

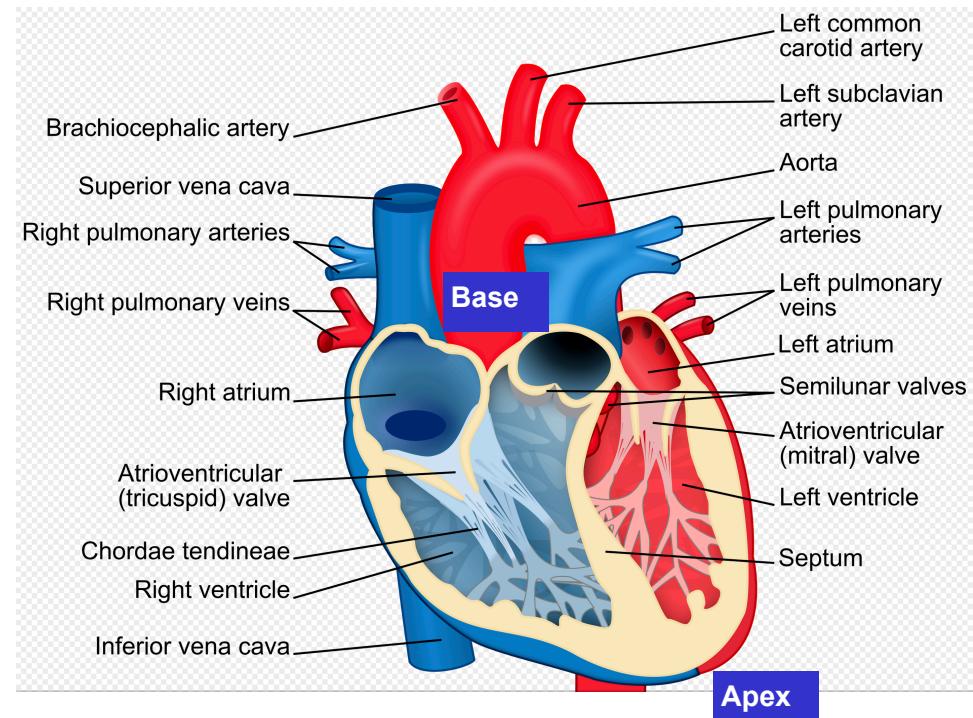
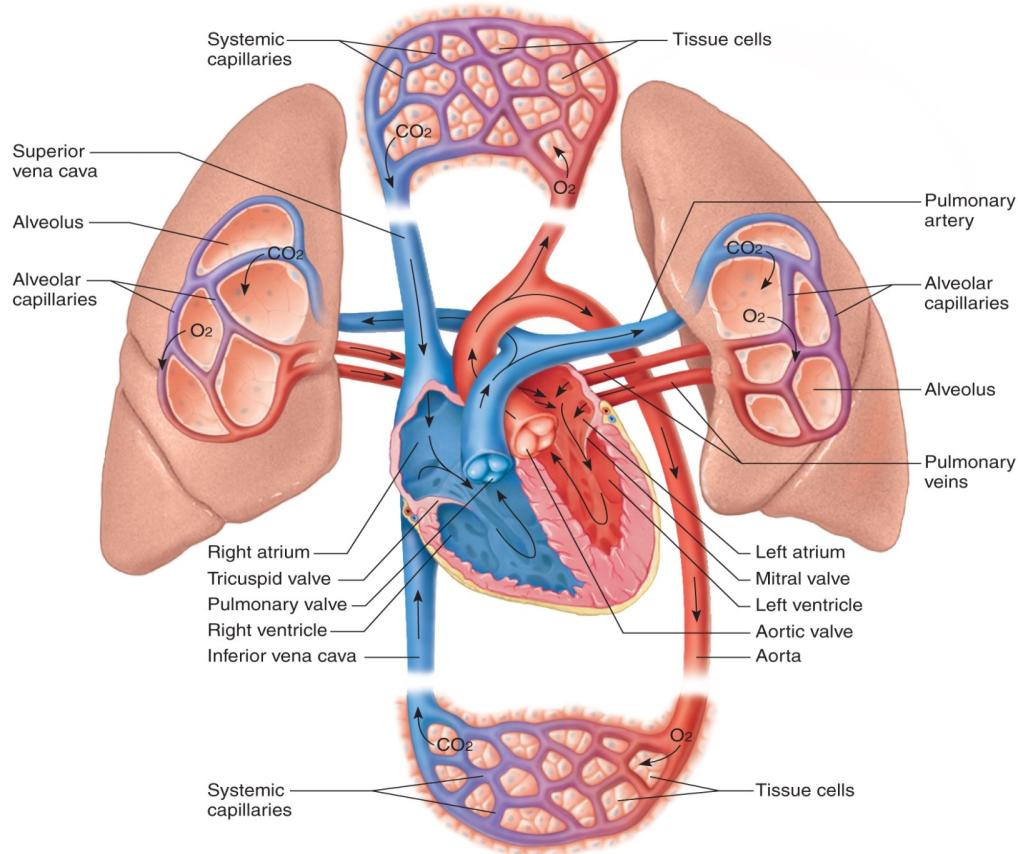
Cardiac Pump Function

Andrew McCulloch

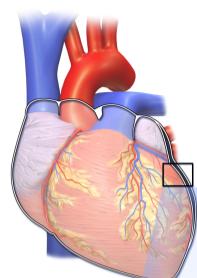
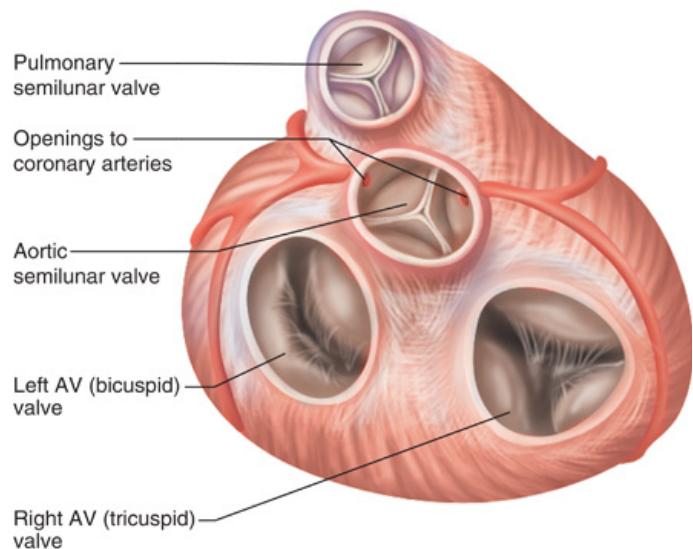
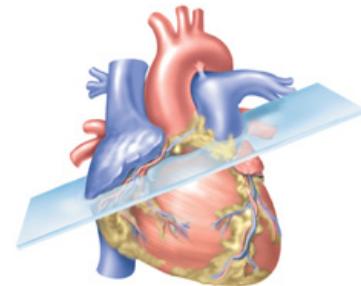
Shu Chien Chancellor's Endowed Chair in Engineering and Medicine
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- Cardiac Functional Anatomy
- Cardiac Cycle and the Wiggers Diagram
- Ventricular Pressure-Volume Relations
- Effects of Altered Preload and Afterload
- Contractility/Inotropy
- Ventricular Energetics
- Ventricular-Vascular Coupling

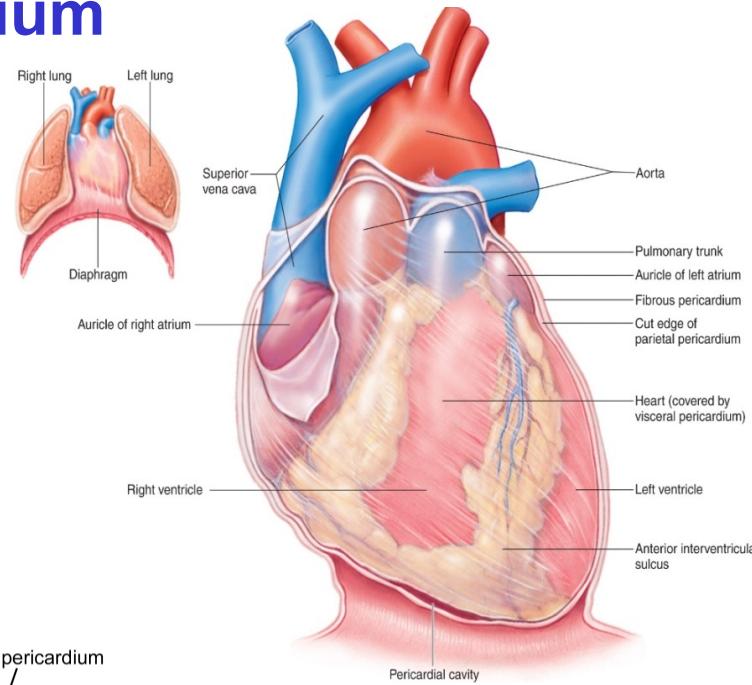
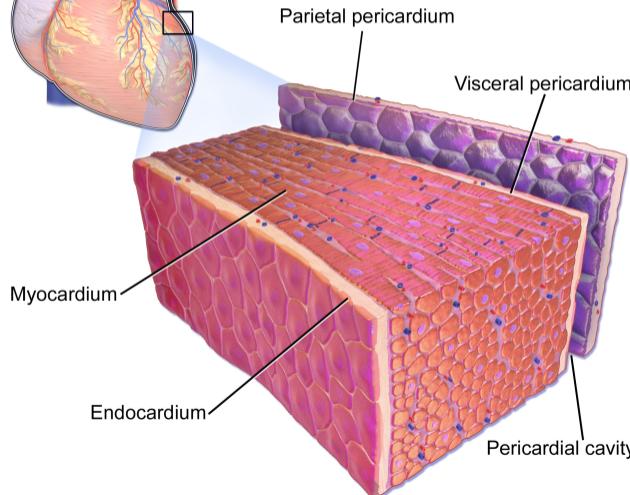
The Heart and Circulation



