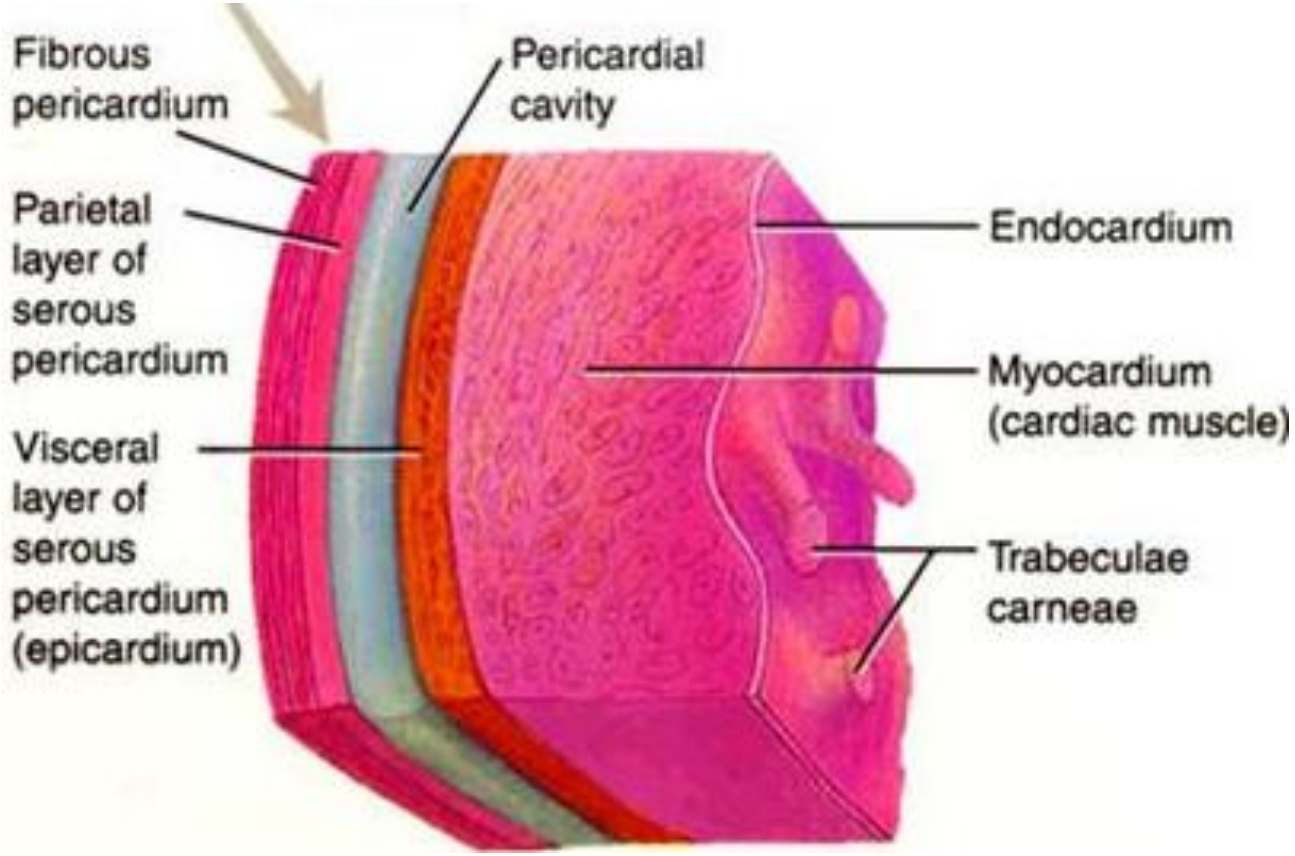
Normal Pericardial Physiology



- Normal pericardium contains 20-30 ml of lymphoid fluid

- lubricating function that facilitates normal myocardial rotation and translation during each cardiac cycle in that the mesothelial layers of the pericardium produce a serous fluid that serves as a lubricant.

- secrete biochemical substances (e.g., prostacyclin) that may play a role in sympathetic neural regulation, coronary vascular tone and cardiac contractility.

Physiology of the normal pericardium

Mechanical function

1. Relatively inelastic cardiac envelope
    - limitation of excessive acute dilatation
    - protection against excessive ventriculoatrial regurgitation
    - maintenance of normal ventricular compliance
    - hydrostatic system
2. ventricular interaction; relative pericardial stiffness
3. maintenance of functionally optimal cardiac shape
4. Provision of closed chamber with subatmospheric pressure

Membranous function

Ligmentous function

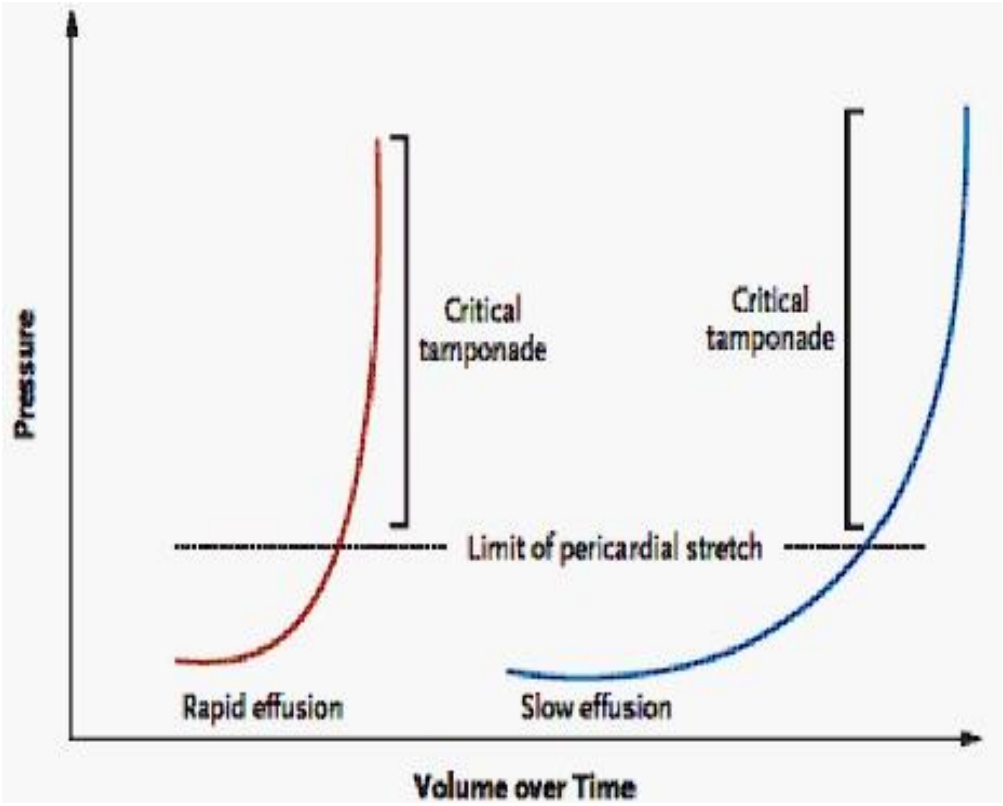
Mechanical function=>"Pericardial restraint"

- The compliance of the pericardium varies with the volume
- Initially the pericardium is supple and can expand with minimal increase in intrapericardial pressure with cardiac filling
- At higher volumes, the intrapericardial pressure rises, and impedes systemic and pulmonary venous return

