PESI Health Care Seminar
All Things Cardiac: Day 2

IMPACTING OUTCOMES
ONE PROFESSIONAL
AND ONE PATIENT
AT A TIME

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www.cardionursing.com

Heart Failure
Pathophysiology
Heart failure is a complex clinical syndrome resulting from any structural or functional cardiac disorder impairing the ability of the ventricle to either fill or eject.

Clinical Syndrome Resulting in Clinical Manifestations

- Dyspnea and fatigue
  - May limit exercise tolerance

AND / OR

- Fluid overload
  - May lead to pulmonary congestion and peripheral edema

Impaired functional capacity and quality of life
# Classifying Heart Failure

### Preserved (HFpEF) LVEF

### Reduced (HFrEF) LVEF

## Heart Failure Stages

New York Association Classification

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## Definition of Heart Failure

<table>
<thead>
<tr>
<th>Classification</th>
<th>Ejection Fraction</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Heart Failure with Reduced Ejection Fraction (HFrEF)</td>
<td>≤40%</td>
<td>Also referred to as systolic HF.</td>
</tr>
<tr>
<td>II. Heart Failure with Preserved Ejection Fraction (HFpEF)</td>
<td>≥50%</td>
<td>Also referred to as diastolic HF. Several different criteria have been used to further define HFpEF. The diagnosis of HFpEF is challenging because it is largely one of excluding other potential non-cardiac causes of symptoms suggestive of HF.</td>
</tr>
<tr>
<td>a. HFpEF, Borderline</td>
<td>41% - 49%</td>
<td>These patients fall into a borderline or intermediate group. Their characteristics, treatment patterns, and outcomes appear similar to those of patients with HFpEF.</td>
</tr>
<tr>
<td>b. HFpEF Improved</td>
<td>&gt;40%</td>
<td>It has been recognized that a subset of patients with HFpEF previously had HFrEF. These patients with improvement or recovery in EF may be clinically distinct from those with persistently preserved or reduced EF.</td>
</tr>
</tbody>
</table>
HFrEF - Systolic Dysfunction

- 50% of HF Population
- **Hallmark:** Decreased LV ejection fraction (LVEF) ≤ 40%
  - Impaired wall motion and ejection; dilated chamber
- Coronary artery disease is cause in 2/3 of patients
- Remainder: Other causes of LV dysfunction/cardiomyopathy

![Diagram of normal and dilated LV]

Note: Cardiomyopathy can exist without the clinical syndrome of HF

HFpEF - Diastolic Dysfunction

- 50% of the population
- Filling impairment
  - Normal chamber size
  - Normal or increased LVEF
- Caused by
  - Ventricular hypertrophy (Fig D)
  - Hypertension
  - Restrictive myopathy (Fig C)
  - Ischemic heart disease
  - Valve disease
  - Idiopathic

![Diagram of normal LV]

Primarily disease of elderly women with hypertension
Stages, Phenotypes and Treatment of HF

**STAGE A**
At high risk for HF but without structural heart disease or symptoms of HF
- Patients with:
  - HTN
  - Atherosclerotic disease
  - DM
  - Obesity
  - Metabolic syndrome
  - Using cardio toxins
  - With family history of cardiomyopathy

**STAGE B**
Structural heart disease but without signs or symptoms of HF
- Patients with:
  - Previous MI
  - LV remodeling including LVM and low EF
  - Asymptomatic valvular disease

**STAGE C**
Structural heart disease with prior or current symptoms of HF
- Patients with:
  - Known structural heart disease and HF signs and symptoms

**STAGE D**
Refractory HF
- Patients with:
  - Marked HF symptoms at rest
  - Recurrent hospitalizations despite GDMT

**THERAPY**

**Goals**
- Control symptoms
- Improve HRQOL
- Prevent hospitalization
- Prevent mortality

**Strategies**
- Identification of comorbidities
- Drug treatment for NYHA class III/IV symptoms
- Advanced care measures

**Drugs**
- ACEI or ARB as appropriate
- Beta blockers as appropriate
- In selected patients
  - ICD
  - Revascularization or valvular surgery as appropriate

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**THERAPY**

**Goals**
- Control symptoms
- Improve HRQOL
- Reduce hospital readmissions
- Establish patient’s end-of-life goals

**Options**
- Advanced care measures
- Heart transplant
- Cardiac assist devices
- Temporary or permanent MCS
- Experimental surgery or drugs
- Palliative care and hospice
- ICD deactivation

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**Stages of Heart Failure**

**Stage A**
- At high risk for HF but without structural heart disease or symptoms of HF

**Stage B**
- Hypertension
- CAD
- Diabetes
- Obesity
- Metabolic syndrome

**Stage C**
- Using cardio toxins
- Family history of cardiomyopathy

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**Heart Failure**
- Hypertension
- CAD
- Diabetes
- Obesity
- Metabolic syndrome

**Stage D**
- Refractory HF
- Marked HF symptoms at rest
- Recurrent hospitalizations despite GDMT

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**Patients with**
- Marked HF symptoms at rest
- Recurrent hospitalizations despite GDMT

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**ASYMPTOMATIC**
<table>
<thead>
<tr>
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ASYMPTOMATIC

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<th>Stage C</th>
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<td>• Structural heart disease with prior or current symptoms of HF</td>
<td>• Known structural heart disease and HF signs and symptoms</td>
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<tr>
<td></td>
<td>Divides patients into two groups:</td>
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<tr>
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<td>HFpEF HFrEF</td>
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</table>
Stages of Heart Failure

**Stage D**
- Refractory HF

**Patients with**
- Marked HF symptoms at rest
- Recurrent hospitalizations despite Guideline Directed Medical Therapy (GDMT)

**SYMPTOMATIC on treatment**

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**NYHA Functional Classification**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
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<tbody>
<tr>
<td>I</td>
<td>No limitation of physical activity. Ordinary physical activity does not cause symptoms of HF.</td>
</tr>
<tr>
<td>II</td>
<td>Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in symptoms of HF.</td>
</tr>
<tr>
<td>III</td>
<td>Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes symptoms of HF.</td>
</tr>
<tr>
<td>IV</td>
<td>Unable to carry on any physical activity without symptoms of HF, or symptoms of HF at rest.</td>
</tr>
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</table>
PATHOPHYSIOLOGY OF LEFT VENTRICULAR FAILURE: REDUCED LVEF

The Real Culprit: Neurohormonal Response
(Sympathetic Nervous System and Renin Angiotensin Aldosterone System)
Autonomic Nervous System

Sympathetic

Beta 1
- ↑ HR
- ↑ Contractility
- ↑ Conductivity
- Bronchodilation
- Arterial Vasodilation

Beta 2
- Arterial Vasodilation

Alpha 1
- Arterial Vasoconstriction

Parasympathetic

Vagal Response
- ↓ HR

Activation of SNS

• First responder
  – Decreased cardiac output (CO) → ↓ BP → activates baroreceptors and vasomotor regulatory centers in medulla

• Increase circulating catecholamines
  – Stimulates alpha1 and beta receptors
    • Increased HR
    • Peripheral vasoconstriction
    • Increased contractility

Positive effect: ↑ CO and BP

Negative effect: ↑ O₂ demand → ischemia, arrhythmias, sudden death
**Chronic Stimulation of SNS**

- Circulating norepinephrine is cardiotoxic
  - Down regulation of B1 receptor sites (less sensitive)
  - Decreases heart’s ability to respond to sympathetic stimulation
    - Contributes to decreased exercise tolerance
  - Also leads to ventricular remodeling

Be aware of your patient’s heart rate response to activity

**Activation of RAAS**

- Kidney’s response to decreased perfusion due to decreasing cardiac output
- Concentrations of angiotensin II and aldosterone rise as end result
  - Potent vasoconstriction
  - Sodium / water absorption increases

**Result**
- Increased preload and increased afterload
- Increased myocardial oxygen demand
Enhanced preload increases end-diastolic volume dilating the LV
- LV becomes overstretched
- LV changes size and shape (ventricular remodeling)

Contractility decreases
Congestive symptoms develop

Process of pathological growth
Can occur from
- Prolonged activation of SNS / RAAS
- Pathological substrates
- Involves
  - Hypertrophy of myocytes
  - Increased pressure (HFpEF)
    - Thicken myocytes (concentric)
  - Increased volume (HFrEF)
    - Elongate myocytes (eccentric)
The Good Guys: Natriuretic Peptides

- Cardiac hormones secreted by myocytes
  - *Atrial natriuretic peptide (ANP)*
    - Produced in atria
  - *Brain natriuretic peptide (BNP)*
    - Produced in ventricles in response to increased ventricular pressure/stretching
    - Stronger release than ANP
- Promote venous and arterial vasodilatation
  - Preload and afterload reduction
- Reduce sodium/water retention
- Reduce production / action of vasoconstrictor peptides
- Plasma concentrations elevated in patients in fluid overload

HFpEF - Diastolic Dysfunction

- Diagnosis is made when rate of ventricular filling is slow
- Elevated left ventricular filling pressures when volume and contractility are normal

**IN PRACTICE:**
The diagnosis is made when a patient has typical signs and symptoms of heart failure and has a normal or elevated ejection fraction with no valvular heart disease.
Normal LV

LV Hypertrophy

Left versus Right Sided Heart Failure

- Two sides of the heart form a circuit, neither side can pump significantly more blood than the other for long
- Signs/symptoms of failure reflect each respective ventricle
RV Dysfunction Secondary to LV Dysfunction

- Chronic left HF is most common cause of RV failure
  - RV dysfunction more common in non ischemic etiology
  - RVEF is a predictor of mortality in patients with left HF
- LV failure leads to increased RV afterload – first from elevated pulmonary venous pressures then ultimately elevated pulmonary artery pressures
- Same pathophysiology resulting in LV failure may affect RV
- Ischemia may affect both ventricles
- LV dysfunction may lead to decreased systolic driving pressure of RV coronary perfusion
- Ventricular interdependence due to septal dysfunction may occur
- LV dilatation in a confined pericardial space may restrict RV diastolic function

Pathophysiology of Right Ventricular Failure

- In response to increased pulmonary artery (PA) pressures
  - Initial adaptive hypertrophy; followed by progressive contractile dysfunction
  - Chamber enlargement allows for compensation for increased preload and maintenance of stroke volume (SV)
  - Pulmonary hypertension may cause
    - RV ischemia
    - Microvascular endothelial cellular dysfunction
    - Apoptosis of myocytes
### Causes of Right Heart Failure (Examples)

<table>
<thead>
<tr>
<th>Pressure Overload</th>
<th>Volume Overload</th>
<th>Ischemia and Infarction</th>
<th>Intrinsic Myocardial Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>● Left sided HF</td>
<td>● Tricuspid regurgitation</td>
<td>● RV myocardial infarction (RV is very resistant to irreversible ischemia, can regain systolic function)</td>
<td>● HF</td>
</tr>
<tr>
<td>● Mitral stenosis</td>
<td>● Pulmonic regurgitation</td>
<td></td>
<td>● Arrhythmogenic RV dysplasia</td>
</tr>
<tr>
<td>● Pulmonary embolus</td>
<td>● Mitral regurgitation</td>
<td></td>
<td>● Sepsis</td>
</tr>
<tr>
<td>● Other causes of pulmonary hypertension</td>
<td>● Atrial septal defect</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Inflow Limitation</th>
<th>Congenital</th>
<th>Pericardial Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>● Tricuspid stenosis</td>
<td>● Ebstein's anomaly</td>
<td>● Constrictive pericarditis</td>
</tr>
<tr>
<td></td>
<td>● Tetralogy of Fallot</td>
<td></td>
</tr>
<tr>
<td></td>
<td>● Transposition of great arteries</td>
<td></td>
</tr>
</tbody>
</table>

### Response to Failing RV

- Neurohormonal, endothelin and cytokine activation
  - SNS adversely affects RV remodeling
  - RAAS activation affects RV remodeling and contributes to fluid retention
  - Endothelin I levels in pulmonary arterial hypertension are associated with more severe RV dysfunction and decreased exercise capacity
  - Increased levels of tumor necrosis factor and endotoxin are associated with more symptomatic disease
Decompensated RV Failure

- Rising filling pressures
- Diastolic dysfunction of LV
  - RV dilation or pressure overload causes leftward shift of septum and impacts LV function
- Decreased cardiac output
  - PA pressures may actually decrease in severe RV failure due to decreased CO of RV
- Tricuspid valve regurgitation
  - Annular dilatation and poor leaflet coaptation
  - Aggravates volume overload and further decreases RV forward flow