Heart Valves and Pericardium

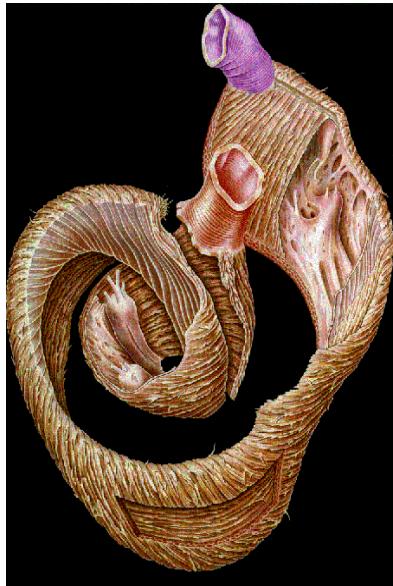


The Heart Wall



Cardiac Muscle Fiber Architecture

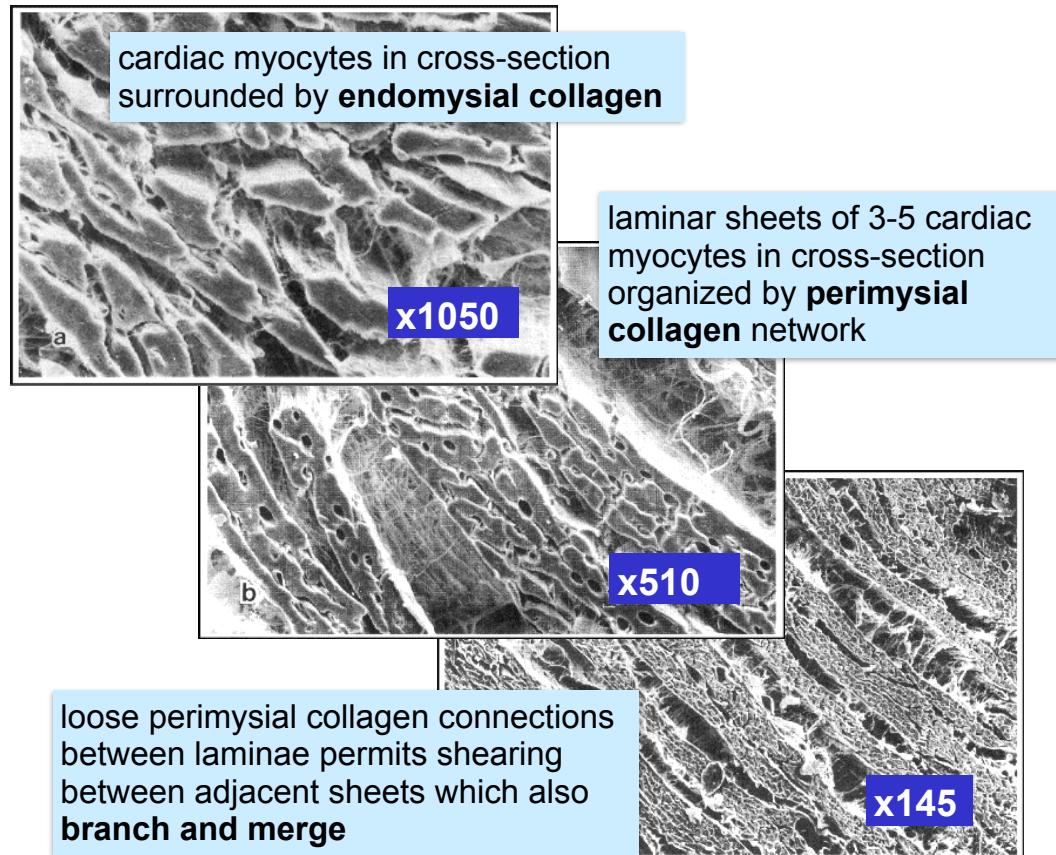
Classical Muscle Band Model



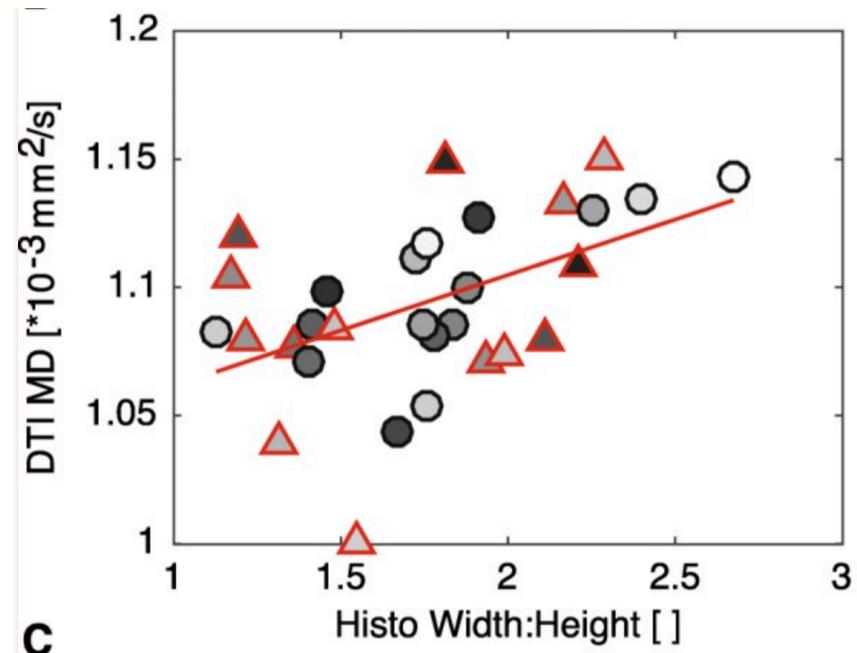
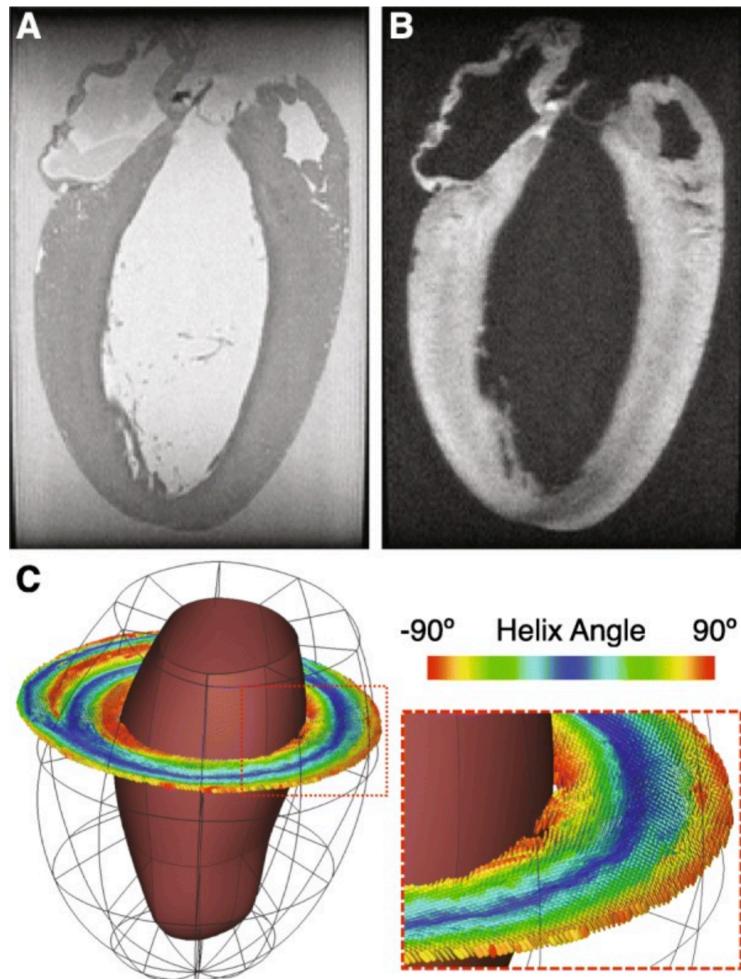
Torrent-Guasp, 1995

Streeter et al. (1969)

Laminar Myofiber Sheet Structure

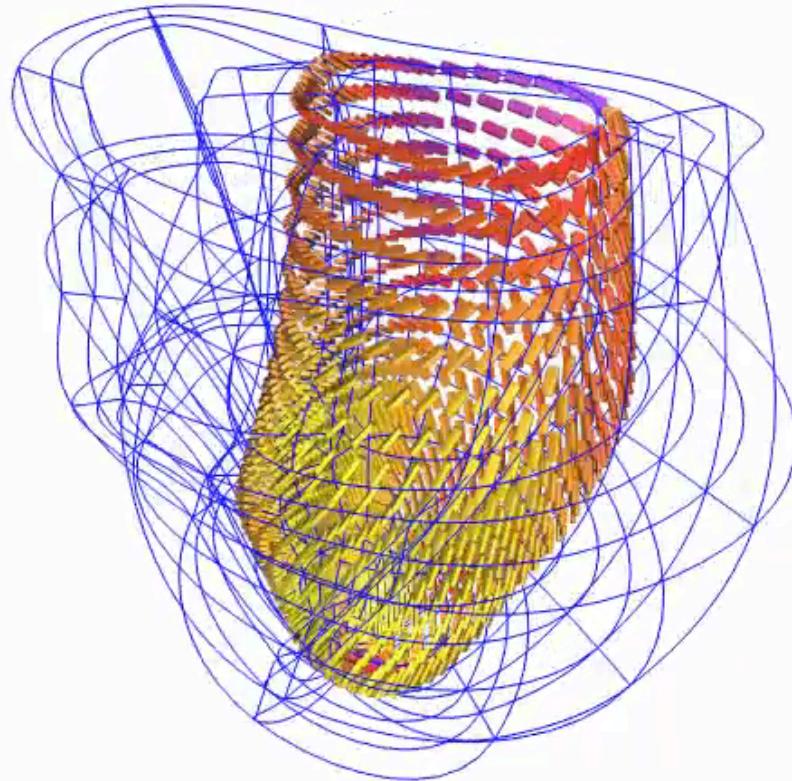
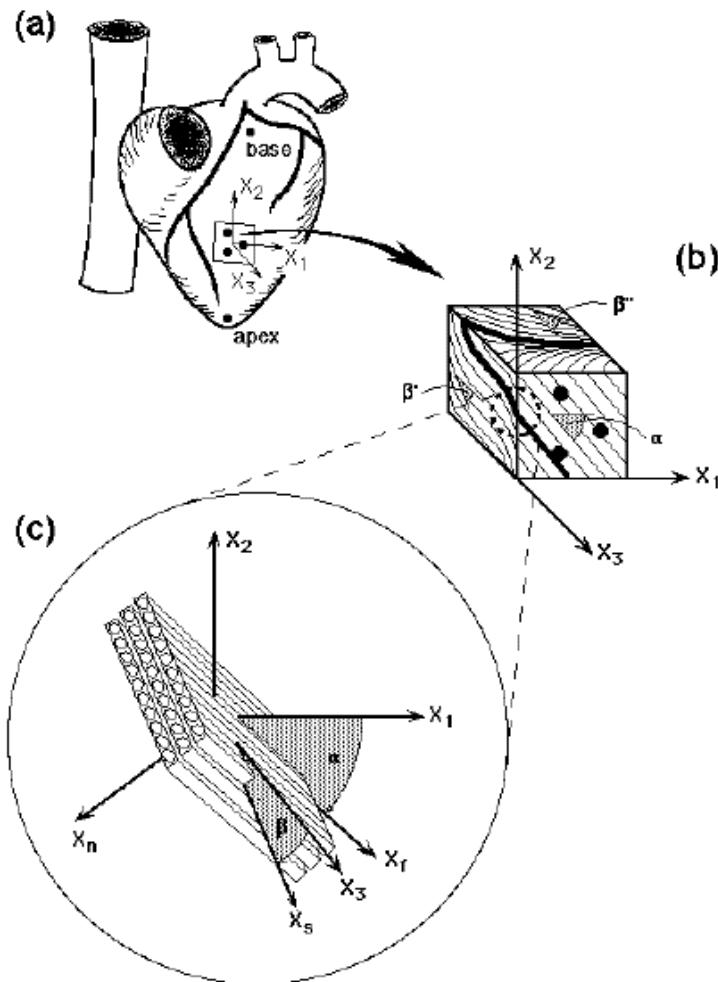