- Volume of the fluid
- Rate of fluid accumulation

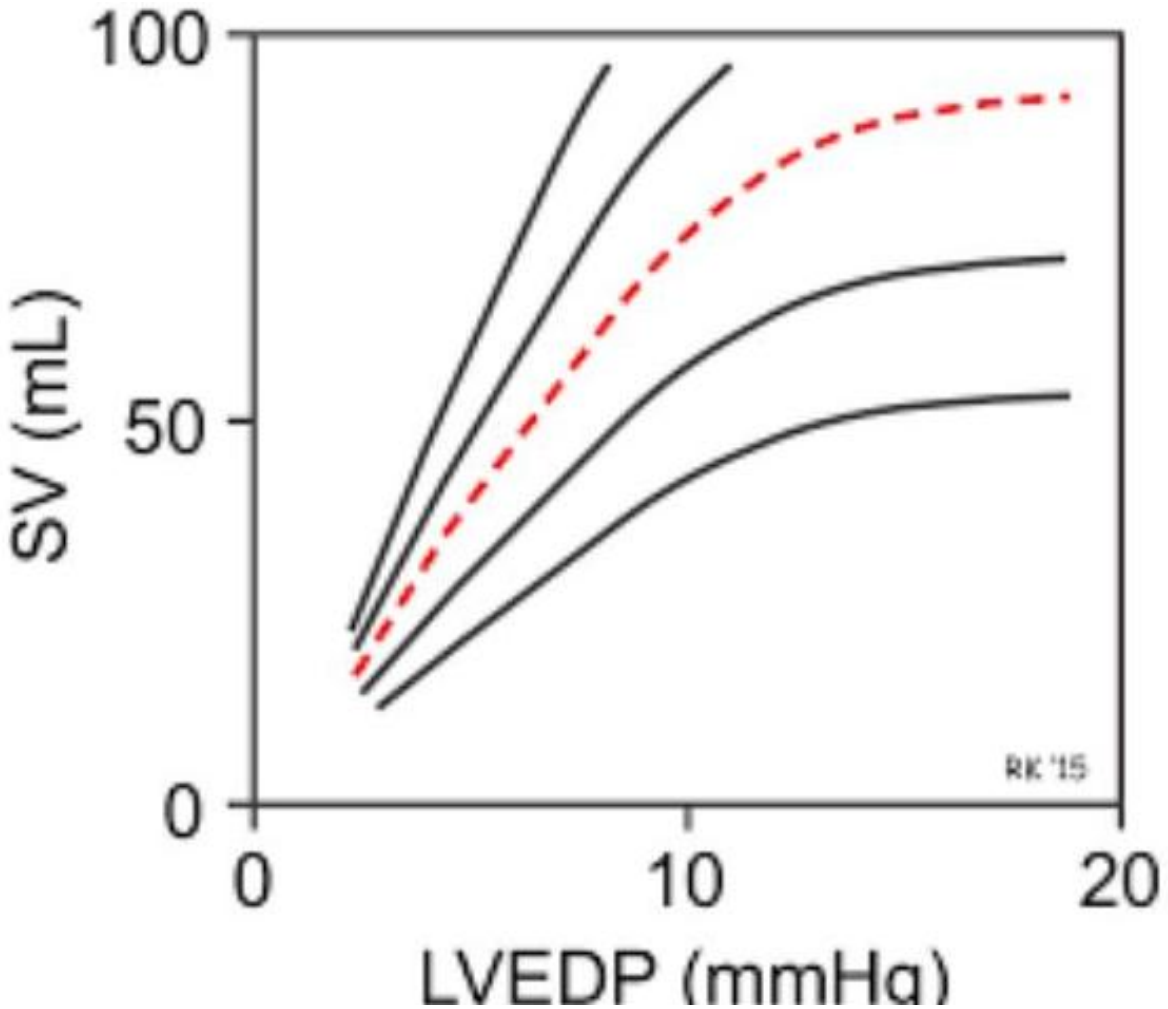
  Slowly accumulation >1L

  Rapid accumulation; 50-100 cc



Ralph Shabetai, M.D. Heart 2004. March 90(3) 255-6.

Defense of the integrity of the Starling curve: Starling mechanism operates uniformly at all intraventricular pressures because of the presence of pericardium



Membranous Function: Shielding the Heart

- Reduction of external friction due to heart movements
- Barrier to inflammation from contiguous structures
- Buttressing of thinner portions of the myocardium

  Atria/ Right ventricle
- Defensive immunologic constituents in pericardial fluid
- Fibrinolytic activity in mesothelial lining

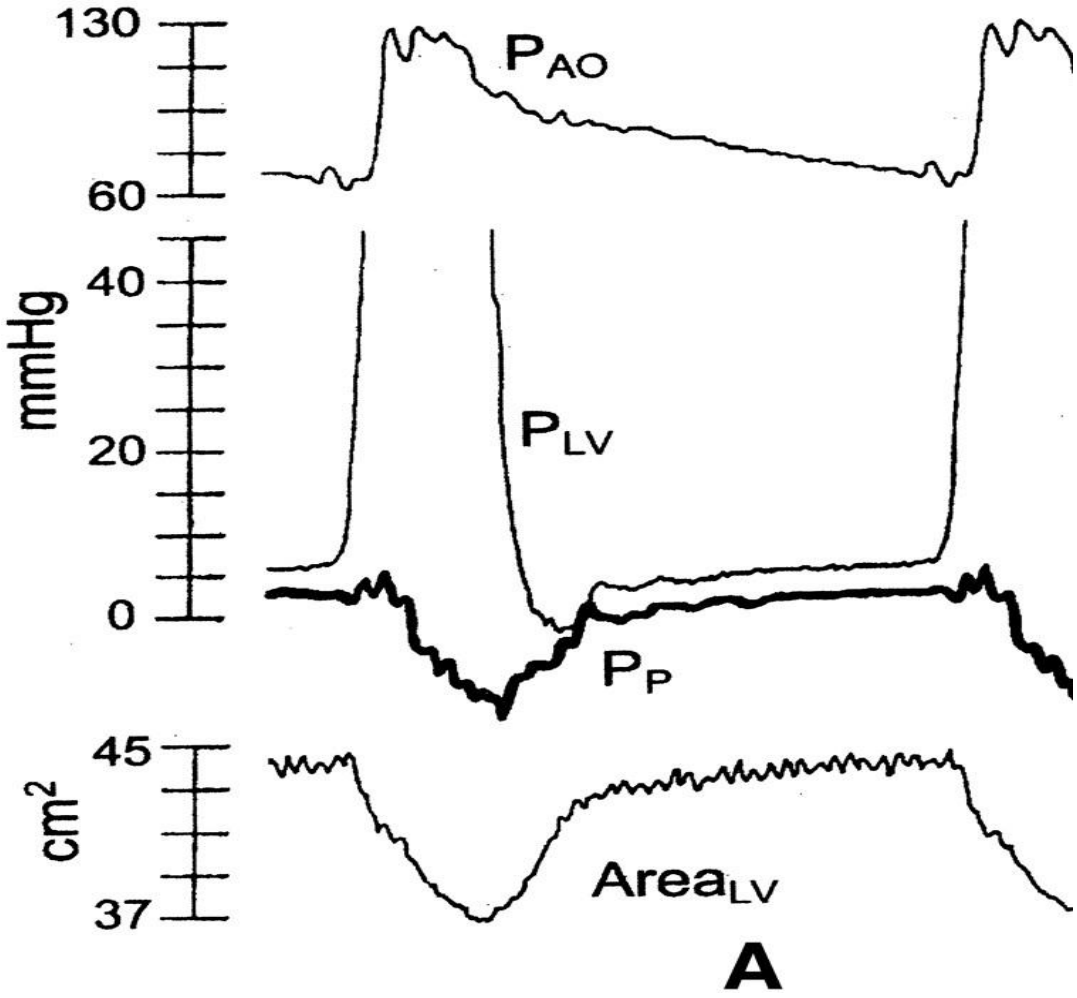
Respiratory-cardiac coupling

- Normal pericardial pressure is subatmospheric (negative)

Transmural pressure ; actual chamber distending (filling) pressure

(Intracavitary pressure) - (Intrapericardial pressure)

Normally negative pericardial pressure produces a distending pressure higher than cavitary pressure



Pericardial pressure (Pp) recorded over the LV free wall using an orthogonal catheter.