Manifestations of RV Failure

- Fluid retention
  - JVD
  - + Hepatopressor Reflex
  - Peripheral edema
  - Ascites
    - Cardiac cirrhosis in severe cases
    - Anasarca
- Decreased systolic reserve (decreased C.O.)
  - Decreased exercise tolerance
    - Exercise capacity may be one of most important prognostic factors
  - Fatigue
- Cardiac arrhythmias
  - Atrial most common
- Protein losing enteropathy
  - Severe loss of proteins into the intestine
RV Dysfunction and Prognosis

• RV function is the most important determinant of longevity in patients with pulmonary arterial hypertension (PAH)

• In left ventricular failure, RV failure may be final common pathway and thus sensitive indicator for impending decompensation and poor prognosis

• Severity of tricuspid regurgitation correlates with worse survival

Heart Failure Symptoms

- Exercise intolerance (hallmark) – Assess NYHA
- Paroxysmal nocturnal dyspnea
  - Attacks of severe shortness of breath and coughing that generally occur at night; It usually awakens the person from sleep, and may be quite frightening
- Orthopnea
  - Shortness of breath (dyspnea) which occurs when lying flat
- Bendopnea / Flexopnea
  - Shortness of breath when bending forward for 30 seconds
- Cough
- Post nasal drip
- Frequent night urination with less during the day
- Weight change/increase abdominal girth/peripheral edema
- Decreased appetite / early satiety
- Chest pain
- Worsening renal function
- Confusion/altered mental status
Physical Exam Findings

- General appearance
  - Resting dyspnea, cyanosis, cardiac cachexia
- Weight gain
- JVD
- Hepatojugular reflux
- Edema
- Displaced apical impulse
- RV heave
- S3/S4
- Murmurs – MR, AS, AI, TR
- BP/HR
  - Include orthostatic pressures
- Abnormal lung sounds

Preload Assessment: Volume

**Right Ventricular Preload**

- JVD
- Hepatojugular reflux

- Less specific
  - Peripheral edema
  - Weight

**Left Ventricular Preload**

- Dyspnea / increased work of breathings
- Hypoxemia (diffusion abnormality)
- Orthopnea / bendorpnea (flexopnea)
- CXR
- BNP / NT-proBNP
- Lungs sounds
  - Role of lymph drainage
  - Clear lungs do not rule out volume overload
- S3 or S4
- Less specific
  - Blood Pressure
  - Urine Output
  - Weight
Diastolic Filling Sounds
S3 - Ventricular Gallop

• Early diastolic filling sound
• Caused by increased pressure and resistance to filling
• Most frequently associated with systolic dysfunction
• Associated with:
  – Fluid overload state
  – Right or left ventricular failure
  – Ischemia
  – Aortic regurgitation
  – Mitral regurgitation

Diastolic Filling Sounds
S3

• Patient position: left lateral decubitus position
• Location:
  – Left-sided S3 – mitral area
  – Right-sided S3 – tricuspid area
• Intensity
  – Left-sided heard best during expiration
  – Right-sided heard best during inspiration
• Duration: short
• Quality: dull, thud like
• Pitch: low
• May be normal in children, young adults (up to 35-40) and in the 3rd trimester of pregnancy
Diastolic Filling Sounds

\( S_4 \) - Atrial Gallop

- Late diastolic filling sound
- Caused by atrial contraction and the propulsion of blood into a noncompliant (stiff) ventricle
- Most frequently associated with diastolic dysfunction
- Associated with:
  - Fluid overload state
  - Systemic hypertension
  - Ischemia
  - Aortic stenosis
  - Restrictive cardiomyopathy
  - Hypertrophic cardiomyopathy
- May be normal in athletes

Diastolic Filling Sounds

\( S_4 \)

- Patient position: left lateral decubitus position
- Location
  - Left-sided \( S_4 \) – mitral area.
  - Right-sided \( S_4 \) – tricuspid area
- Intensity
  - Left-sided louder on expiration.
  - Right-sided louder on inspiration
- Duration: Short
- Quality: Thud like
- Pitch: Low
Chest X Ray in Volume Overload

- Prominent vascular markings: upper lung fields
- Peribronchial thickening
- Patchy alveolar filling in a perihilar distribution – progressing to diffuse infiltrates
- Kerley B lines

Kerley B Lines
When assessing volume also assess for volume depletion: skin turgor, mucous membranes, and orthostatic blood pressures.

**Diagnosis – Initial Lab Studies**

- CBC
  - Anemia
  - Infection as precipitator for decompensation
- UA
  - Urine protein
- Electrolytes – including magnesium and calcium
- BUN / Creatinine
- Glucose / A1C
  - Concern for osmotic diuresis
- Fasting lipid profile
- Liver function studies
  - Right sided HF
- Albumin
  - Nutritional status
- Thyroid stimulating hormone
  - Diagnose new disease or evaluate the effectiveness of current treatment
- Troponin
  - Rule out ACS

**BNP / NT proBNP**
Diagnosis and Prognosis
### BNP versus NT-proBNP

<table>
<thead>
<tr>
<th>BNP</th>
<th>NT-proBNP</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Lower levels than NT-proBNP</td>
<td>• Higher levels than BNP</td>
</tr>
</tbody>
</table>
| • Cleared more quickly from the circulation  
  – 20 minutes | • Cleared more slowly from the circulation  
  – 120 minutes |
| • Cleared by natriuretic peptide receptors | • Cleared by various organs  
  – Skeletal tissue, liver, kidneys |

- Both equally cleared by kidneys
- Both equally useful in the diagnosis of acute decompensated heart failure
- Both may be elevated for reasons other than HF

### Natriuretic Peptides

- Good to assess in patients with dyspnea being evaluated for heart failure
- Should not be used as the sole tool to diagnose HF
- Must be used in concert with signs and symptoms
- Low values have strong negative predictive value
- Adds to prognostic information – marker of risk
- Predictor of increased risk
  – If levels do not fall after aggressive HF care, risk for death or hospitalization from HF is significant
**Ranges**

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<th>BNP</th>
<th>NT-proBNP</th>
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<tbody>
<tr>
<td>• &lt; 100 pg/mL - HF unlikely</td>
<td>• &lt; 300 pg/mL - HF unlikely</td>
</tr>
<tr>
<td>• &gt; 400 pg/mL - HF likely</td>
<td>• Age &lt; 50 years,</td>
</tr>
<tr>
<td>• 100-400 pg/mL - use clinical judgment</td>
<td>– &gt;450 pg/mL - HF likely</td>
</tr>
<tr>
<td></td>
<td>• Age 50-75 years</td>
</tr>
<tr>
<td></td>
<td>– &gt;900 pg/mL – HF likely</td>
</tr>
<tr>
<td></td>
<td>• Age &gt;75 years</td>
</tr>
<tr>
<td></td>
<td>– &gt;1800 pg/mL – HF likely</td>
</tr>
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- Levels increase as age increase
- Levels increase as renal function decreases
- Levels decrease as BMI increase

**HOWEVER:** Elevated levels are a marker of risk

**Causes of Elevated Naturetic Peptide Levels**

<table>
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<tr>
<th>Cardiac</th>
<th>Non-cardiac</th>
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<tbody>
<tr>
<td>• Heart failure, including RV syndromes</td>
<td>• Advancing age</td>
</tr>
<tr>
<td>• Acute coronary syndrome</td>
<td>• Anemia</td>
</tr>
<tr>
<td>• Heart muscle disease, including LV hypertrophy</td>
<td>• Renal failure</td>
</tr>
<tr>
<td>• Valvular heart disease</td>
<td>• Pulmonary: obstructive sleep apnea, severe pneumonia, pulmonary hypertension</td>
</tr>
<tr>
<td>• Pericardial disease</td>
<td>• Critical illness</td>
</tr>
<tr>
<td>• Atrial fibrillation</td>
<td>• Bacterial sepsis</td>
</tr>
<tr>
<td>• Myocarditis</td>
<td>• Severe burns</td>
</tr>
<tr>
<td>• Cardiac surgery</td>
<td>• Toxic-metabolic insults, including cancer, chemotherapy and environmental</td>
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Non-Invasive Imaging

**Echocardiogram**
- Serial studies important to
  - assess response to therapy
  - determine candidacy for devices
  - referrals for advanced therapies
- Routine studies without changes in therapy or condition not recommended

**Other Measures**
- MRI
  - LV volume and LVEF
  - Myocardial perfusion and viability
  - Myocardial fibrosis
  - Identify congenital disease
- CT Scan
  - Cardiac structure & function including coronaries
- Stress test
  - For those with known CAD and no angina to assess for ischemia

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**Echocardiogram**
- M-Mode echocardiography first available in 1953
- Early goal was to have a tool to quantitatively identify mitral stenosis and mitral regurgitation
- M-Mode (Motion Mode)
  - Clinically assess valve motion, chamber size, artic root size, wall thickness and ventricular function
- Doppler ultrasound
  - Determines velocity of blood within the heart which includes chamber pressure and gradients
- Color flow Doppler
  - Shows the direction of flow in respect to the transducer head.
    - Red: Towards the transducer
    - Blue: Away from the transducer
    - Yellow or green: areas of turbulent flow
Transthoracic Echo (TTE)

- Obtained in 4 views
  - Parasternal (long and short axis), apical, subcostal, suprasternal notch
- Valve function
  - Regurgitant volumes
  - Valve gradients
- Right and left atrial size
- Right and left ventricular function / size
  - Wall thickness
  - Systolic and diastolic abnormalities
  - Regional wall motion abnormalities
- Pulmonary artery pressures
- Pericardial abnormalities

(A) Parasternal long-axis view.
(B) Parasternal short-axis view.
(C) Apical four-chamber view.
(D) Apical two-chamber view (intercommissural plane).

Ao = ascending aorta;
LA = left atrium;
LV = left ventricle;
LAA = left atrial appendage;
RV = right ventricle;
TV = tricuspid valve.
Dilated Left Ventricle
Stages, Phenotypes and Treatment of HF

**STAGE A**
At high risk for HF but without structural heart disease or symptoms of HF

- e.g., Patients with:
  - HTN
  - Atherosclerotic disease
  - DM
  - Obesity
  - Metabolic syndrome or Patients with:
    - Using cardiotoxins
    - With family history of cardiomyopathy

**THERAPY**
- Goals
  - Heart healthy lifestyle
  - Prevent vascular, coronary disease
  - Prevent LV structural abnormalities

- Stages
  - ACEI or ARB in appropriate patients for vascular disease or DM
  - Statins as appropriate

**STAGE B**
Structural heart disease but without signs or symptoms of HF

- e.g., Patients with:
  - Previous MI
  - LV remodeling including LVH and low EF
  - Asymptomatic valvular disease

**THERAPY**
- Goals
  - Control symptoms
  - Improve HRQOL
  - Prevent hospitalization
  - Prevent mortality

- Drugs
  - ACEI or ARB as appropriate
  - Beta blockers as appropriate
  - In selected patients:
    - KI
    - Renal revascularization or vascular surgery as appropriate

**STAGE C**
Structural heart disease with prior or current symptoms of HF

- e.g., Patients with:
  - Known structural heart disease and HF signs and symptoms

**THERAPY**
- Goals
  - Control symptoms
  - Prevent hospitalization
  - Establish patient's end-of-life goals

- Drugs
  - ACEI or ARB as appropriate
  - Beta blockers as appropriate
  - In selected patients:
    - KI
    - Renal revascularization or vascular surgery as appropriate

**STAGE D**
Refactory HF

- e.g., Patients with:
  - Marked HF symptoms at rest
  - Recurrent hospitalizations despite GDMT

**THERAPY**
- Goals
  - Control symptoms
  - Improve HRQOL
  - Prevent hospitalization
  - Prevent mortality

- Drugs for routine use
  - ACEI or ARB
  - Beta blockers
  - Aldosterone antagonists

- Drugs for use in selected patients
  - Atrioventricular pacemakers
  - Inotropes
  - Tachyarrhythmia ablation
  - Digoxin

- In selected patients:
  - ICD
  - LV revascularization or vascular surgery as appropriate

**Refactory HF symptoms of HF at rest, despite GDMT**

**At Risk for Heart Failure**
- Patients with:
  - Marked HF symptoms at rest
  - Recurrent hospitalizations despite GDMT

**Heart Failure**
- Patients with:
  - Previous MI
  - LV remodeling including LVH and low EF
  - Asymptomatic valvular disease

- Patients with:
  - HTN
  - Atherosclerotic disease
  - DM
  - Obesity
  - Metabolic syndrome or Patients using cardiotoxins

- Patients with family history of cardiomyopathy

Development of symptoms of HF

Yancy et al. 2013 ACCF/AHA guideline for the management of heart failure. JACC, 62(16), e147-e239.
Stage A Treatment Strategies

