

Cardiovascular response & acute vasodilatation

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Anaphylaxis Pathophysiology

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<https://veltman.org/education/notre-dame-vasodilatation-talk/>

Outline

- Cardiac Physiology
 - Definitions and examples.
- Anaphylaxis
 - Definition
 - Diagnosis
 - Management

Definitions

Hypotension

Vasodilation

Shock

Hypotension

A low systolic blood pressure.

Typically < 90 mmHg in an adult.

Mostly not a problem

Clinically significant Hypotension

Symptoms or signs of
hypo-perfusion:

Conscious state (dizziness -> coma)

Functional capacity (fatigue -> blackouts)

Causes

- Three causes for a fall in blood pressure
 - Less filling of the heart.
 - Less strength in contraction
 - Less tone in the arterial tree
- Many factors play into this
 - Renal function
 - Reflex regulation of blood pressure

Vasodilation

- A reduction in the vascular resistance of the whole vascular tree
 - Regional dilation involves part of body

- $SVR = MAP / CO$

SVR = Systemic Vascular Resistance

MAP = Mean Arterial Pressure

CO = Cardiac Output

Shock

- Inadequate tissue perfusion to meet the metabolic demands of the body
- Clinical syndrome with:
 - Inadequate oxygen and nutrient delivery
 - Inability to meet the metabolic needs of tissues
 - Inadequate cellular metabolism and function

Physiology

Cardiac Output and
Perfusion

Preload/Contractility/
Afterload

Pressure volume loops

Cardiac Output

Cardiac output = Heart Rate x Stroke Volume

Stroke Volume determined by:

- Preload
- Contractility
- Afterload

The heart is a slave

- In a healthy person, the heart just responds to its environment
- Basically, what goes in will be pumped out.

Preload

- The amount of filling in the heart before contraction starts

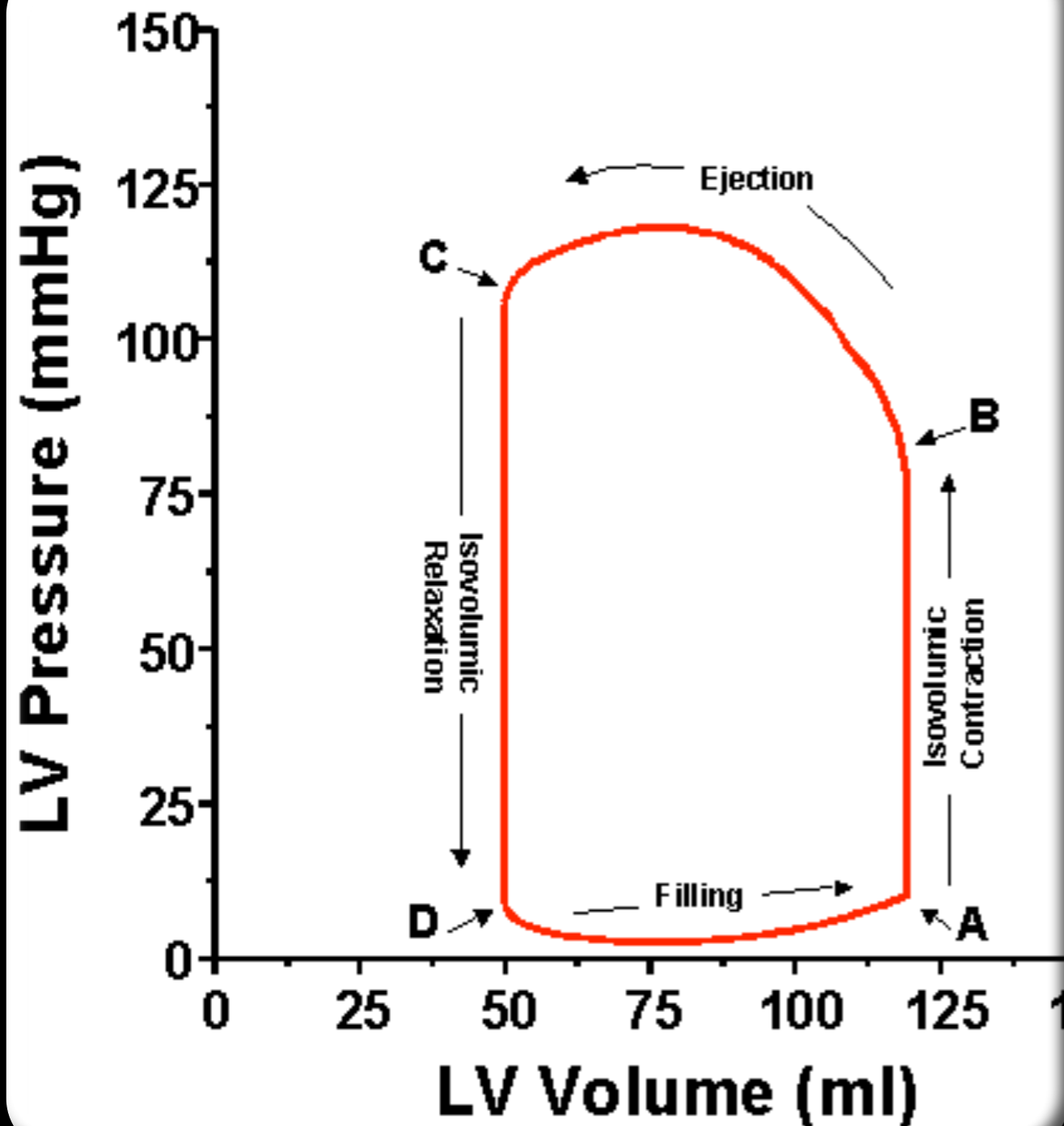
Contractility

- The strength of contraction (relative to a fixed preload)

Afterload

- The resistance to ejection of blood from the heart.