Gwyneth deVries et al. Am J Physiol Heart Circ Physiol 2001;280:H2815-H2822

Respirophasic variation

-6mmHg at the end inspiration
-3mmHg at the end expiration
as measured by a fluid filld non-balloon tipped catheter

For example)
Actual filling pressure spontanesously breathing patient
RA pressure 6mmHg
Intrapericardial pressure -6mmHg

6-(-6) mmHg during inspiration

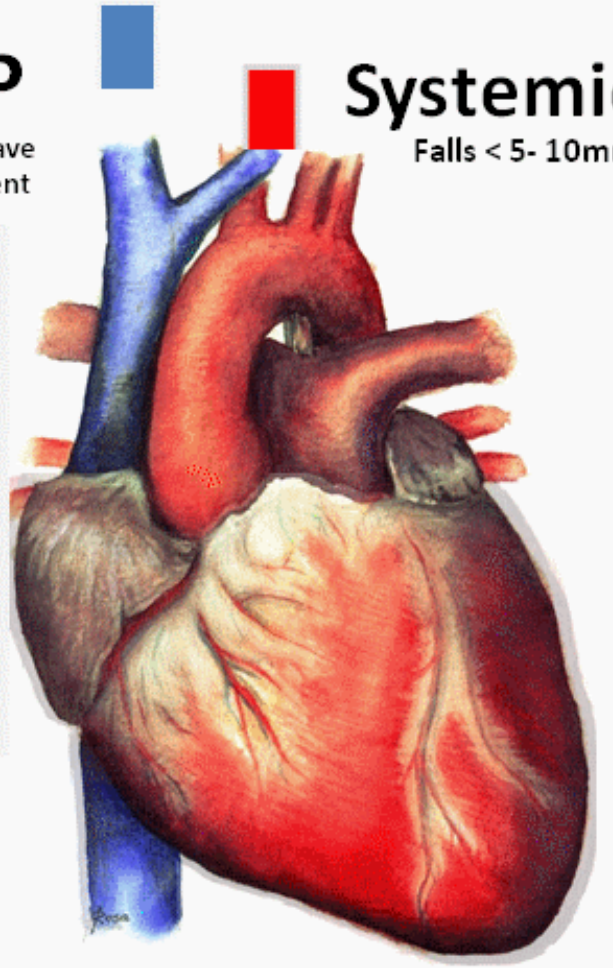
Normal response of JVP and BP to Inspiration

JVP

Mean pressure falls .Wave forms become prominent

Systemic BP

Falls < 5- 10mmhg



Mechanism

Inspiratory transmission of negative Intra thoracic,intra pleural , Intra-pericardial and Intra cardiac pressures help the right heart chambers to suck the blood from extra-thoracic venous reservoir IVC*

*(*Which is uninfluenced by swinging Intra thoracic pressure Hence a positive gradient towards the right heart is created.)*

Mechanism

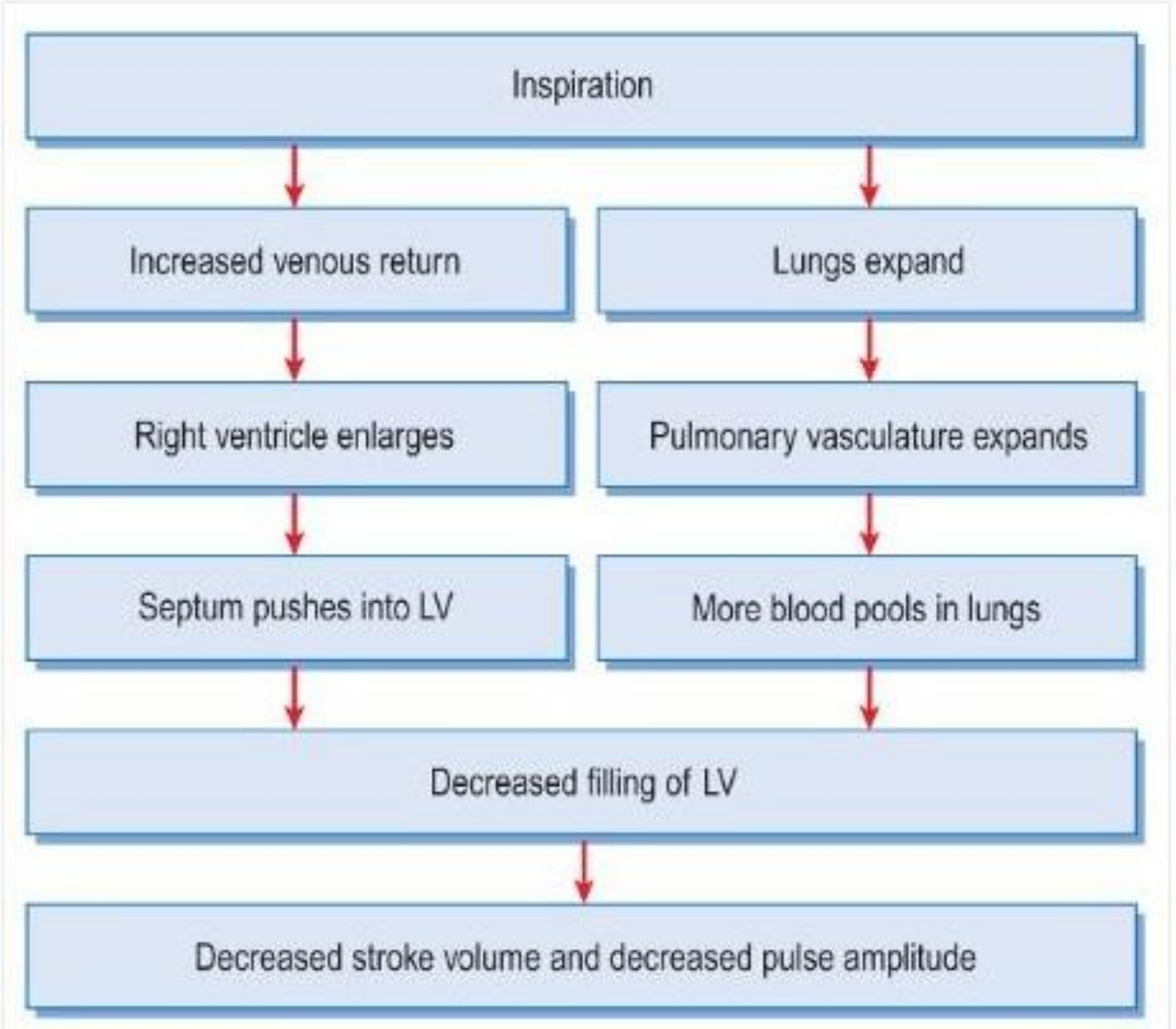
- During inspiration there is relative pooling of blood volume in lungs.

