ENERGIZING THE HEART: FOCUSING ON HF MANAGEMENT THROUGH DEVICES

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Objectives
- Review definition and classifications of heart failure.
- Identify and discuss optimal pharmacological management for non-acute heart failure.
- Identify non-pharmacological (nursing interventions) to optimize heart failure management.
- Identify the role of resynchronization pacing and ICD therapy in heart failure management.
- Determine proper resynchronization (biventricular) pacing function in pacemakers and ICDs.

Heart Failure: United States

- Annual Incidence (New Cases)
- Heart Failure Prevalence (estimate at a given point)
- Annual Mortality

Go, AS, et. al. for AHA Statistics Committee and Stroke Statistics; Heart Disease and Stroke Statistics—2014 Update
www.americanheart.com
http://www.cdc.gov/heartdisease/statistics.htm

HF Stats
- Hospital discharges unchanged from 2000 to 2010 (1.0 ⇒ 1.02 million)
- Approximately 80% of men and 70% women <65 yrs with HF die within 8 yrs
- Most common causes: CAD, HTN, DM
- HF increases SCD risk by 6-9 fold

American Heart Association. 2009 Heart and Stroke Statistical Update
**HF Stats**
- 75% of HF cases have hypertension (BP>160/90 = double risk)
- Majority of HF patients have impairment of left ventricular function
- At age 40, lifetime risk of HF (MI) = 1 in 5 (men & women). No MI = 1 in 9 (men), 1 in 6 (women)
- 2012 total cost of HF in U.S.= $30.7 million (68% direct medical)
- Part of CMS quality measures (LV assessment and use of ACEI/ARBs)

**Heart Failure**
- Definition: Heart unable to pump blood to meet demands/needs of periphery
  - Syndrome characterize by: SOB, fatigue
  - Classified according to physiologic symptoms:
    - **Systolic** (decrease CO-pump problem): decreased LVEF (<40%); also referred to as LV failure
    - **Diastolic** (filling problem): elevated venous pressures; LVEF probably normal(>40%); also referred to as RV failure

**5 Million Lives: New interventions targeted at harm**
- Prevent Harm from High-Alert Medications... starting with a focus on anticoagulants, sedatives, narcotics, and insulin
- Reduce Surgical Complications... by reliably implementing all of the changes in care recommended by SCIP, the Surgical Care Improvement Project (www.medqic.org/scip)
- Prevent Pressure Ulcers... by reliably using science-based guidelines for their prevention
- Reduce Methicillin-Resistant Staphylococcus aureus (MRSA) infection... by reliably implementing scientifically proven infection control practices
- Deliver Reliable, Evidence-Based Care for Congestive Heart Failure... to avoid readmissions

**Heart Failure Definition**
- **“Acute”**
  - Sudden onset
  - Exacerbation of symptoms
  - Life threatening
- **“Chronic”**
  - Stable or progressive worsening of symptoms
  - Decompensated (may also be acute)
Pathophysiology

- Starling’s Law: Force of contraction is proportional to length (amount) of myocardial muscle fiber stretch
- Heart compensates by lengthening fiber and/or increasing diastolic ventricular volume
- Results in dilated ventricles, back pressure into pulmonary & systemic venous systems

Relationship of Determinants to CO

- CO
- HR
- SV
- Preload
- Afterload
- Contractility

Pathophysiology

- **Stroke volume** – the amount of blood ejected with each cardiac cycle
- **Preload** – the amount of stretch on the cardiac muscle fibers produced by the volume of blood in the ventricles at the end of diastole (depends on the volume of venous return to the heart)
- **Afterload** – the amount of resistance that the left ventricle must overcome in order to pump or eject blood. (the greater the afterload the harder the myocardium will have to pump)

Pathophysiology

- **Contractility** – the ability of the heart muscle fibers to contract or shorten. The degree of preload and afterload that the fibers must work against to eject blood, and the ability of the fibers to contract to produce the force of ejection determine the stroke volume.
Heart Failure
Risk Factors and Causes

- Risk Factors
  - Noncompliance with medical therapies
  - HTN
  - Arrhythmias (VT/AF)
  - Endocrine (hyperthyroid)
  - Pulmonary (PE, infection)
  - Renal/liver disease
  - Alcohol/cocaine