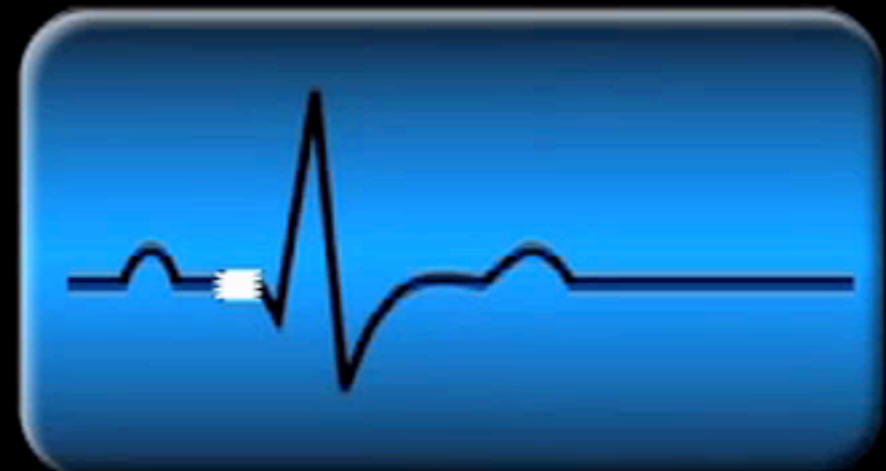
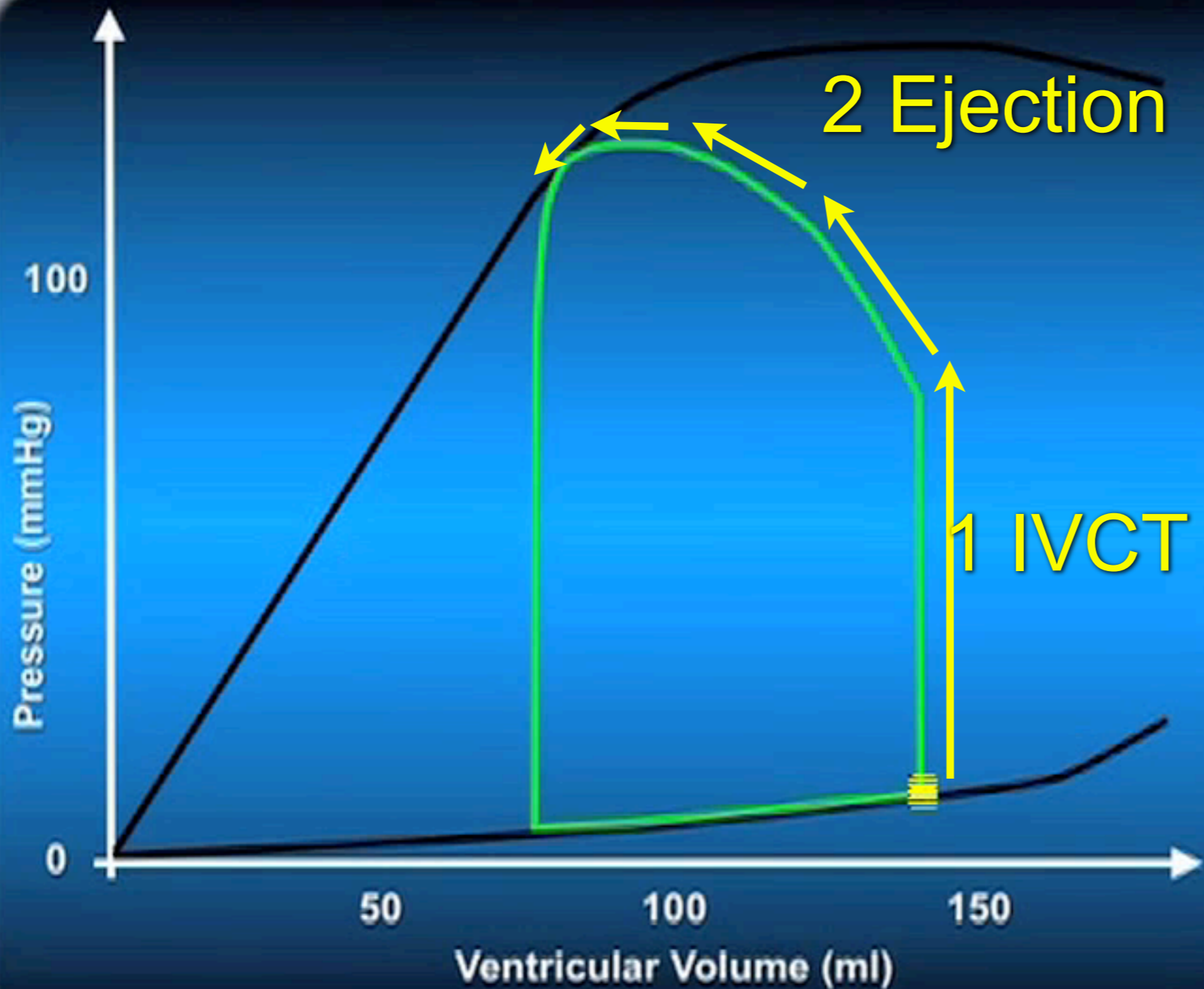
Pressure Volume Loops