(Note :Since both pulmonary circuit and the left heart are intra-thoracic structures Inspiratory gradient is not established between them unlike right side.)

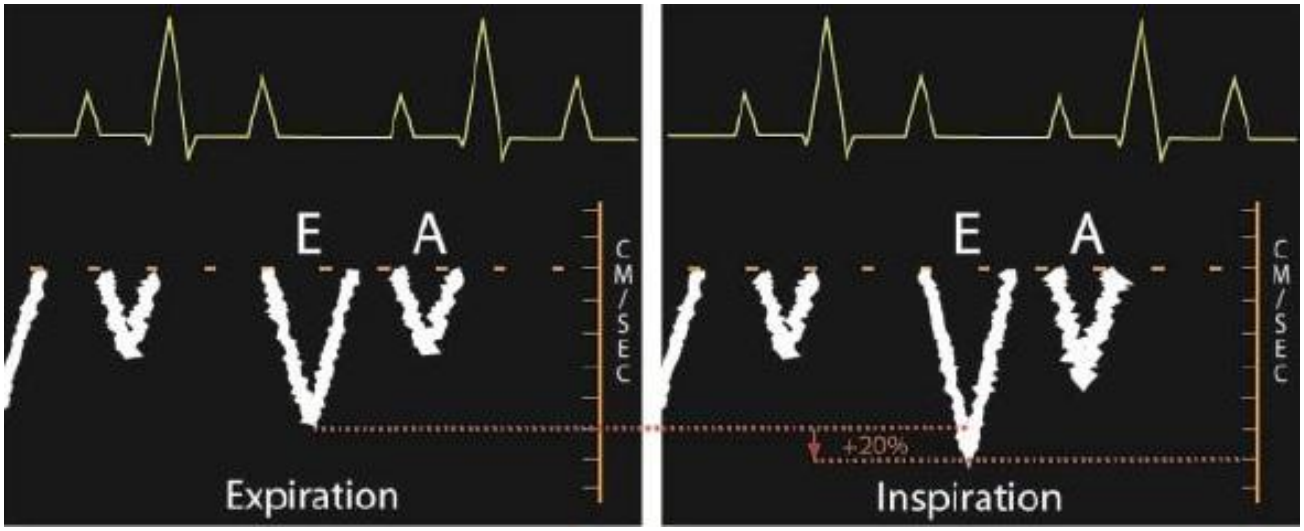
- Septal movement towards LV cavity interferes with LV stroke volume (Reverse Benhiem effect/Ventricular interdependence) Pericardial restraint also augments this.

- Importantly , direct transmission of negative intra-thoracic pressure into the central arteries also contribute to momentary reduction Aortic afterload & BP by few mmhg.

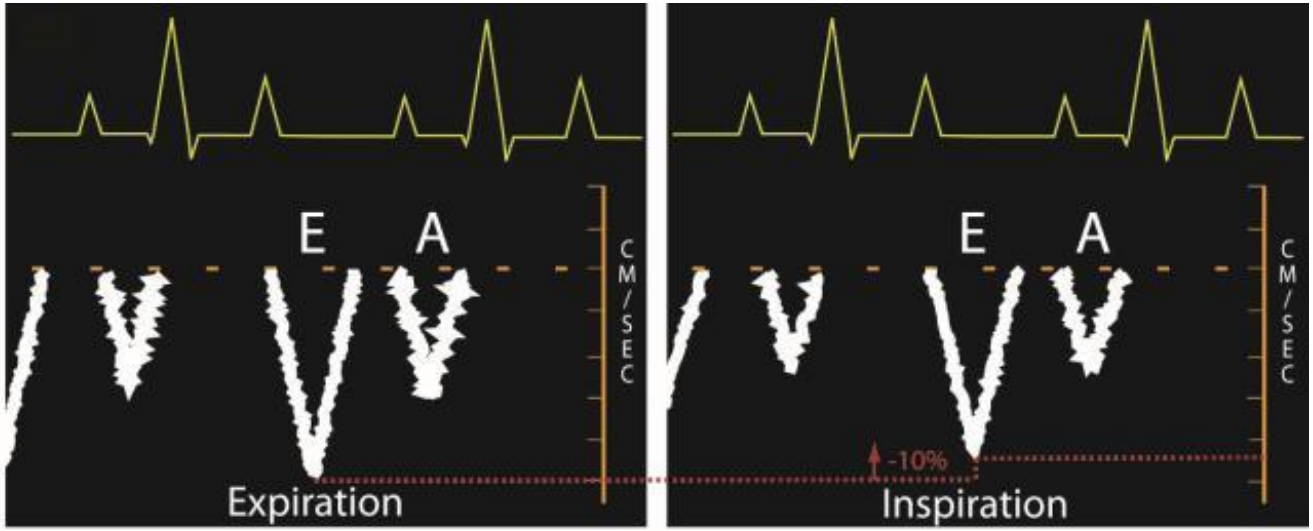
Inspiration


Inspiration
Increased venous return
Lungs expand
Right ventricle enlarges
Pulmonary vasculature expands
Septum pushes into LV
More blood pools in lungs
Decreased filling of LV
Decreased stroke volume and decreased pulse amplitude

