

Pressure - Volume Loop of the left ventricle may be used to derive many values. This is frequently a question in exams and vivas.

Cardiac Cycle Events

Valve Openings and Closings (A = MV opening, B = MV closes, C = Aortic Valve opens, D = Aortic Valve closes)
 Isovolaemic Contraction and Relaxation, systolic blood ejection and diastolic ventricle filling

Measurable Values

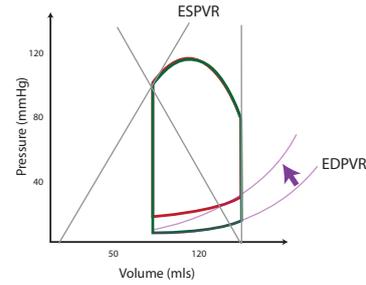
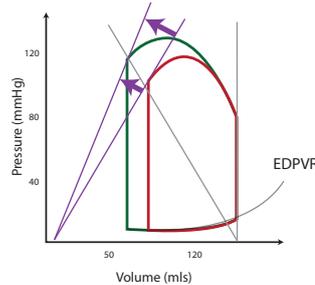
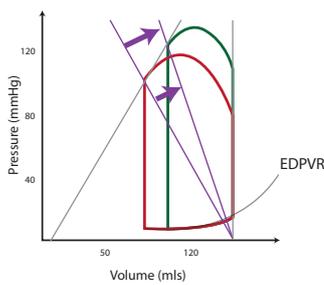
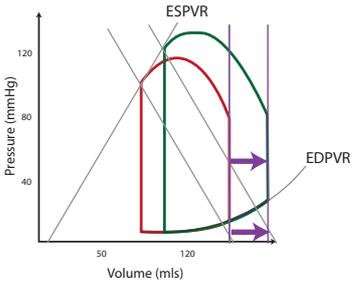
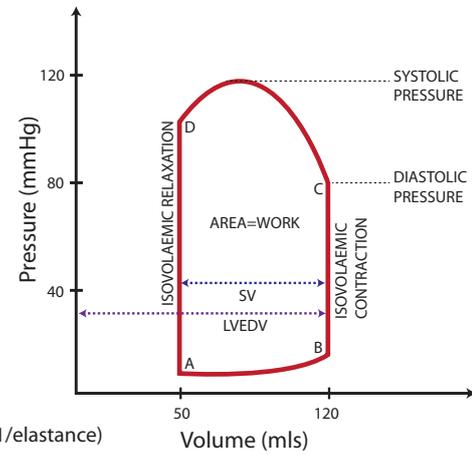
The **Diastolic** and **Systolic pressures**,
 The **Stroke Volume (SV)**
Left Ventricular End Diastolic Volume (LVEDV)

Derived values

The area of the loop represents **External Work**
Ejection Fraction = SV/LVEDV

Surrogate Markers (see below)

The LVEDV point on the abscissa (x-axis) represents a surrogate marker of **Preload**
 The **Afterload** is the angle formed between the Preload and the End Systolic Point when AV closes (D)
 The **Contractility** is the angle formed by the End Systolic Pressure Volume Relationship ESPVR.
 The **Elastance** relates to the End Diastolic Pressure Volume Relationship EDPVR, **compliance** may be inferred (1/elasticity)



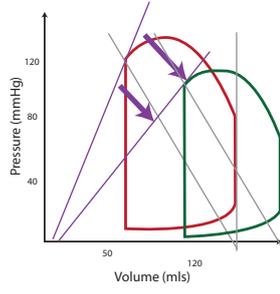
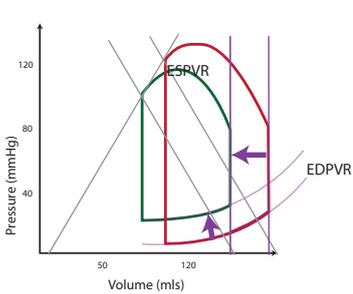
Increased Preload is demonstrated by a shifting along the EDPVR curve resulting in an increased LVEDV. The afterload and contractility is unchanged.

Increased Afterload note the increase in the angle of the afterload line. The contractility is the same (ESP on same line) the preload is the same because the LVEDV point is the same.

Increased Contractility note that the ESP is not on the same line. The preload is the same (LVEDV is the same) and the afterload is also constant (the afterload lines are parallel).

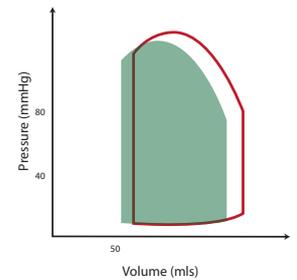
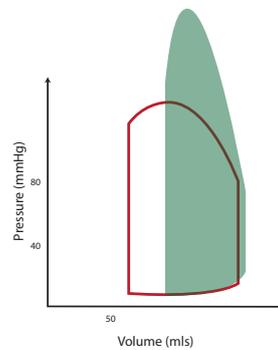
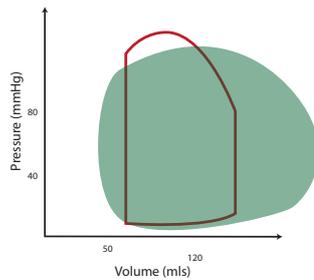
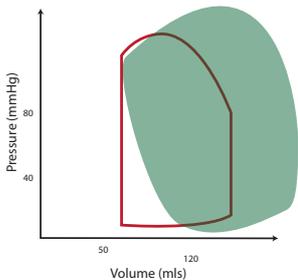
Increased Elastance by definition the elastance is the $\Delta P / \Delta V$ ie the slope of the EDPVR. Often this is erroneously referred to as compliance in several textbooks. The End Diastolic Pressure Volume Relationship is steepened, but other values held constant.

Disease States



Diastolic Heart Failure due to LVH causes a **decreased compliance** (represented by increased elastance) and **decreased LV filling**. The result is a new EDPVR curve and reduced preload. Afterload and contractility may be unchanged and ejection fraction is often maintained.

Systolic Heart Failure the primary pathological process is a **loss of inotropy (contractility)**. The heart compensates by increasing preload. The result is lower systolic and diastolic pressures and a decreased EF. Compliance is not necessarily changed nor is afterload.



Aortic regurgitation there is a **loss of isovolaemic relaxation** as the blood regurgitates. This leads to an increased LVEDV (and therefore preload). In accordance with the Frank Starling mechanism this leads to increased pressures hence the raised systolic pressure.

Mitral regurgitation there main pathological proces is a **loss of isovolaemic contraction** as the blood regurgitates. As some of the blood backflows, there is increased filling in distole therefore increased LVEDV. Unlike AR however the systolic pressure is reduced due to only part of the blood ejecting from the aorta.

Aortic stenosis the main pathological problem is increased LV resistance to ejection and subsequent **increase in afterload**. The result is a wide pulse pressure with a high systolic pressure. The heart may compensate, but if the AS is severe the result will be a decrease in SV.

Mitral Stenosis the main pathological process is an **impairment of LV filling**. This leads to **decreased Preload** and by the F-S Mechanism reduced CO and aortic pressure (the accompanying decrease in afterload partially compensates).