Goal
• Heart healthy lifestyle
• Prevent vascular, coronary disease
• Prevent LV structural abnormalities

Treatments
• HTN screening / treatment
• ACE I or ARB in appropriate patients with vascular disease or DM
• Statins per recommendations for primary prevention
• Risk factor modification
  – Diet
  – Exercise
  – Tobacco cessation

Mortality Benefit

Stage B Treatment Strategies

Goals
• Structural heart disease but without signs or symptoms of HF

Treatments
• Medications to prevent ventricular remodeling
  – Beta blockers
  – ACE inhibitors / ARBs
  – Aldosterone antagonist
• ICD
• Revascularization
• Valvular surgery

Mortality Benefit
Stage C HFrEF Treatment Strategies

**Goals**
- Control symptoms
- Patient education
- Prevent hospitalization
- Prevent mortality

**Treatments**
- Diuretics for fluid retention
- Beta blockers
- ACE I or ARB
  - Entresto (Neprilysin inhibitor (sacubitril) with ARB (valsartan))
- Aldosterone blockers
- Hydralazine/isosorbide dinitrate
- Digoxin
- Corlanor (ivabradine)
- CRT
- ICD
- Revascularization or valvular surgery as appropriate
- Palliative care partnering with guideline directed medical therapy

Mortality, reduced hospitalization and symptom benefit

Medications to Avoid

**No Benefit**
- Nutritional supplement as treatment for HF
  - Coenzyme Q10
  - Carnitine
  - Taurine
  - Antioxidants
- Hormone therapies outside correcting deficiencies
  - Growth hormone
  - Thyroid hormone

**Harm**
- Antiarrhythmics
- Calcium channel blockers
  - Myocardial depressant
  - Except amlodipine – neither harm nor benefit in HF – OK for HTN / ischemia control
- NSAIDs
  - Cause sodium and water retention
- Thiazolidinediones
  - Associated with increase incidence of HF
**Diuretics**: Used in HFrEF, HFpEF, isolated right ventricular failure, and biventricular failure

- Decrease congestive symptoms
  - No mortality benefit
- First line: Loop diuretics
  - Thiazide diuretic may be added
- Potassium and magnesium monitoring
- Use with moderate NA restriction
- Fluid restriction criteria

- Monitor response to therapy
  - Adequate diuresis
    - BNP or NT-pro BNP goal
    - JVP assessment
    - Orthopnea
  - Over diuresis
    - Hypotension
    - Dizziness
    - Orthostatic BP
Loop Diuretics

- **Equivalents**
  - Furosemide 40 mg
  - Torsemide 20 mg
  - Bumetanide 1 mg

- **Dosing**
  - Adequate to relieve symptoms
  - Threshold medication

**Bumetanide (Bumex)**

**Furosemide (Lasix)**

**Torsemide (Demadex)**

---

**Differences in Loop Diuretics**

<table>
<thead>
<tr>
<th>Bumetanide</th>
<th>Furosemide</th>
<th>Torsemide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lack of randomized control data with comparison to furosemide</td>
<td>BID Dosing when GFR is low</td>
<td>2 randomized trials comparing Torsemide and Furosemide</td>
</tr>
<tr>
<td>Better pharmacokinetic profile (oral bioavailability) than furosemide but torsemide has evidence of more efficacy and more safety</td>
<td></td>
<td>Torsemide associated with reduction in HF and CV readmission in systolic HF with a trend towards reduction of all cause mortality.</td>
</tr>
<tr>
<td>Oral Bioavailability 80%</td>
<td>Oral Bioavailability 50%</td>
<td>Oral Bioavailability 80-100%</td>
</tr>
<tr>
<td>Max dose 10mg / day</td>
<td>Max dose 600mg / day</td>
<td>Max dose 200mg / day</td>
</tr>
<tr>
<td>Onset 30-60min</td>
<td>Onset 60min</td>
<td>Onset 60min</td>
</tr>
<tr>
<td>Peak 1-2 hours</td>
<td>Peak 1-2 hours</td>
<td>Peak 1-2 hours</td>
</tr>
<tr>
<td>Duration 4 hours</td>
<td>Duration 6-8 hours</td>
<td>Duration 6-8 hours</td>
</tr>
<tr>
<td>May repeat every 4-5 hours</td>
<td>May repeat every 6-8 hours</td>
<td>May repeat every 6-8 hours</td>
</tr>
</tbody>
</table>
Thiazide Diuretics

– Inhibit reabsorption of Na+ and Cl-
  • In the distal convoluted tubule
  • More sodium loss than loop diuretics
– Delayed onset but longer duration of action than loop diuretics
  • Give 30 minutes before a loop diuretic

– LOW CEILING DIURETICS
– Less potent diuretic than loop diuretics
– Diminished effectiveness in presence of renal failure

Thiazide Diuretics

<table>
<thead>
<tr>
<th>Drug</th>
<th>SIDE EFFECTS:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bendrofluazide (Naturetin)</td>
<td>Blood Chemistry changes::</td>
</tr>
<tr>
<td>Benthiazide (Aquatag, Exna)</td>
<td><strong>Hyponatremia</strong> (↓ Na⁺)</td>
</tr>
<tr>
<td>Chlorothiazide (Diuril)</td>
<td>Hypokalemia (↓ K⁺)</td>
</tr>
<tr>
<td>Chlorthalidone (Hygroton)</td>
<td>Hypomagnesemia (↓ Mg⁺)</td>
</tr>
<tr>
<td>Cyclothiazide (Anhydron)</td>
<td>Hyperglycemia (↑ blood sugar)</td>
</tr>
<tr>
<td>Hydrochlorothiazide (HCTZ)</td>
<td>Hyperuricemia (↑ uric acid)</td>
</tr>
<tr>
<td>Hydroflumethazide (Saluron, Diucardin)</td>
<td>Hypercalcemia (↑ Ca⁺⁺)</td>
</tr>
<tr>
<td>Indapamide (Lozol)</td>
<td>Decreased glomerular filtration in kidneys (↑ BUN, creatinine)</td>
</tr>
<tr>
<td>Metolazone (Zaroxolyyn)</td>
<td>↑ cholesterol</td>
</tr>
<tr>
<td>Polythiazide (Renese)</td>
<td>↑ triglycerides</td>
</tr>
<tr>
<td>Trichlormethiazide (Metahydrin, Naqua)</td>
<td>↓ HDL cholesterol</td>
</tr>
<tr>
<td></td>
<td>OTHER SIDE EFFECTS:</td>
</tr>
<tr>
<td></td>
<td>Impaired glucose tolerance</td>
</tr>
<tr>
<td></td>
<td>Gout</td>
</tr>
<tr>
<td></td>
<td>Impotence</td>
</tr>
<tr>
<td></td>
<td>Ventricular arrhythmias (↓ K⁺)</td>
</tr>
<tr>
<td></td>
<td>Nausea, dizziness, headache</td>
</tr>
</tbody>
</table>
Cardiac Resynchronization Therapy (CRT)

- Treatment modality for heart failure not just pacing
- Treatment modality in conjunction with drug therapy
- Goals:
  - Improve hemodynamics by restoring synchrony of ventricular contraction
  - Improve quality of life
  - Decrease mortality and morbidity
Normal Ventricular Depolarization

Mitral valve closed to prevent regurgitation

Septum moves leftward and functions as part of LV to eject blood

Papillary muscles contract with LV

Ventricular Depolarization with LBBB

A

Reduced ejection of blood into aorta

Abnormal electrical activation

B

Mitral regurgitation

Septum bulges into RV

Abnormal mechanical function
Indications for CRT Therapy

- Patient with cardiomyopathy on GDMT for >3 mo or on GDMT and >40 d after MI, or with implantation of pacing or defibrillation device for special indications
- LVEF <35%
- Evaluate general health status
- Comorbidities and/or frailty limit survival with good functional capacity to <1 y
- Acceptable noncardiac health
- Evaluate NYHA clinical status

NYHA class I
- LVEF ≤30%
- QRS ≥150 ms
- LBBB pattern
- Sinus rhythm
- Ischemic cardiomyopathy
- Non-LBBB pattern

NYHA class II
- LVEF ≤35%
- QRS ≥150 ms
- LBBB pattern
- Sinus rhythm
- LVEF ≤35%
- QRS ≤150 ms
- Non-LBBB pattern
- Sinus rhythm
- LVEF ≤35%
- QRS ≤150 ms
- Non-LBBB pattern
- Sinus rhythm
- LVEF ≤35%
- QRS ≤150 ms
- Non-LBBB pattern
- Sinus rhythm

NYHA class III & Ambulatory class IV
- LVEF ≤35%
- QRS ≥150 ms
- LBBB pattern
- Sinus rhythm
- LVEF ≤35%
- QRS ≤150 ms
- Non-LBBB pattern
- Sinus rhythm
- LVEF ≤35%
- QRS ≤150 ms
- Non-LBBB pattern
- Sinus rhythm
- LVEF ≤35%
- QRS ≤150 ms
- Non-LBBB pattern
- Sinus rhythm

Special CRT Indications
- Anticipated to require frequent ventricular pacing (>40%)
- Atrial fibrillation, if ventricular pacing is required and rate control will result in near 100% ventricular pacing with CRT

Colors correspond to the class of recommendations in the ACCF/AHA Table 1.

Benefit for NYHA class I and II patients has only been shown in CRT-D trials, and while patients may not experience immediate symptomatic benefit, late remodeling may be avoided along with long-term HF consequences. There are no trials that support CRT pacing (without ICD) in NYHA class I and II patients. Thus, it is anticipated these patients would receive CRT-D unless clinical reasons or personal wishes make CRT pacing more appropriate. In patients who are NYHA class III and ambulatory class IV, CRT-D may be chosen but clinical reasons and personal wishes may make CRT pacing appropriate to improve symptoms and quality of life when an ICD is not expected to produce meaningful benefit in survival.

CRT

- Goal: Force biventricular pacing
- Goal: Ventricular Pacing 90% of time or greater
- Causes of Loss of Bi V pacing:
  - Long AV Delays
  - Prolonged PVARP
  - ST with 1 degree AV Block
  - Lead dislodgement
Automatic Implantable Cardioverter Defibrillators

Functional status / 1 year

Turning off
ICD Device

- **Pulse Generator**
  - Single chamber, dual chamber, or biventricular pacing
  - Back up pacing
  - Antitachycardia pacing
  - Implanted subcutaneously – same as pacemaker

- **Defibrillator lead**
  - Detects arrhythmias
  - Delivers therapy
  - Defibrillator lead capable of pacing and defibrillating
  - Placed in right ventricle

ICD Functions

- **ATP-Anti tachycardia Pacing**
  - **Tiered Antiarrhythmic Therapies**

[Burst and Ramp diagrams]
ICD Functions

— Cardioversion Shock
  • Delivers shocks from 0.1 to 30 joules synchronized on the R wave

— Defibrillating Shock
  • Delivers high energy (20-34 joules) unsynchronized shock for VF
Internal Monitoring with Devices

- Heart rate variability / Night heart rate
- Patient activity
- Impedance / Volume assessment
- Atria fib burden
- Ventricular arrhythmias

Routine re-evaluation of pacing burden is important in the treatment of HF.

Data from ICD
CardioMEMs
CHAMPOIN Trial (2011)

- **Clinical Trial Indications:** Patients 18 years older with NYHA Class III heart failure for at least 3 months, irrespective of left ventricular ejection fraction and a hospitalization for heart failure within the past 3 months. *Current manufacturer recommendations (post FDA approval):* NYHA Class III HF hospitalized in the past year for heart failure.

- Randomized to CardioMEMS or standard treatment for heart failure. During the entire follow-up (mean 15 months [SD 7]), the treatment group had a 37% reduction in heart-failure-related hospitalization compared with the control group (158 vs 254, HR 0.63, 95% CI 0.52–0.77; p<0.0001).

- **Conclusion:** The addition of information regarding pulmonary artery pressure to clinical signs and symptoms improves heart failure management and reduces heart failure readmissions. Quality of life data is also available.

- **Current Practice:** Current practice is representative of the control group in this trial which includes monitoring signs and symptoms (reporting of symptoms and daily weights) as in the control group in the CHAMPION Trial.

