Heart Failure = Pump Failure

Can Develop Rapidly (‘Acute’)
- M.I.
- Infection
- Post bypass surgery
Heart Failure = Pump Failure

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<td>HTN/AS / AR/AS</td>
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<tr>
<td>• Adaptive Remodeling</td>
</tr>
<tr>
<td>• Functional Decline</td>
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Heart Failure = Pump Failure

Myocardial Damage

Coronary Artery Disease
- Ischemia
- Necrosis/Fibrosis
- Diminished Function

Failure to relax properly

Systolic Failure
- Can’t Pump Enough

Diastolic Failure
- Can’t Fill Enough

Overload
- +Afterload
  - HTN
  - AS

Remodeling
- +Preload
  - MR
  - AR

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Adaptations / Remodeling

+ Afterload
Greater force needed to overcome greater load

Concentric

Normal
Adaptations / Remodeling

+ Afterload
Greater force needed to overcome greater load

Concentric

Normal

+ Preload
Chamber expands to accommodate larger volume

Eccentric

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Impacts of Adaptations and Remodeling Process
Adaptation to +Afterload – Concentric LVH

Hypertension
Aortic Stenosis

Increased
Wall thickness

Increased # of
Parallel sarcomeres

Increased
Wall stress tends
to be maintained

\[ \sigma = \frac{pr}{w} \]

Decreased
Ventricular Compliance

\[ C = \frac{dv}{dp} \]

\[ C = \frac{1}{[E \times (w/r)]} \]

Heart Failure

Filling Problems
Further elevation
In EDP

Diastolic Dysfunction

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Adaptation to +Preload (+ EDV) – Eccentric LVH

Volume Overload
MR or AR

EDV1
Systolic Dysfunction
Abnormally Decreased Contractility

Ischemia Infarction Myopathies Other

EDV2
Chamber Dilation
Increased # of Sarcomeres in series

Wall stress tends to increase
\[ \sigma = \frac{pr}{w} \]
- Increased O\(_2\) demand
- Reduced EF
- Heart Failure

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Pressure/Volume Overload

Adaptive Hypertrophy

PRESSURE +Muscle Mass

VOLUME +Chamber Size

Concentric Hypertrophy

Eccentric Hypertrophy

Normal  LVH (AS)  Dilated (AR)
Systolic and Diastolic Dysfunction Summary
Systolic Dysfunction

- Myocardial Contractility
  ↓
  Impaired Contraction
  ↓
  -SV
  ↓
  +EDV
  ↓
  +EDP
  → IF LV involved: Pulmonary congestion & edema
  IF RV involved: Peripheral edema and ascites

Intrinsic e.g. Deficit of Contractile Apparatus
Loss of Viable Contracting Muscle e.g. M.I.
Diastolic Dysfunction

- Ventricle Compliance
  - EDV
  + EDP
  - SV

Pulmonary Congestion

Concentric Hypertrophy
  + Muscle Mass
  + Wall Thickness

Reduced Lusitropy

Reduced removal of calcium from SR

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Review of Physiological Processes and Underpinnings
Decreased Contractility $\rightarrow$ Reduced Velocity

Tx $\rightarrow$ Reduce Afterload $\rightarrow$ Vasodilator

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Decreased Contractility $\rightarrow$ Increased Preload

Tx $\rightarrow$ +Inotropy $\rightarrow$ Cardiac Glycoside (digitalis)

Cardiac Function Curves Shift

$SV$ (ml)

$LVEDP$ (mmHg)

Na/K-ATPase

$+[Na^+]$

$Na^+/Ca^{++}$

$+[Ca^{++}]$

+Contractility

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Decreased Contractility $\rightarrow$ Increased Preload

Tx $\rightarrow$ +Inotropy $\rightarrow$ Cardiac Glycoside (digitalis)

P-V Loop Shifts

- SV
- EF
- SW
+ EDV
Diastolic Dysfunction \rightarrow Reduced Compliance

- EDV
- SV
- SW
- EDP

Due to:
- Concentric Hypertrophy
- Negative Lusitropy
**Reduced Lusitropy → Decreased Compliance**

**Excitation-Contraction Coupling**

Cardiac Myocyte

- **TN-C = Troponin-C = Ca^{++} binding site**
- **Actin**
- **Myosin**
- **Tropomyosin**

**Trigger Ca^{++}**

- **Ca^{++}**
- **SR**

- **SR removes Ca^{++} from TN-C vicinity**
- **Actin-Myosin disengage**

**Reduced Ca^{++} removal rate and/or amount reduces relaxation rate and/or amount**

**Ventricular Relaxation**
Thanks for your attention! QUESTIONS?