Diffusion Tensor MRI: Diffusivity Correlates with Myocyte Shape

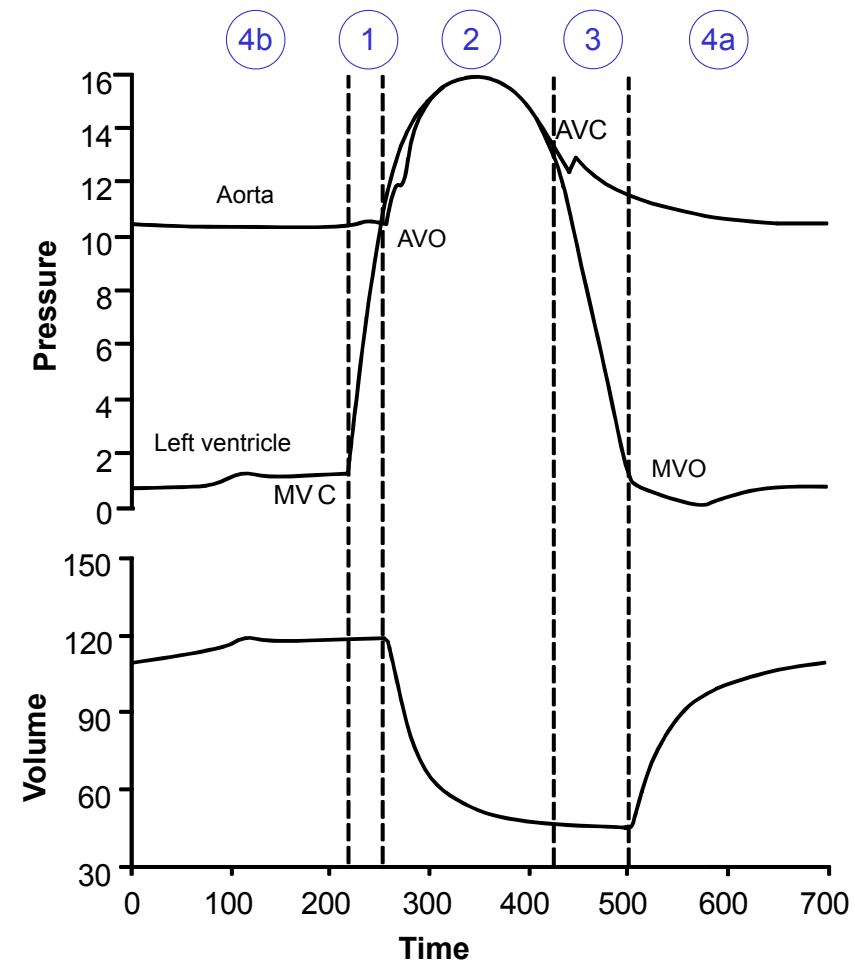
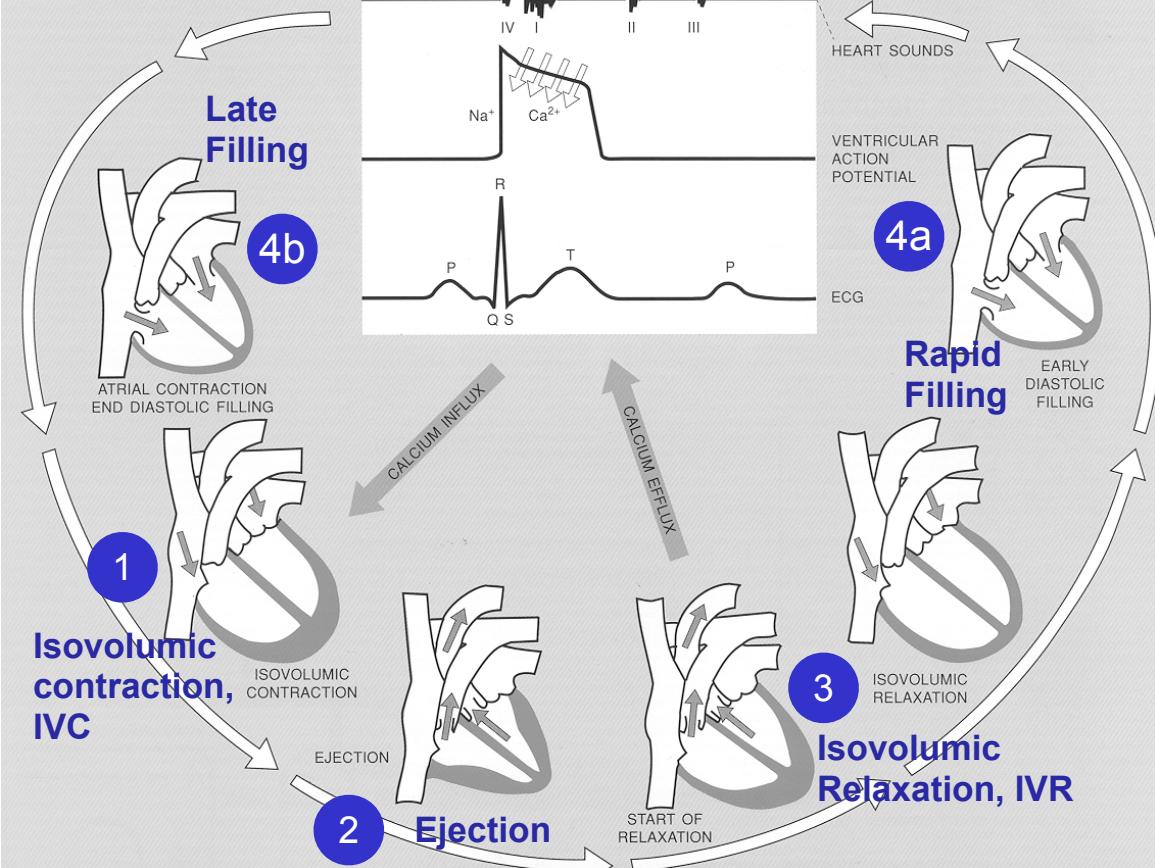


Carruth ED, Teh I, Schneider JE, McCulloch AD, Omens JH, Frank LR. Regional variations in ex-vivo diffusion tensor anisotropy are associated with cardiomyocyte remodeling in rats after left ventricular pressure overload. *J Cardiovasc Magn Reson.* 2020;22(1):21

Laminar Fiber Sheet Model

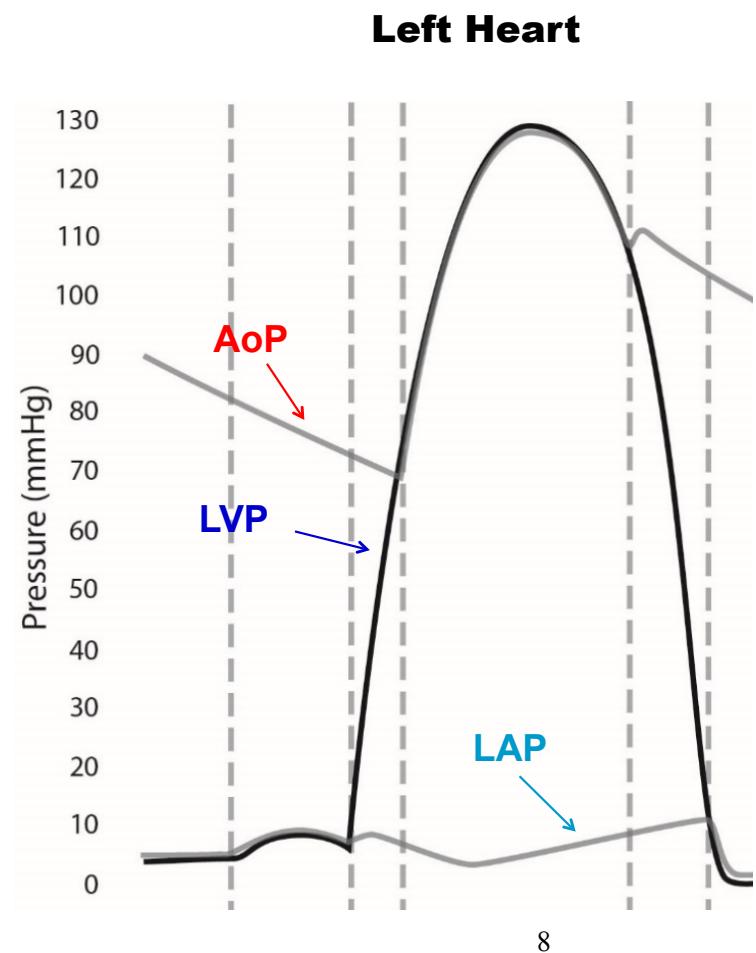
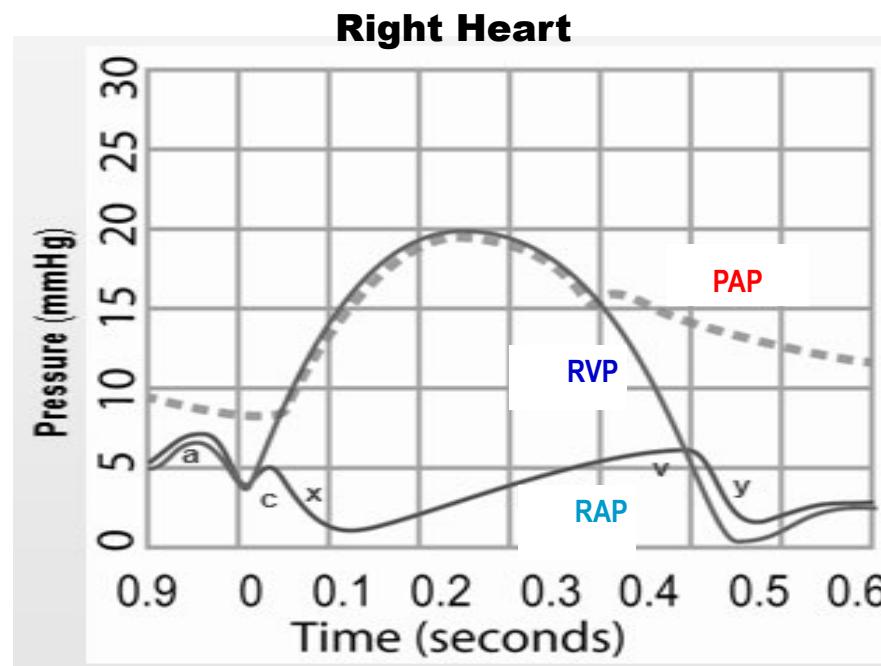