- **Safety:** 98.6% free of device complications.
Stage C HFpEF Treatment Strategies

**Goals**
- Control symptoms
- Improve quality of life
- Prevent hospitalization
- Prevent mortality

**Treatments**
- Diuresis to relieve symptoms of congestion
- Identify comorbidities
- Follow guideline driven indication for comorbidities
  - HTN, AF, CAD, DM, sleep apnea, anemia
- Revascularization or valvular surgery as appropriate

Mortality, reduced hospitalization and symptom benefit

HFpEF

- No evidence based medical therapy
- ARBs, aldosterone antagonists, and sildenafil have all been tested
- ARBs may reduce hospitalizations but not mortality
- Aldosterone antagonists may reduce hospitalization and mortality
- TOPCAT Study
- Focus on co-morbid conditions:
  - HTN
    - Blood pressure control is imperative to prevent flash pulmonary edema
  - Sleep apnea
  - Atrial fibrillation
    - Rhythm control may be required to assure adequate preload
  - Anemia
  - CAD
  - Diabetes
ACUTE DECOMPENSATED HEART FAILURE

Acute Decompensated Heart Failure (ADHF)

- Sudden or gradual onset of the signs and symptoms of heart failure requiring unplanned office visits, emergency room visits, or hospitalizations
- Associated with pulmonary and systemic congestion due to increased left and right heart filling pressures

Acute Decompensated HF represents a sentinel prognostic event. Readmission rate predicted to be 50% at 6 months. 1-year mortality of approximately 30% of ADHF admissions
Common Precipitating Factors of ADHF

- Non adherence with
  - Medications
  - Dietary sodium intake
  - Fluid intake
- Excessive alcohol or drug use
- ACS
- Arrhythmias
- Persistent hypertension
- Valvular heart disease

- Recent addition of negative inotrope
- Nonsteroidal anti-inflammatory drugs
- Worsening renal function
- Endocrine abnormality
- Concurrent infection
- New anemia
- Pulmonary embolism

Hospitalization Recommended

Evidence of severe ADHF, including:
- Hypotension
- Worsening renal function
- Altered mentation

Dyspnea at rest
- Typically reflected by resting tachypnea
- Less commonly reflected by oxygen saturation <90%

Hemodynamically significant arrhythmia - including new onset of rapid atrial fibrillation

Acute coronary syndromes
Treatment Goals

- Improve symptoms, especially congestion and low-output symptoms
- Optimize volume status
- Identify etiology
- Identify and address precipitating factors
- Optimize chronic oral therapy
- Minimize side effects
- Identify patients who might benefit from revascularization
- Identify patients who might benefit from device therapy
- Identify risk of thromboembolism and need for anticoagulant therapy
- Educate patients concerning medications and self management of HF
- Consider and, where possible, initiate a disease management program

3 Clinical Presentations

Patient 1: Volume overload (Backwards Failure)

Patient 2: Profound depression of cardiac output—hypoperfusion (Forwards Failure)

Patient 3: Signs and symptoms of both fluid overload and hypoperfusion (Cardiogenic Shock)
Evaluation Guides Treatment Decisions

• Determine
  – Volume Status
  – Perfusion Status
  – Role of / or presence of precipitating factors and/or comorbidities
  – Ejection fraction
    • HFPeF
    • HFReF

Hypoperfusion vs. Volume Overload

• **Hypoperfusion**
  – Narrow pulse pressure
  – Resting tachycardia
  – Cool Skin
  – Altered mentation
  – Decreased urine output
  – Increased BUN/Creatinine
  – Cheyne Stokes Respiration

• **Intravascular Volume Overload**
  – Elevated jugular venous pressure
  – Hepatocellular reflex
  – Orthopnea
  – Dyspnea
  – Crackles
  – Weight gain
  – Peripheral edema
Backwards Failure
Pulmonary Congestion
Wet Patient

Forwards Failure
Hypoperfusion
Dry / Hypoperfused Patient

<table>
<thead>
<tr>
<th>Warm and Dry</th>
<th>Cold and Dry</th>
</tr>
</thead>
</table>
| Normal Perfusion
No Congestion | Low Perfusion
No Congestion |

<table>
<thead>
<tr>
<th>Warm and Wet</th>
<th>Cold and Wet</th>
</tr>
</thead>
</table>
| Normal Perfusion
Congestion | Low Perfusion
Congestion |
Pulmonary Edema

- Extra vascular accumulation of fluid in the lungs (cardiac or non-cardiac)
  - Results in impaired diffusion of oxygen due to increase in interstitial space
  - Results in decreased V/Q ratio due to poorly ventilated fluid filled alveoli
  - Fluid in alveoli also impacts compliance of lungs and therefore ventilation

- Capillary endothelium more permeable to water and solute than alveolar endothelium

- Edema accumulates in the interstitium before the alveoli

Pulmonary Edema

- Fluid in pulmonary interstitium is removed by lymphatic drainage of the lung

- Volume of lymph flow from the lung can increase ten fold in pathological conditions

- Only when this large safety factor is taxed does pulmonary edema occur
**Pulmonary Edema**

- Increase in pulmonary capillary hydrostatic pressure (includes cardiac)
  - Left sided heart failure
  - Excessive fluid administration
  - Occlusion of pulmonary vein

- Loss of integrity of alveolar capillary membrane (non cardiac)
  - Infection
  - Inhaled toxins
  - Oxygen toxicity

Other: Blockage of lymphatic system

- Cardiac pulmonary edema is treated as acute decompensated heart failure

- Non-cardiac pulmonary edema is treated like ARDS

**Treatment for Acute Decompensated HF**

**Congestion with Adequate Perfusion**
- Subset II
- Reduce Preload

**Hypoperfusion with No Congestion**
- Subset III
- Increase contractility
  - Assure adequate preload

**Hypoperfusion with Congestion**
- Subset IV
- Reduce Afterload
Acute Decompensated Heart Failure

**Reduce Preload**
- Diuretics
- Venous vasodilators
  - Low dose NTG
  - Nesiritide
- Ultrafiltration

**Reduce Afterload**
- Arterial vasodilators
  - High dose NTG
  - Nitroprusside
  - Nesiritide
- Intra aortic balloon pump

**Increase Contractility**
- Positive Inotropes
  - Dobutamine
  - Milrinone
  - Dopamine

**Assure adequate volume**

Ultrafiltration

**UNLOAD Trial**
- Veno-venus ultrafiltration (UF) vs standard IV diuretic therapy for hypervolemic HF
- 200 patients randomized
- UF with statistical significance for: greater weight loss (48 hours), greater fluid loss (48 hours), less 90-day resource utilization for HF.
- No statistically significant difference in dyspnea scores or creatinine levels (safety endpoint)

**CARESS-HF Trial**
- Treatment of ADHF, worsening renal function, persistent congestion with stepped pharmacologic approach vs ultrafiltration
- 188 patients randomized
- UF: inferior to pharmacologic therapy and associated with adverse events.
Sodium Restriction

• Sodium restriction is reasonable for patients with symptomatic HF to reduce congestive symptoms (IIc)

• Clinicians should consider some degree of sodium restriction (< 3 grams /day) for patients with Stage C and D HF

Fluid Restriction

• Dietary Sodium Restriction
  – Water follows sodium

• If hyponatremic
  – Serum sodium < 130 mEq/L
    • 2 liters per day
  – Serum Sodium < 125 mEq/L
    • Stricter fluid restriction may be considered

• If persistent fluid overload
  – Assure sodium restriction in conjunction with fluid restriction
Foley Catheter

- Foley Catheter
  - Not recommended routinely in heart failure
  - If need to closely monitor hourly urine output
  - Possible outlet obstruction
    - High risk patients include those with BPH and or right sided volume overload

Oxygen

- Oxygen therapy is recommended if the patient exhibits hypoxemia
- If not hypoxemic no need for oxygen therapy

BiPap/CPAP

- Use of non-invasive positive pressure ventilation may be considered for severely dyspneic patients with clinical evidence of pulmonary edema.
Invasive Monitoring

- Routine use not recommended
- When to consider:
  - Refractory to initial therapy
  - Volume status and cardiac filling pressures are unclear
  - Pulmonary and systemic pressures unclear
  - Clinically significant hypotension (SBP < 80 mm Hg)
  - Worsening renal function

Stage D Treatment Strategies

**Goals**
- Control symptoms
- Improve quality of life
- Prevent hospitalization
- Prevent mortality

**Options**
- Advanced care measures
- Heart transplant
- Temporary or permanent mechanical circulatory support
- Chronic inotropes
- Experimental surgery or drugs
- Palliative care and hospice
- ICD deactivation

Quality of Life
Indicators for Advanced Heart Failure

- Severe symptoms of HF with dyspnea and/or fatigue at rest or with minimal exertion (NYHA class IIIb or IV) on optimal medical therapy
- Repeated episodes of fluid retention (pulmonary and/or systemic congestion, peripheral edema) and/or reduced cardiac output at rest (peripheral hypoperfusion) on optimal medical therapy
- Objective evidence of severe cardiac dysfunction shown by at least 1 of the following on optimal medical therapy:
  - LVEF <30%
  - Mean PCWP >16 mmHg and/or RAP >12 mmHg by PA catheterization
  - High BNP or NT-proBNP plasma levels in the absence of non-cardiac causes
Indicators for Advanced Heart Failure

• Severe impairment of functional capacity while on optimal medical therapy shown by 1 of the following:
  – Inability to exercise
  – 6-Minute walk distance < 300 m
  – Peak Vo2 <12 to 14 mL/kg/min
• Repeated (≥2) hospitalizations or ED visits for HF in the past year or > 1 hospitalization for heart failure
• Progressive deterioration in renal function
• Progressive decline in serum sodium, usually to <133 mEq/L
• Weight loss without other cause
• Intolerance to ACE inhibitors due to hypotension and/or worsening renal function
• Intolerance to beta blockers due to worsening HF or hypotension
• Frequent systolic blood pressure <90 mm Hg
• High diuretic requirements to maintain volume status (i.e. furosemide equivalent dose >160 mg/d and/or use of supplemental metolazone therapy)
• Frequent ICD shocks

Prognostic Models

• Heart Failure Survival Score
  – All cause mortality
• Seattle Heart Failure Model
  – All cause mortality, urgent transplantation or LVAD implant
• EVEREST Risk Model
  – Combined endpoint of mortality or persistently poor quality of life over the 6 months after discharge
• EFFECT
  – 30-day and 1-year mortality
• ADHERE
  – In-hospital mortality
• ESCAPE Discharge Score
  – 6 month mortality
Risk Factors for Mortality

> 2 Referral for Advanced Treatment

- >2 Prompt Referral for Advanced Rx
- Hospitalization for HF on oral HF therapy
- Inability to take ACEI/ARB/BB
- BUN > 45, Creat > 2.5, CrCl < 45 cc/min
- BNP > 4 x’s upper limit of normal
- Na+ < 136
- Malnutrition/Cachexia
- VO2 < 55% predicted
- LVEDD > 7.0 cm

Mechanical Circulatory Support (MCS) in ADHF

- Bridge to transplant (BBT) for those who are transplant eligible
- Destination therapy (DT) for those who are not transplant eligible.
- Bridge to Decision (BTD)
- Careful consideration for all therapies
  - Some patients may be too ill with multisystem issues to benefit from MCS
  - Some decisions are best made in the hands of the most experienced centers
IABP: Counterpulsation Therapy

- Intra Aortic Balloon (IAB) is inflated during diastole and deflated during systole
- The IAB is a volume displacement device

IAB Placement
- Descending thoracic aorta
- 1 to 2 cm below the subclavian artery origin
- Above renal and mesenteric arteries
Hemodynamic Impact of IAB Pumping

- Increased diastolic aortic pressure
- Increased coronary blood flow
- Increase cardiac output / ejection fraction / forward flow
- Increased cerebral and renal blood flow
- Increased systemic perfusion
- Increased coronary and systemic oxygen supply
- Increased hemodynamic pulse rate

Hemodynamic Impact of IAB Pumping

- Decreased systolic aortic pressure
- Decreased afterload
  - Decreased MVO2
- Decreased LV wall tension
- Decreased preload
  - Decreased pulmonary congestion
- Decreased HR

Decrease LV Afterload
Increase coronary perfusion
**Indications / Contraindications**

**Indications**
- Cardiogenic shock.
- Recurrent ischemia / extending myocardial infarction.
- Unstable angina.
- Intractable ventricular dysrhythmias.
- Support for high risk intervention.
- Bridging device in acute cardiac failure.
- Mechanical defects such as acute mitral regurgitation.
- Post-operative myocardial dysfunction.

**Absolute Contraindications**
- Aortic valve insufficiency / regurgitation
- Dissecting aortic aneurysm
- Aortic stents

**Relative Contraindications**
- Calcific aortic iliac disease
- Peripheral arterial disease
- Abdominal aortic aneurysm
- Thrombocytopenia

**Goals of Inflation**

- **Increase coronary perfusion pressure**
- Increase systemic perfusion pressure and peripheral oxygen supply
- Increase baroreceptor response and decrease SNS stimulation
  - Decrease SVR
  - Decrease HR
Goals of Deflation

- **Decrease afterload**
  - Decrease MVO2
  - Decrease assisted peak systolic pressure (APSP)
  - Increase cardiac output and ejection fraction (increase forward flow)

**Inflation Timing:**
The IAB is inflated immediately upon closure of the aortic valve.

**Deflation Timing:**
The balloon must be deflated before the full onset of systole.
Why Inflation Works:
Inflation of the IAB during diastole increases aortic volume and pressure.

