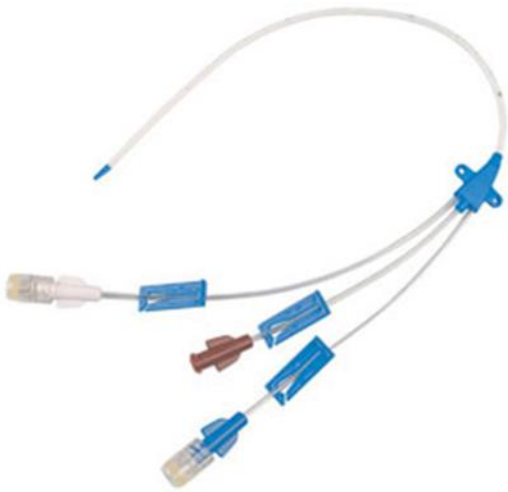
- Pressure of blood in the right atrium (RV) or superior vena cava (SVC)
- It represents the Right Ventricular End-Diastolic Pressure (RVEDP) or RV preload
- Obtained via a Central Venous Catheter (CVP)



CVP Effects



- **Increases with:***
  - ⇑ venous return (hypervolemia)
  - ⇑ Intrathoracic Pressures (PPV)
  - RV failure
- **Decreases with:***
  - ⇓ venous return (hypovolemia)
  - ⇓ Intrathoracic Pressures (spontaneous breathing)
  - ⇑ myocardial contractility



Fick Cardiac Output

Q is Cardiac Output:

The amount of blood from the heart in 1 min

Represents O_2 Delivery

(a – v)O_2 Difference is:

The difference in the amount of oxygen in arterial blood, compared to venous blood.