Systole:
 1. IVC
 2. Ejection
Diastole:
 3. IVR
 4. Filling

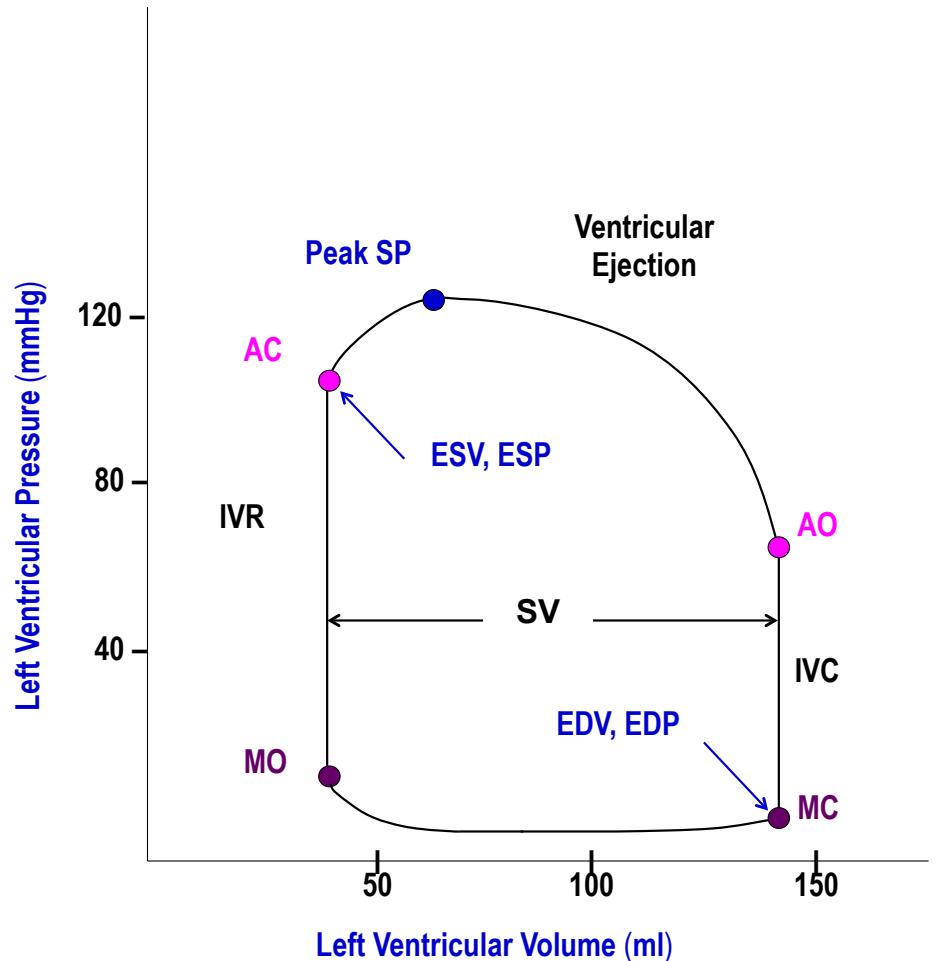
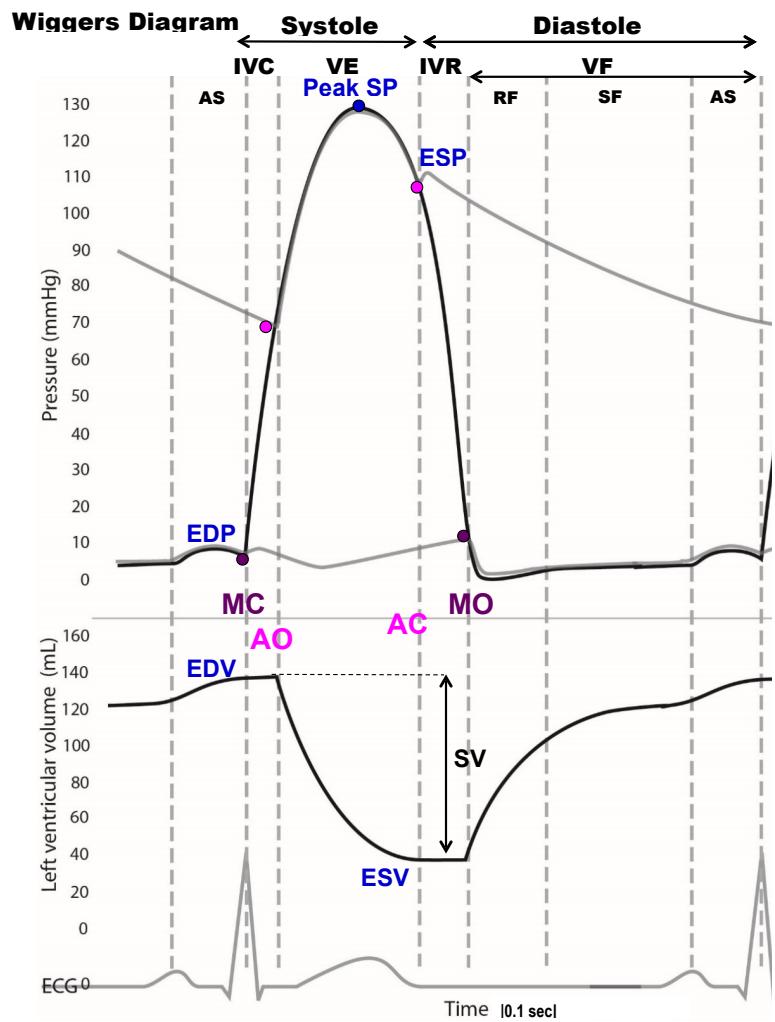


Right vs. Left Heart

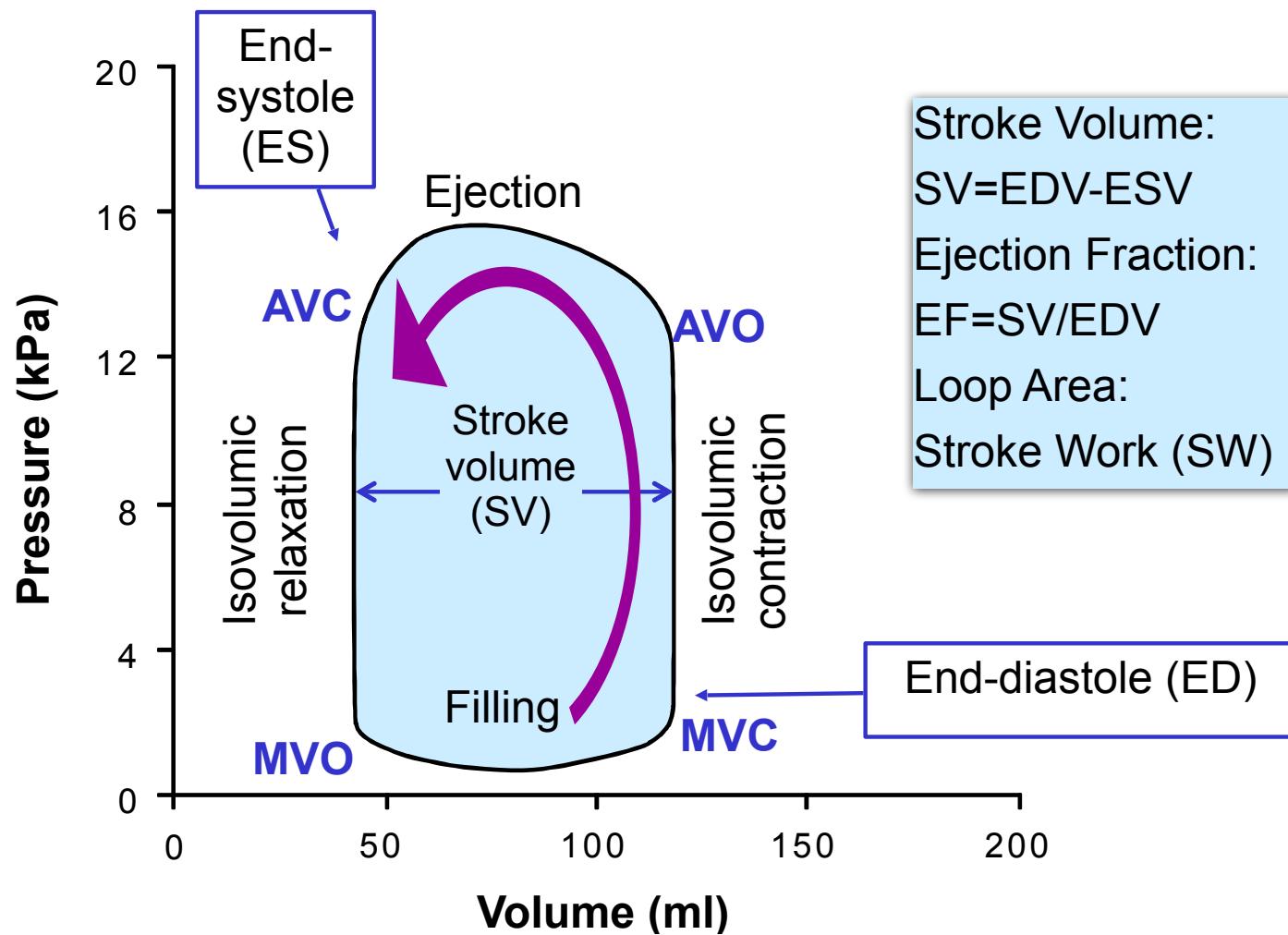
Ventricular Pressures (mmHg)		
	RVP	LVP
Peak systole	15-30	100-140
End-diastole	2-8	3-12