Why Deflation Works:
IAB deflation just prior to systole creates a potential space in the aorta. This reduces aortic volume and pressure.

Discussion of Key Nursing Considerations

- Pressure assessment for optimization of therapy
- Balloon mobility
- Left radial pulse assessment
- Urine output
- Distal pulse assessment
- Groin care
- Platelets
- Other complications: aortic dissection
Impella

• Pulls blood from the left ventricle and expels blood into the ascending aorta.
• Inserted via femoral artery, into the ascending aorta, across the valve and into the left ventricle.
• Produces CO of 2.5 – 5.0 L/Min (2 different devices)

Impella

• Percutaneously inserted catheter-based cardiac assist device
• Inserted via the femoral artery
• Positioned across the aortic valve with the tip in the LV and the outlet area in the aorta.
• Small motor on the catheter pulls blood from the left ventricle through an inlet area near the tip of the catheter and ejects it into the aorta – non pulsatile
• Device unloads the left ventricle, reduces myocardial workload and oxygen consumption, and increases cardiac output, coronary and peripheral perfusion.
• Complex patient
• Monitor for limb ischemia – at least hourly
• Anticoagulation required – maintain APTT for 45-55 seconds
• Monitor for hemolysis
  – Decreased HGB/HCT, haptoglobin
  – Hemoglobinuria may develop and AKI
  – Impella Console
    • P8 – performance level 8 is standard with 50,000 revolutions per minute to provide a flow rate of 1.9-2.5 L/min of cardiac output

• Physiologic Impact
  – Increase forward flow
  – Unloads LV
  – Augments cardiac output
  – Increases mean arterial pressure

• Contraindications
  – Mechanical aortic valve
  – Moderate to severe aortic disease
  – Left ventricular thrombus
  – Moderate to severe peripheral arterial disease
ECMO

• Venous – arterial
  – allows for gas exchange and provides hemodynamic support by bypassing the lungs and heart.
  – can also work alongside native circulation allowing for a portion of the blood to flow naturally through the heart and lungs.
  – Indicated in refractory cardiogenic shock or as salvage strategy during cardiac arrest after 10 minutes of unsuccessful advanced cardiac life support.

• Venous – venous
  – facilitates gas exchange but does not provide for hemodynamic support because the blood is returned to the right side of the heart before it enters pulmonary circulation.
  – Pump is necessary to pump venous blood through the membrane oxygenator.
  – Used as an alternative strategy in adults with ARDS to rest the lungs and avoid insult of mechanical ventilation.

• Arterial – venous
  – a pumpless circuit
  – blood flows from the femoral artery through a membrane and returns to the femoral vein.
  – Hemodynamic support comes from the patient’s own cardiac output.
  – Absence of a pump makes this mode easier for transport. However, cardiac function must be well preserved.
ECMO

TandemHeart
### Absolute and Relative Contraindications for Durable MCS

<table>
<thead>
<tr>
<th>Absolute Contraindications</th>
<th>Relative Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Irreversible hepatic disease</td>
<td>* Hypertrophic, infiltrative, or restrictive cardiomyopathy</td>
</tr>
<tr>
<td>Irreversible renal disease</td>
<td>Uncorrectable moderate or greater aortic insufficiency</td>
</tr>
<tr>
<td>Irreversible neurological disease</td>
<td>Age ≥80 y (for destination therapy)</td>
</tr>
<tr>
<td>Major coagulopathy</td>
<td>Obesity or malnutrition</td>
</tr>
<tr>
<td>Right sided heart failure (unless candidate for biventricular support)</td>
<td>MS disease that impairs rehabilitation</td>
</tr>
<tr>
<td>Medical non-adherence</td>
<td>Active systemic infection</td>
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<tr>
<td>Severe psychosocial limitations</td>
<td>Prolonged intubation</td>
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<tr>
<td></td>
<td>Untreated malignancy</td>
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<tr>
<td></td>
<td>Severe PVD</td>
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<tr>
<td></td>
<td>Active substance abuse</td>
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<td></td>
<td>Impaired cognitive function</td>
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<tr>
<td></td>
<td>Unmanaged psychiatric disorder</td>
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<tr>
<td></td>
<td>Lack of social support</td>
</tr>
</tbody>
</table>

* May be a relative contraindication

Source: Peura et al., 2012; Slaughter et al., 2010.
Long Term Therapy

**Bridge to Transplant**
- Extracorporeal MCS
  - Thoratec pVAD II
- Implantable MCS
  - Heart Mate II
  - HeartWare HVAD
- Total Artificial Heart
  - CardioWest
  - Abiomed: Abiocor II

**Destination Therapy**
- Heart Mate II
- HeartWare HVAD
- Investigational Devices
Heart Mate II

HeartWare HVAD
Components of effective shared decision making include:

- Establishing trust
- Identifying patient values, preferences, and goals for care early in the course of treatment
- Using the framework “Ask-Tell-Ask” to determine both what patients know and what they want to know
- Understanding the reasons why there are conflicts regarding decisions of care
- Using numeric data in a clear and understandable way as a decision aid
- Respecting that patient’s may change their goals as the disease progresses

Allen, 2012.
Palliative Care

• An approach that improves the quality of life of patients and their families facing the problem associated with life-threatening illness, through the prevention and relief of suffering by means of early identification and impeccable assessment and treatment of pain and other problems, physical, psychosocial and spiritual. (World Health Organization)

Palliative Care

• Provides relief from pain and other distressing symptoms;
• Affirms life and regards dying as a normal process;
• Intends neither to hasten or postpone death;
• Integrates the psychological and spiritual aspects of patient care;
• Offers a support system to help patients live as actively as possible until death;
• Offers a support system to help the family cope during the patients illness and in their own bereavement;
• Uses a team approach to address the needs of patients and their families, including bereavement counseling, if indicated;
• Will enhance quality of life, and may also positively influence the course of illness;
• Applicable early in the course of illness, in conjunction with other therapies that are intended to prolong life, such as chemotherapy or radiation therapy, and includes those investigations needed to better understand and manage distressing clinical complications.
• Can help decide when end of life care is appropriate
**Hospice Care**

- Care provided to patients with a life expectancy of six months or less.
- No longer seeking a cure
- Aims to make remaining time as comfortable and as meaningful as possible.
- Relief of pain.
- Emotional support (for patient and family) and help with everyday tasks.
- Goal: Ensure every moment counts, in the last six months of life.
- Medicare / insurance benefit with covered services

**Criteria for Discharge**

- Exacerbating factors addressed
- Near optimal volume status achieved
- Transition from intravenous to oral diuretic successfully completed
- Patient and family education completed, including clear discharge instruction
- LVEF documented
- Smoking cessation counseling initiated
- Near optimal pharmacologic therapy achieved, including ACE inhibitor and beta-blocker (for patients with reduced LVEF), or intolerance documented
- Follow-up clinic visit scheduled, usually for 7 to 10 d
Criteria for Discharge

- Advanced HF Patient or recurrent admission
  - Oral medication regimen stable for 24 h
  - No intravenous vasodilator or inotropic agent for 24 hours
  - Ambulation before discharge to assess functional capacity after therapy
  - Plans for post discharge management (scale present in home, visiting nurse or telephone follow up generally no longer than 3 d after discharge)
  - Referral for disease management, if available

Education and Counseling

- Should be delivered by providers using a team approach in which nurses with expertise in HF management provide the majority of education and counseling
- 1-hour of nurse education using standardized instructions resulted in improved clinical outcomes, increased self-care adherence, and reduced cost of care
- Use “teach back” method

Identify primary care giver / support person and include in ALL education.
Self-Care Maintenance and Self-Care Management

• Self-care maintenance
  – Following the rules and instructions related to the disease process
  – “What to do”

• Self-care management
  – Decision-making process and critical thinking to make decisions in response to changes in the client’s current health status
  – “How to do”

Barriers to Self-Care Management

• Higher acuity
• Multiple needs
  – Co-morbidities
• Shorter LOS
• Noncompliance
• Transportation issues
• Financial concerns
• Depression / anxiety

• Lack of knowledge
• Literacy
• Multiple medications
• Fear of medication side effects
• Living alone (lack of social support)
• Memory problems

Don’t assume a patient has received education or understands because of a “frequent flier” status.
1. Diet and nutrition
2. Discharge medications
3. Activity level
4. Follow up appointments
5. Daily weight
6. Response to symptoms: Who to call

Hospital education should be limited to “essential” education

Readmission Data

- Nearly 2 million Medicare patients readmitted within 30 day of discharge
- Cost of those readmissions = $17.5 BILLION
- National average for readmission 19%
- Readmissions a symptom of an overly expensive and uncoordinated healthcare system
  - Limited connection from inpatient to outpatient
- HF readmissions rate 20-25% at 30 days
  - > 50% at 6 months
  - 35% of 30 day readmissions due to HF
What We Know

- Readmissions are prevalent and costly
- Adverse events associated with hospital discharge are common
  - And about ¼ of them are readmissions
- Patients are not taking ideal medication regimens
- Limited follow up on adjusted medications, lab or other tests and workups is common
- Real room for improving hospital to post-hospital receiver communication
- Creating the perfect in-house discharge process probably won’t make enough difference
- Severity of illness is probably related to readmission risk
  - But the CMS measures do not adjust for it
- Ideal risk identification strategies are unavailable
- Clinicians often have a different perspective on what led to the readmission than patients do

Multidimensional Nursing Roles

- Coordinate care with interdisciplinary team members who can target coexisting medical, social, and financial issues
- Facilitate behavioral strategies that ease patient and caregiver burdens related to adherence to the treatment plan
- Educate on advance directive planning and community services that meet learning needs
- Promote continuity of care between home, HF clinic, or palliative care services
  - Foster collaborative relationships
  - Coach collaborators to use evidence-based therapies
  - Ensure open communication
  - Position patients and caregivers to proactively assess and manage signs and symptoms of worsening condition
- Assess goal progression
- Recognize and target unresolved HF issues
• Systematic review 47 trials

• At 30 days a high intensity home-visiting program reduced all cause readmissions

• At 3-6 months home-visiting programs and multidisciplinary heart failure clinic (MDS-HF) interventions reduced all cause readmissions

• Structured telephone support (STS) reduced HF specific readmissions but not all cause readmissions

• Mortality benefit with MDS-HF clinic, home-visiting programs, and STS

• Based on current evidence, telemonitoring interventions (non structured) and primarily educational interventions are not efficacious for reducing readmissions or mortality

Feltner et al., 2014

CARDIAC MONITORING AND ARRHYTHMIAS
### Table

<table>
<thead>
<tr>
<th>Lead</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>III (or aVF) and V₃ (or V₂)</td>
<td>In patients with suspected ACS who have not yet demonstrated ECG changes or who do not yet have defined coronary anatomy.</td>
</tr>
<tr>
<td>III (or aVF)</td>
<td>In patients with known inferior wall or right coronary artery involvement</td>
</tr>
<tr>
<td>V₃ (or V₂)</td>
<td>In patients with known anterior wall or left anterior descending artery involvement</td>
</tr>
<tr>
<td>V₆</td>
<td>In patients if there is concern for left circumflex artery involvement</td>
</tr>
<tr>
<td>V₅ or V₄</td>
<td>In patients at risk for supply and demand ischemia (high risk patient undergoing non-cardiac surgery and patient with critical medical illness)</td>
</tr>
<tr>
<td>V₄R</td>
<td>In patients presenting with acute inferior wall ST segment elevation MI to assess for co-existing right ventricular myocardial infarction because right ventricular infarct has special treatment considerations.</td>
</tr>
<tr>
<td>II</td>
<td>Not recommended for ST segment monitoring</td>
</tr>
</tbody>
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**AACN Practice Alert:**

**Accurate Dysrhythmia Monitoring in Adults**

*Critical Care Nurse Vol 36, No. 6, DECEMBER 2016*

August 2004 Original Author:
- Barbara Drew, RN, MS, PhD, FAAN, FAHA, CNS-BC

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Bedside Cardiac Monitoring

- ✓ V1 and V6 are gold standard monitoring leads for ectopy versus aberrancy
  - ✓ Bundle branch block patterns and ventricle ectopy can be differentiated by using the morphology of these leads.

DON’T rely on Lead II !!

