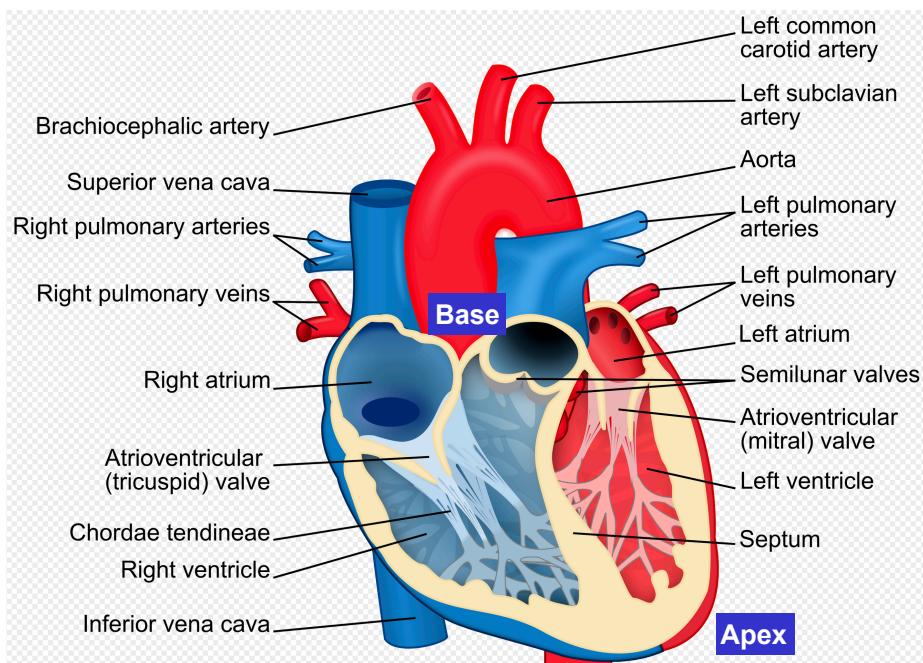


Ventricular Pump Function

- Cardiac Functional Anatomy
- Cardiac Cycle
- The Wiggers Diagram
- Ventricular Pressure-Volume Relations
- Effects of Altered Preload and Afterload
- Contractility/Inotropy
- Ventricular-Vascular Coupling
- Ventricular Energetics

Cardiac Functional Anatomy



The Cardiac Cycle

Defined by valve events

Systole:

1. Isovolumic contraction
2. Ejection

Diastole:

3. Isovolumic relaxation
4. Filling

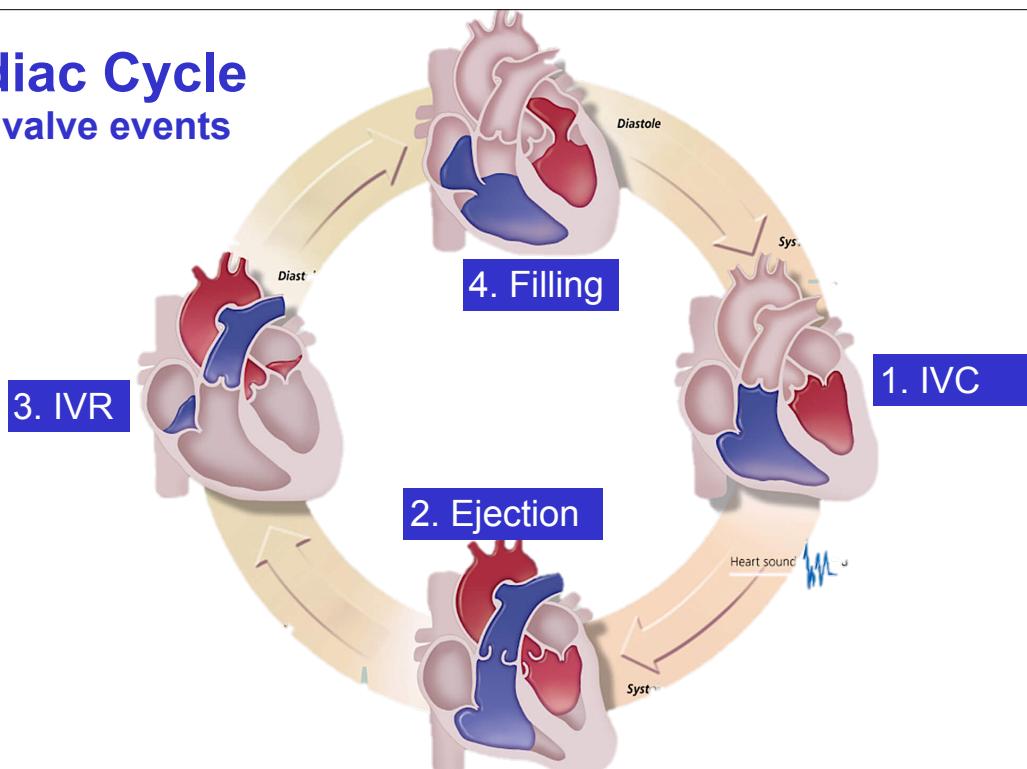
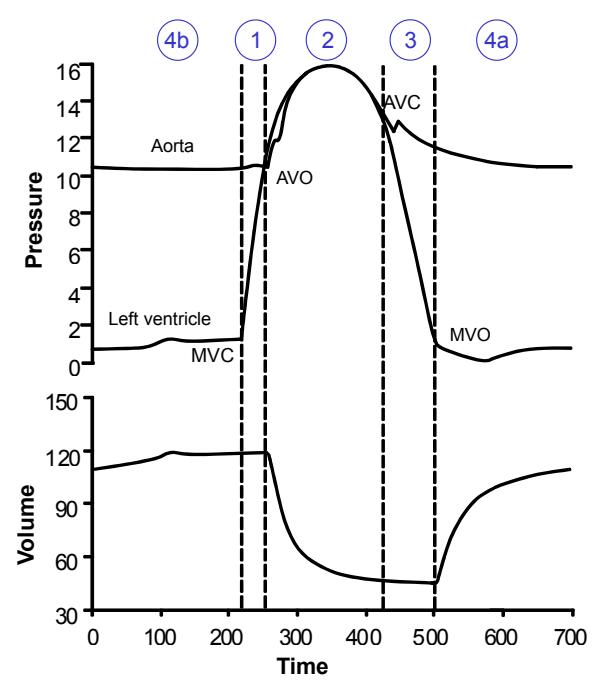
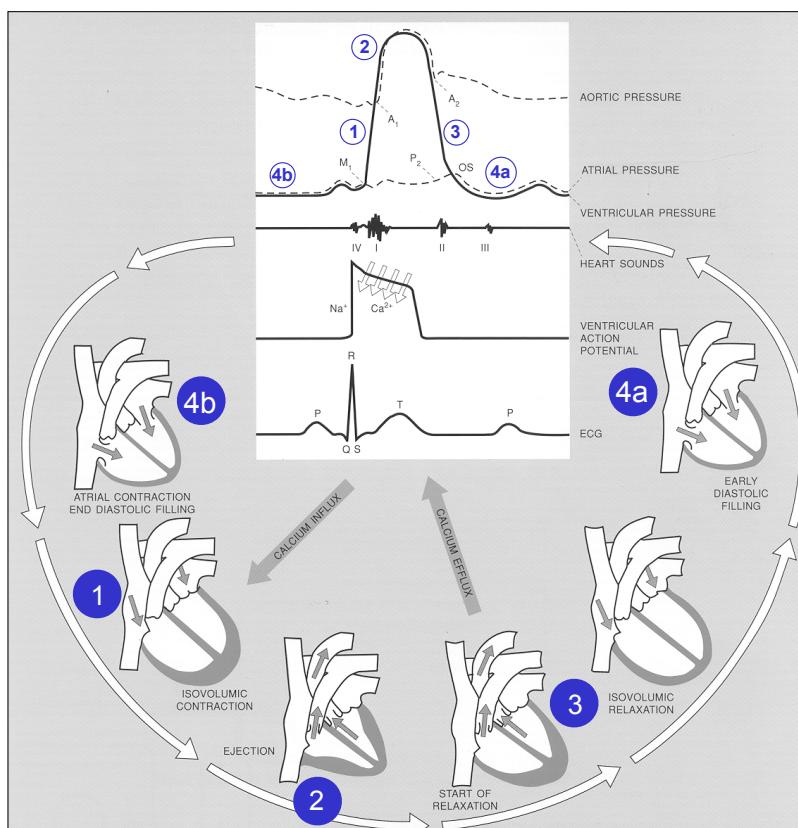