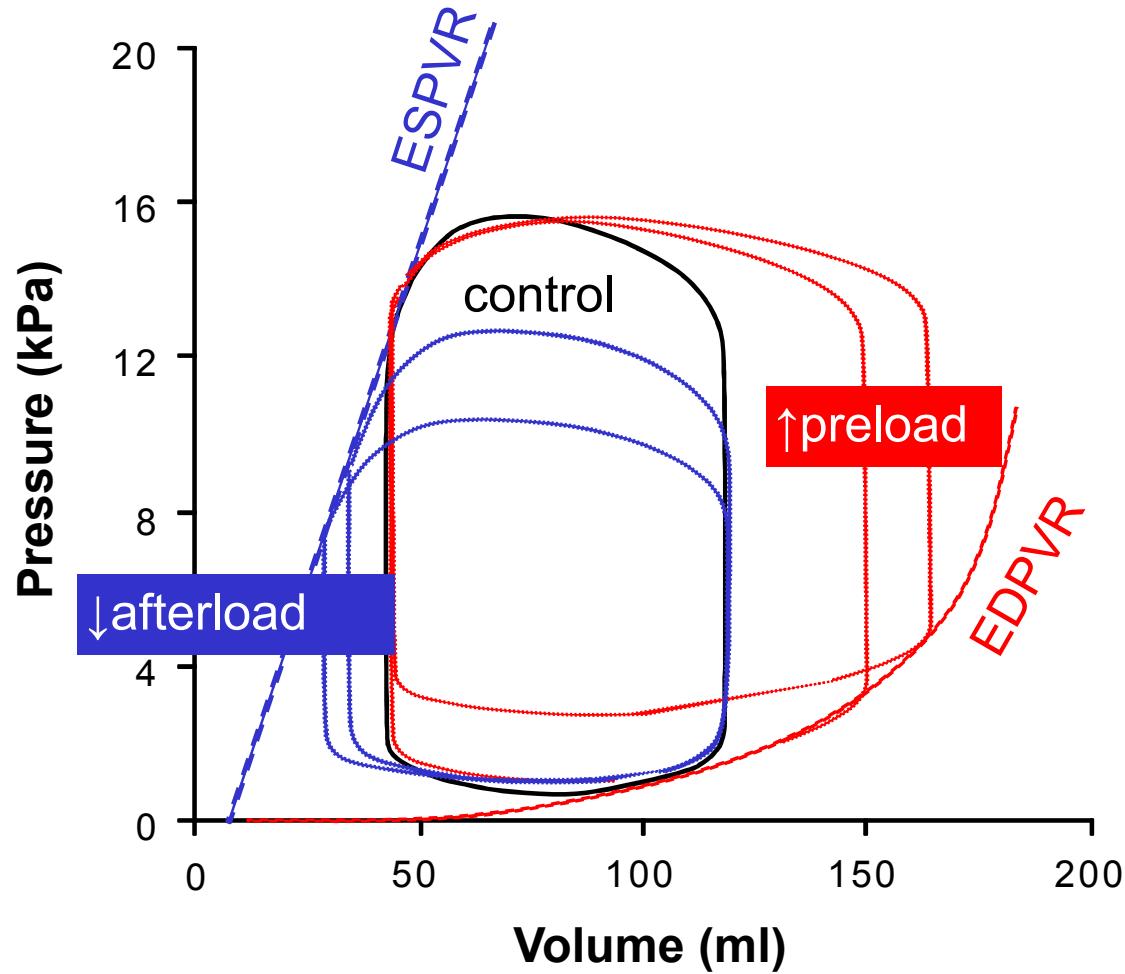
Pressure-Volume (PV) Loop



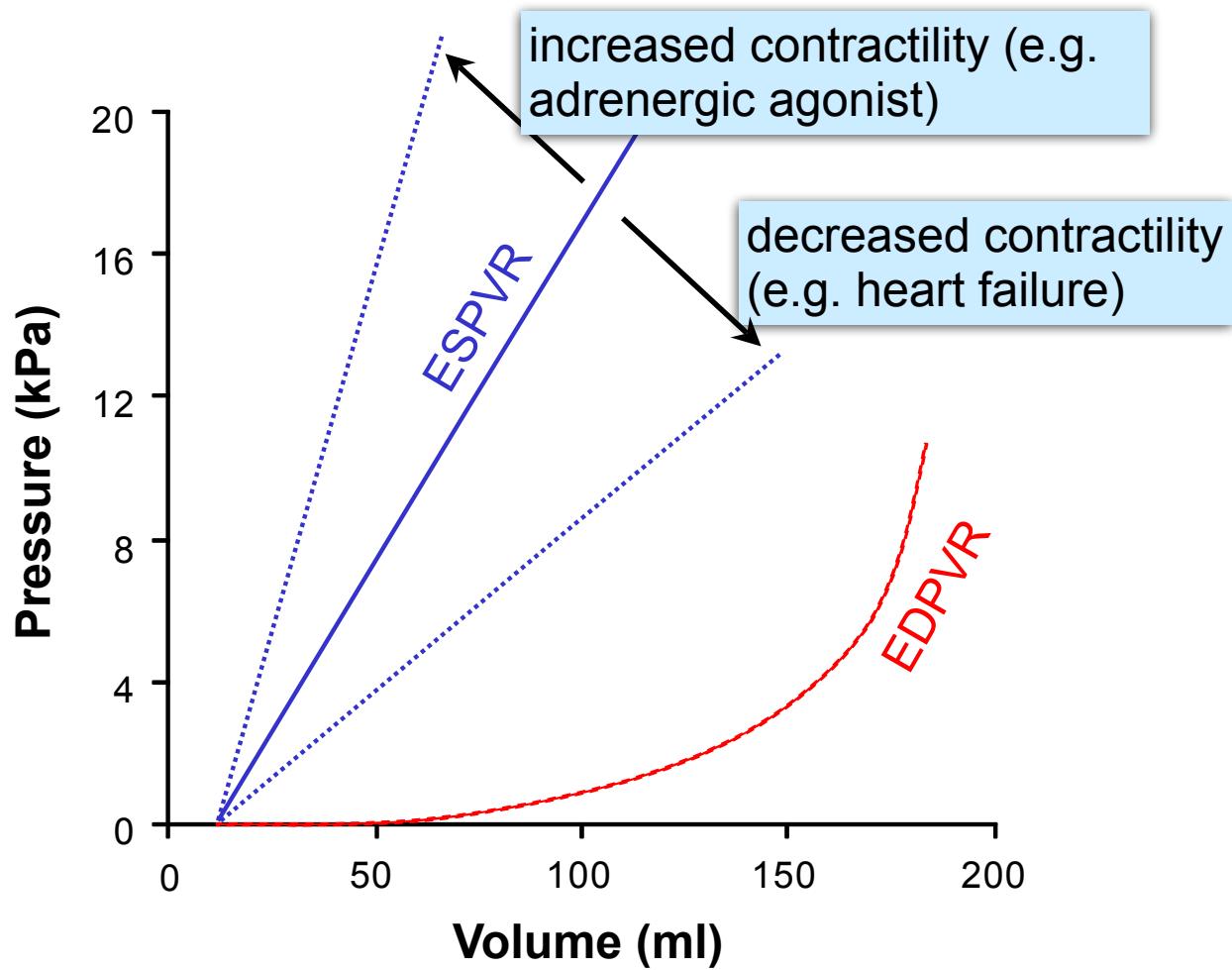
The Pressure-Volume Loop



Preload and Afterload

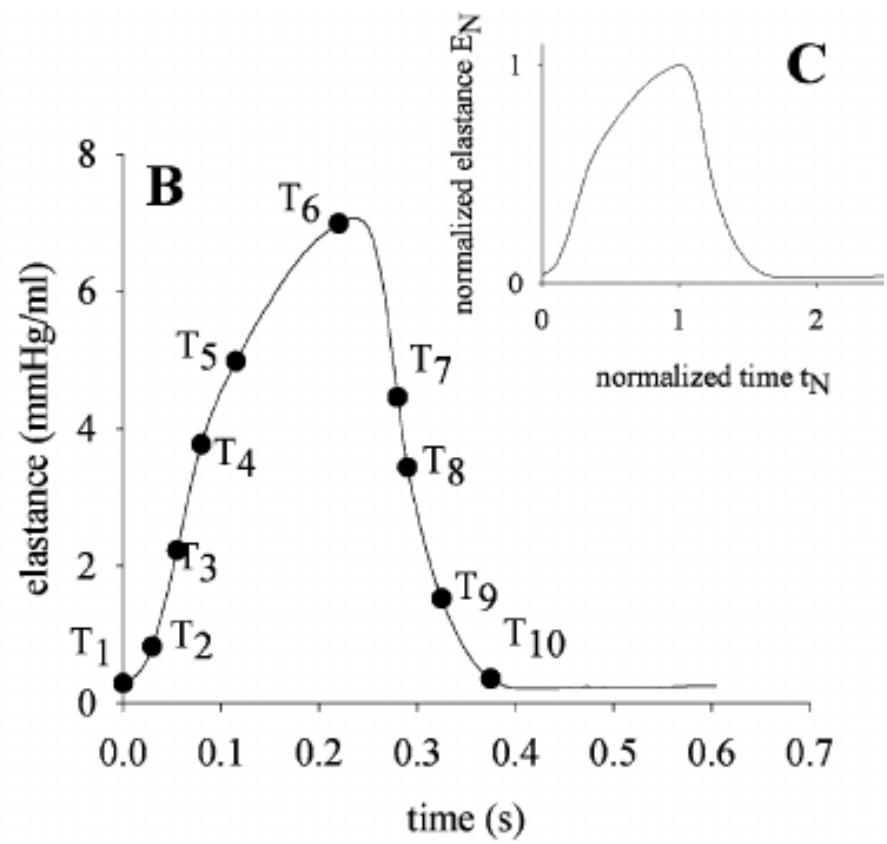
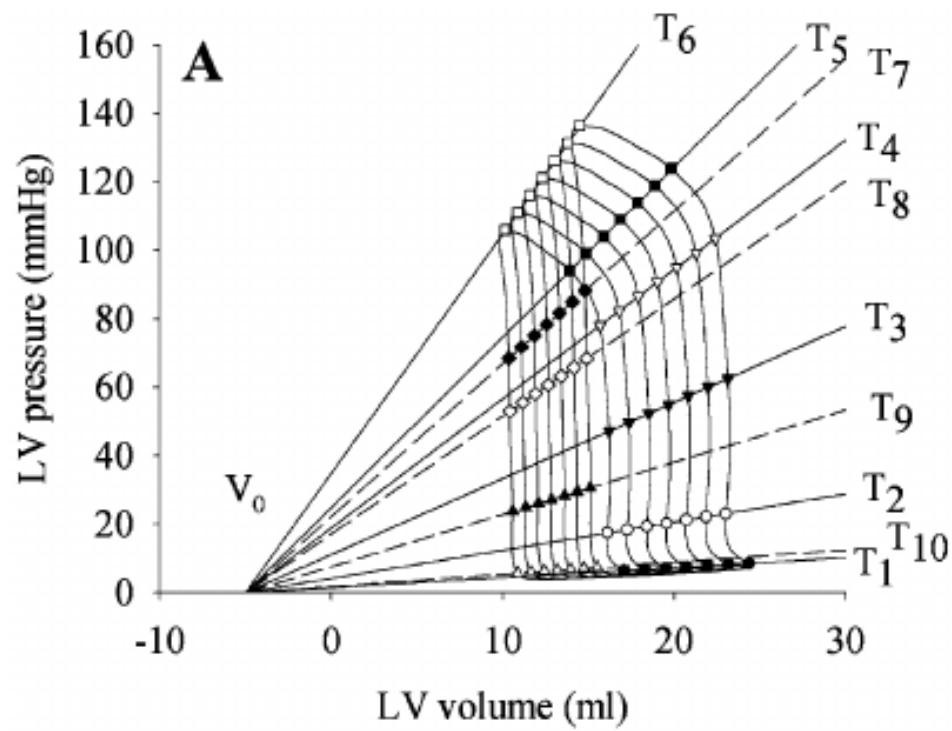


Contractility (Inotropic State)