AACN Practice Alert:

Ensuring Accurate ST-Segment Monitoring

Critical Care Nurse Vol 36, No. 6, DECEMBER 2016

August 2004 Original Authors:
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The Lewis Lead

When P waves are not clearly seen in a rhythm strip (see lead 3 above), the Lewis lead can be very helpful in assessing for the presence of atrial activity.

As seen in the Lewis lead below, this patient is clearly in an atrial flutter. The atrial flutter is not as obvious in the lead III rhythm strip.
Heart Blocks: AV Blocks

- Classification
  - 1st Degree
  - 2nd Degree
    - Type I (Wenckebach)
    - Type II
  - High Grade
  - Third Degree

When the term heart block is used, clinicians are referring to block within, around, or below the AV node.
THE DEFINITION OF 2ND DEGREE AV BLOCK IS..................

**One** P Wave at a time fails to conduct to ventricle.

After you determine it is second degree heart block:

THEN YOU CAN ASK IF IT IS TYPE 1 OR TYPE 2.
What’s the difference between 2nd degree Type 1 and Type 2?

Not only is the rhythm strip criteria different. It also has to do with the most common physiological location of the block:

**Type 1 block** is usually within the AV node.  
**Type 2 block** is below the AV node and usually involves both bundle branches.

---

2 Types of 2nd Degree Heart Block

**Type I (Wenckebach)**

- Most often conduction fails within the AV node
  - Therefore: There will be a problem with the PR interval.
  - The QRS remains narrow (unless there is an existing BBB) because the block is in the AV node and does not involve the His Perkinje system.

**Type II**

- Conduction fails below the AV node and usually involves both bundles
  - There will be no problem with the PR interval (AV node not involved)
  - QRS complex is usually wide (His Perkinje system involved)
    - Can be narrow if the block only involves the Bundle of His
Wenckebach (2nd Degree AV Block Type I)

- Sinus node fires regularly
- Disease is in AV node
- Group beating is noted
- First P-R of group of often longer than normal with progressive lengthening of the P-R until a beat is not conducted
  - PR problem because of physiological location of the block
- In absence of BBB QRS is normal
  - Normal QRS width because of physiological location of the block
- Conduction ratios may be 2:1, 3:2, 4:3 etc.
- May develop 2:1 conduction if sinus rate increases
  - Verify the block is still type I
  - P-R longer than normal
  - Absence of prolonged QRS
- Treatment: Often none
  - Acutely with symptoms: Atropine or TTVP
  - Atropine will work because of physiological location
  - Atropine blocks the parasympathetic nervous system. Atropine will only work where there are parasympathetic nervous system fibers. These fibers are present in the sinus node, AV node, and throughout the atria. They are only sparse in ventricles.

Note:
- Progressive lengthening of the PR interval (problem in the AV node)
- Normal QRS width (no problem in the His Perkinge System)
2nd Degree AV Block Type II

- No progressive lengthening of P-R interval
  - Physiological problem does not involve AV node
  - P-R interval is fixed with normally conducted beats

- Disease within the Bundle of His or below Bundle of His
  - QRS: wide – when below the Bundle of His

- Treatment: if symptomatic - pacemaker

Both of these examples:
1. One P wave at a time fails to conduct
2. There is a fixed PR interval
3. There is a wider than normal QRS
Heart Blocks - High Grade AV Block

- Two or more consecutive atrial impulses are blocked.
- P waves: Regular, but 2 or > in a row fail to conduct to the ventricles.
- This is not 2\textsuperscript{nd} degree heart block by definition because more than one P wave in a row fails to conduct.
- In the strip below every third P wave is conducted. This differentiates High Grade AV Block from Complete Heart block which has no conducted P waves.

Third Degree AV Block – Complete

- No atrial impulses are conducted to the ventricles
- One form of AV dissociation
- Ventricular Rate: Maintained by ventricular escape (wide QRS) or by pacemaker coming from His bundle (narrow QRS – less common)
- Symptomatic if develops acutely
  - May be well tolerated if develops overtime
  - Treatment: Permanent Pacemaker
Remember – Patients in atrial fibrillation can develop complete heart block. The R to R interval becomes regular because the escape pacemaker is now in control.

Narrow QRS Tachycardias:
AV Nodal Passive

Rate Control:
Ca++ blockers
(verapamil, diltiazem)
Beta blockers

Adenosine is not appropriate

Atrial Tachycardia
- P waves visible in front of QRS or may be hidden in T wave
- Atrial rhythm is regular: 150-250
- Ventricular rate usually same (unless AV block present)

Atrial Flutter
- Atrial rate 250-350 with flutter waves
- Ventricular rhythm regular or irregular
- Ventricular rate depends on amount of AV block: 150 or lower

Atrial Fibrillation
- Atrial rate very fast with irregular fib waves
- Ventricular rhythm always irregular
- Ventricular rate depends on amount of AV block: can be up to 200 or so
Narrow QRS Tachycardias:
AV Nodal Active

- Vagal maneuver
- Adenosine – drug of choice
  - Beta blockers
  - Ca** blockers

**AV Nodal Reentry Tachycardia**
- Regular at rates 140-200
- P waves usually not seen (sometimes peek out at end of QRS)
- Term “SVT” is appropriate
- AV node is part of reentry circuit that maintains the tachycardia

**AV Reentry Tachycardia**
- Regular at rates 140-280
- P waves not easily seen – appear in ST segment when seen
- Term “SVT” is appropriate
- AV node is part of reentry circuit that maintains the tachycardia

---

AV Nodal Re-entrant Tachycardia (AVNRT) – Typical

- A PAC initiates atrial depolarizations which travel via the slow AV nodal pathway
  - the fast pathway is refractory (blocked) due to previous SA node depolarization
- At the AV node exit depolarizations travel antegrade to depolarize the ventricles and retrograde up the fast pathway to depolarize the atria.
- This cycle repeats.

---

Slow – Fast
Typical AV Nodal Re-entrant Tachycardia
Typical AV Nodal Re-entrant Tachycardia

Typical AV Nodal Re-entrant Tachycardia
Koch’s Triangle: Location of fast pathway; an anatomical area located in the endocardium of the right atrium which has the coronary sinus at one angle, and the tendon of Todaro and the septal leaflet of the tricuspid valve as two sides. The slow pathway is located inferior and posterior to the AV node tissue.
AV Nodal Reentrant Tachycardia (Typical)

- Most common supraventricular tachycardia
- 60% occur in women
- Palpitations most common symptom
  - Also polyuria from higher atrial pressures (ANP)
- Least likely to be life threatening
  - Greatest fall in blood pressure during first 10 to 30 seconds
  - HR alone not responsible for hemodynamic compromise

- **Narrow QRS**
  - Unless pre-existing BBB or development rate dependent
  - BBB * usually RBBB

- **No visible P waves**
  - Simultaneous depolarization
  - Or, P waves are so close to QRS they look like part of it (pseudo R waves in V1 and pseudo R waves in inferior leads)

---

Pseudo R Wave: Short R-P Tachycardia

![ECG Image]

- V1
Pseudo S Wave: Short R-P Tachycardia

SVT

Pseudo S

Pseudo S

Sinus Rhythm

Pseudo S

Pseudo R'
AVNRT
Treatment for AVNRT

- Vagal (teach patient)
  - Valsalva
  - Carotid massage
  - Facial cold water immersion
- Adenosine or non-dihydropyridine calcium channel blockers (stable)
  - Adenosine preferred
- DC Cardioversion (unstable)
- Beta blockers or calcium channel blockers if ablation is declined

Ablation: AVNRT is most common reason for cardiac ablation.
Adenosine (Adenocard)

- Slows conduction through the AV Node
- Vasodilator
- Interrupts reentry pathways through the AV node and restores sinus rhythm
- Uses: Paroxysmal SVT, AVNRT, Drug stress testing
- Side Effects: Headache, arrhythmias (blocks), SOB, chest pressure

**Nursing Considerations:**
- Use cautiously in patients with asthma – could cause bronchospasm
- Onset IV: Immediate
- Peak: 10 sec
- Duration 20-30 seconds
- Dosing for conversion of arrhythmia:
  - 6mg IV rapid push
  - If no change within 1-2 minutes repeat with 12mg rapid push
  - Not indicated in WPW

Ablation lesions are made in the inferior or mid part of the triangle of Koch. Initial success rates are greater than 95%.

Cryo or radiofrequency.

Minimal or no fluoroscopy.

Ablation is definitive treatment.
Atrioventricular Reciprocating Tachycardias (AVRT)

• Requires the presence of a bypass tract or accessory pathway
  – Mean onset of age is younger than for AVNRT

• Most common: Kent bundles in “Wolf Parkinson White” Syndrome

• Left lateral free wall, right lateral free wall, and posterior septum

Concept of Pre-excitation

• Termed Pre-excitation because some conduction occurs via the Kent bundles in addition to the normal pathway; because conduction via the Kent bundles is faster than via the AV node the ventricles are pre-excited
  – Manifest pathway if it allows conduction antegrade
  – Present in 0.1 to 0.3% of population

• This produces a “delta wave” on the EKG
• Fusion beat
  – Short PR
  – Wider than normal QRS
Delta Wave of Pre-excitation Syndrome

- 60 to 70% of WPW shows evidence in SR

Left sided accessory pathway:
Positive delta wave in V1

Right sided accessory pathway:
Negative delta wave in V1
Concealed pathways only allow retrograde conduction
Arrhythmias of WPW (AVRT or CMT)

Orthodromic Tachycardia: 90 to 95% of AVRTs

Orthodromic tachycardia occurs when the wave of electrical activation enters the ventricle normally through the AV node and returns to the atrium (retrograde) via the accessory pathway.

This allows the electrical impulse to re-enter the AV node and stimulate the ventricles once again.
Orthodromic Tachycardia

Negative P’ in lead 1 = left sided accessory pathway
Positive P’ in lead 1 = right sided accessory pathway
Orthodromic AVRT or AVNRT

- **AVNRT**
  - Simultaneous depolarization
  - P' waves buried
  - Initial P'-R interval prolonged (.38 second)
  - Atypical will have a distinct P'
    - Long RP
    - P' waves negative in II, III, and aVF
    - Can mimic orthodromic tachycardia

- **Orthodromic AVRT**
  - Sequential Depolarization
  - Distinct P' waves
  - Initial P'-R interval normal
  - Faster rate
  - Accessory pathway required

---

![EKG Image](image-url)
Antidromic Tachycardia

- The less common form of atrioventricular reentrant tachycardia
- The path of tachycardia passes from the atrium to the ventricle via the accessory pathway (Kent bundles) and returns to the atrium via the AV node
- The QRS complex is wide because antegrade conduction bypasses the AV node
- Antidromic tachycardia is very difficult to distinguish from ventricular tachycardia because ventricular depolarization begins where the accessory pathway enters the ventricle
  - Negative concordance will not be antidromic tachycardia
Antidromic Tachycardia

Diagram showing the cardiac anatomy and the pathway of electrical conduction in antidromic tachycardia.
Atrioventricular Reentrant Tachycardia (AVRT)

- **Orthodromic**
  - Traveling down the AV junction and up an accessory pathway
  - Sequential depolarization
  - Narrow because travel via the AV node
  - More common than antidromic tachycardia

- **Antidromic**
  - Activation of the ventricles is initiated by impulses descending via an accessory pathway
  - Ventricular depolarization begins at an ectopic site in the myocardium and returns via the AV node

Presence of pre-excitation on 12 lead and paroxysmal palpitations.
Unique Forms of AVRT

A permanent form of junctional reciprocating tachycardia (PJRT)
- Type of orthodromic tachycardia
- A concealed accessory pathway that has decremental conduction properties
- Usually located in the posteroseptal region and allows retrograde conduction
- Deep inverted P waves in leads II, III, and aVF with a long R-P interval
- Incessant in nature and can lead to a tachycardia mediated cardiomyopathy

Mahaim fiber
- Atriofascicular fiber that connects the right atrium to the distal right bundle branch
- Conducts anterograde only and has decremental conduction properties
- Left bundle branch morphology

Note: Any form of SVT can conduct over an accessory pathway. This is called bystander pathway.

WPW and Atrial Fibrillation

- Mechanism of Action
  - Development of Atrial Fibrillation in WPW
    - 10-32% of patients
  - Refractory period of accessory pathway

- Danger
AF in WPW

Accessory Pathway

The risk of SCD greatest in the first two decades of life.