Figure modified from Scientific Publishing
Physiology series



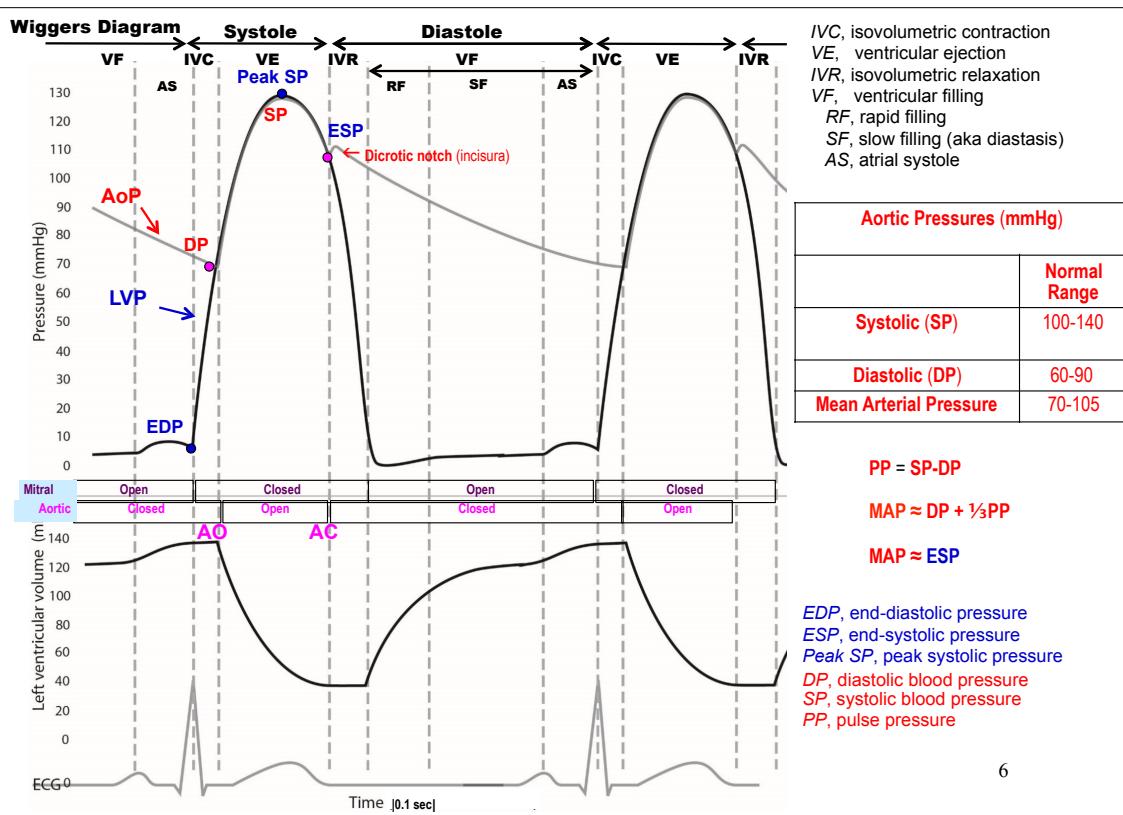
Wiggers Diagram



Carl J. Wiggers (1883-1963)

- Physician and physiologist
- Developed diagram for teaching cardiovascular physiology at Case Western Reserve School of Medicine

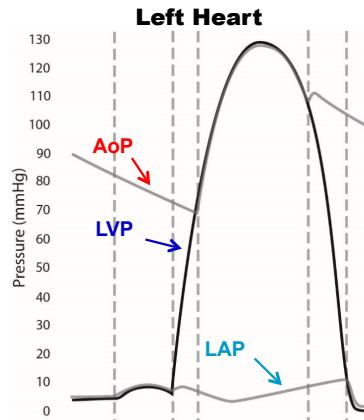
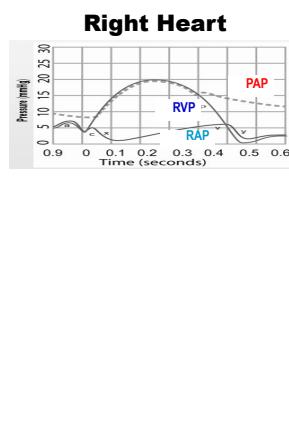
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6

Wiggers Diagram

Right Heart vs. Left Heart



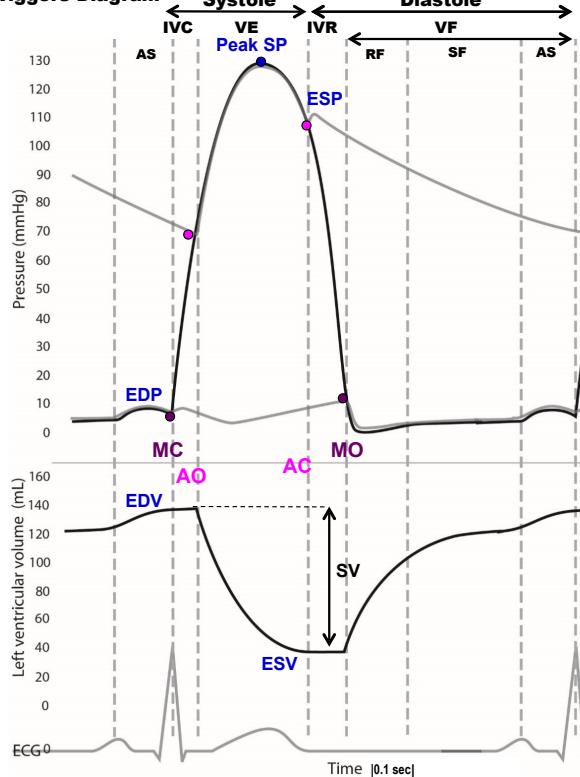
Great Vessel Pressures (mmHg)		
	PAP Normal Range	AoP Normal Range
Systolic (SP)	15-30	100-140
Diastolic (DP)	4-12	60-90
Mean	9-18	70-105