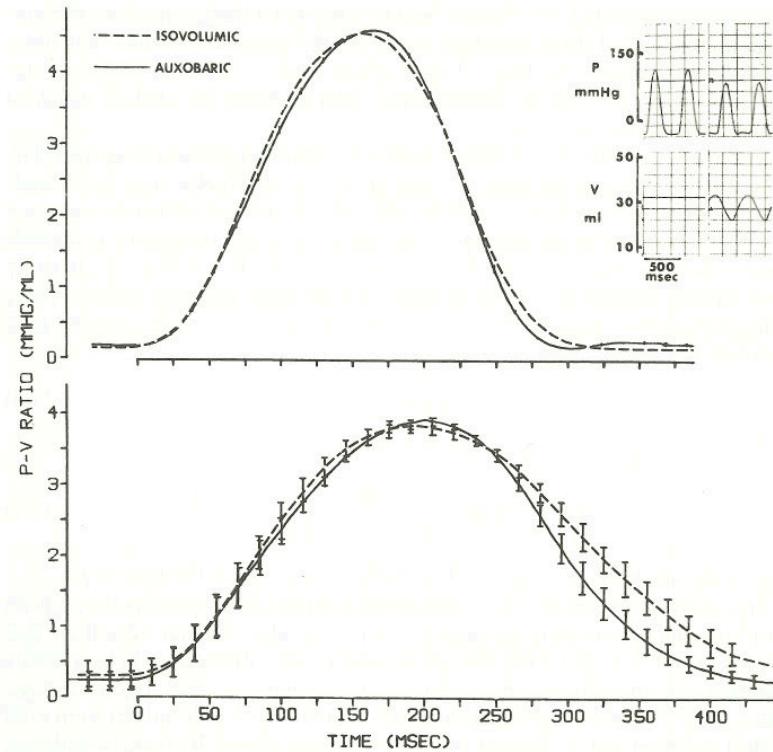
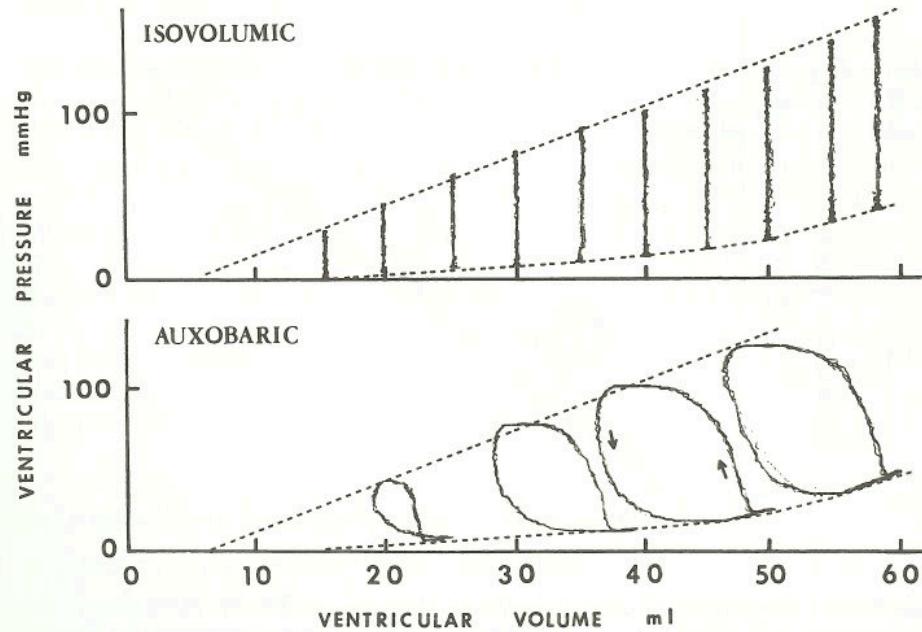


Time-Varying Elastance Model

$$P(t) = E(t)\{V(t) - V_0\}$$



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

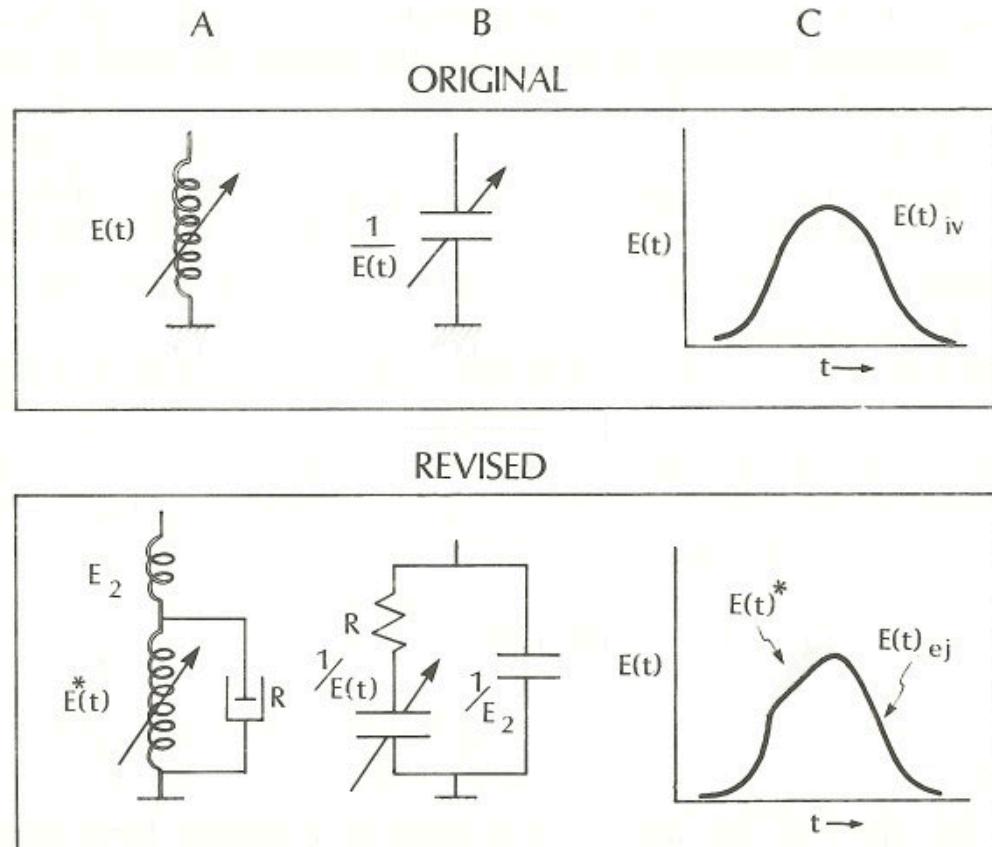


There are both positive and negative effects of shortening on pressure development though they are small.

Extended Models

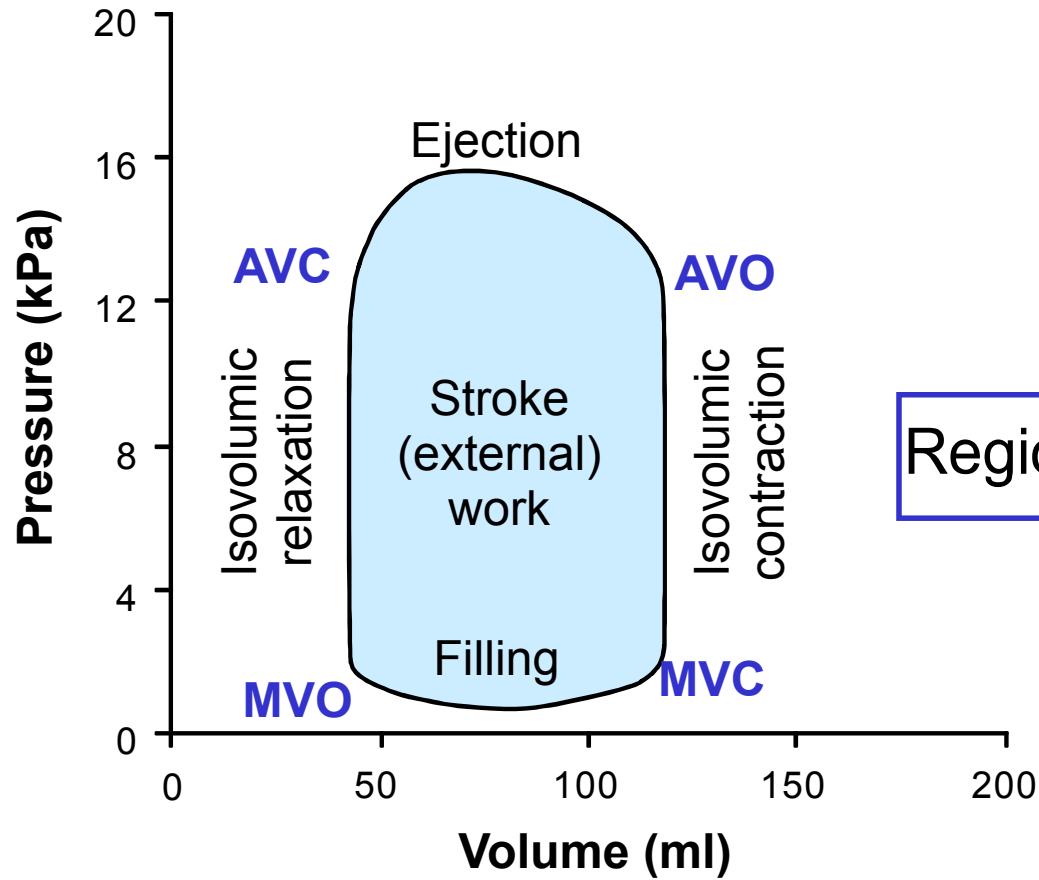
Time-varying elastance model has been modified to include:

- Inertial effects
- Viscous resistance
- Force deactivation terms



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

Stroke Work



$$EW = \int_{EDV}^{ESV} P(t) dV$$

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

Myocardial Oxygen Consumption and Coronary Blood Flow

- Since 95% of ATP in myocytes is normally produced by aerobic metabolism, myocardial oxygen consumption (MVO_2) is used to determine cardiac energy utilization by multiplying coronary blood flow by the arterio-venous O_2 difference:
- Energy generated per unit oxygen is fairly constant and similar to glucose and lactate ~ 20 J/ml O_2