Risk features for SCD include multiple accessory pathways, history of symptomatic tachycardia, and a pre-excited R to R interval of < 250 msec during atrial fibrillation.
Example of WPW Atrial Fib
(antegrade conduction via accessory pathway)

Example of WPW Atrial Fib
(antegrade conduction via accessory pathway)
Treatment for WPW Tachycardias

- AV Reentrant (orthodromic): Adenosine
- AV Reentrant (antidromic)
- Atrial Fib with antegrade conduction over accessory pathway: NO diltiazem, verapamil, beta blockers, or digoxin

Slow conduction over accessory pathway:
- Amiodarone
- Procainamide – may be used IV
- Flecainide – alternative to ablation
- Sotalol
- Propafenone – alternative to ablation
- Ibutilide – may be used IV

New 2014 Atrial Fibrillation Guidelines

CHANGE IN RECOMMENDATION REGARDING AMIODARONE FOR PATIENTS WITH PRE-EXCITATION
Lethal Outcome After Intravenous Administration of Amiodarone in Patient with Atrial Fibrillation and Ventricular Preexcitation

MUJOVIĆ NEBOJŠA M.D., SIMIĆ DRAGAN M.D., ANTONIJEVIĆ NEBOJŠA M.D. and ALEMPIJEVIĆ TAMARA M.D.

Journal of Cardiovascular Electrophysiology

Volume 22, Issue 9, pages 1077–1078, September 2011

Article first published online: 18 FEB 2011
DOI: 10.1111/j.1540-8167.2011.02013.x
Classification of Ventricular Arrhythmia by Electrocardiography

- Nonsustained ventricular tachycardia (VT)
  - Monomorphic
  - Polymorphic
- Sustained VT
  - Monomorphic
  - Polymorphic
- Bundle-branch re-entrant tachycardia
- Bidirectional VT
- Torsades de pointes
- Ventricular flutter
- Ventricular fibrillation

ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death
Ventricular Flutter
Spontaneous conversion to NSR (12-lead ECG)

VF with Defibrillation (12-lead ECG)
Common Cause of Monomorphic VT

Bundle Branch Reentrant Ventricular Tachycardia
✓ Malignant
✓ Treated with ablation
Acute Management of Ventricular Arrhythmias

- Wide complex tachycardia presumed to be VT if diagnosis is unclear
- DC cardioversion with sedation if hemodynamically unstable
- Don’t assume VT cannot be well tolerated!
- The rate, size of the heart and presence of additional complications are often more important than the source of the tachycardia

✓ Check the patient (need to defib?)
✓ Check the blood pressure (need to cardiovert?)
✓ Check the ECG (determine the rhythm)
Arrhythmias with ACS: ACC/AHA

• V-fib early in ACS
  • Increases hospital mortality
  • Does not increase long term mortality
• Lidocaine prophylaxis: Not indicated
  • Decreases V-fib
  • Increases mortality due to bradycardia
• Beta-blockers prophylaxis
  • Decreases V-fib
• Always correct potassium and magnesium abnormalities

Monomorphic VT: ACC/AHA

• DC cardioversion with sedation if unstable
• IV procainamide
  • Effective in stable VT
  • Use with caution with CHF or severe LV dysfunction
• IV amiodarone
  • Indicated in hemodynamically unstable VT
  • Indicated in VT refractory to shock
• TTVP for pace termination
• Lidocaine can be effective if ischemia is etiology
• Class III: Calcium channel blockers in wide complex of unknown origin; especially if myocardial dysfunction
**Repetitive Monomorphic VT: ACC/AHA**

- IV amiodarone, beta-blocker, procainamide
- If idiopathic VT it is most likely from RV outflow tract
  - May be provoked by exercise
  - Beta-blockers or calcium channel blockers may be effective
  - Ablation is successful treatment option

**Clinical Pearls for Ventricular Arrhythmias**

- V-fib seldom is seldom preceded by warning arrhythmias
- R on T PVCs are typically only important first 24 hours of myocardial infarction
- Although not routinely treated - bigeminy may need treated if cardiac output effected
- Ventricular ectopy (as infrequent as 15% burden) can result in heart failure
Clinical Pearls for Ventricular Arrhythmias

• Potential reversible causes
  – Hypokalemia: K < 3.2 mEq/L (cause or result)
  – Magnesium < 1.5 mEq/dL
  – Ischemia
  – Use of inotropic agents

Polymorphic VT with normal QT:

• Seen frequently in ischemic conditions
  – Think revascularization
  – Think beta blockers
Special Considerations:
Polymorphic VT (normal QT)

ACC/AHA

• DC cardioversion with sedation when unstable
• IV beta-blockers if ischemia suspected
  • Improve mortality
• IV amiodarone in absence of abnormal repolarization
  – Amiodarone better than placebo
  – Magnesium not better than placebo
• Urgent angiography to exclude ischemia
• Lidocaine may be reasonable if ischemia suspected
• Check electrolytes
• Consider any other potential reversible cause

Expected QTc Intervals

<table>
<thead>
<tr>
<th></th>
<th>1 to 15 Years</th>
<th>Adult Males</th>
<th>Adult Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt; .44 seconds</td>
<td>&lt; .43 seconds</td>
<td>&lt; .45 seconds</td>
</tr>
<tr>
<td>Borderline</td>
<td>.44 to .46 seconds</td>
<td>.43 to .45 seconds</td>
<td>.45 to .47 seconds</td>
</tr>
<tr>
<td>Prolonged</td>
<td>&gt; .46 seconds</td>
<td>&gt; .45 seconds</td>
<td>&gt; .47 seconds</td>
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</tbody>
</table>


QTc .50 sec (500 msec or more is dangerous and should be considered an ominous sign of impending Torsade's de Pointes.
QT Interval

• Measured from beginning of QRS complex to the end of the T wave

• Reflects both ventricular depolarization (QRS) and ventricular repolarization (T wave)

• Used most specifically to reflect ventricular repolarization

U Waves and Biphasic T waves.
Heart Rate Adjustment

- QT interval needs to be adjusted for HR
- QT does not adjust to HR on a beat to beat basis

- **Dynamic changes are most important**
- Abnormal findings are uncovered during abrupt changes in the R to R

- **Irregular heart rhythms (i.e. atrial fibrillation) remain a clinical challenge**
What are Early After Depolarizations?

- Right after repolarization (or during) there is a transient sub threshold depolarization
  - Can occur during Phase II or III of the cardiac action potential
  - If an early after depolarization reaches threshold a second upstroke occurs and a **triggered** beat follows
  - The triggered beat may have its own after depolarization that reaches threshold – thus causing another triggered beat
- Thought to be etiology of Torsade's de Pointes
  - Acquired
  - Congenital
More on Early After Depolarizations

- Precipitating Factors
  - Hypokalemia
  - Hypomagnesemia
  - Heightened sympathetic tone
  - Slow heart rate
  - Prolonged repolarization (QT interval)

![Cardiogram](image_url)
Cardiac Ion Channel Abnormalities

- Long QT Syndrome (LQTS)
- Brugada disease
- Idiopathic short QT
  - < 300 to 340 msec
- Diagnosed by family history and ECG

Note: Patients with heart failure can develop channelopathies

LQTS

- QTc > 450 ms
- Genetic defect in either potassium (LQT1 or LQT2) or sodium (LQT3) channels
  - KCNQ1 (LQTS type 1), KCNQ2/HERG (LQTS type 2) and SCN5A (LQTS type 3)
  - Autosomal dominant trait
  - 1 in 2500
  - Delayed repolarization (1 and 2)
  - LQT1 and LQT2 = 95%
    - Beta blockers
  - LQT3 = 5%
    - Beta blockers may be harmful
    - Events during sleep or episode of bradycardia
- QT prolongation important risk factor for SCD
  - Untreated mortality of 50%
    - QTc < 440 ms / < 5%
    - QTc 460 to 500 ms / 20%
    - QTc > 500 ms / 50%
Each Type of Congenital QT Looks Differently in Terms of T Wave Morphology

• Interestingly – some acquired Torsade's may be preceded by T wave morphology looking like congenital LQTS

• Long QT 1: wide, broad-based T waves
• Long QT 2: low amplitude, often notched T waves
• Long QT 3: long ST segment and tall, peaked T waves

Resting ECG may not show QT prolongation.
LQT1 episodes in response to exercise of exertion: particularly swimming. Prepuberty males more events than females; female adults more than adult males.

11 year old male LQT1 patient ECG showing a normal T wave pattern and average QTc of about 480 msec.

28 year old female with LQT2. Bifid T waves are evident in leads II, III, AVF and particularly V4. Events typically associated with auditory triggers.
Brugada Syndrome

- Inherited ion channelopathy.
  - Disorder of cardiac sodium channel (20%)
- Autosomal dominant
  - Most common in Southeast Asian countries
  - 90% of patients are male
- Predispose to Syncope or sudden cardiac death (SCD)
  - Impacts action potential
  - Events occur more commonly at rest or during sleep
  - Events occur in 3rd or 4th decade of life
  - Increased risk for SCD
    - Syncopal episode
    - Early repolarization pattern on ECG
    - Family history of SCD
    - Asymptomatic patients at low risk for SCD
- Treatment
  - ICD
  - Quinidine or isoproterenol for VT Storm

Diagnosis of Brugada Syndrome

- 3 characteristic ECG patterns identified
- If type 1, 2 or 3 ECG findings are present one of the following must also be present to consider a diagnosis of the BS:
  - Documented ventricular fibrillation
  - Self-terminating polymorphic ventricular tachycardia
  - Family history of sudden cardiac death at < 45 years
  - Type 1 ST-segment elevation in family members
  - Electrophysiologic inducibility of VT
  - Unexplained syncope suggestive of a tachyarrhythmia
  - Nocturnal agonal respiration
| Type 1 | Coved ST elevation  
ST gradually descends to an inverted T wave  
Present in more than one right precordial lead V1-V3. |
|-------|--------------------------------------------------|
| Type 2 | T wave remains positive or biphasic  
The terminal portion of the ST-segment is elevated ≥ 1 mm  
Present in more than one right precordial lead V1-V3 |
| Type 3 | T Wave is positive  
The terminal portion of the ST-segment is elevated < 1 mm  
Present in more than one right precordial lead V1-V3 |

Source: Chaturvedi et al., 2011.
Torsade's De Pointes

• Recognition of this life-threatening arrhythmia is important because it is not treated like other VTs
• Two groups: Acquired and congenital
• Acquired
  • Drugs prolonging repolarization
    – Most often as a result of blocking the potassium channel
  • Electrolyte abnormalities
    – Low potassium
    – Low magnesium
• Severe bradycardias / pauses
More on Drugs that Prolong Repolarization (blocking of potassium channel efflux)

- www.QTdrugs.org
- www.torsades.org

- Class Ia and Class III antiarrhythmics
- Antihistamines
- Antibiotics
- Antipsychotics
- Antidepressants
- Sedatives
- Gastric motility agents
- Anticancer agents
- Opiate agonists

Other Risk Factors for Torsade's de Pointes

- Rapid (IV) administration of QT prolonging agent
- Renal or hepatic dysfunction
- Female gender (particularly for drug induced)
- Advanced age
- Anorexia
- Heart disease
- Poly pharmacy
Class I: Na\(^+\) Channel Blockers

Class III: K\(^+\) Channel Blockers

Class IV: Calcium Channel Blockers

---

Class I
Slow conduction (widen QRS).
Some prolongation of refractory period (prolong QT interval).

Class III
Marked prolongation of refractory period (prolong QT interval).
Warning signs for Torsades de Pointes

- Increase QTc from predrug baseline of 60 ms,
- Marked QTc interval prolongation .500 ms
- T-U wave distortion that becomes more exaggerated in the beat after a pause
- Visible (macroscopic) T-wave alternans
- New-onset ventricular ectopy, couplets
- Nonsustained polymorphic ventricular tachycardia initiated in the beat after a pause.
Torsade's de Pointes

- Class I
  - Discontinue offending drugs
    - Note: Class IA drug induced TdP usually appears soon after the initial administration of the drug
  - Correct electrolytes
    - Magnesium
    - Potassium
  - Increase HR
    - Isoproterenol
      - 2 mcg/min then titrate to HR of 100 beats per minute
    - Temporary pacing at rate of 100 to 110
    - Permanent pacing if bradycardia or CHB cannot be resolved.
- Defibrillation if sustained
  - However, continue to assess for and treat cause

Magazine is considered treatment of choice.

More on Magnesium in Torsade's de Pointes

- 2 Gm IV bolus over 1-2 minutes
  - Followed in 15 minutes by another bolus if necessary
  - May start continuous infusion at rate of 3-20 mg/min
- Benefit occurs without shortening of QT interval and in presence of normal Magnesium level
Case Example

QT Interval Monitoring Case Example

- Patient admitted for syncope after having motor vehicle crash while driving.
- Long standing history of paroxysmal atrial fibrillation – on dofetilide (Tykosin) for several years.
- Recent chemotherapy for breast CA resulting in a reduction of EF.
- Recent increase in carvedilol and lisinopril per general cardiology to improve EF.

