

Evaluation of Left Ventricular Systolic Function using TEE

Tamas Seres, M.D.
UCHSC

Myocardial Remodeling

- Cardiac remodeling is thought to be an important aspect of disease progression in HF regardless of cause.
- It is manifested clinically by changes in cardiac size, shape, and function in response to cardiac injury or increased load.

Myocardial Remodeling

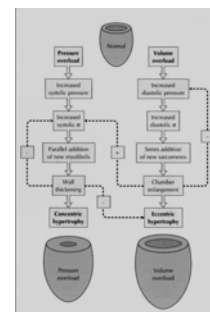
- Remodeling can be a physiologic or pathologic condition:
 - Physiologic remodeling is a compensatory change in the proportions and function of the heart; this type of remodeling is seen in athletes.

- Pathologic remodeling may occur:
 - After myocardial infarction
 - With pressure overload (eg, aortic stenosis, hypertension)
 - Inflammatory myocardial disease (myocarditis)
 - With idiopathic dilated cardiomyopathy
 - With volume overload (eg, valvular regurgitation)

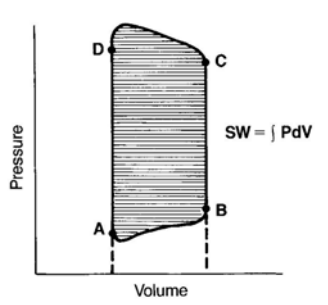
Myocardial Remodeling

- Altered loading conditions (eg, increased preload) stretch cell membranes and increase wall stress, which may play a role in inducing the expression of hypertrophy associated genes.
- In cardiac myocytes, this may lead to the synthesis of new contractile proteins and the assembly of new sarcomeres.

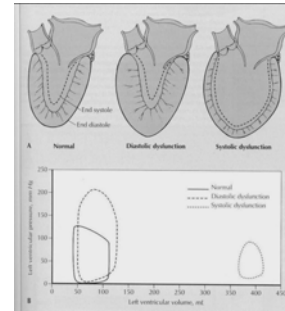
Myocardial Remodeling



Pressure-Volume Loop



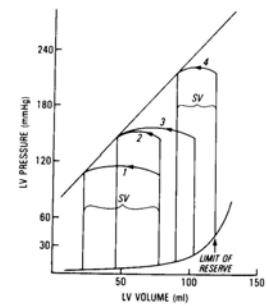
Myocardial Remodeling



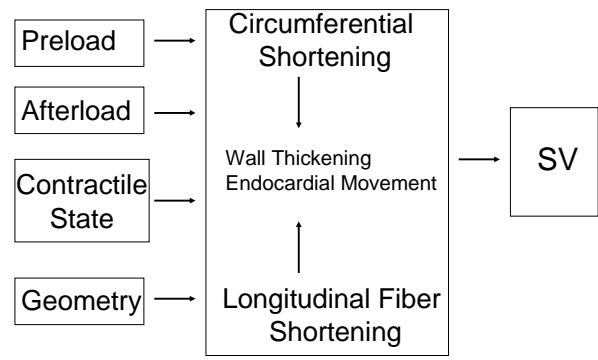
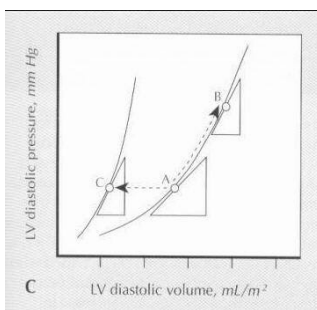
Myocardial Remodeling

- Systolic heart failure (SHF), is characterized by progressive chamber dilation, eccentric remodeling, and abnormalities in systolic function.
- Diastolic heart failure (DHF), is characterized by normal LV volume, concentric remodeling, normal LV systolic properties, and abnormalities in diastolic properties.