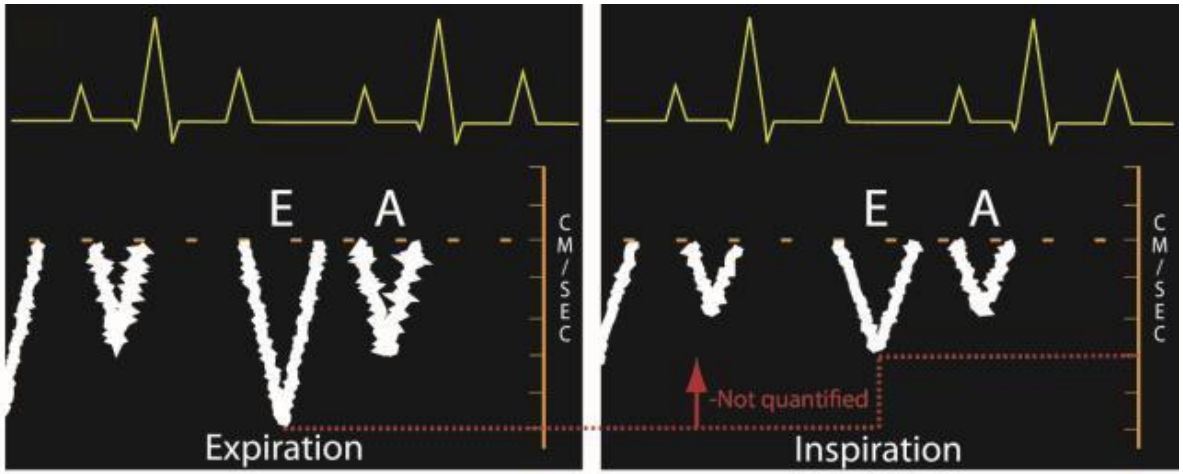
Spontaneous Respiration: Transtricuspid



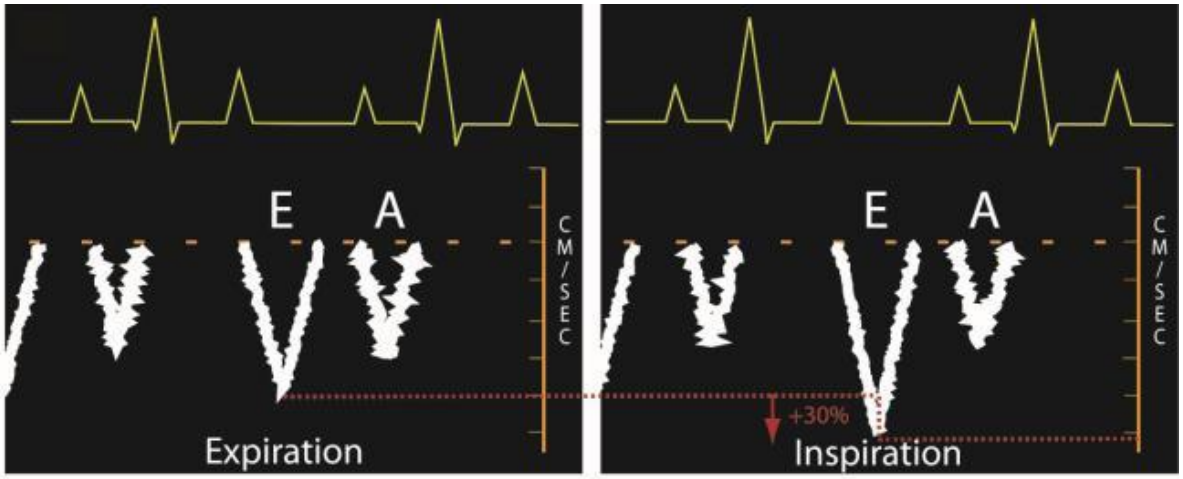
Spontaneous Respiration: Transmitral



IPPV: Transtricuspid



IPPV: Transmitral



- The absolute values of these velocities
    - affected by several physiologic variables
        - age
        - heart rate, rhythm
        - preload, volume flow rate
        - ventricular systolic function, diastolic function
        - atrial contractile function.

The transmission of intrathoracic pressures to the intrapericardial structures appears blunted

- pericarditis or pericardial effusions severe enough to elicit tamponade physiology

TAMPONADE Physiology

Impairment of diastolic filling of the LV during inspiration, caused by abnormally elevated intrapericardial pressure.

With increasing intrapericardial pressure

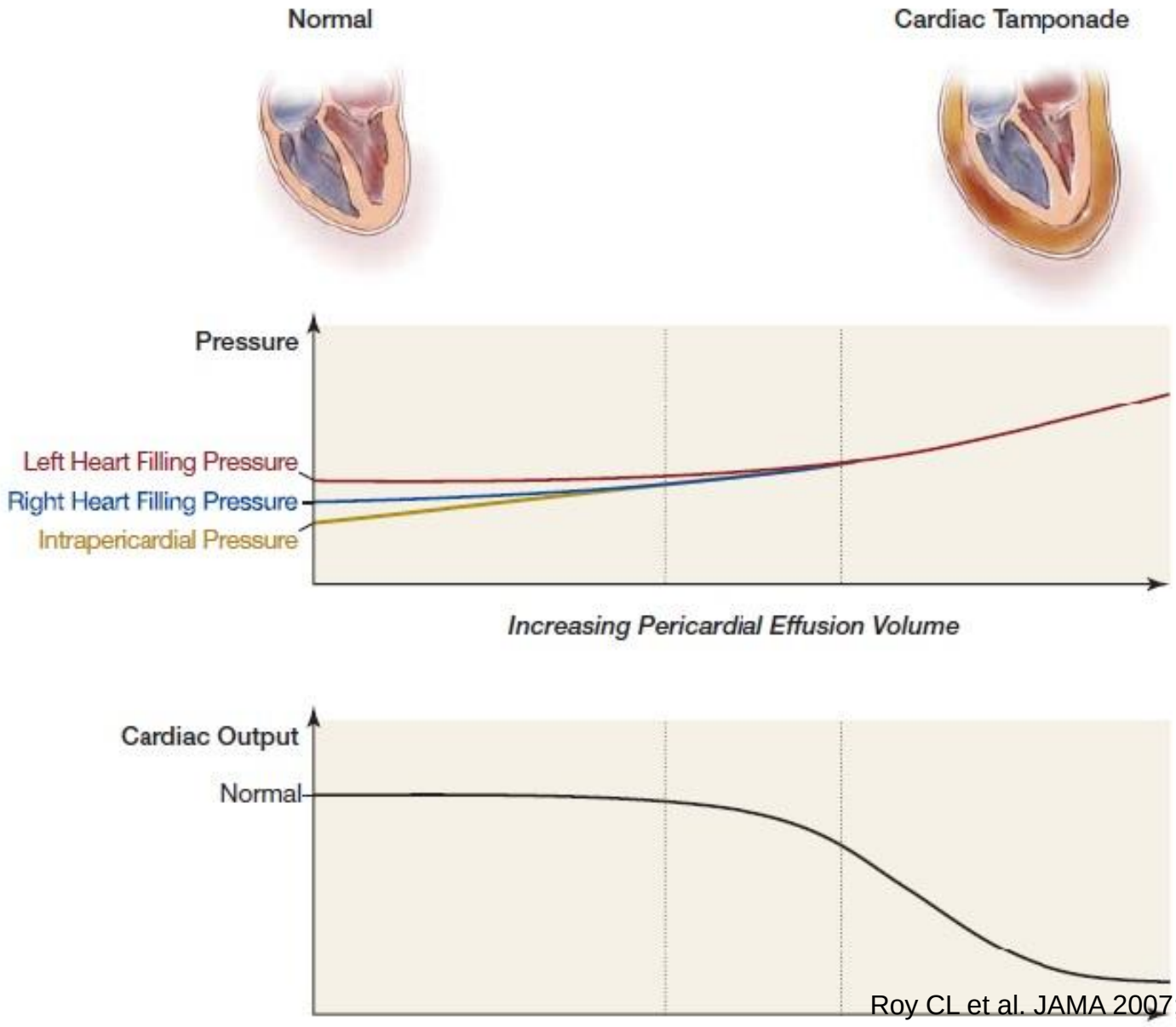
i.e., negative → positive

(Intracavitary pressure) - (Intrapericardial pressure)