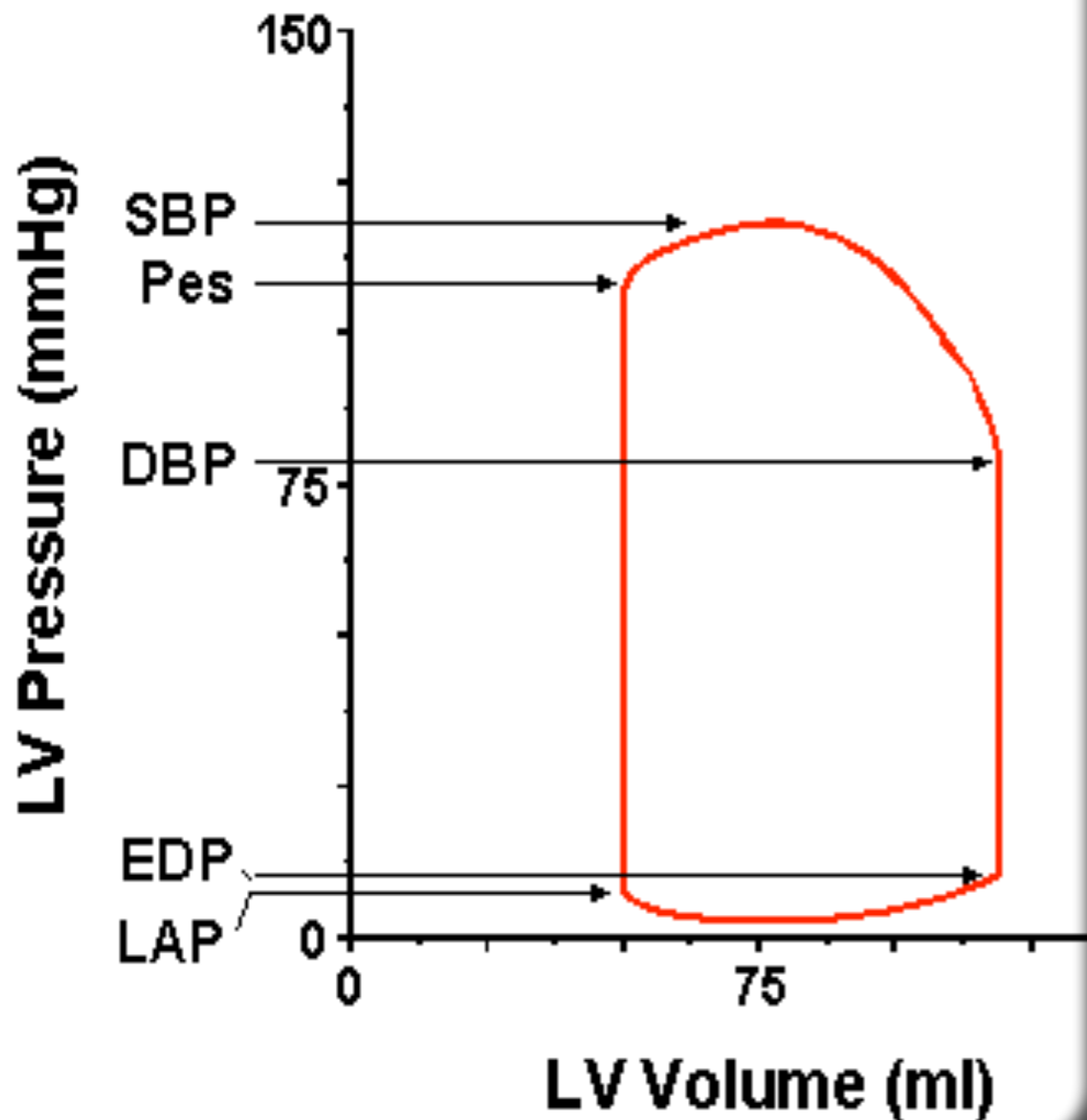
- Preload
- Contractility
- Afterload

Cardiac Cycle Left Ventricle

Phases of Systole

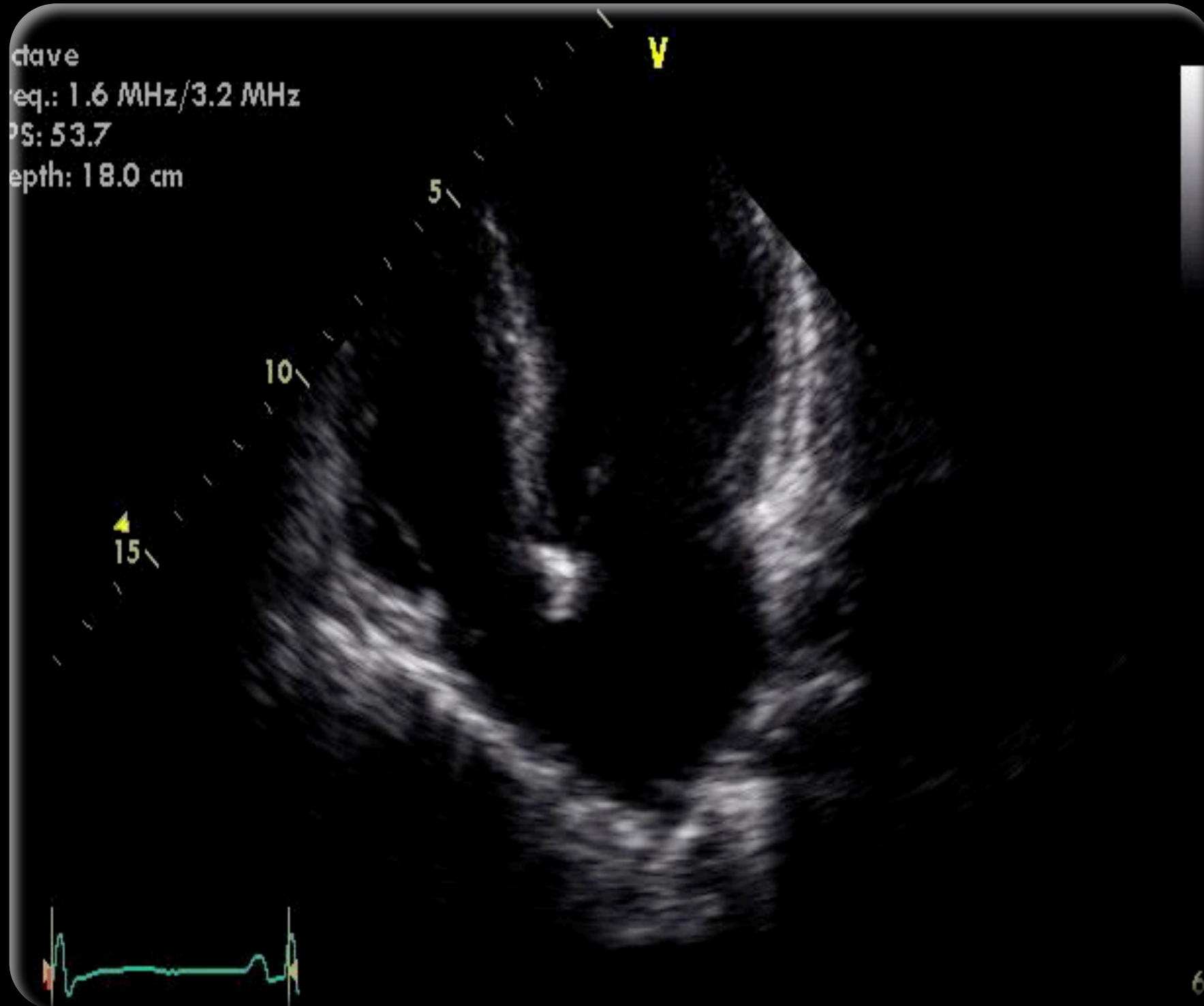


Ventricular Pressure



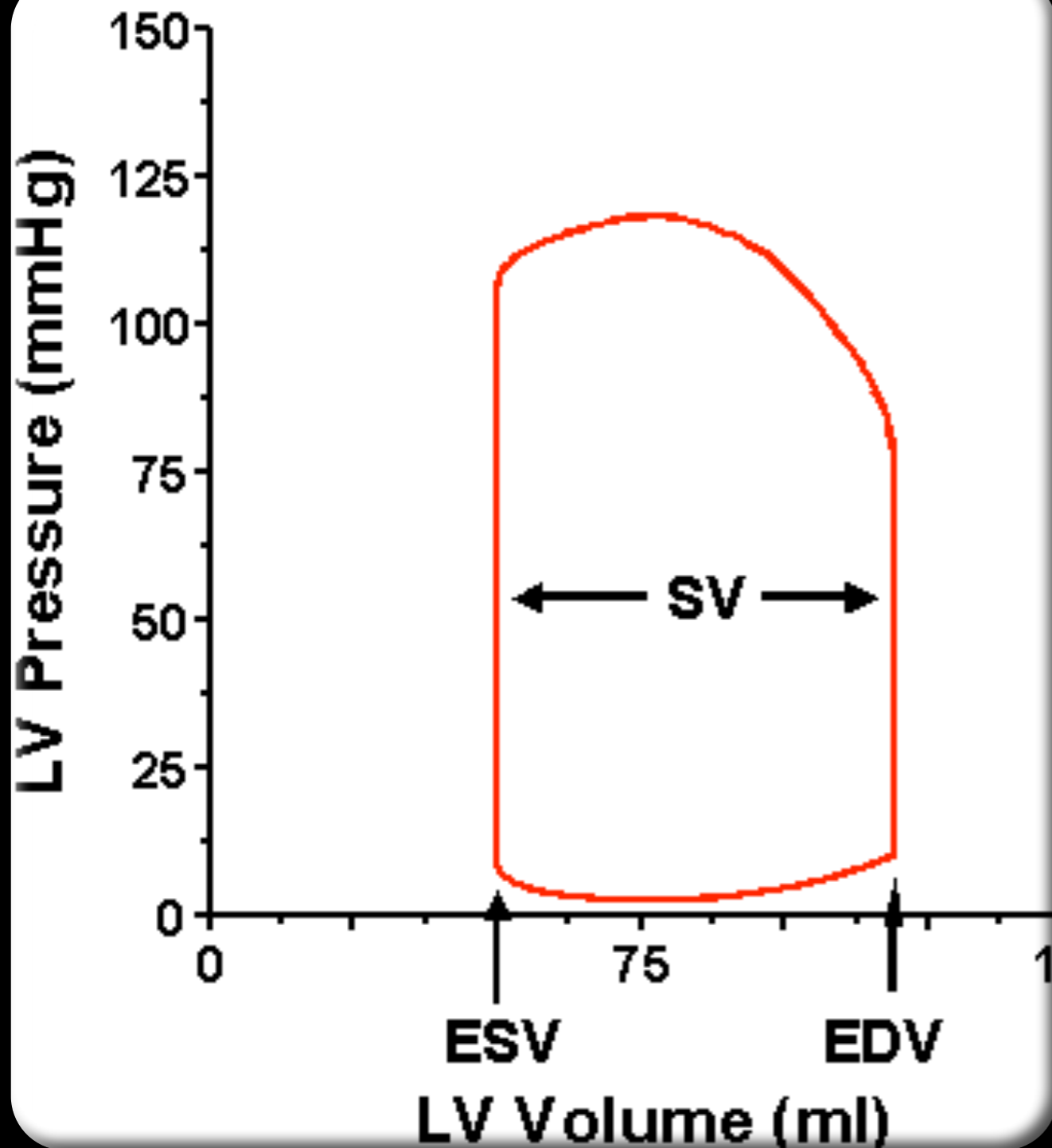
- Pressures are on the Y-Axis
- LV pressure is very close to arterial blood pressure
 - When aortic valve is open
 - If aortic valve is normal