Systolic Pressure-Volume Relationship



Diastolic Pressure-Volume Relationship



Evaluation of the LV Systolic Function

- Fractional Shortening: 28-41 %
- Fractional Area Change: 36 - 64 %
- Ejection Fraction: 45 - 75 %
- Stroke Volume: 36-76 ml/m²
- Cardiac Output: 5 l/min

Teicholz Method

- $V = [7.0 / (2.4 + D)] (D^3)$

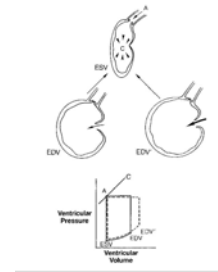
V = LV volume

D = LV internal dimension

Preload

- Evaluation of end-diastolic volume

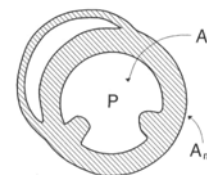
The Effect of Increasing Preload



Afterload

- LV wall stress: $\sigma = 1.33 P (A_c / A_m)$ (dynes/cm²)
- σ = end systolic wall stress
P = cuff systolic BP
A_c = LV end systolic cavity area
A_m = myocardial area (end-systolic epicardial area – end-systolic endocardial area)

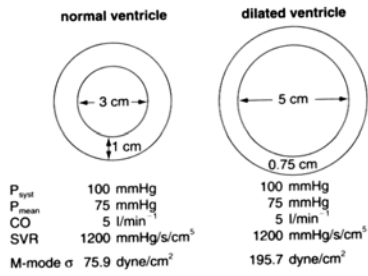
LV wall stress measurement



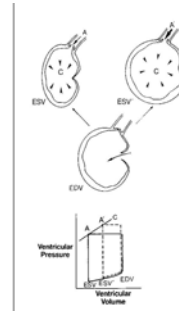
$$\sigma_{\square} = 1.33P(A_c / A_m) \times 10^3 \text{ dynes/cm}^2$$

Normal values: 60-100 dynes/cm²

Comparison of vascular resistance and end-systolic wall stress in evaluation of afterload



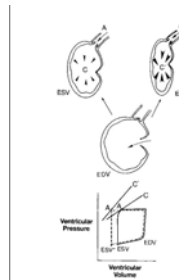
The Effect of Increasing Afterload



Contractility

- Maximal rate of LV pressure rise measured from mitral regurgitant jet

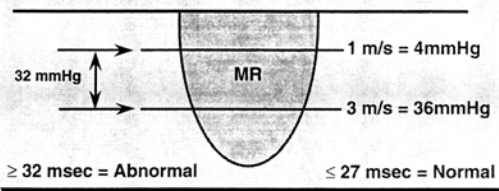
The Effect of Increasing Contractility



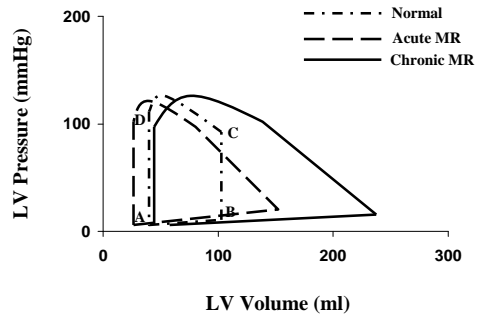
Isovolumetric Phase Index of LV Systolic Performance (dP/dt)

LV Systolic Performance (dP/dt)

Time (msec) it takes for the LV pressure to rise 32 mmHg



Normal: < 27 msec or > 1200 mmHg/s Abnormal: > 32 msec or < 1000 mmHg/s



Geometry

- Wall motion abnormalities

Wall motion:	Endocardial movement	Myocardial thickening
Normal:	normal	>30%
Mild hypokinesis:	decreased	10-30 %
Severe hypokinesis:	slight	<10%
Akinesis:	none	none
Dyskinesis	outward movement	thinning

Tissue Velocity Imaging

- Measuring the velocity of the myocardium during systole and diastole.
- The systolic maximal velocity characterizes the longitudinal contraction.
- The diastolic velocity pattern characterizes the diastolic function.

Tissue Tracking

- Measures distance of the movement of the myocardium.
- Velocity time integral of the tissue velocity curve.

Strain Imaging

- Measures deformation of the myocardium between two points.
- The percent of longitudinal deformation of the myocardium (%).