Ventricular Pressures (mmHg)		
	RVP Normal Range	LVP Normal Range
Peak systolic	15-30	100-140
End-diastolic (EDP)	2-8	3-12

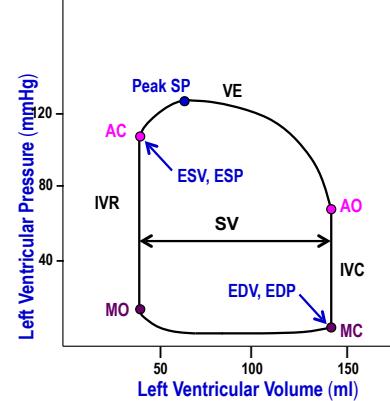
Atrial Pressures (mmHg)		
	RAP Normal Range	LAP Normal Range
a wave	2-10	3-15
v wave	1-10	7-15
Mean	2-8	2-10

Wiggers Diagram

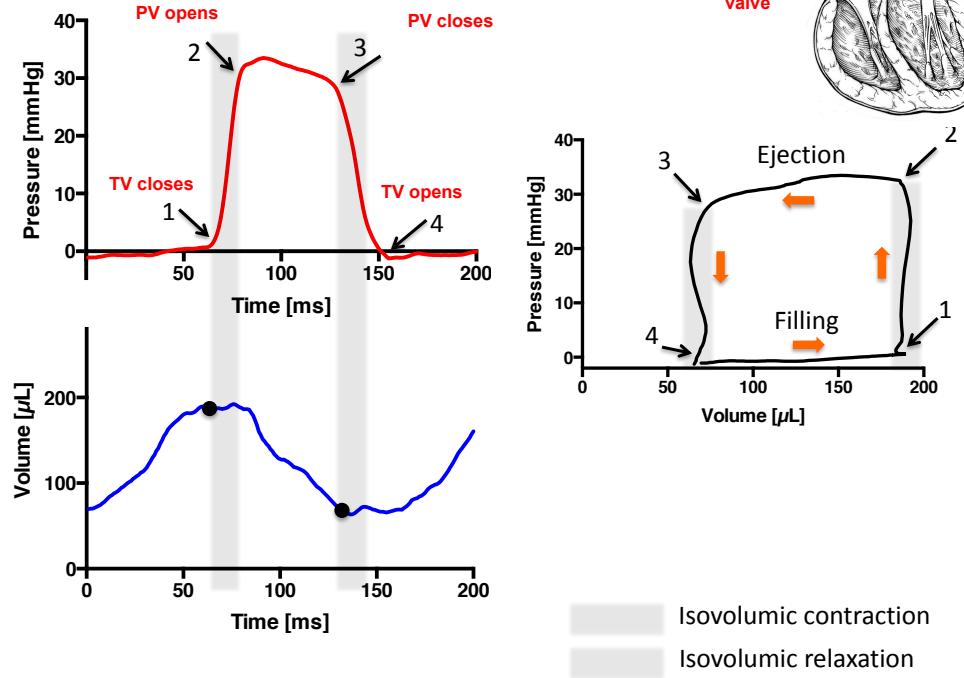
Systole Diastole



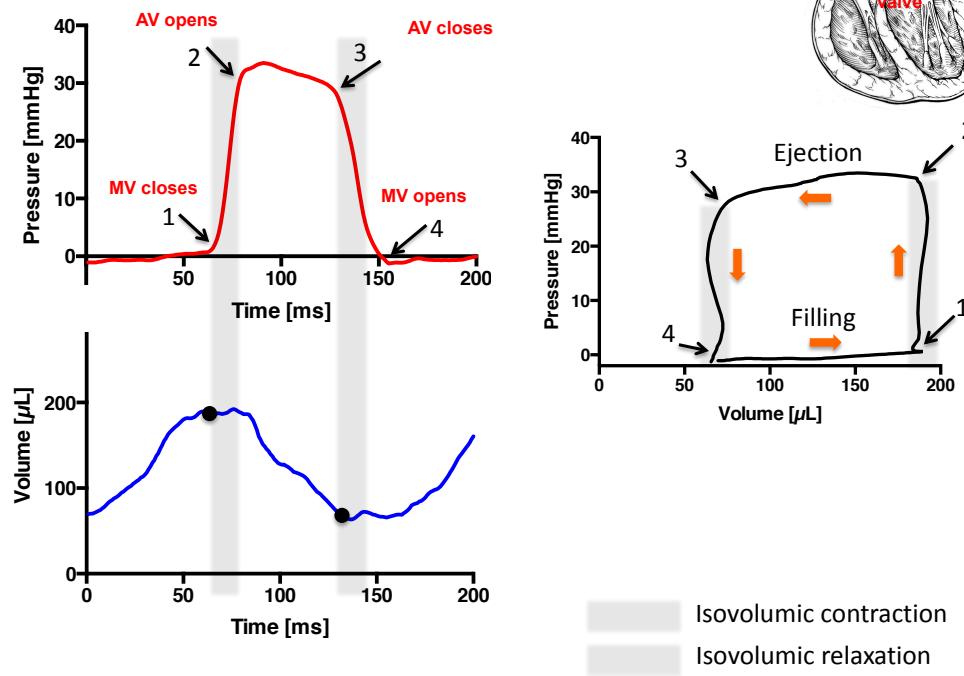
Pressure-Volume (PV) Loop



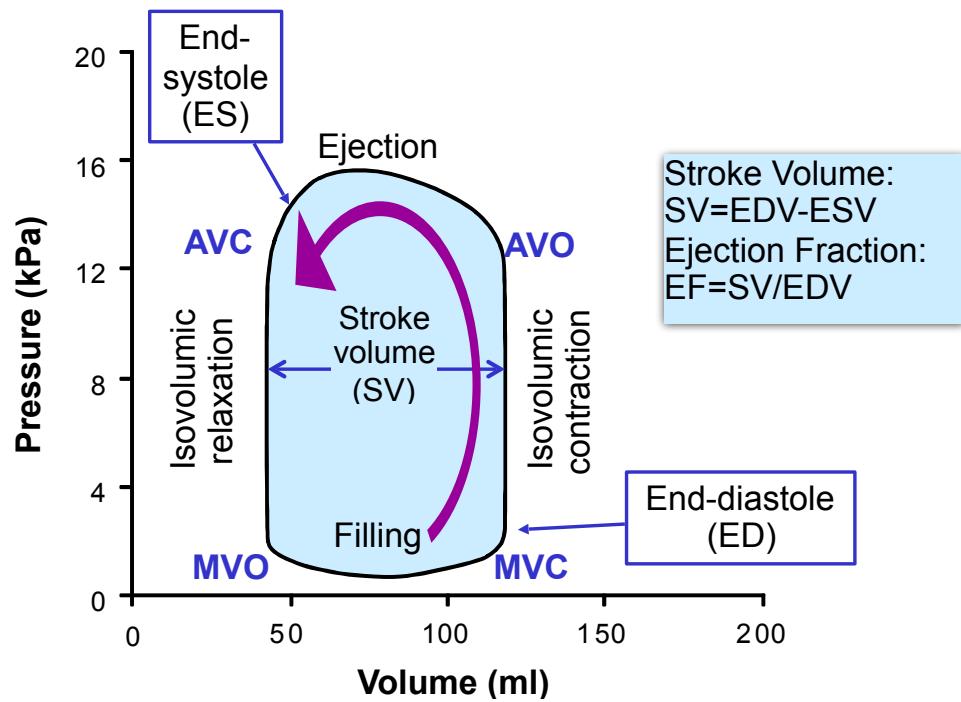
RV Hemodynamics



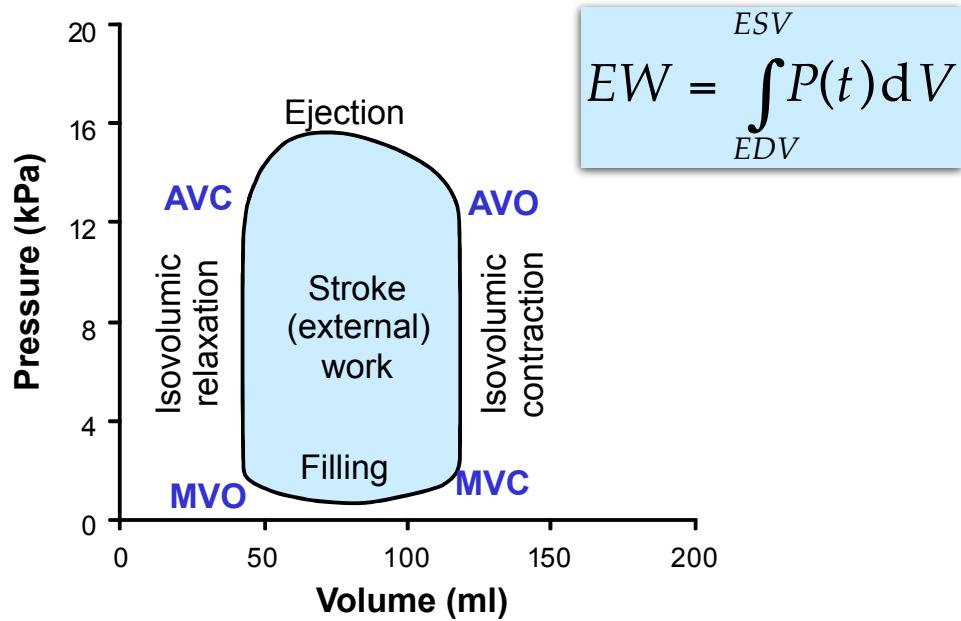
LV Hemodynamics



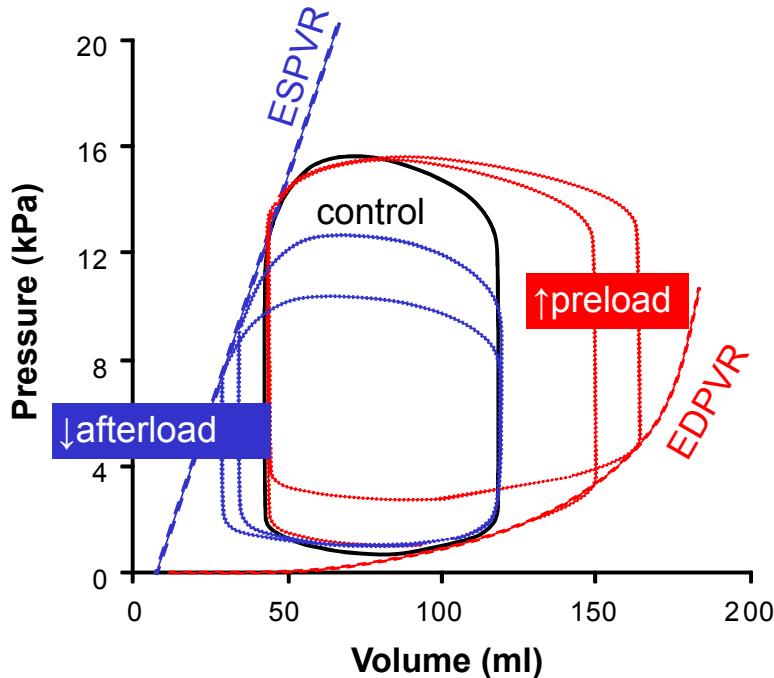