Represents how much of the O_2 is actually used by the tissue

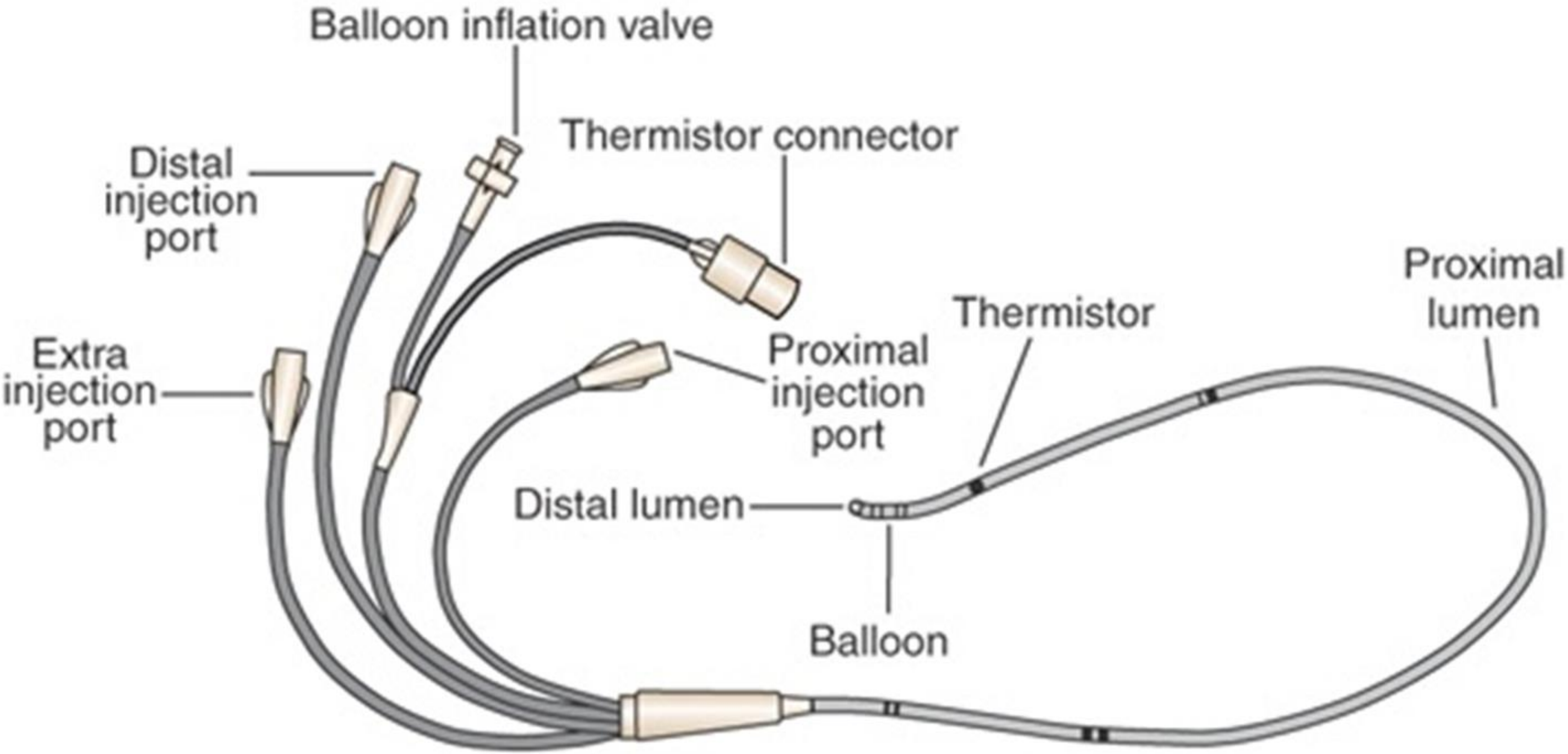
Fick Method Continued

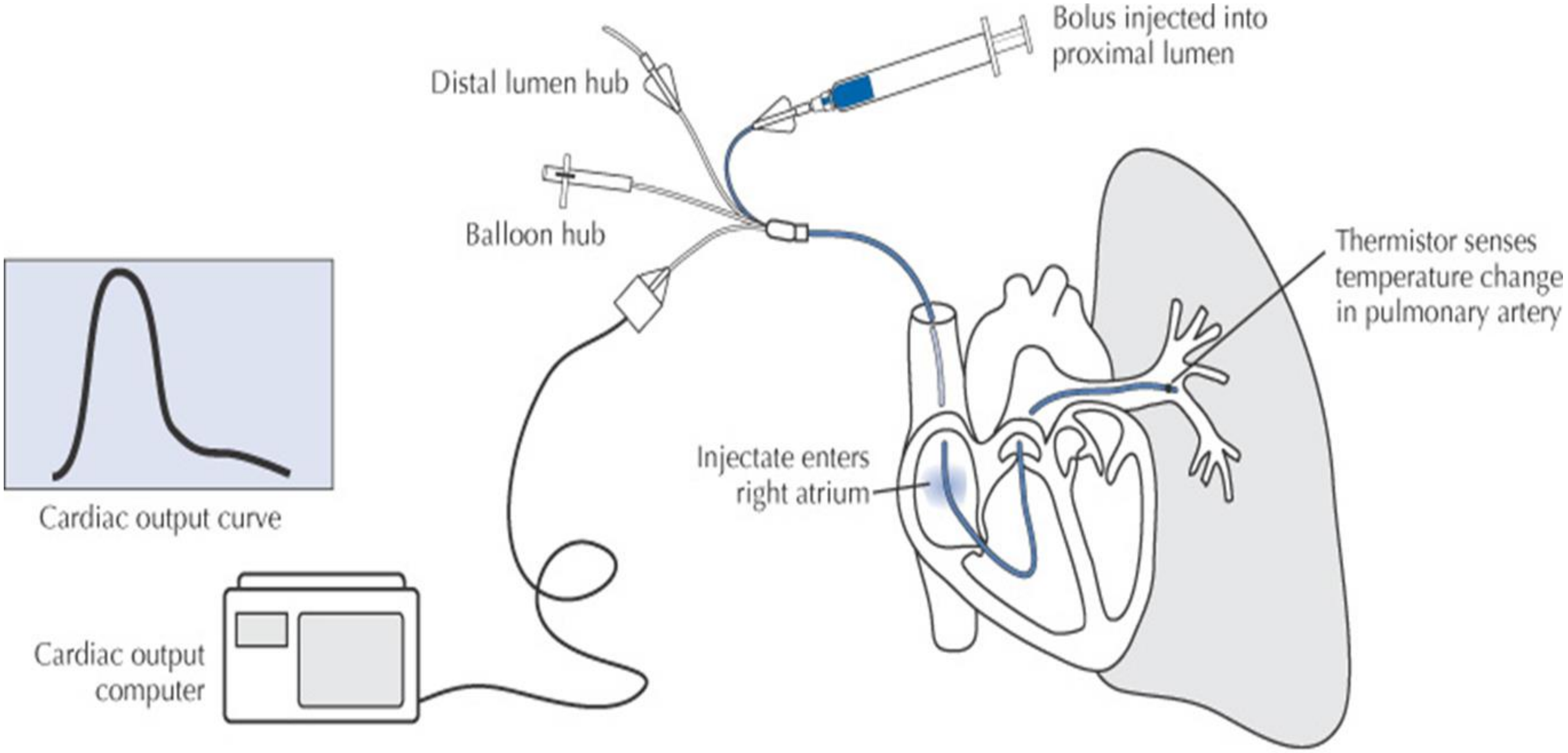
Normal O2 Consumption Range = 180 – 290 mL/min