- Primary Causes
  - IHD/CAD
  - Uncontrolled HTN
  - Cardiomyopathy

Signs and Symptoms of HF

- Decreased activity tolerance
- Fatigue
- Dyspnea (PND)
- Orthopnea
- Cough
- Edema: peripheral or pulmonary
- Arrhythmias

Assessment - Systolic

- Echocardiogram -- EF will help determine type
- EKG -- evidence of ischemia or MI
- Labs
  - CBC, Basic chem, thyroid, lipids, UA, BNP
  - CXR (note cardiac size, pulmonary congestion)
- If no h/o CAD -- PTST or cath to r/o ischemia (if negative, then most likely diastolic HF)

Classification of HF: Signs/Symptoms

<table>
<thead>
<tr>
<th>NYHA Functional Class System</th>
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<tbody>
<tr>
<td>Class</td>
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<tr>
<td>Class I</td>
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<tr>
<td>Class II</td>
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<tr>
<td>Class III</td>
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<tr>
<td>Class IV</td>
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</tbody>
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1. Able to run upstairs
2. Short of breath after climbing stairs
3. Pauses during climbing of stairs
4. Unable to climb stairs
### ACC/AHA Classification of Heart Failure (based on progression)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
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<tbody>
<tr>
<td>A</td>
<td>At risk, but no structural dz or symptoms of HF</td>
</tr>
<tr>
<td>B</td>
<td>(+)structural dz, no symptoms of HF</td>
</tr>
<tr>
<td>C</td>
<td>Structural dz with prior or current symptoms of HF</td>
</tr>
<tr>
<td>D</td>
<td>Refractory HF (end stage), needs specialized interventions</td>
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### BNP (B-Type Natriuretic Peptide)

#### Interpretive Guide:

<table>
<thead>
<tr>
<th>BNP level</th>
<th>Interpretation</th>
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<tbody>
<tr>
<td>≤ 100 pg/mL</td>
<td>Negative</td>
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<tr>
<td>&gt;100 pg/mL</td>
<td>Positive</td>
</tr>
<tr>
<td>&gt;200 pg/mL</td>
<td>High correlation with LV failure</td>
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#### BNP Key Points

- Use in conjunction with physical exam.
- BNP secreted in response to ventricular volume and pressure.
- Circulating BNP is proportionate to severity of heart failure.

### NT-proBNP (N-Terminal pro-B Type)

<table>
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<tr>
<th>Age</th>
<th>Rule IN Value</th>
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<tbody>
<tr>
<td>&lt; 50 years</td>
<td>450</td>
</tr>
<tr>
<td>50-75 years</td>
<td>900</td>
</tr>
<tr>
<td>&gt; 75 years</td>
<td>1800</td>
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Rule out value ≤ 300
Factors Affecting BNP

- Age – baseline increases with age
- Sex – women > men
- Cirrhosis – baseline can be 3X> than healthy subjects
- Renal failure – baseline > before dialysis
- LV hypertrophy – correlates with age related changes
- Other: myocarditis, cardiac allograft rejection, Kawasaki dz, primary pulmonary HTN, hyperaldosteronism, Cushing syndrome

Heart Failure: Treatment

- Goals of therapy for systolic and diastolic HF:
  - Symptom management
  - Maintain/improve quality of life
  - Prolong survival/delay progression

Treatment Guidelines

- HFSA Comprehensive Heart Failure Practice Guideline 2010
- Link to practice guideline website
  - www.hfsa.org
  - www.guideline.gov
- www.americanheart.org

Treatment

- Treat underlying cause for both types
- Systolic Dysfunction
  - Pharmacological Interventions (determined by functional class)
    - Class I
      - ACEI (even if asymptomatic)
      - Asymptomatic hypotension is not contraindication
      - Start low dose and increase gradually
      - Captopril 6.25 mg bid; Enalapril 2.5 mg bid; Lisinopril 2.5 mg qd
      - ARB
      - Valsartan, Candesartan
### Treatment

#### Systolic Dysfunction

- **Class I**
  - Pharmacological
    - Diuretics (if edema or wt. gain)
    - *Asymptomatic hypotension not contraindication
    - HCTZ 25-50 mg QD or BID

- **Class II-IV**
  - Pharmacological
    - ACEI (isosorbide + hydralazine if don’t tolerate ACEI)
    - ARB (angiotensin receptor blockers)
    - Diuretics (symptom management) Spironolactone
    - Digoxin (? Benefits in recent studies, not first line)
    - Beta-blockers (Metoprolol succinate, Carvedilol, Bisoprolol)
      - For class IV patients
      - Optimize diuretics prior to initiating B-blockers
      - Decreases cardiac remodeling
      - Anticoagulation not support in recent studies (? If EF<10%)