Normal filling



- Note size of LV in diastole

Preload and Stroke Volume

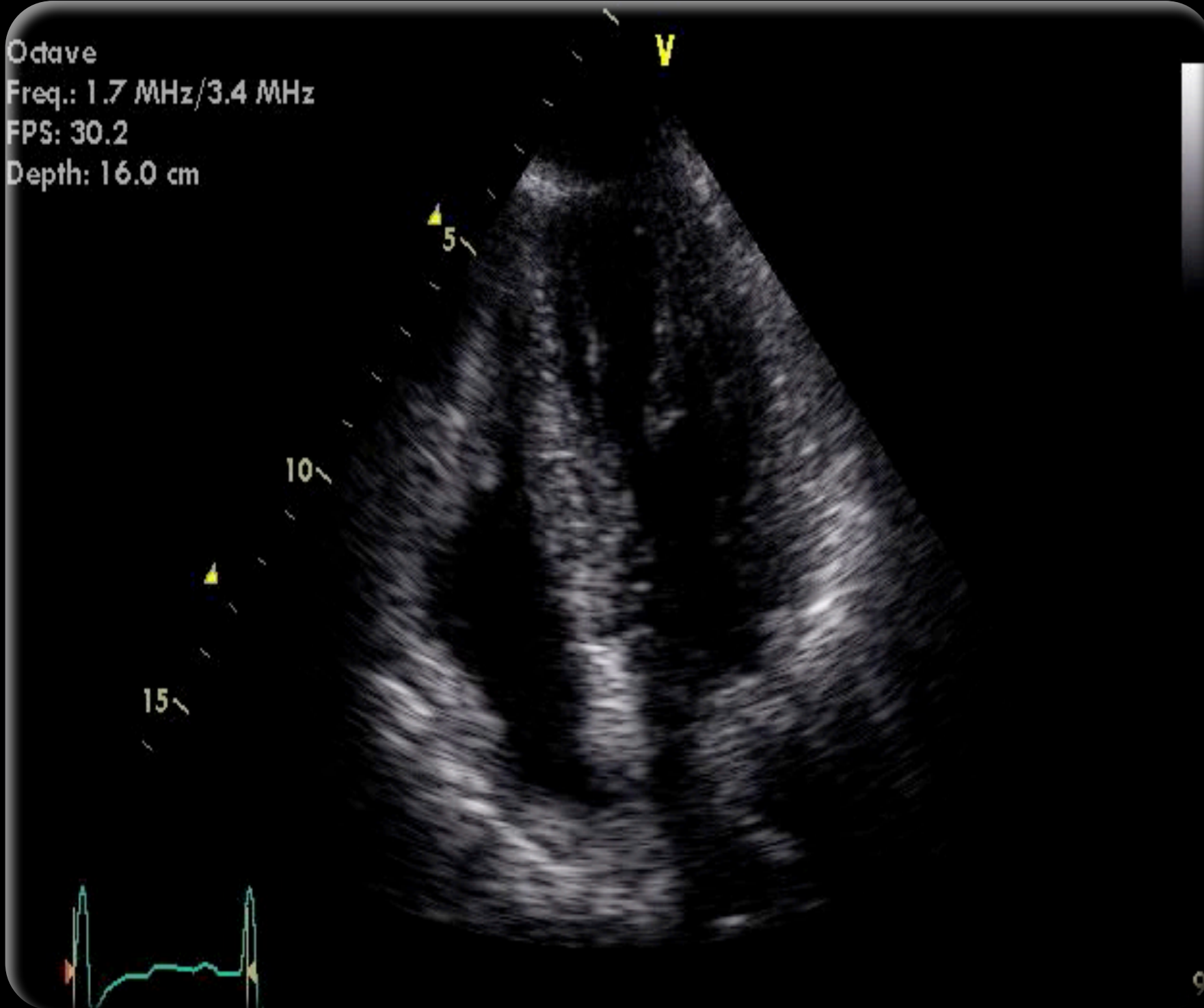


- The difference in the X-axis represents the volume of blood ejected
 - This is known as the stroke volume (SV)