Normal $C(a-\bar{v})O_2$ **Range = 3-5.5 Vol % (> 5.5 ↓ CO)**

Thermodilution Method Procedure

- Measures the temperature change of blood following injection of a solution of a difference temperature.
- A specific quantity of saline or DSW at iced or room temperature is injected rapidly into the proximal (RA) port of a Thermodilution PA catheter.
- The temperature drop of blood is measured at the distal tip at the thermistor. The temperature is plotted and CO is calculated.
- The following 2 slides illustrate


Balloon inflation valve
Thermistor connector
Distal injection port
Extra injection port
Proximal injection port
Thermistor
Proximal lumen
Distal lumen
Balloon


Bolus injected into
proximal lumen
Distal lumen hub
Balloon hub
Thermistor senses
temperature change
in pulmonary artery
Injectate enters
right atrium
Cardiac output curve
Cardiac output
computer

COMPLICATIONS

- Trauma (hemothorax, pneumothorax, nerve damage, etc,.)
- Dysrhythmias
- Heart or PA perforation
- Embolus, thrombus, hematoma, infection.
- Pulmonary infarction
- Dislodgment and migration

Alternative- Less Invasive Methods

- Pulmonary Artery Catheters and Central Venus Catheters are also **plagued by complication risks and inconsistencies**
- Therefore, ***less invasive*** commercial products have been introduced.
- They include:
  - Pulse pressure variation
  - Stroke volume variation
  - Oximetric waveform variation
- One other method that warrants discussion is **thoracic bioreactance**. Thoracic bioreactance uses noninvasive electrodes and an alternating current voltage across the thorax to obtain a signal which correlates with aortic flow to calculate:
  - CO
  - SV
  - Cardiac index
  - Volume responsiveness.
- However, complete evaluation of all available products is beyond the scope of this Presentation.

When to Monitor?

- Unstable Cardiogenic Pulmonary Edema
- Hemodynamically unstable ARDS patients
- Major cardiac and thoracic surgery
- Cardiogenic or septic shock