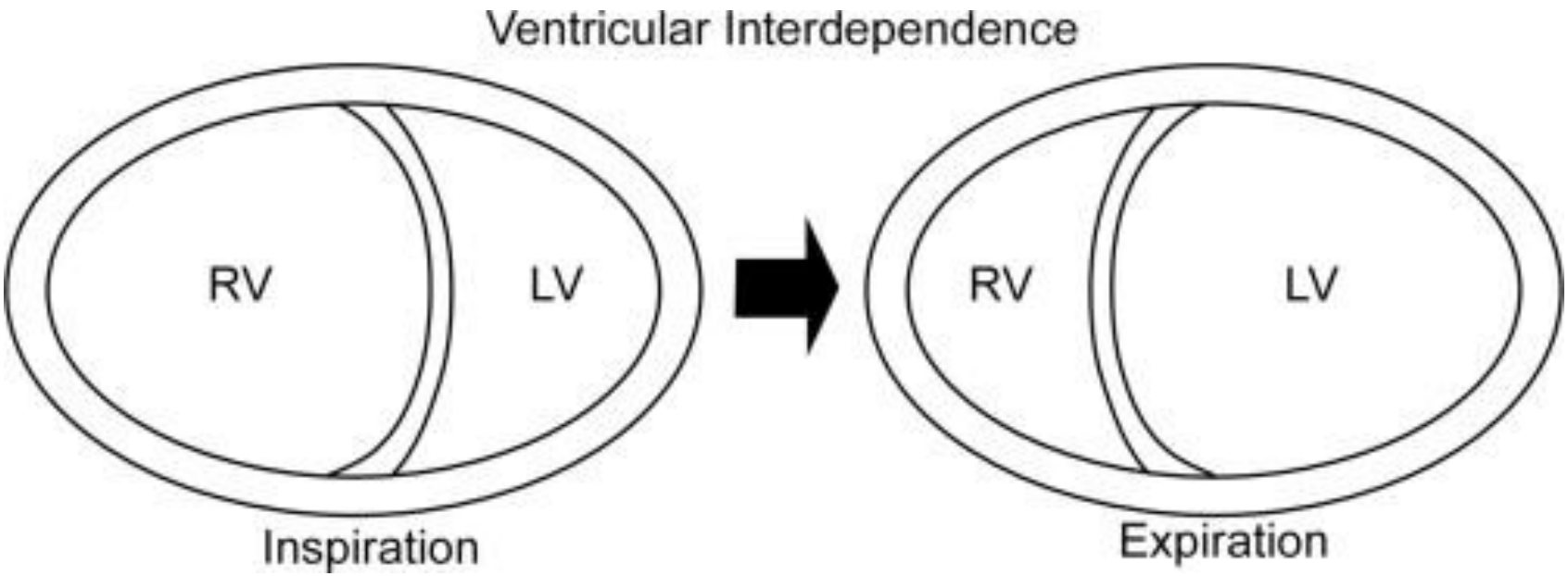
local transmural gradient becomes negative

=> cavity collapse occurs

Relationship Between Intracardiac Filling Pressures and Intrapericardial Pressure and Cardiac Output in Cardiac Tamponade



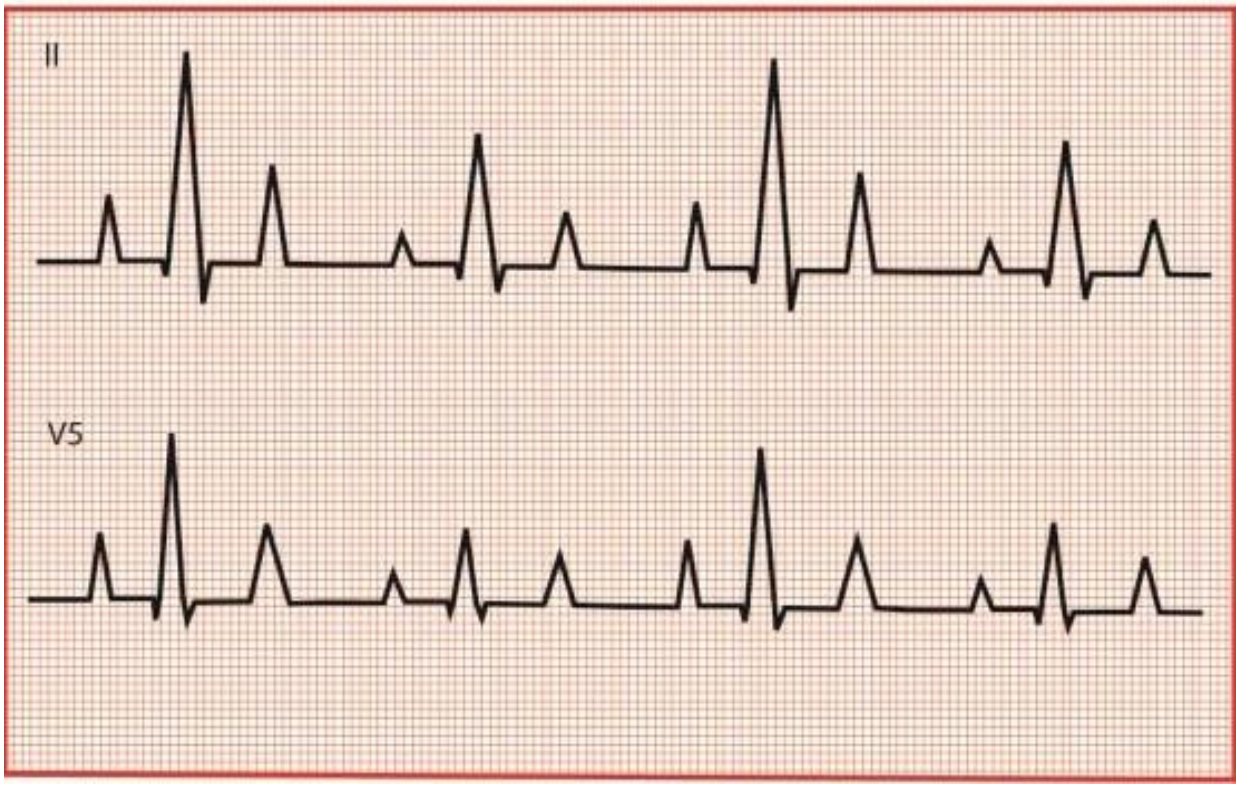
Roy CL et al. JAMA 2007;297:1810-8.

