Cardiovscular Changes with Ventilation

**During Regular Breathing**

When the diaphragm contracts, the intrapleural pressure (Pip) decreases. This reason for this Is the volume expansion in the pleural cavity, which decreases pressure in accordance with boyle’s law. When Pip decreases, the transmural

**Differences between spontaneous- and mechanical breathing**

Fig 2 shows the difference in the waveforms between spontaneous breaths and breaths induced by MV.

In spontaneous breathing, the waveform oscillates between positive and negative pressure. The reason for this, is that when the diaphragm contracts, the decreased Pip causes the lungs to expand, and thus pressure in the lungs falls below atmospheric pressure. Conversely, during expiration, the volume of the lungs decreases, which in turn creates positive pressure of the lungs.

During positive pressure MV, the pressure is solely provided by the MV. This means, that the lungs don’t inflate in response to a negative Pip, but as a result of the added pressure, in accordance with the patient’s Crs.

**A diagram of a mechanical breakdown

Description automatically generated**

*Fig 2 – Differences in waveforms of spontaneous and mechanical breaths*

**End-Systolic and End-Diastolic pressure-volume relationships**

Fig 3 shows the pressure volume loops for the LV during the cardiac cycle. It illustrates, that during diastole when the mitral valve (MTV) opens, the LV is filled with blood. Then, the MTV closes, and a contraction without change in volume opens the aortic valve (AV), initiating systole. Then, due to increase in LV pressure. blood flows from the LV into the aorta, decreasing LV volume and increasing pressure. The AV closes, and the LV slowly expands, decreasing pressure, until the MTV again opens. The area from C to D represents stroke volume (SO).

**Diagram of a pressure volume loop

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***Fig 3 Presure volume loop for the left ventricle. A) Denotes opening og the mitral valve, B) Denotes closening of the mitral valve, C) Denotes Opening of the aortic valve and D) denotes closening of the aortic valve***

Fig 4 shows the End-Systolic (Esv) and End-Diastolic (Ed) pressure-volume relationships, and the effects of afterload on these relationships.

The top dotted line represents Esv. During normal pressure, Esv lies around 110mmHg. However, when afterload increases, Esv increases to ~140mmHg, and subsequently at decreased afterload, Esv decreases to ~70mmHg,

Ed remains the same iif the initial preload condition is unchanged. However, The area from C to D increases at decreased afterload, and reversely at increased afterload. This mechanism shows, that afterload directly effects SO.

*A diagram of a diagram showing the volume of a patient

Description automatically generated with medium confidence  
Fig 4 Illustration of the End-Sytolic and End-Diastolic pressure-volume relationships, and the effects of afterload on these*

For the same diastolic conditions, and the same preload conditions, afterload will be proportional to cardiac output.

Conversely, at the same conditions, changes in preload will affect changes in cardiac output.

**LV preload and mechanical ventilation**

1. Decreased RV preload will decrease LV preload. The underlying mechanism for this interaction, is that the volume of blood returned to the RV through the vena cava (VC), is the volume returned to the LV after pulmonary ventilation.
2. Increased RV afterload decreases LV preload. The reason being, that the higher pressure needed to pump blood from the RV into the pulmonary vein, the less volume will go through pulmonary ventilation, resulting in less LV preload.