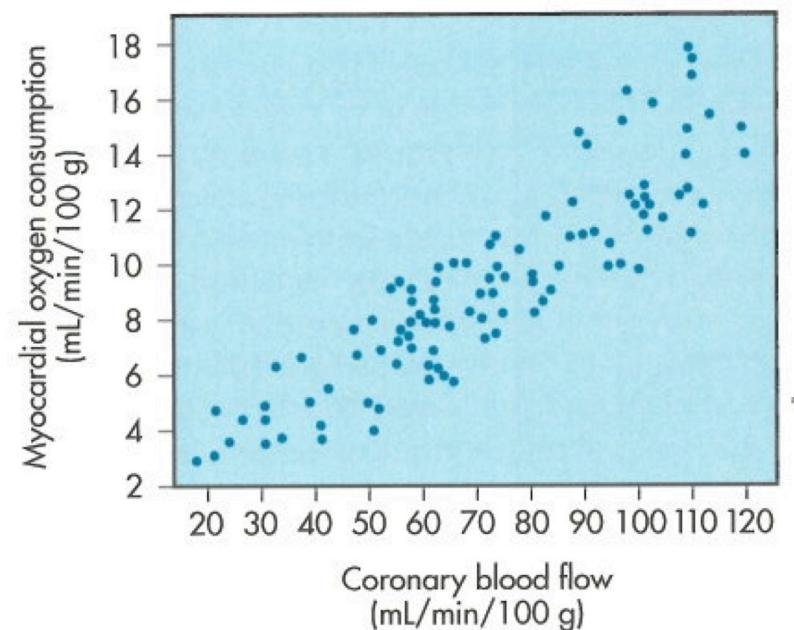
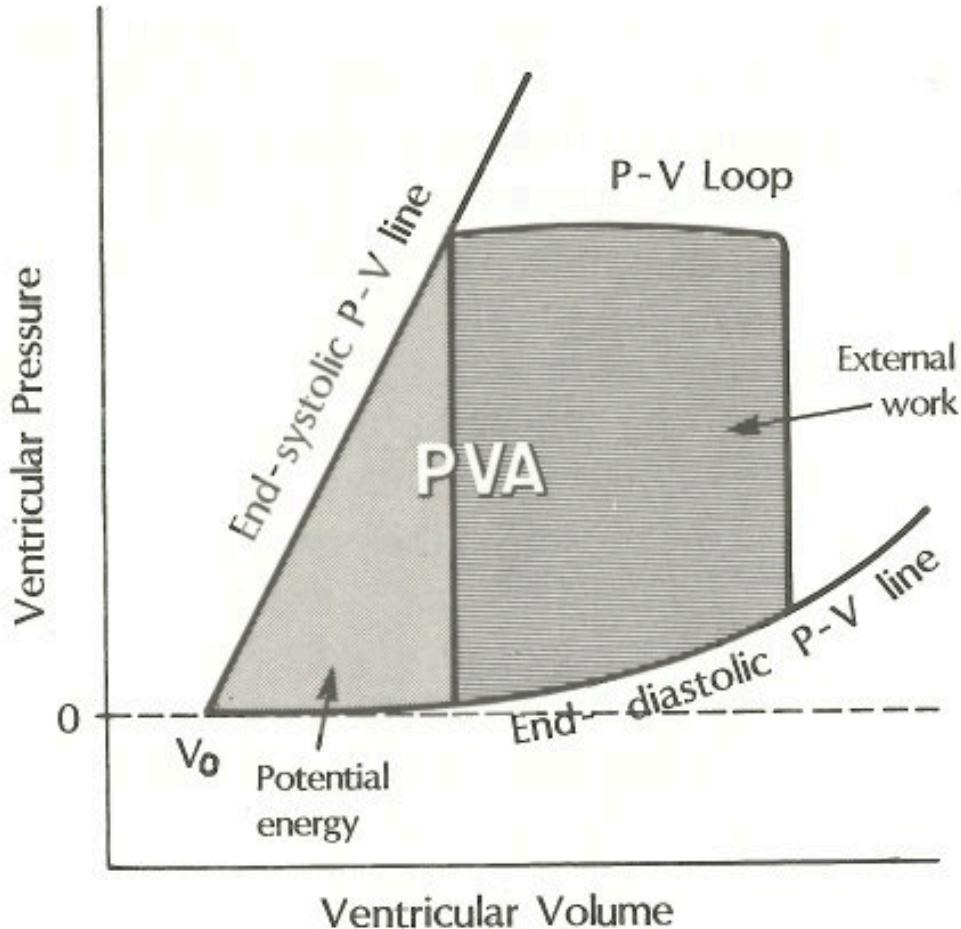


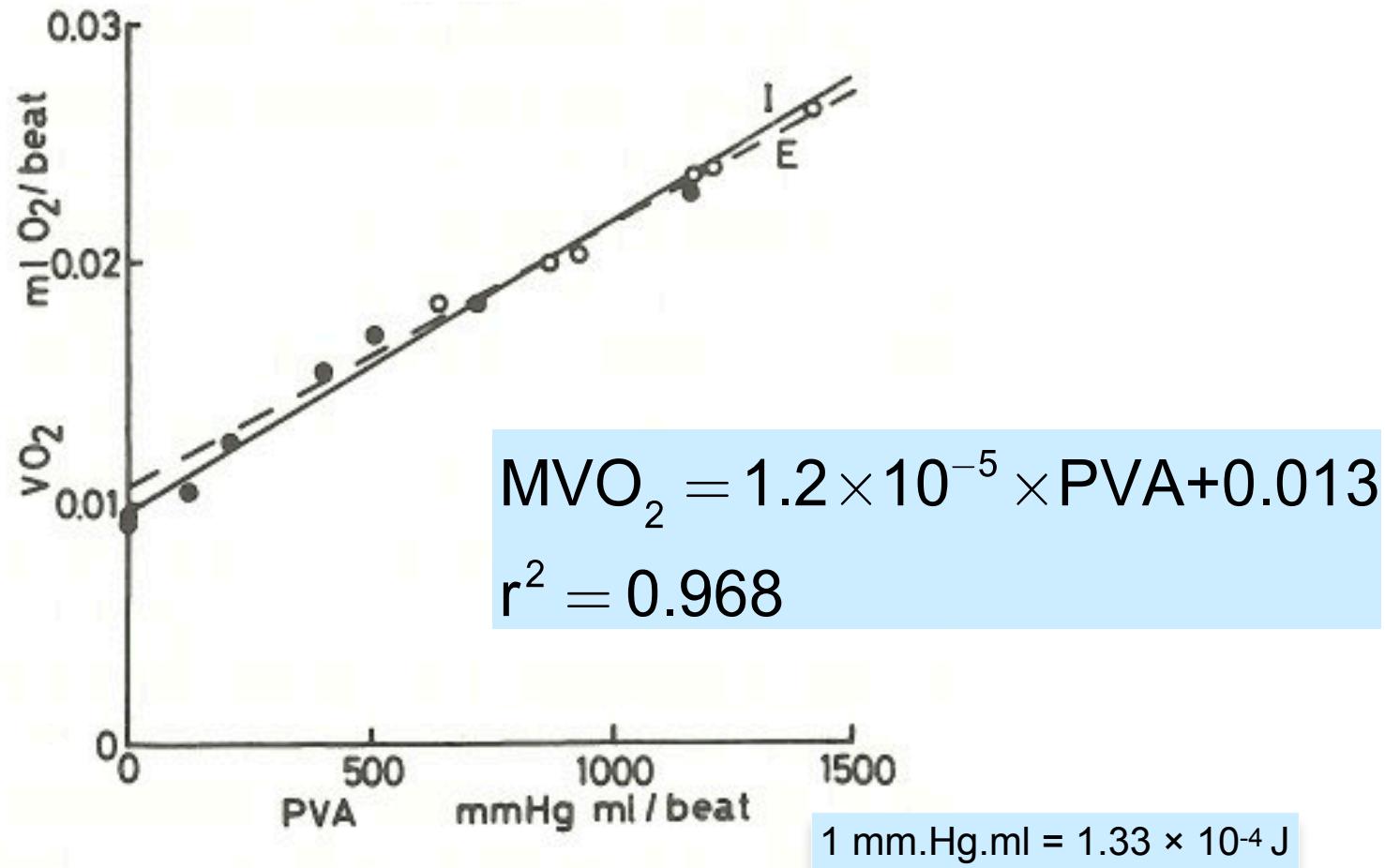
FIGURE 11-5 ■ Relationship between myocardial oxygen consumption and coronary blood flow during a variety of interventions that increased or decreased myocardial metabolic rate. (Redrawn from Berne RM, Rubio R: Coronary circulation. In: *Handbook of Physiology, Section 2: The Cardiovascular System—The Heart, vol 1*. Bethesda, Md, 1979, American Physiological Society.)

Pressure-Volume Area (PVA)

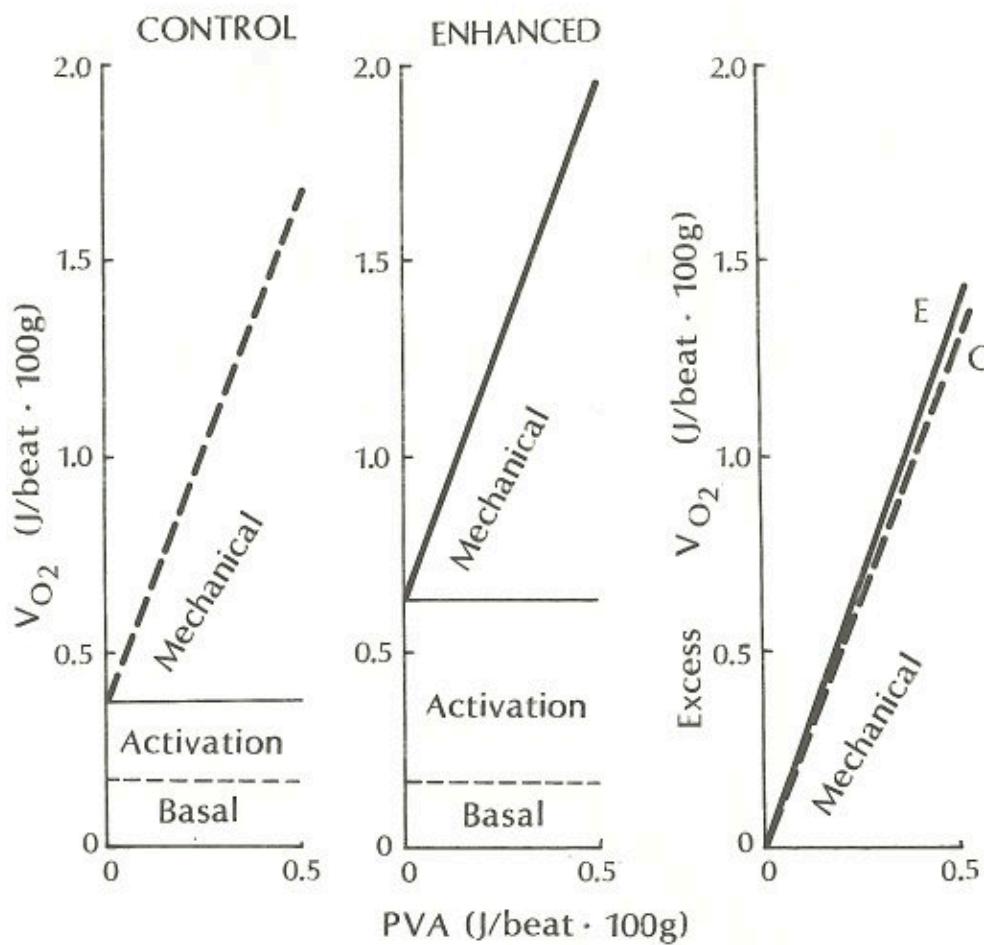


- Suga (1979) considered the elastic potential energy generated during an isovolumic contraction
- He realized that this pressure development required metabolic energy though it did no external work.
- Rather it must be dissipated as heat.
- Hence he defined the **pressure-volume area (PVA)** as the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