#### Diastolic Dysfunction

- **Pharmacological**
  - Diuretics & Nitrates
  - Beta-Blockers -- slow heart rate, decrease BP
  - ACEI (watch for hypovolemia)
  - Nonselective beta-blockers like Carvedilol
  - Ca channel blockers
    - (? , except Amlodipine)

### Non-pharmacological

- **Patient Education**
  - Wt. gain
  - Side effects of meds
  - Self adjustments according to symptoms
  - Limit Alcohol intake
  - Home BP monitoring
  - Adequate rest (naps are OK !)

- **Cardiac rehab/exercise**
- **Diet** -- sodium and H2O restriction
- **Support groups**
- **Manage depressive symptoms (women)**
- **Advance Directives/end-of-life planning**

*Song EK, Moser DK, Lennie TA. Relationship of depressive symptoms to the impact of physical symptoms on functional status in women with heart failure. AJCC. 2009; 18(4):348-356.*
Other Therapies/Interventions

**Prevention:**
- Pacing
- Dual chamber to preserve atrial kick
- Atrial overdrive to minimize atrial fibration
- Limit RV pacing

**Intervention:**
- Resynchronization therapy – if maximized on medical therapies and still symptomatic; QRS > 130 ms.
- Surgery (transplant, CABG, Valve)

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Permanent Pacing for Prevention of Atrial Fibrillation

- Evidence that AAI pacing is associated with less atrial fibrillation than VVI pacing.\(^1\)
- Chronic dual-site right atrial pacing may also prevent recurrent atrial fibrillation.\(^2\)

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Atrial Pacing Preference (APP)/AF Suppression

- Paces the atrium at a rate slightly higher than the underlying sinus rhythm in an effort to reduce atrial tachyarrhythmias

![Diagram of atrial pacing preference](image)

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Preventing Pacemaker Mediated Cardiomyopathy

Managed Ventricular Pacing
- {Medtronic}
  - AAI (R) \(\leftrightarrow\) DDI (R)

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\(^1\) Andersen HR. Lancet. 1994;344:1523-1528.
\(^2\) Saksena S. J Am Coll Cardiol. 1996;28(3);687-694.
DAVID

- Study results are consistent with the pacing literature.
  - AAI was associated with slightly better survival and lower rate of severe CHF compared to VVI pacing mode in patients with SSS.
  - QOL was better in elderly patients with sinus node disease with VVI compared to DDD pacing.
  - More than 40% ventricular pacing was associated with increased CHF hospitalizations.
  - The benefit of DDDR pacing was most evident in patients who needed continuous pacing.

Conclusions

- Bradycardia pacing operation in dual-chamber ICDs should be optimized for individual patients.
  - RV pacing in patients with LV dysfunction and no bradycardia indication for pacing can be harmful.
  - Programming of dual chamber devices to backup ventricular pacing is justified in this patient population.

MOST Sub-study

Conclusions: CHF

- Higher rates of CHF hospitalization were associated with higher Cum% VP:
  - Cum % VP<10% was associated with the lowest rates of CHF hospitalization (DDDR 2%, VVIR 7%).
  - Cum % VP >90% was associated with the highest rates of CHF hospitalization (DDDR 12%, VVIR 16%).

- Ventricular pacing in the DDDR mode more than 40% confers a 3-fold increased risk of heart failure hospitalization but can be reduced to about 2% if ventricular pacing is minimized.

Novel Pacing Algorithms to Optimize Ventricular Pacing

- Minimal ventricular pacing modes can be used in all patients, but are most effective in SND (sinus node dysfunction) patients with reliable AV conduction and normal ventricular activation.
- Development will continue on new pacing algorithms which have been identified as an important means of minimizing ventricular pacing.

References:
MVP Basic Operation

AAI(R) Mode
Atrial based pacing allowing intrinsic AV conduction

PR Intervals are only restricted by the underlying atrial rate or sensor rate; VS events simply need to occur prior to the next AS or AP.

MVP Basic Operation

Ventricular Backup
Ventricular pacing only as needed in the presence of transient loss of conduction

MVP Basic Operation

DDD(R) Switch
Ventricular support if loss of A-V conduction is persistent