Cardiac output = SV x heart rate

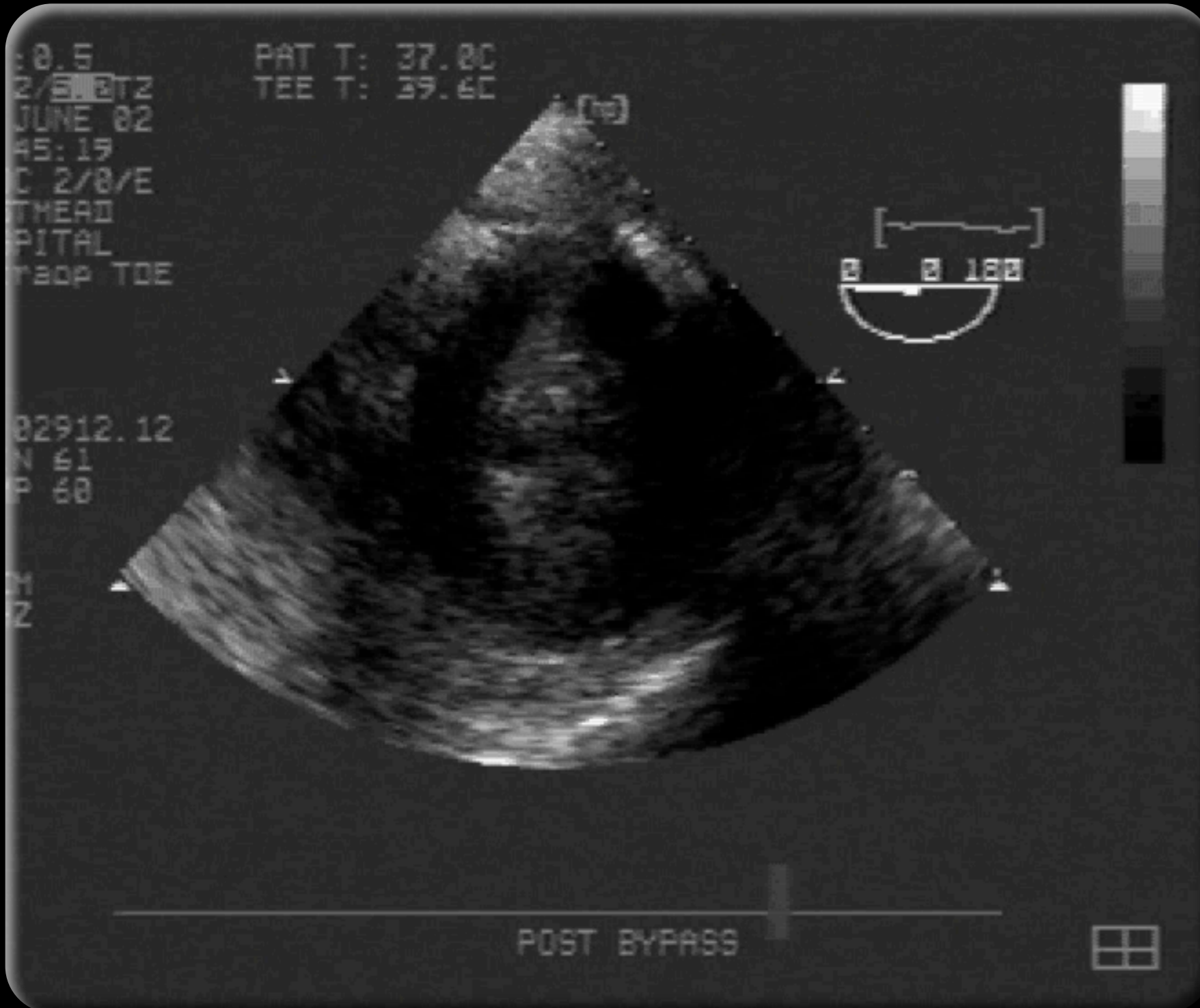
Reduced Preload Hypovolaemia

Octave
Freq.: 1.7 MHz/3.4 MHz
FPS: 30.2
Depth: 16.0 cm



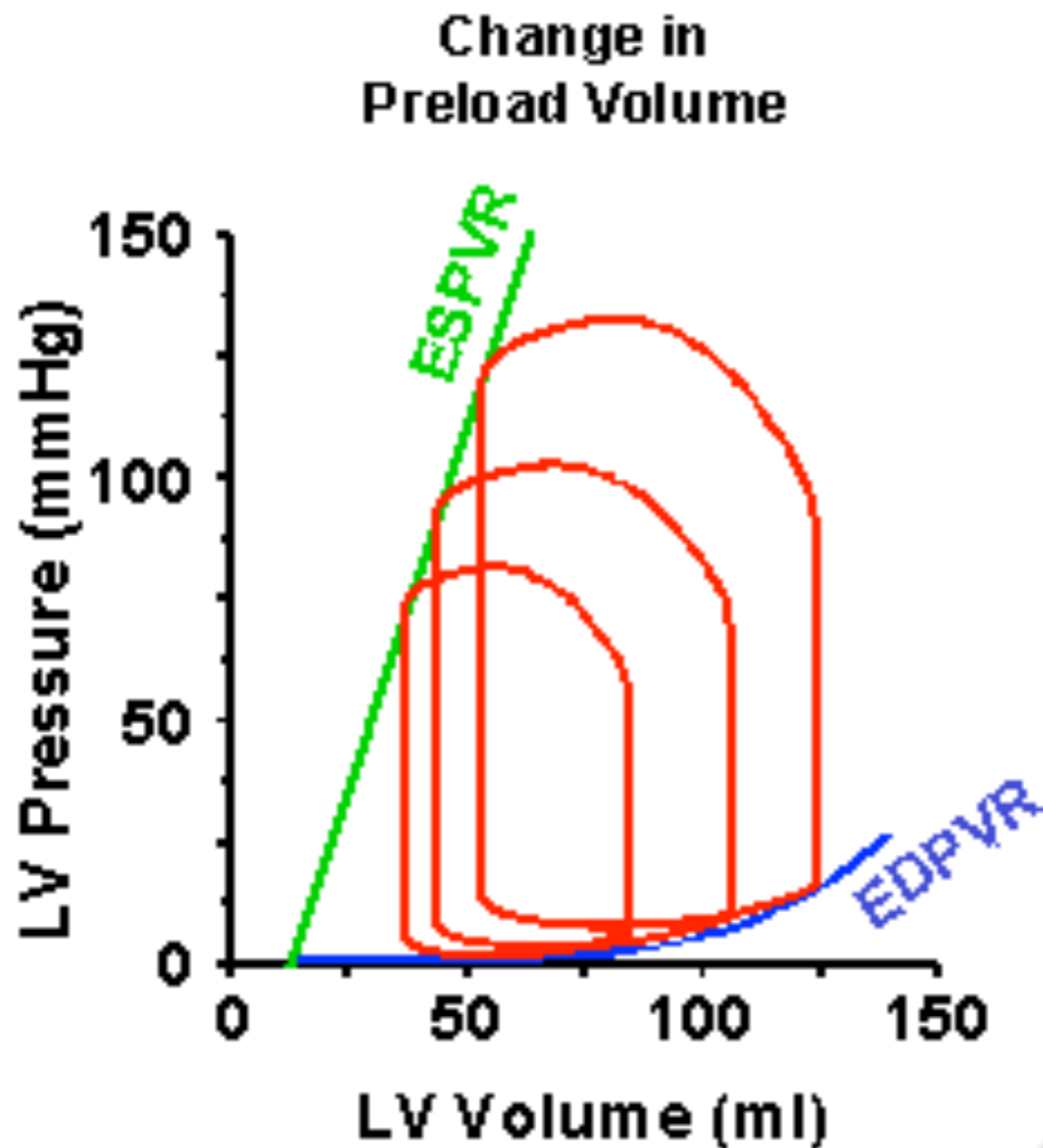
- Empty heart
 - Walls close
 - Papillary muscles touch

Hypovolaemia



- Ventricle contracts to empty

Changes in filling



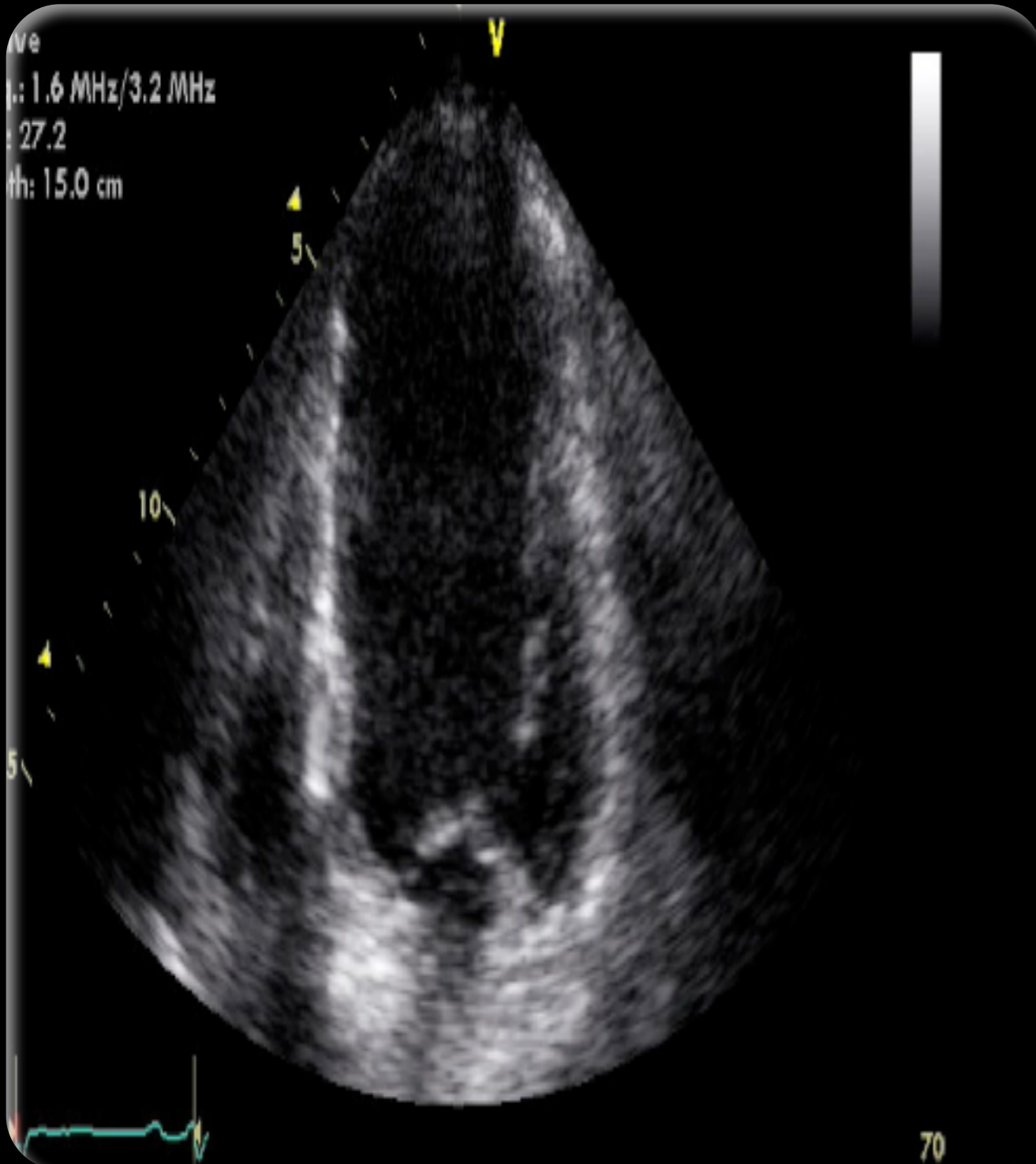
- Uncompensated reduction in filling causes:
 - Stroke volume and cardiac output
 - Systolic and diastolic pressures