The Pressure-Volume Diagram



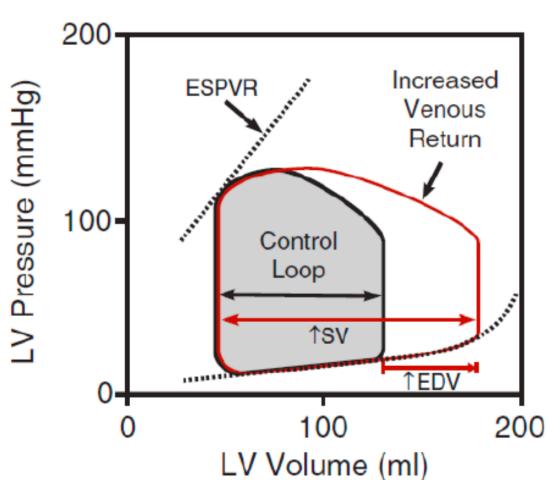
The Pressure-Volume Diagram



Preload and Afterload



Effects of Increasing Venous Return on LV Pressure-Volume Loops

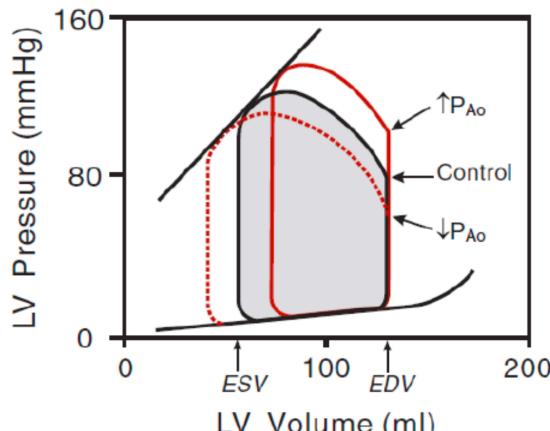


Source: Klabunde, RE, [Ventricular Pressure-Volume Relationship](#)

- This diagram shows the acute response to an increase in venous return.
- It assumes no cardiac or systemic compensation and that aortic pressure remains unchanged
- Increased venous return increases end-diastolic volume (EDV) but it normally does not change ESV; therefore, stroke volume (SV) is increased.