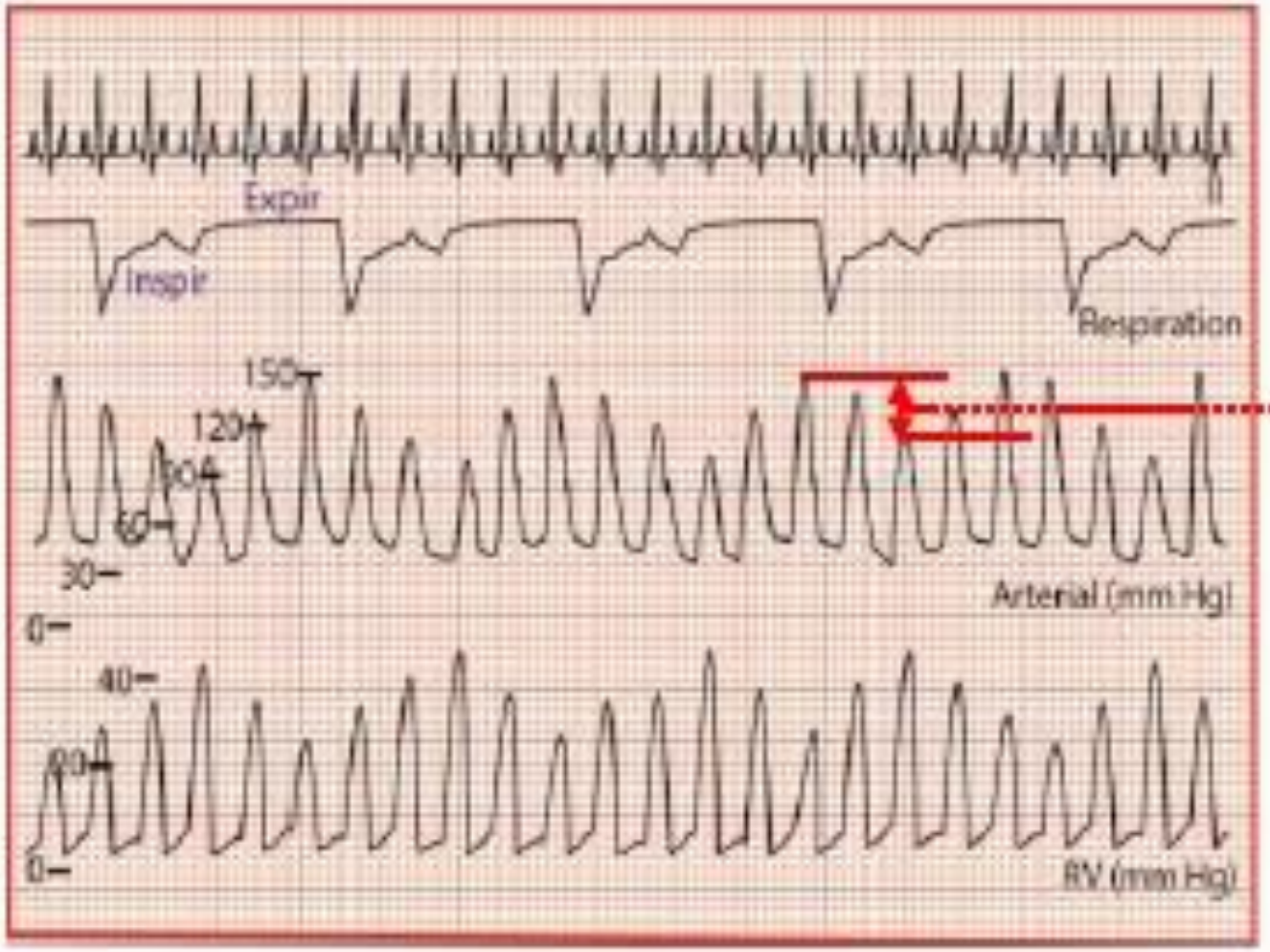

Ventricular Interdependence
RV
LV
RV
LV
Inspiration
Expiration

Filling of the chamber is competitive

- respiratory changes in venous pressure difference alternately favoring RV and LV filling
- Filling of one ventricle and resultant shifting septum impede filling of the other ventricle

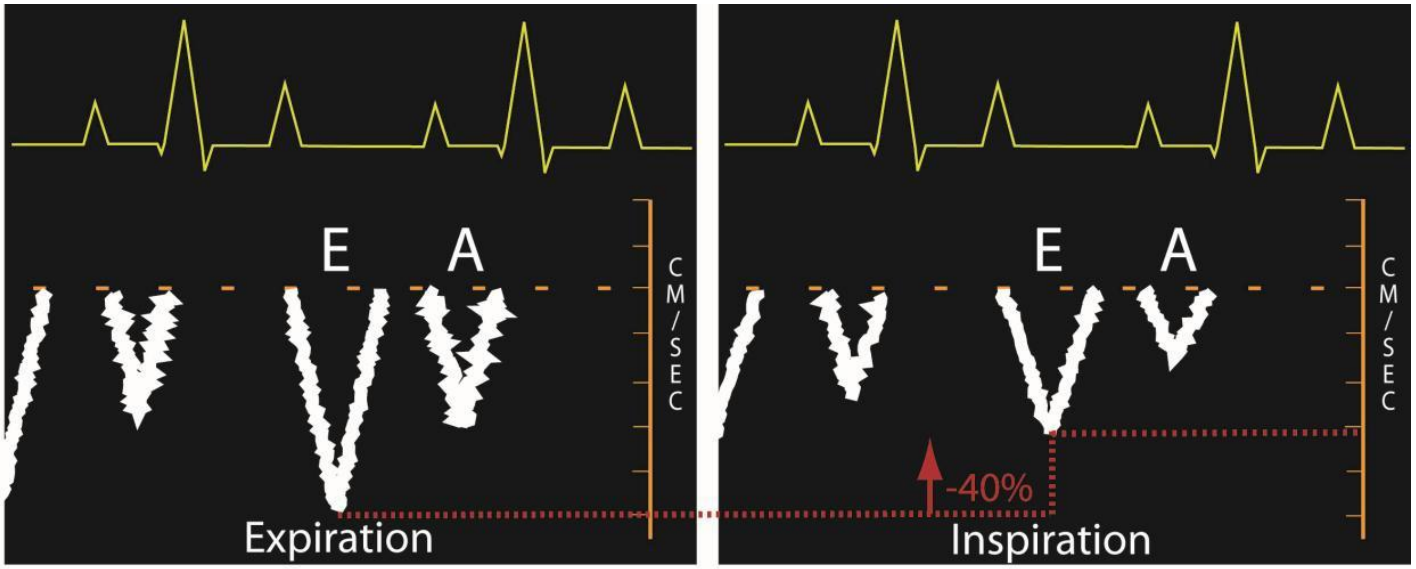
Large, chronic effusion are associated with excessive antero-posterior heart motion as well as counterclockwise rotation in the horizontal plane.
Effusions can lead to cardiac translation within the pericardial space *electrical alternans*



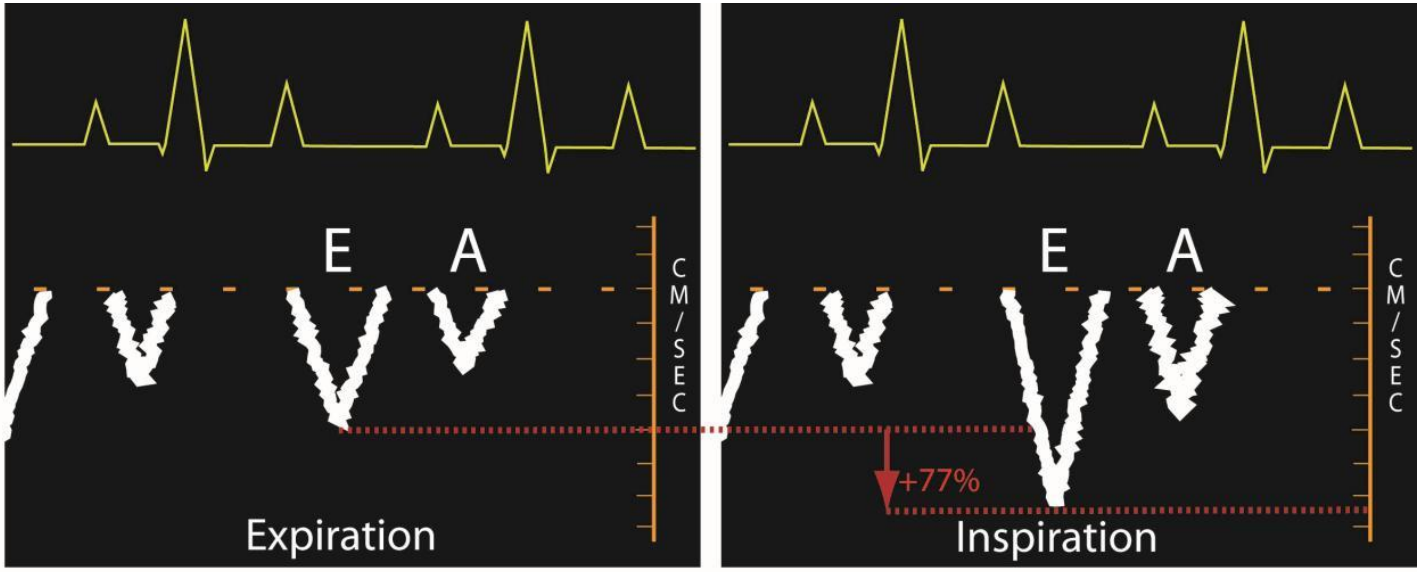


Avery EG 2010[1].