Normal function



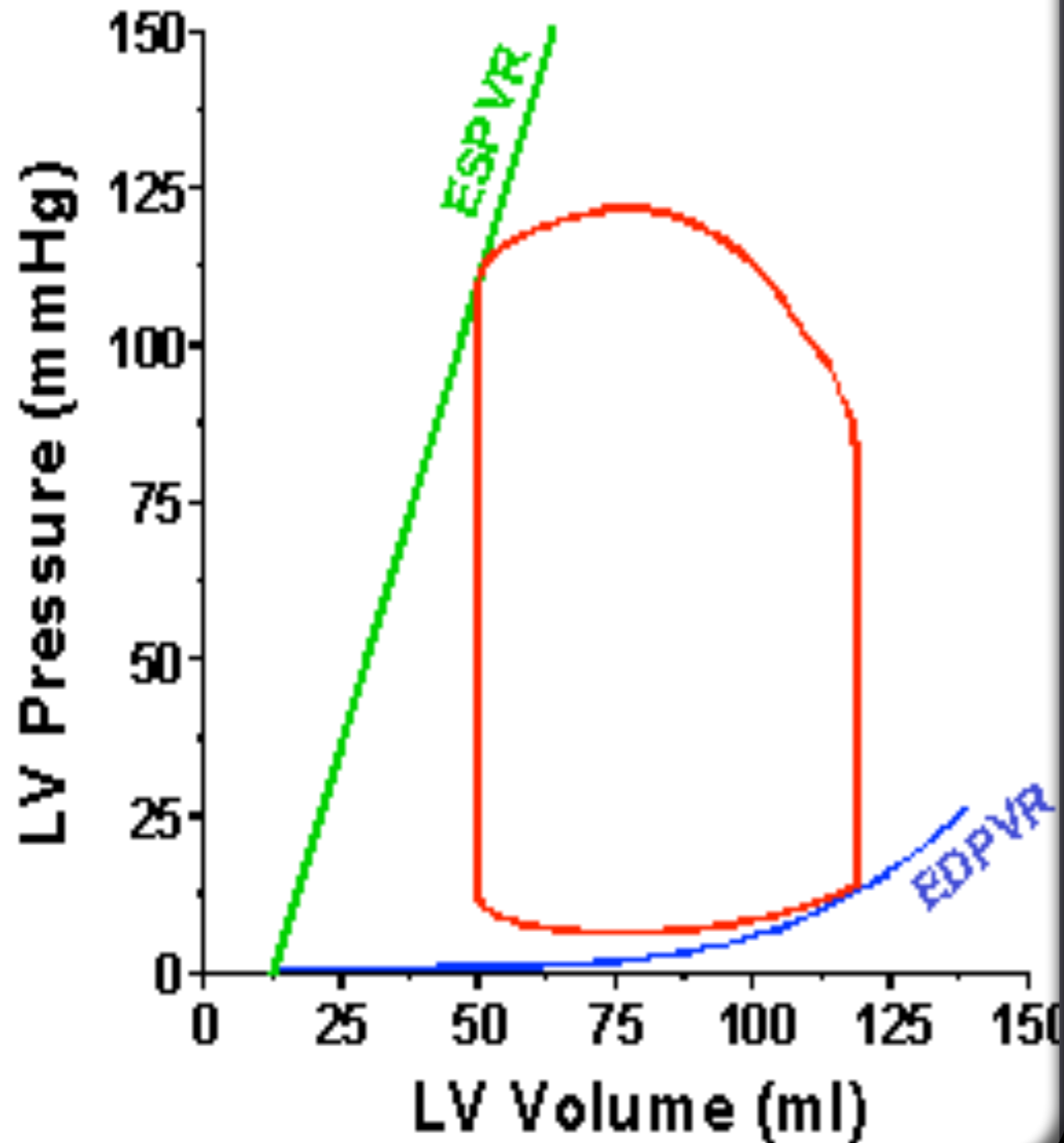
- Note how much the left ventricle empties.