MVO₂ increases linearly with PVA



Basal and activation energy



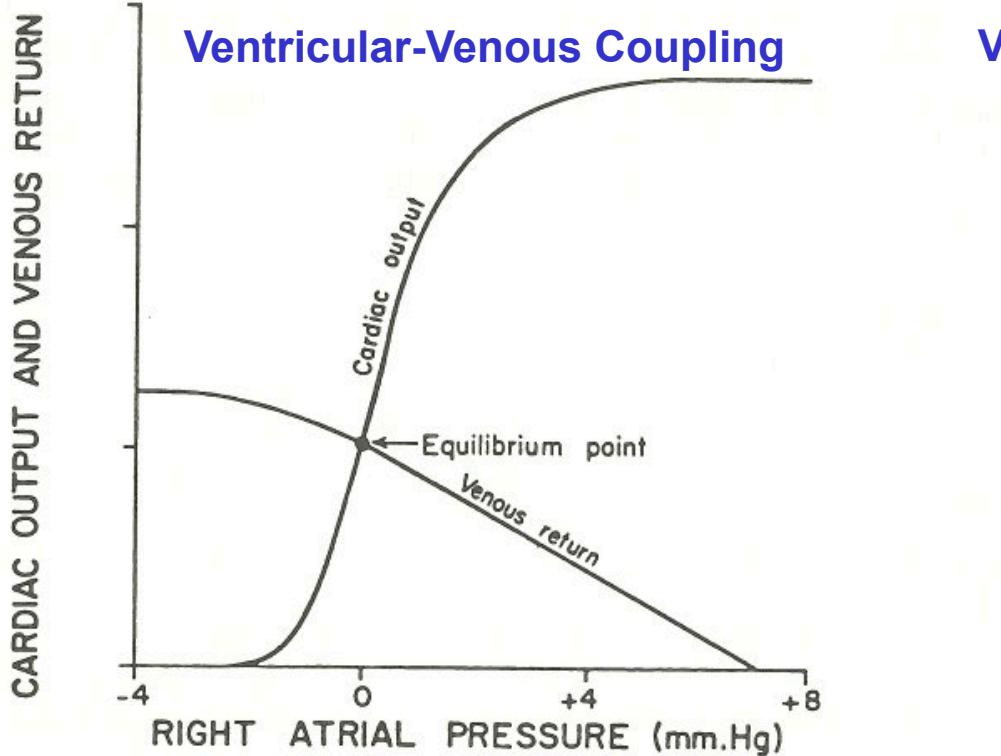
$$\text{Mechanical efficiency} = \frac{\text{External Work}}{\text{Total } MVO_2}$$

$$\text{Conversion efficiency} = \frac{\text{Pressure-Volume Area}}{\text{Total } MVO_2}$$

$$\text{Myofibrillar efficiency} = \frac{\text{Pressure-Volume Area}}{MVO_2 - \text{unloaded } MVO_2}$$

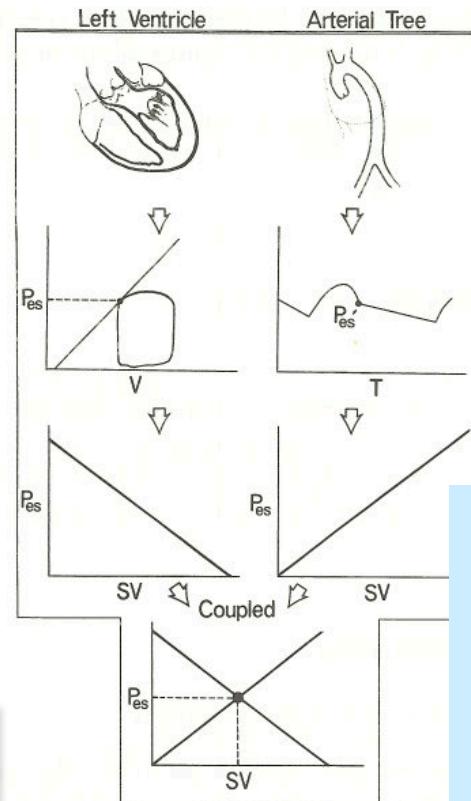
- Mechanical efficiency = 15-25%
- Conversion efficiency = 25-40%
- Myofibrillar efficiency = 40-50%

Ventricular-Vascular Coupling

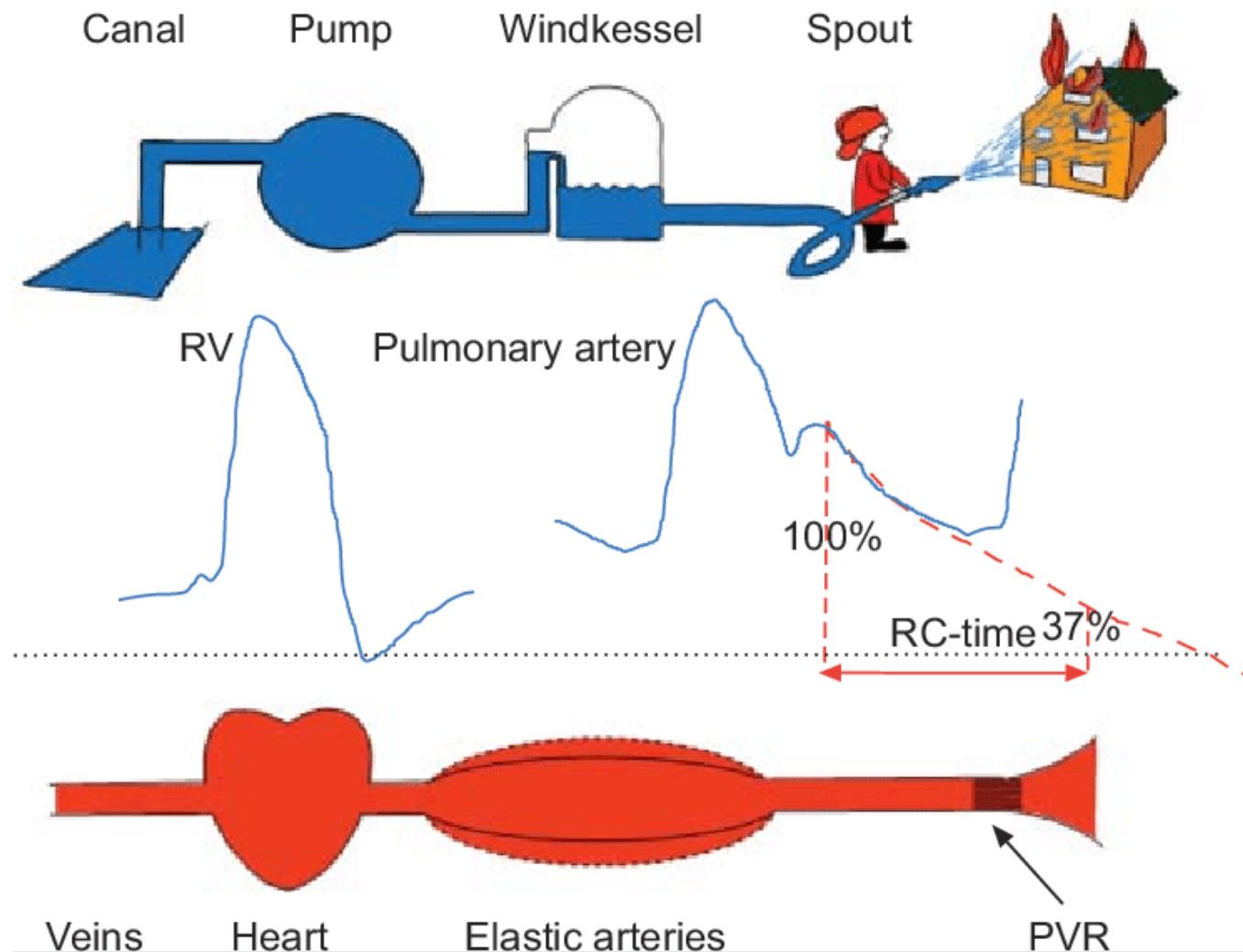


Guyton's equilibrium diagram for RA pressure, venous return and cardiac output. Normal cardiac output is determined by the intersection of the venous return curve and the cardiac output curve.

Ventricular-Arterial Coupling

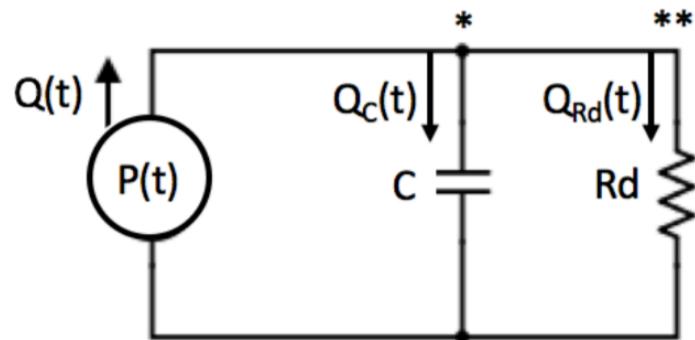
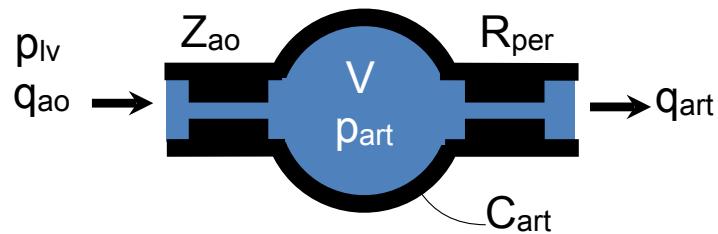
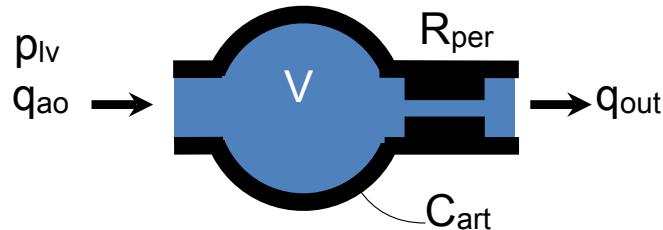


As end-systolic pressure increases, stroke volume decreases, and aortic volume increases (owing to its compliance).

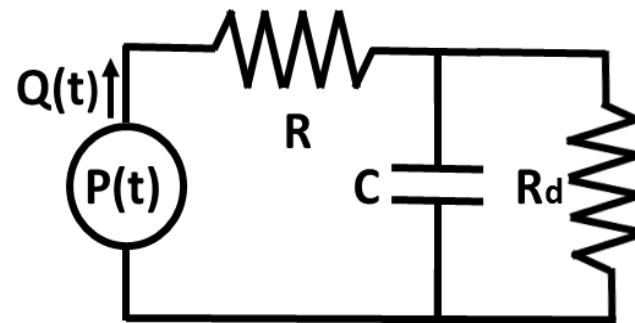


N. Saouti et al. Eur Respir Rev 2010

2-element vs. 3-element Windkessel Models



$$\frac{dP(t)}{dt} = \frac{Q(t)}{C} - \frac{P(t)}{R_d C}$$



$$\frac{dP}{dt} R R_d + P = \frac{dQ}{dt} R R_d C + Q(R + R_d)$$

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*)
- 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*
- *Myocardial oxygen consumption* is proportional to pressure-volume area (external work plus potential energy)
- Depending on the definition *ventricular energy efficiency* is 15-50%

Next Cardiac Muscle Mechanics: The Physiological Basis of Starling's Law