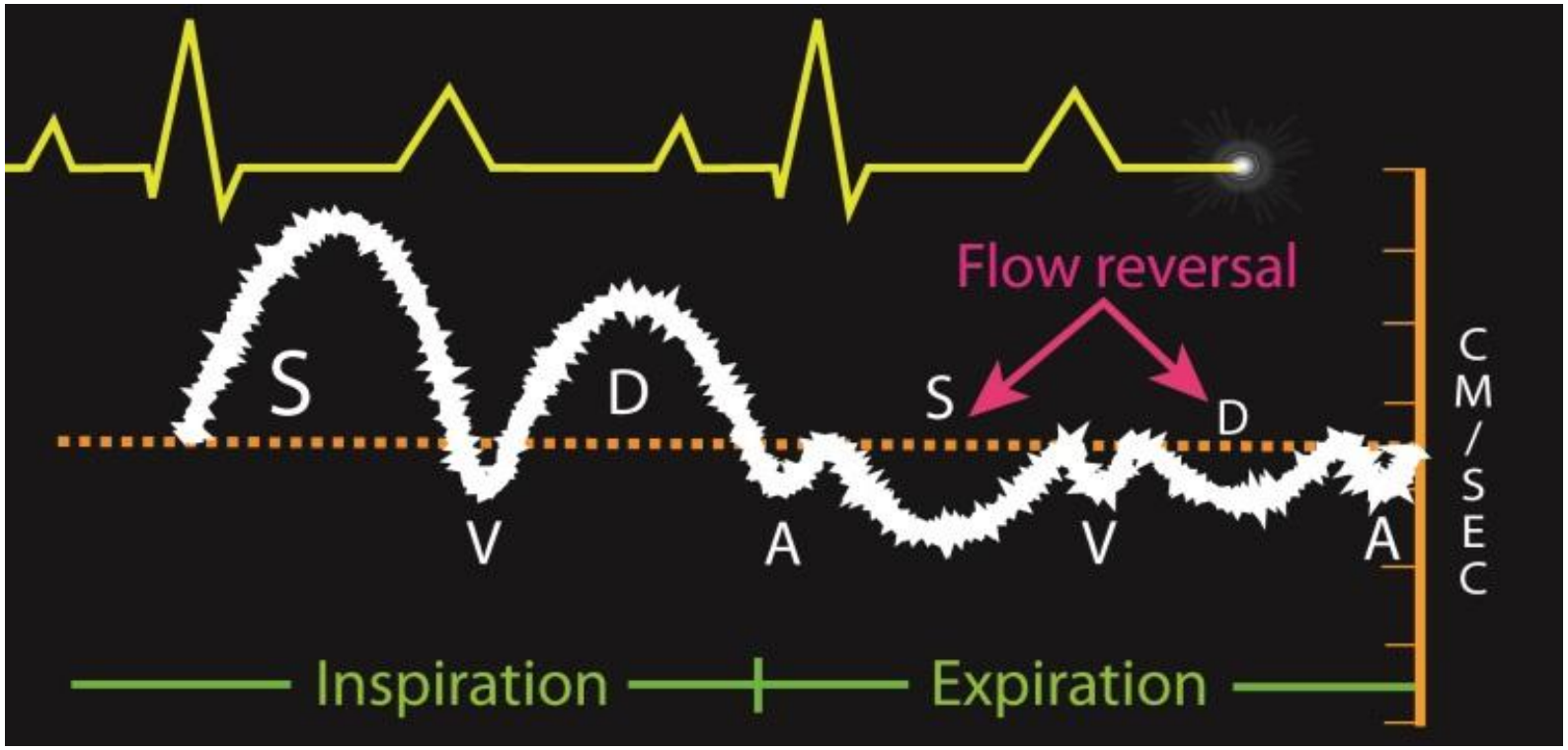
Spont. Respiration: Tamponade – Transmitral



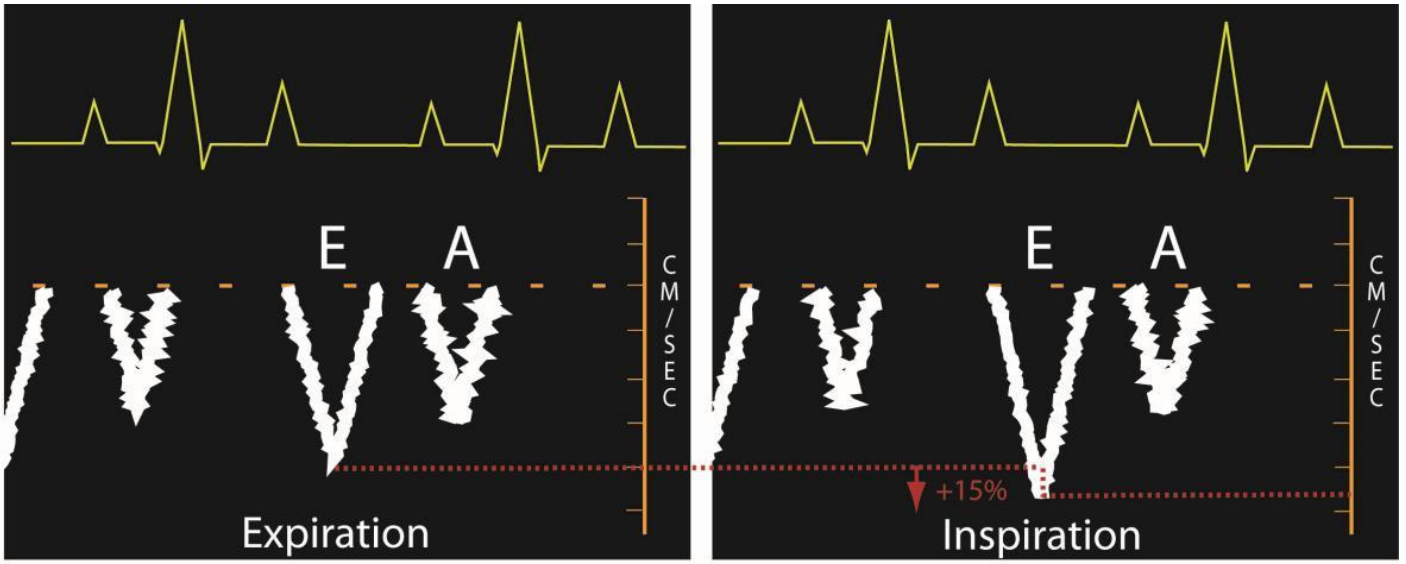
Spont. Respiration: Tamponade - Transtricuspid



Spontaneous Respiration: Tamponade – Transhepatic



IPPV: Tamponade - Transmitral



Percent change in Doppler Flow Velocity with Inspiration

	Pandian[81]	Leeman[83]	Appleton[84]
Control			
Mitral	−10	−8	−4
Tricuspid	17	5	14
Aortic	—	−3	−4
Pulmonic	—	9	5
Effusion			
Mitral	−12	−3	−5*, −31†
Tricuspid	17	21	32*, 74†
Aortic	—	−7	−17
Pulmonic	—	11	49
Tamponade			
Mitral	−42	−35	E −43 ± 9%; A −28 ± 12%
Tricuspid	117	80	E 85 ± 53%; A 58 ± 25%
Aortic	—	−33	−26
Pulmonic	—	86	40 ± 25%
Left ventricular isovolumic relaxation time	—	—	85 ± 14%
Left ventricular ejection time	—	—	−21 ± 3%