Reduced contractility



- Severe hypokinesia

Systolic Function

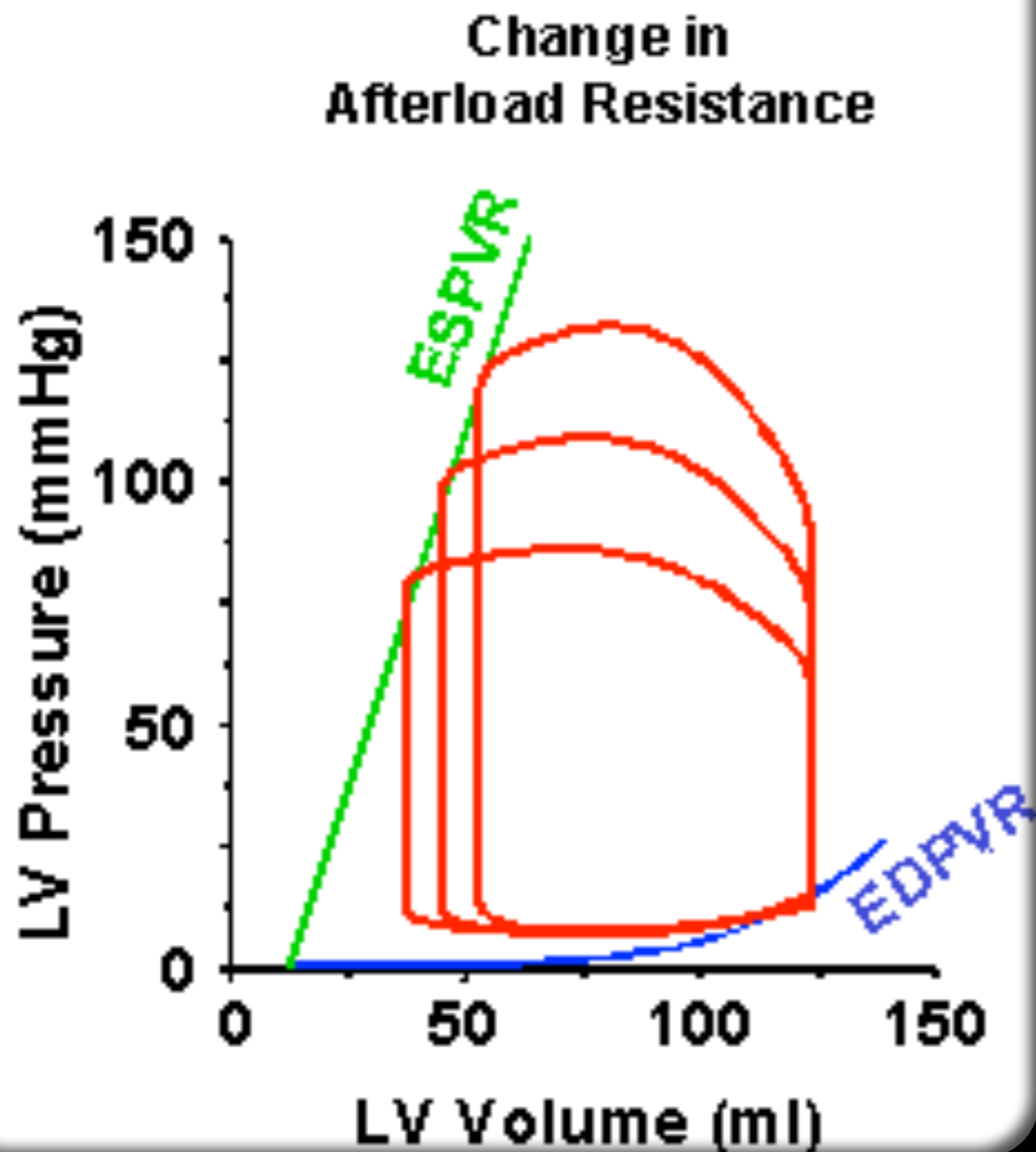


Normal function



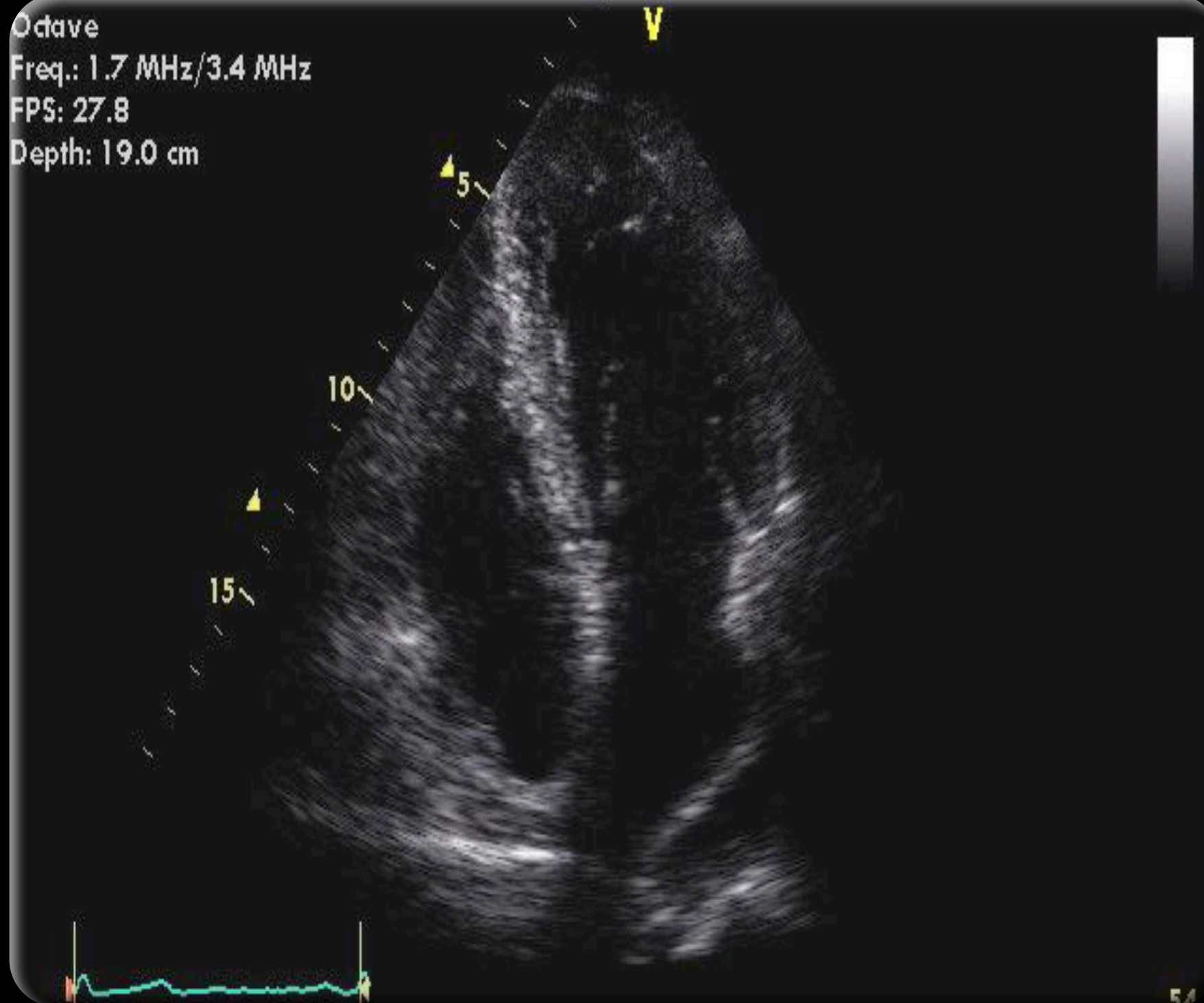
- Note how much the left ventricle empties.

Changes in afterload



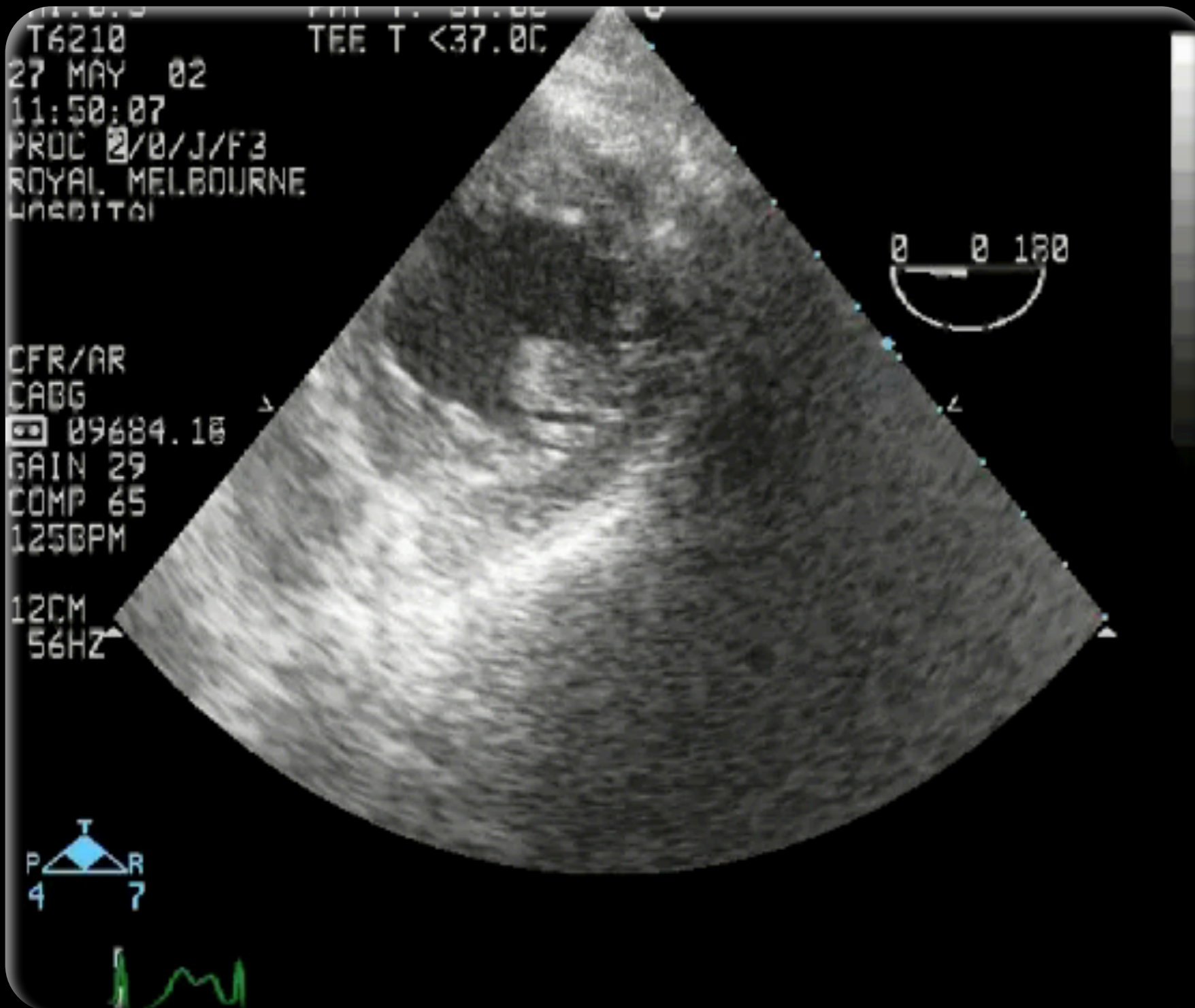
- As after load falls
 - Arterial pressures fall
 - Stroke volume increases
 - Output increases

Decreased after load



- Empties more but from a normal starting volume.

Decreased Afterload



- Heart is
 - Empty
 - Ejecting well

Response to shock

Response to Hypoperfusion

Inadequate oxygen delivery



Catecholamine response



Anaerobic metabolism



Cell dysfunction



Cell death

- Reflex to fall in mean arterial pressure
 - ↑ Sympathetic activation
 - ↑ Adrenaline/Nor-Adr
 - ↑ HR, ↑ BP ↑ Contractility
- Activation of Renin-Angiotensin System
 - Salt/Water retention

Catecholamine Response: Cardiac physiology

- Catecholamine affects:
 - SA & AV node
 - Conducting pathways
 - Myocardial function
- Net result is the heart:
 - Pumps harder and faster

β_1 :

- ↑ Contractility
- ↑ Relaxation
- ↑ Heart Rate
- ↑ Conduction velocity

Catecholamine Response: Vascular Tree

- Catecholamines affect:
 - Splanchnic
 - Renal
 - Non-essential organs
- Net result is:
 - Coronary and Cerebral flow is maintained.