ESPVR, end-systolic pressure-volume relationship.

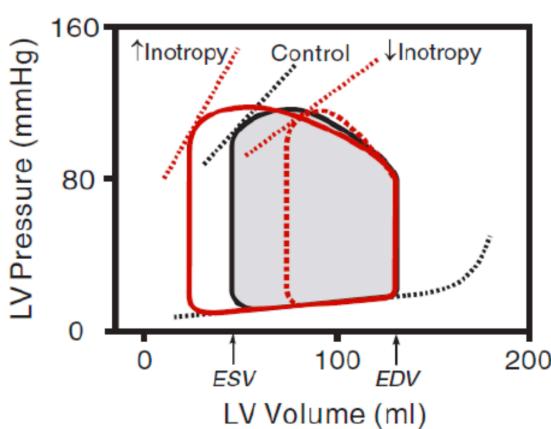
Effects of Changes in Afterload on LV Pressure-Volume Loops



Source: Klabunde, RE, [Ventricular Pressure-Volume Relationship](#)

Increased aortic pressure (solid red loop) decreases stroke volume (width of loop) and increases end-systolic volume (ESV), whereas decreased aortic pressure (AO dashed red loop) increases stroke volume and decreases end-systolic volume. Preload and inotropy are held constant in this illustration.

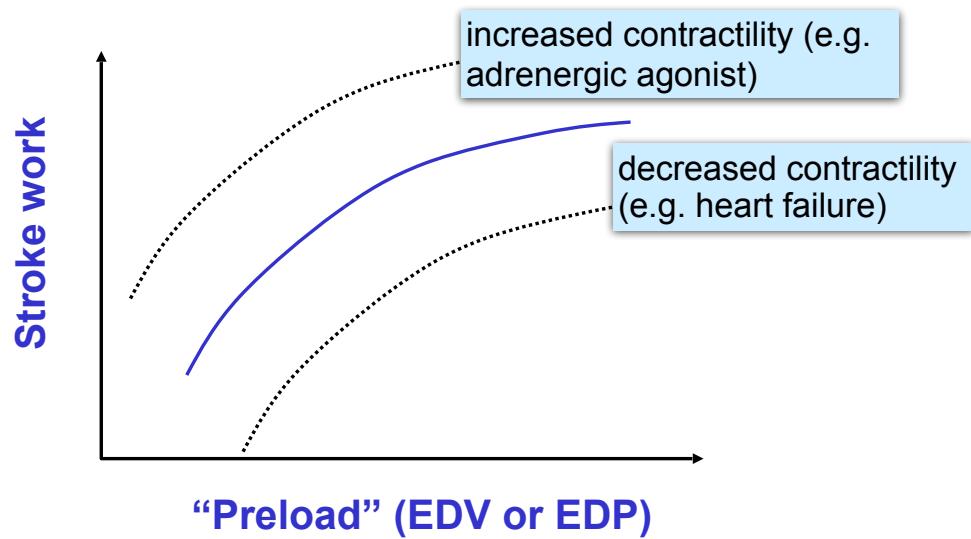
Effects of increasing contractility on LV Pressure-Volume Loops



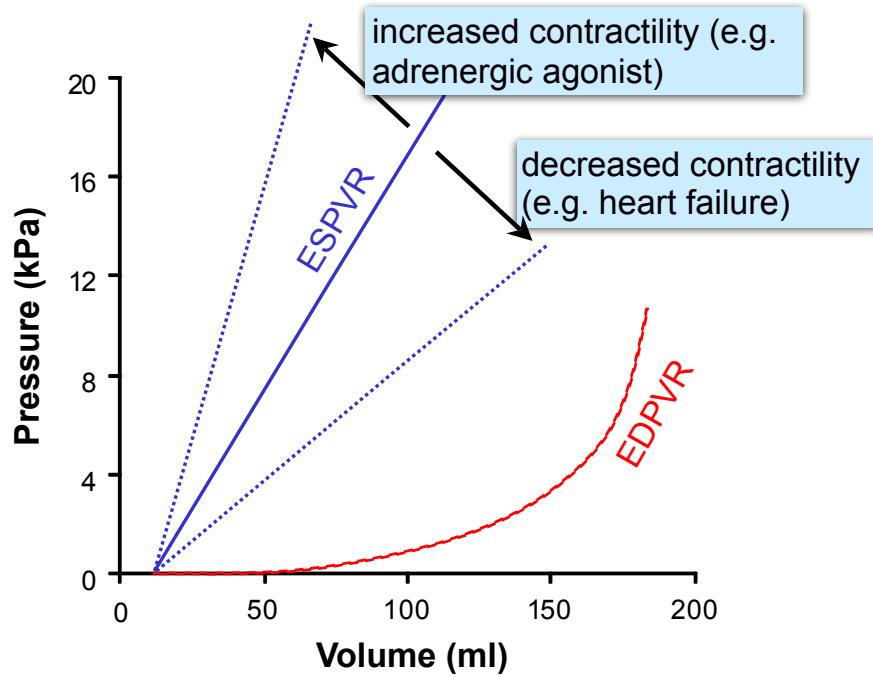
Source: Klabunde, RE, [Ventricular Pressure-Volume Relationship](#)

Increased inotropy shifts the ESPVR up and to the left, thereby increasing stroke volume and decreasing end-systolic volume (ESV). Decreased inotropy shifts the end-diastolic pressure-volume relationship down and to the right, thereby decreasing stroke volume and increasing end-systolic volume. Preload and aortic pressure are held constant in this illustration.