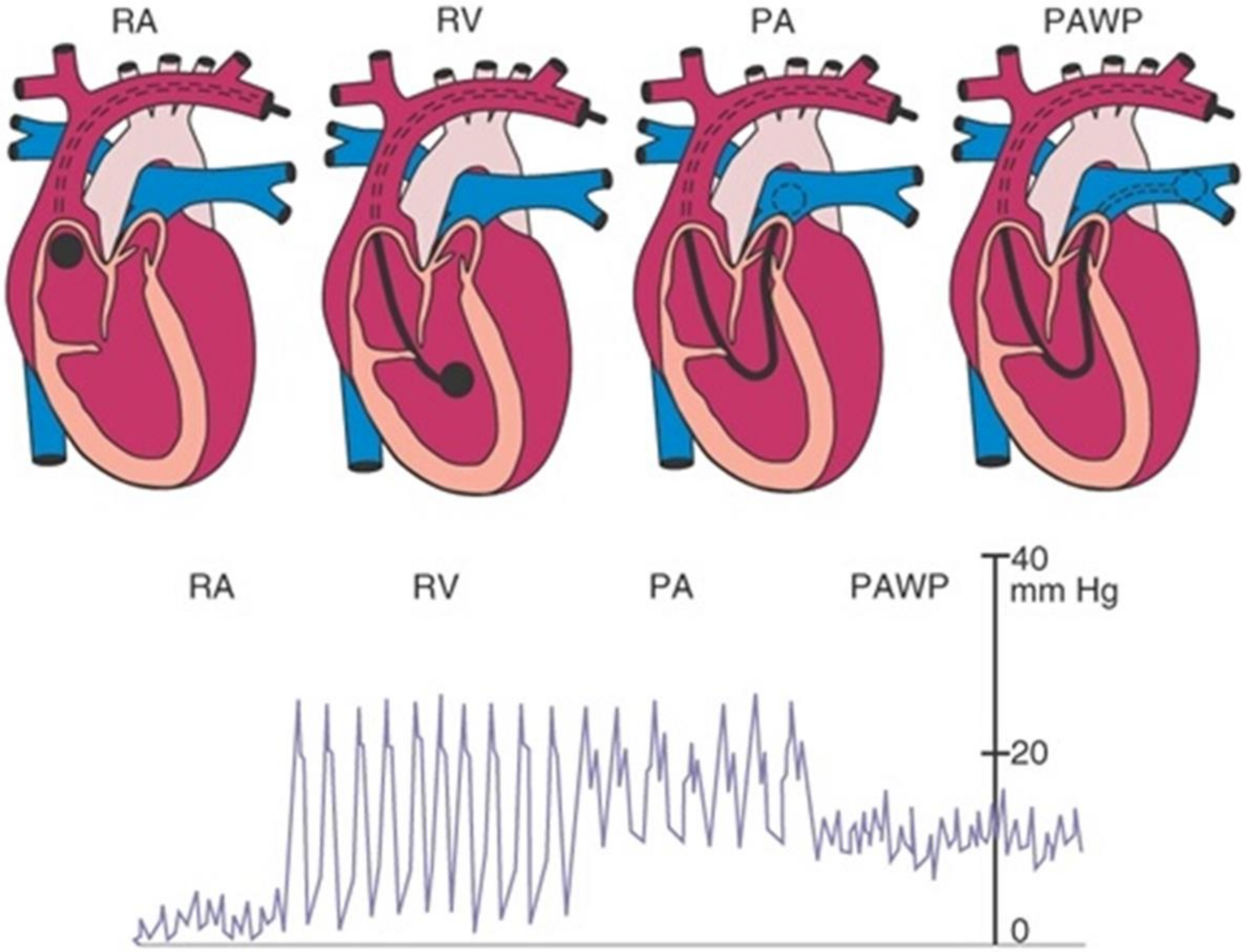
CVP			PAP PCWP	LAP		Arterial BP	
Venous System	RA	RV	Lungs	LA	LV	Aorta	Systemic Circulation
Blood Flow			Alterations in Resist. And Flow are Reflected Backwards				

Filling Pressures

- Right heart:
  - The filling pressure for the Right heart is the **Right Atrial** pressure, aka **CENTRAL VENOUS PRESSURE-(CVP)**
  - Normal CVP value: 2- 6 mm Hg

- Left heart:
  - The filling pressure for the Left heart is the left Atrial pressure, aka **PULMONARY CAPILLARY WEDGE PRESSURE (PCWP)**
  - Normal PCWP value: 6-12 mm Hg

Waveform



Right atrium	Right ventricle	Pulmonary artery	Pulmonary capillary wedge pressure
Normal pressure: 2-6 mm Hg	Normal pressure: 20-30 / 0-5 mm Hg	Normal pressures: 20-30 / 6-15 mm Hg Mean pressures: 10-20 mm Hg	Normal pressure: 4-12 mm Hg

PAP Interpretation

- ⇑ PVR caused by:
  - PE (obstruction)
  - Acute or chronic lung diseases (vasoconstriction caused by Hypoxia)
  - Cardiac Tamponade
  - ⇑ Intrathoracic pressures (PPV)
  - LHF
  - Mitral valve regurgitation
  - Equipment

–⇓ PVR caused by:

- ⇓ venous return
- Hypovolemia
- Vasodilation
- ⇑ LV contraction
- Equipment

PCWP Interpretation

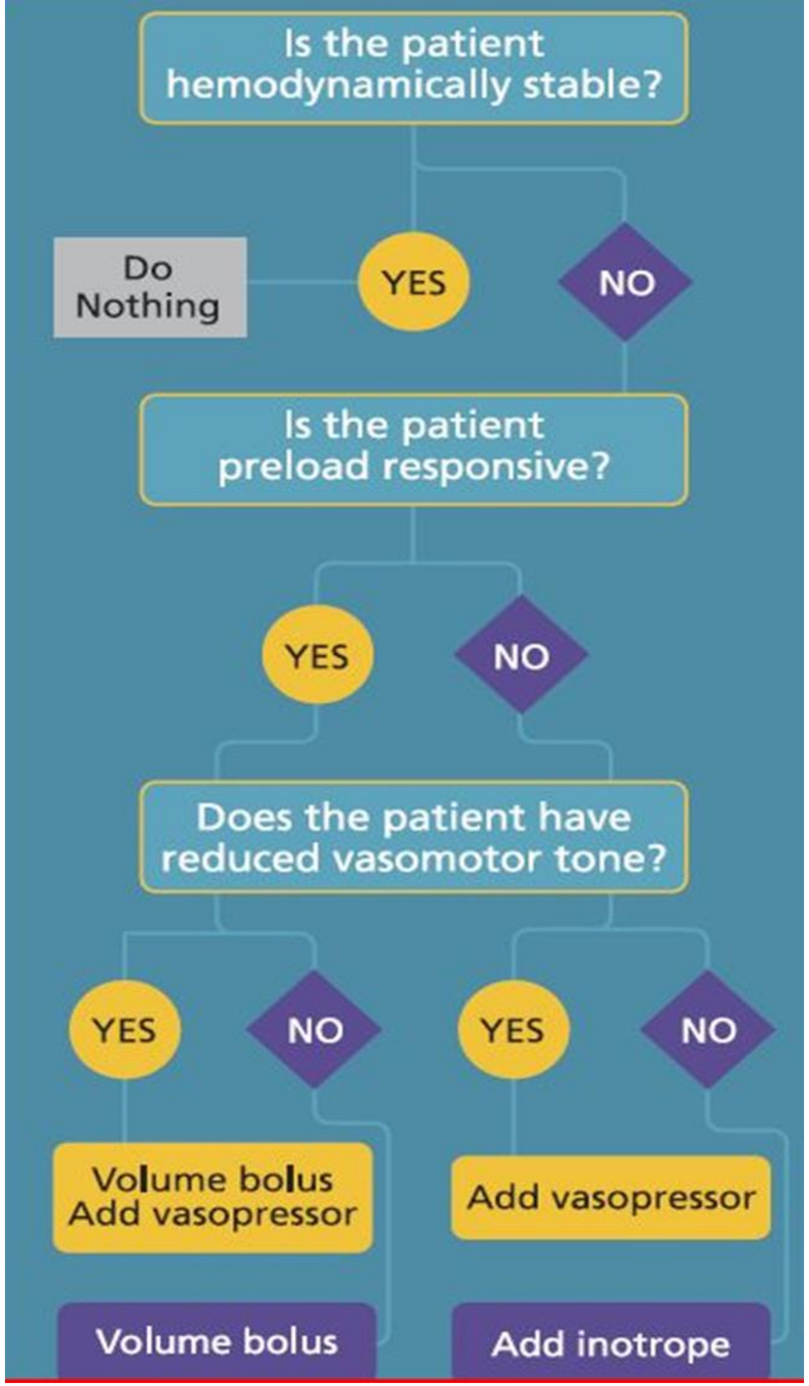
- ❑ LEFT VENTRICULAR FAILURE
- ❑ MITRAL VALVE DEFECTS
- ❑ HYPO/HYPERVOLEMIA
- ❑ PEEP
- ❑ EQUIPMENT

Note: >18 → Mild pulmonary congestion
>18 → Acute pulmonary edema

Up Up Up!

Right Ventricular Failure	Increased CVP ↑	Increased Vascular Congestion ↑	
Left Ventricular Failure	Increased PCWP ↑	Increased PAP ↑	Increased CVP ↑
Increased Lung Pressures	Increased PAP ↑	Increased CVP ↑	Increased Vascular Congestion ↑

Hemodynamic Management



Selected References

- AARC.org
- Egan's Fundamentals of Respiratory Care, ed 12, Kacmarek, Stoller & Heuer, 2021.
- Clinical Assessment in Respiratory Care, ed. 8, Heuer & Scanlan, 2018.
- Respiratory Disease: A Case Study Approach to Patient Care, ed 3, 2007.
- Pubmed
- Medline