α_1 - Vasoconstricts

β_2 - Vasodilates

β_1 - Vasodilates
(Renal)

α_2 - Vasoconstricts
(Coronary/Skin)

Anaphylaxis

Definition

Diagnosis

Management

Anaphylaxis Outline



- Definition
- Diagnosis
- Management

Anaphylaxis



- Anaphylaxis is
 - A severe sudden activation of the immune response
 - involves preformed antibodies
- Involves multiple systems
 - Respiratory
 - Cardiovascular

Anaphylaxis Causes

- Drugs
 - Antibiotics
 - Sulphur based agents
 - Muscle Relaxants (esp Rocuronium)
 - Colloids (Haemacell, Gelofusine)
 - Pretty much any drug
- Foods
- Environmental antigens

Pathophysiology

- Type 1 immune hypersensitivity reaction
 - IgE mediated degranulation of mast cells & basophils
- Release of pro inflammatory mediators
 - Tryptase (mast cell specific)
 - Nitric Oxide (NO)
 - Platelet activation factor (PAF)
 - Prostaglandins / Leukotrienes

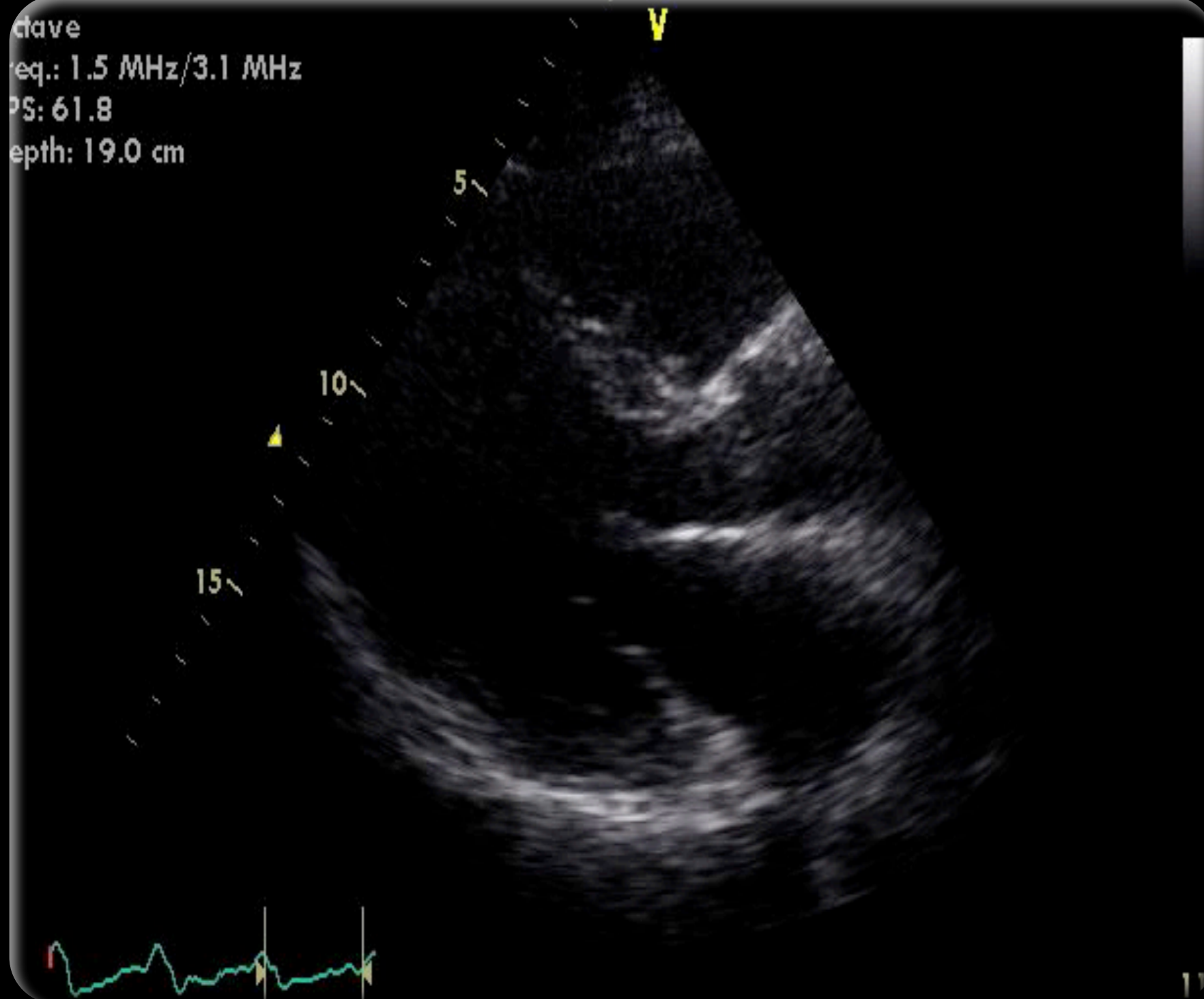
Pathophysiology

1. Vasodilatation leads to reductions in:
 1. TPR and effective blood volume
 2. Venous return
2. Increased capillary permeability
 1. Fluid loss to the interstitium and oedema
 2. Further reduction in intravascular volume

Pathophysiology

1. Mixed distributive & hypovolaemic shock
 1. ↓ central blood volume
 2. ↓ Venous pressure ↓ SV ↓ CO
 3. ↓ SBP ↓ DBP ↓ MAP ↓ Pulse pressure

Tachycardia



- Some impairment in contractility also seen here

Anaphylaxis Features

- Respiratory (50% of cases)
 - Bronchospasm & Wheeze
 - Difficulty in ventilation - Hypoxia and desaturation
- Cardiac (90% of cases)
 - Vasodilation and hypotension
 - Tachycardia
 - Oedema
- Rash (30% of cases)

Anaphylaxis

First Signs



- Difficulty breathing
- Rash
- Dizziness

Grading Severity

- Mild
 - Skin and Subcutaneous tissues only
- Moderate
 - Respiratory, CVS and GI involvement
- Severe
 - Hypoxia, Hypotension and CNS compromise

Brown SGA 2005

Clinical features and severity grading of anaphylaxis

J Allergy Clin Immunol 114 :371-376*

Anaphylaxis Management

- Urgent attention required - Get help.
 - Discontinue drug which caused reaction
 - Lay Flat / Elevate legs

- Basic life support

Anaphylaxis Management

- Airway/Breathing:
 - 100% O₂, +/- Ventilate
- Circulation: Maintain blood pressure
 - Adrenaline +/- Fluids
- Bronchodilate
 - Adrenaline and other beta agonists

Adrenaline (Epinephrine)

- How much to give:
 - 0.5mg IM if no venous access
 - 25-50-100 mcg if IV access
- Different from a cardiac arrest
 - 1mg IV every 3-5 minutes

Anaphylaxis Management

- Treat inflammatory response
- Stabilise mast cells
 - Adrenaline (IV or Nebulised)
- Treat Bronchospasm
 - Ventolin/Salbutamol
- Treat Hypotension
 - Other Pressors (Metaraminol)

Anaphylaxis

Longer Term Therapy

- Immunosuppress
 - Steroids – IV high dose.
 - Dexamethasone
- Test for anaphylaxis
 - Mast cell tryptase
 - Allergy / Skin testing.
- Followup – Medic alert bracelet

Anaphylaxis Summary



- Definition
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Summary

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