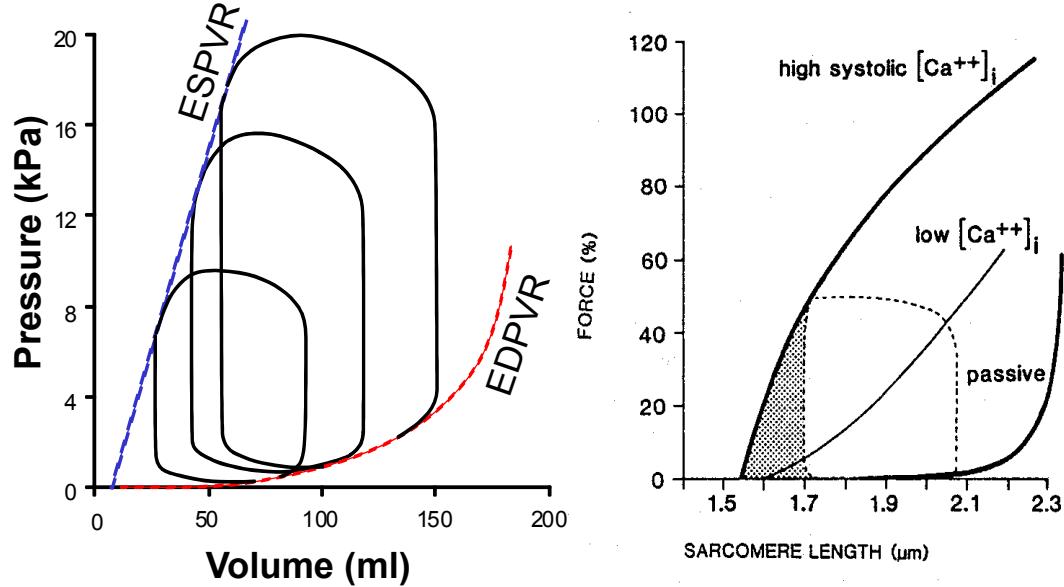
Starling's Law of the Heart (The Frank-Starling Mechanism)



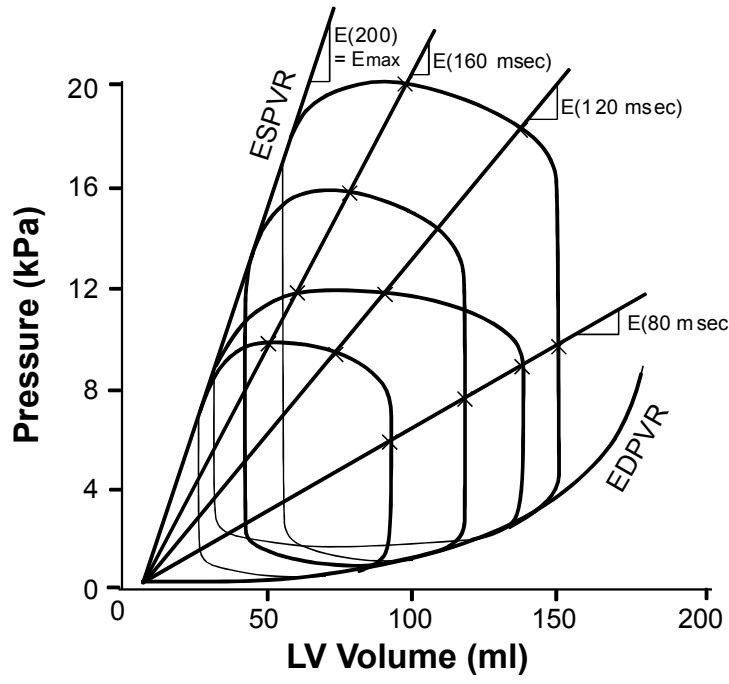
Contractility (Inotropic State)



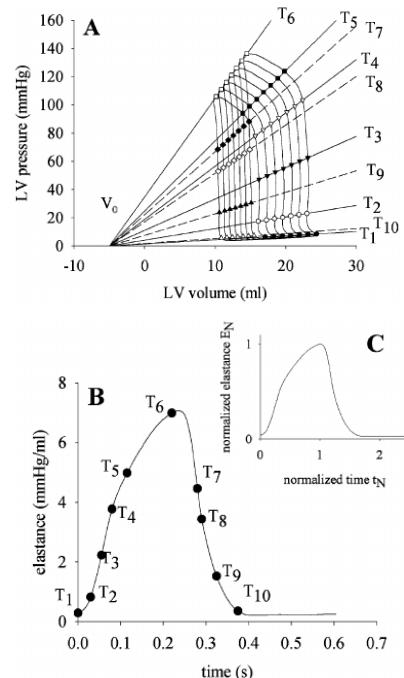
Physiological Basis of Starling's Law



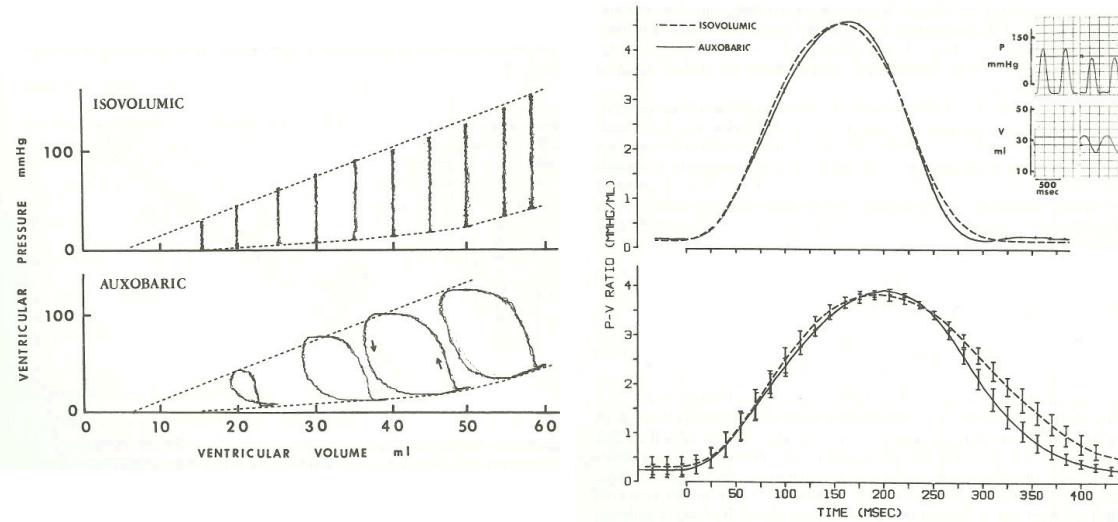
Time-Varying Elastance



$$P(t) = E(t)[V(t) - V_0]$$



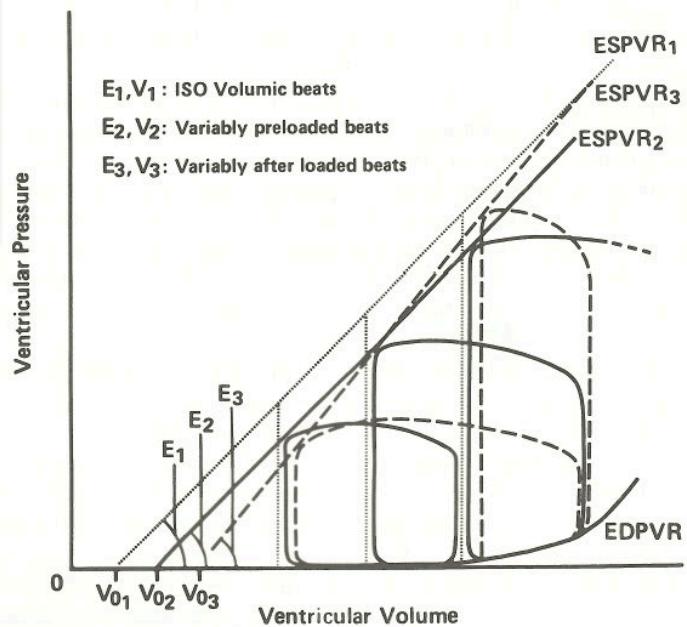
How Well Does Time-Varying Elastance Work? Ejecting vs Isovolumic Beats



There are both positive and negative effects of shortening on pressure development though they are small.

Effects of preload and afterload on ESPVR

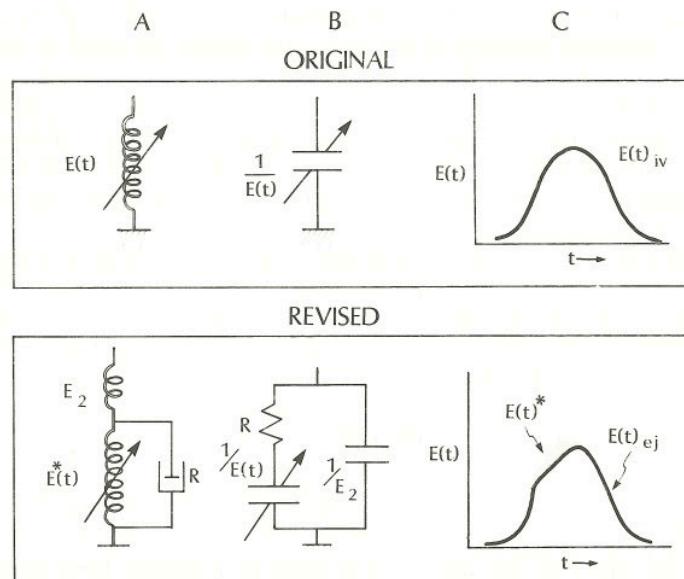
Larger ejecting beats decreased end-systolic pressure by up to 15-20% from isovolumic



Extended Models

Time-varying elastance model has been modified to include:

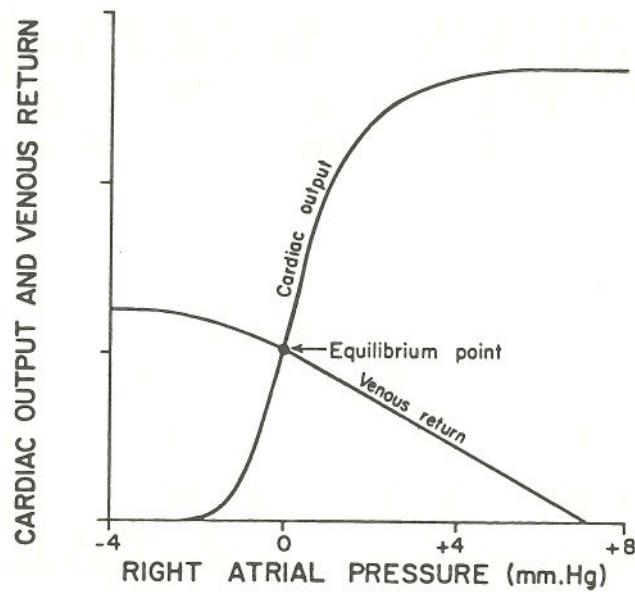
- Inertial effects
- Viscous resistance
- Force deactivation terms



$$P_V(t) = M\ddot{V}(t) + R\dot{V}(t) + E(V(t) - V_0)$$

Ventricular-Venous Coupling

Guyton's equilibrium diagram for RA pressure, venous return and cardiac output. Normal cardiac output is determined by the intersection of the venous return curve and the cardiac output curve.



Ventricular-Arterial Coupling

ESPVR:

$$P_{ES} = E_{ES} (V_{ED} - V_{ES}) = E_{ES} (V_{ED} - SV - V_0)$$

Mean arterial pressure-flow relation:

$$P_m = R_T Q_m$$

$$P_m \approx P_{ES} \approx \frac{R_T SV}{T} = E_a \cdot SV$$

where T is cycle length and

$$E_a = \frac{R_T}{T}$$

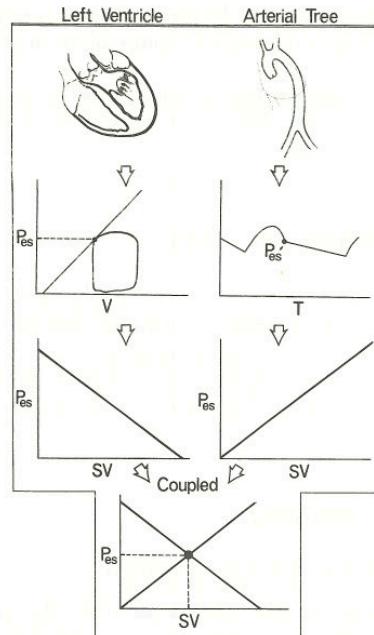
is effective arterial elastance. For Windkessel model:

$$R_T = \frac{P_m}{Q_m} = \frac{A_T / T}{SV / T} = \frac{A_T}{SV}$$

where A_T is area under arterial pressure pulse.

Ventricular-Arterial Coupling

Both the ventricular system and the arterial systems are characterized by the relationship of end-systolic pressure to stroke volume. The intersection between these two relations gives the stroke volume of the coupled system.

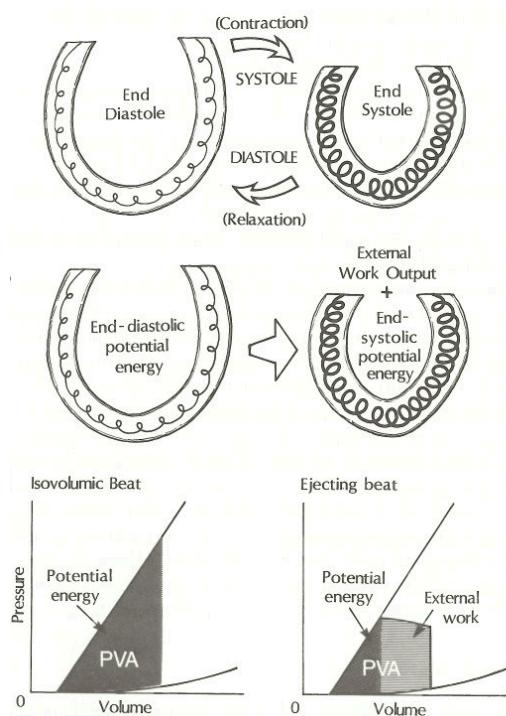


Myocardial Oxygen Consumption

- Since 95% of ATP in myocytes is normally produced by aerobic metabolism (oxidative phosphorylation), myocardial oxygen consumption (MVO_2) is often used to determine cardiac energy utilization by multiplying coronary blood flow by the arterio-venous O_2 difference:
$$MVO_2 = CBF \times AV\Delta O_2$$
- While the energy generated by the oxidation of 1 mole of substrate varies with substrate, the energy generated per unit oxygen is fairly constant and similar to that for glucose and lactate $\sim 20 \text{ J/ml } O_2$
- External work is related to regional work done by the myocardium:

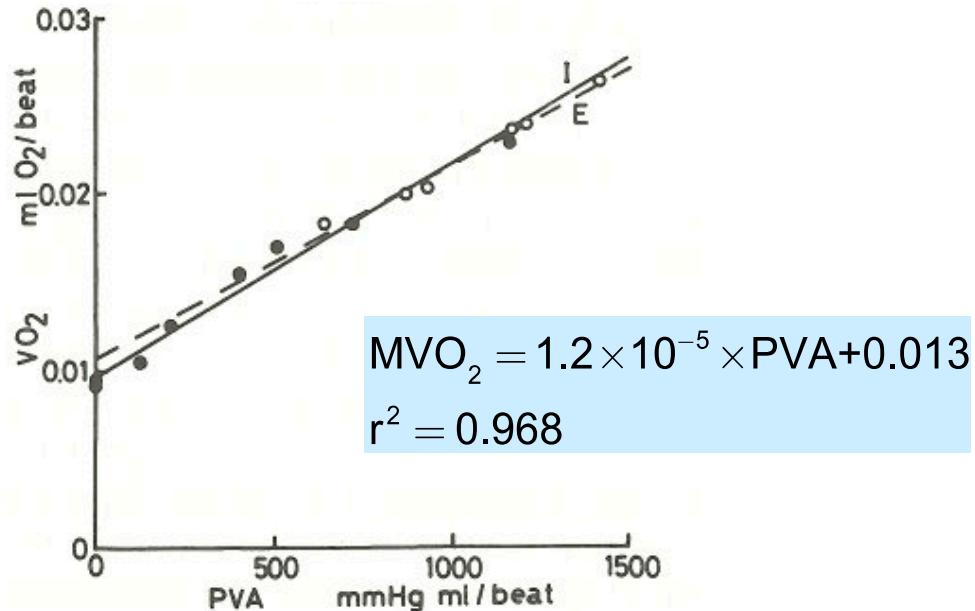
$$\text{Regional Work} = - \int T_{ij} dE_{ij} \cong \int T_a d\ell$$

Pressure-Volume Area (PVA)

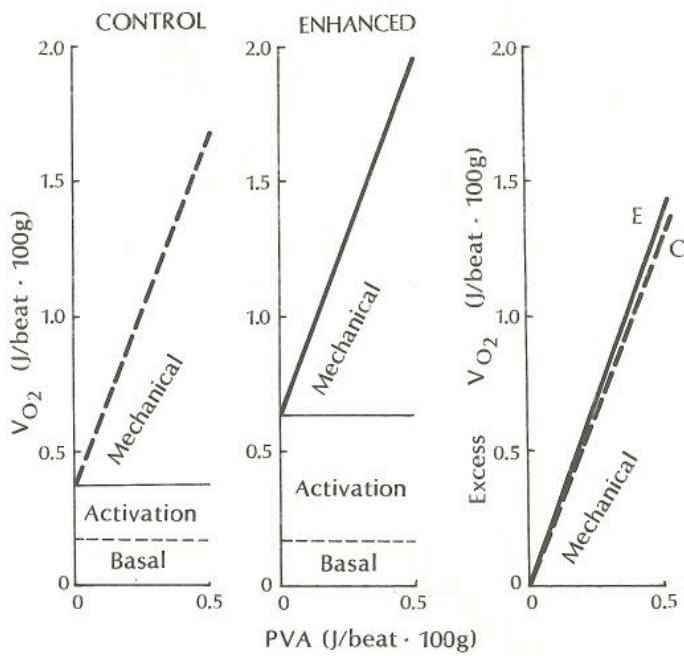


- Suga (1979) considered the elastic potential energy generated during an isovolumic contraction
- He realized that this pressure development required metabolic energy though it did no external work.
- Rather it must be dissipated as heat.
- Hence he defined the **pressure-volume area** (PVA) as the sum:
$$PVA = PE + EW$$

MVO₂ increases linearly with PVA

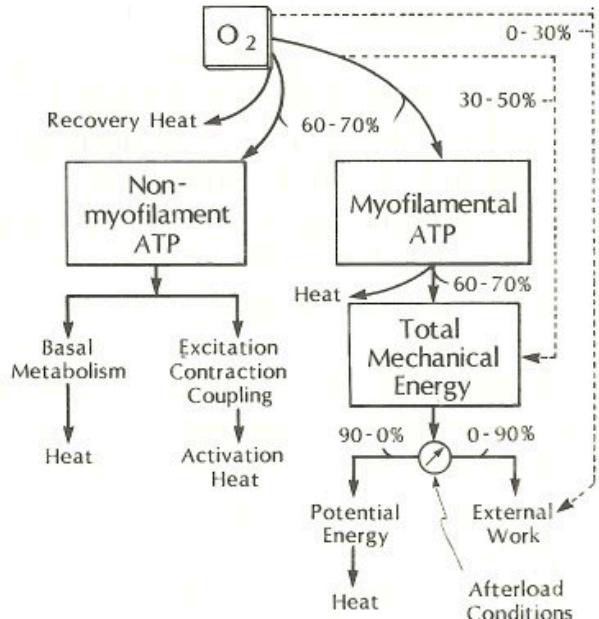


Basal and activation energy



- Basal energy is oxygen consumption when heart is not beating
- Activation energy increases with contractility and is mainly the energy of calcium cycling

Efficiency



$$\text{Mechanical efficiency} = \frac{\text{External Work}}{\text{Total MVO}_2}$$

$$\text{Conversion efficiency} = \frac{\text{Pressure-Volume Area}}{\text{Total MVO}_2}$$

$$\text{Myofibrillar efficiency} = \frac{\text{Pressure-Volume Area}}{\text{MVO}_2 - \text{unloaded MVO}_2}$$

- Conversion efficiency = 10-30%
- Myofibrillar efficiency = 30-40%

Ventricular Function: Summary of Key Points

- *Ventricular anatomy* is 3-D and complex
- *Systole* consists of isovolumic contraction and ejection
- *Diastole* consists of isovolumic relaxation and filling
- Area of the pressure-volume loop is *ventricular stroke work or external work*
- *Stroke work* increases with filling (*Starling's Law*)
- *Myocardial oxygen consumption* is proportional to pressure-volume area (external work plus potential energy)
- Ventricles behave approximately like *time-varying elastances*
- The slope of the end-systolic pressure volume relation is a load-independent measure of *contractility or inotropic state*.