- Next slide is admission ECG. Note the QTc interval.
1. Strip 1: QTc consistent with admission ECG.
2. Strip 2: Marked QTc prolongation when patient asleep.
3. Initial run of ventricular tachycardia initiated by PVC firing at end of T wave,
Same patient with sustained Torsades de Pointes. Treated effectively with 2 grams IV Magnesium (magnesium level was normal at baseline). Magnesium is the drug of choice to stabilize the cardiac membrane. Dofetilide (Tikosyn) was also discontinued.

Note: Although the patient had been on dofetilide (Tikosyn) for several years, the recent change in ejection fraction and increase in beta blocker therapy increased her risk for Torsades de Pointes.

Electrolyte Effects

- Potassium and calcium are the two electrolytes with the most influence on the ECG
- Changes are non specific
  - ECG cannot be considered diagnostic of an electrolyte abnormality
  - Electrolyte abnormalities can occur in the absence of ECG changes
- Magnesium abnormalities aren’t revealed by changes on the ECG
  - Can result in cardiac arrhythmias
  - Magnesium is treatment of choice in Torsades de Pointes
**Action Potential of Cardiac Cells**

- **Phase 0**: Rapid depolarization – **Sodium Influx**  
  (beginning of QRS complex)
- **Phase 1**: Brief, rapid initiation of repolarization
- **Phase 2**: Slowing of the repolarization – **Calcium Influx**  
  – correlates with ST segment

**The Electronics**

- **Phase 3**: Sudden acceleration in the rate of repolarization - **Potassium Efflux**  
  – Correlates with T wave
- **Phase 4**: Resting membrane potential
Hyperkalemia: Signs and Symptoms

Symptoms when K+ > 6.0 mEq/L
Skeletal muscle effects when K+ > 7.0 mEq/L
Neuromuscular effects complicated by acidosis, low sodium, low calcium, high magnesium
* Parathesia
Lower extremity weakness
Hypotension

EKG Changes
Tall narrow peaked T waves
Wide QRS
Prolonged PR and flattened to absent P wave
Dysrhythmias
√ Bradycardia / heart block
√ Sine wave pattern
√ Asystole

Hyperkalemia

![EKG diagram showing tall narrow peaked T waves and wide QRS]
Hyperkalemia

Note: This is not a normal sinus rhythm.

Hyperkalemia

Potassium 8.8
BUN 240
Creatinine 24.4

Note: Prolonged PR interval and flattening of the P wave.
Hyperkalemia

Hypokalemia: ECG Changes

- Mild hypokalemia: delays ventricular repolarization
  - ST depression, flattening of T wave, inverted T wave
  - Heightened U waves, prolonged QT interval
- Increases risk for Torsades de Pointes
- Lowered threshold for ventricular fibrillation and reentrant tachycardias
- Severe hypokalemia
  - Increased PR interval
  - Increased QRS interval
Clinical Pearls

Digoxin

• Hypokalemia increases risk of digoxin toxicity.

Class III Antiarrhythmics

• Hypokalemia increases the risk of Torsades de Pointes with potassium channel blocking medications

Hypokalemia
Hypokalemia

Etiology of Cardiac Arrest

Hypokalemia: Severe
Hypocalcemia: Signs and Symptoms

Most common symptoms due to neuromuscular irritability.

- **Parathesias (common)**
- **Hyperreflexia**
- **Tetany (spasms of face, hands, and feet)**
- **Chvostek’s sign**
  - Tapping of face over facial nerve located below the temple
  - Positive sign results in spasm of lip, nose or face.
- **Trousseau’s sign**
  - Inflate blood pressure above systolic BP and hold for 3 minutes
  - Positive sign results in contraction of fingers or hand.
- **Stridor / wheezing / bronchospasm**
- **For severe deficit:** laryngeal spasm, change in mental status, seizures
- **Chronic:** dry skin and hair and brittle nails; bone pain and risk of fracture

### Cardiovascular effects:

- Decreased contractility
- Hypotension
- **Prolonged QT**
  - ST segment hugging baseline for extended period
  - QT prolongation is not due to delay in ventricular repolarization
  - Torsades de pointes
  - Bradycardia / heart block
  - **Digitalis insensitivity**
  - Heart failure
  - Cardiac arrest

Hypocalcemia results in the prolonged opening of the calcium channels during Phase II of the Cardiac Action Potential: Thus extending the ST segment.

Note the hugging of the ST segment to baseline.
Hypocalcemia

Note the hugging of the ST segment to baseline.

Hypercalcemia: Signs and Symptoms

- Cardiac symptoms:
  - hypertension (may be offset by co-existing dehydration)
  - cardiac ischemia
  - arrhythmias (conduction abnormalities)
  - digitalis toxicity.

- **ECG signs**
  - shortened QT segments (secondary to shortened ST segments)
  - Short ST segments can cause ST to merge with T wave (similar to what occurs with hyperacute T wave in a STEMI)

- Life threatening signs and symptoms are rare unless calcium levels reach > 14 mg/dL.
Hypercalcemia

Note lack of horizontal component of ST segment and abrupt take off of T wave after QRS.
INJURY AND ISCHEMIA WITH BBB

ST T Wave Changes With BBB

In both RBBB and LBBB DISCORDANCE is normal.

DISCORDANCE = END of the QRS and the T Wave are opposite from each other.

NORMAL ST-Twave Changes Right BBB

Right Bundle Branch Block Morphology With Appropriately Discordant T-Waves

ST should be at baseline. Presence of any ST elevation or depression: Abnormal. Presence of concordance: Abnormal.
Identifying Infarction in LBBB

If end of QRS & T wave are DISCORDANT:
ABNORMAL: If ST elevation $\geq 5$ mm and/or disproportionate with the QRS voltage (B).
ABNORMAL: If ST Segment depression $\geq 1$ mm in leads V1, V2 or V3 (C).

If end of QRS & T wave are CONCORDENT:
Abnormal: If ST elevation $\geq 1$ mm (A).
New or Presumed New LBBB

- Left BBB is common reason for delayed or withheld reperfusion

- New or presumed to be new LBBB and clinical signs of AMI are indication for reperfusion therapy

- Old LBBB with increased ST elevation or specific indictors should also receive reperfusion

Admitted for Hyperkalemia
Developed CP During Dialysis the Next Day

RV Paced Rhythm
ECG of Printzmetal’s Angina or Variant Angina

- Precordial ST elevation in most adults
  - Up to 90%
- Early repolarization most common normal variant
  - Roughly 5-13% of population
  - More common in African Americans
  - More common in adolescents and athletes
Historical Characteristics of Early Repolarization

- J wave, J point elevation, and tall symmetrical T waves with concave ST segment elevation in two contiguous leads
- Historically most common in left precordial leads

ECGs over time with early repolarization.

May be confused with or concurrent with LVH.

J Wave

- Also known as Osborne wave
- Deflection (similar morphology to a P wave) at the end of the QRS
- Usually seen in all 12 leads
Early Repolarization and Associated Risk

- Association with idiopathic VT especially with J waves and horizontal ST segment depression.
- Increased risk of sudden cardiac death when seen in inferolateral leads

New Definitions Related to Early Repolarization

- If the ST segment slopes upward and is accompanied by an upright T wave: this is called early repolarization with an ascending ST segment
- If there is horizontal or a downward sloping ST segment: this is called early repolarization with a horizontal or descending ST segment
- ST segment elevation without a slur or notch should not be reported as early repolarization
• Proposed classification of Early Repolarization (ER)
  – Type 1: associated with ER in the lateral precordial leads. This form is common among healthy male athletes and is thought to be largely benign.
  – Type 2: associated with ER in the inferior or inferolateral leads and is associated with a moderate level of risk.
  – Type 3: associated with ER globally in the inferior, lateral, and right precordial leads, and appears to be associated with the highest relative risk (though the absolute risk of sudden death remains small).
Early Repolarization

Early Repolarization
Pericarditis: ECG Findings

- Mimics: anteroinferior; inferolateral; antero-infero-lateral MI
- ST Elevation
  - ST elevation typically greatest in II and V5 (also I and V6)
  - ST elevation may also be in V1 –V4; aVF, III and aVL (least)
  - Upwardly concave ST segments
  - ST elevation usually ≤ 5 mm
**Pericarditis: ECG Findings**

- Other ST changes
  - ST depression in aVR
  - Minimal depression V1, III, aVL may exist

- PR Segment depression
  - PR depression most common in II, aVF and V4 – V6
  - PR elevation > 0.5 mm in aVR

- Electrical Alternans
  - Voltage changes with pericardial effusion or tamponade

**Stages of Pericarditis**

- **Stage I**
  - ST elevation
  - More concave
  - Lasts up to 2 weeks

- **Stage II**
  - ST to baseline
  - Decrease T wave amplitude
  - Lasts from days to several weeks

- **Stage III**
  - T wave inversion
  - Starts at end of second to third week

- **Stage IV**
  - Gradual resolution
  - T wave may stay inverted up to 3 months
Pericarditis

- Diffuse Pericarditis
  - Easiest to differentiate with both pain and ECG assessment
- Localized Pericarditis
  - May have reciprocal changes

Perimyocarditis

- Troponin
- Wall motion abnormalities

Classic Pericarditis
Pericarditis

Dilated Cardiomyopathy
Dilated Cardiomyopathy

• Most common form of cardiomyopathy
  – Idiopathic
  – Genetic disorders
  – Viral / Bacterial Infection
  – Hyperthyroidism
  – Chemotherapy
  – Peripartum Syndrome Related to Toxicity
  – Cardiotoxic Effects of Drugs or alcohol

Hypertrophic Cardiomyopathy
Hypertrophic Cardiomyopathy

- 1 of every 500 (Maron et al, 2003)
- Primary genetic cardiomyopathy
- Effects men and women equally
- Hypertrophy of myocardial muscle mass in the absence of increased ventricular afterload
- Associated with decreased ventricular filling (diastolic dysfunction) and decreased cardiac output
- Most common cause of sudden death in young adults
- Cause unknown
  - 50% transmitted genetically

Hypertrophic Cardiomyopathy

- Disarray of cardiac myofibrils with hypertrophy of myocytes
- Cells take on a variety of shapes
- Myocardial scarring and fibrosis occurs
Hypertrophic Cardiomyopathy

- Usually only effects Left Ventricle
- Changes may be symmetrical
- Asymmetrical septal hypertrophy is more common

Hypertrophic Cardiomyopathy

May involve entire septum or only a portion of septum
Symptoms of HF may develop
Transferred to pulmonary system
Atrial dilatation due to increase in pressure and volume
Atrial kick more essential than normal
Passive filling from the atria is slowed
Stiff walls resist filling (diastolic dysfunction)
Ventricular chamber size decreases as enlarging walls close in on chamber
EF increases to 70-80%
Compensation for decreased filling -> hyperdynamic systolic dysfunction
Mitral Regurgitation
Physiologic Changes with Hypertrophic Cardiomyopathy
Atrial dilatation due to increase in pressure and volume
Transferred to pulmonary system
Symptoms of HF may develop
<table>
<thead>
<tr>
<th>OBSTRUCTIVE Hypertrophic Cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>• 35% of HCM patients have obstruction at rest</td>
</tr>
<tr>
<td>• 35% additionally have obstruction with provocation</td>
</tr>
<tr>
<td>• Obstruction of outflow tract</td>
</tr>
<tr>
<td>• Septal wall enlarges into ventricular cavity</td>
</tr>
<tr>
<td>• Anterior leaflet of mitral valve drawn towards the septum during ejection</td>
</tr>
<tr>
<td>• Early closure of aortic valve, decreased ejection time, decreased cardiac output</td>
</tr>
</tbody>
</table>
Hypertrophic Cardiomyopathy Presentation

- Many asymptomatic for years
- Incidence of sudden death often first presentation
  - Or identified during screening of relative of patient with HCM
- Symptoms related to severity of diastolic dysfunction
- Heart failure
- Dyspnea #1 sign
- Syncope / palpitations with activity
- Chest pain
- Supraventricular arrhythmias
- Development of mitral regurgitation

Subvalvular Left Ventricular Outflow Obstruction Systolic Murmur

- Timing: Mid systolic
- Location: best heard along left sternal boarder
- Radiation: usually does not radiate
- Configuration: crescendo-decrescendo
- Intensity: grade 3/6 to 4/6
- Pitch: medium
- Quality: harsh or rough
Subvalvular Left Ventricular Outflow Obstruction Systolic Murmur

HOCM murmur louder during Valsalva’s maneuver

Decreases venous return to the heart

- Decreased preload $\rightarrow$ ↓ left ventricular filling
- Decreased left ventricular filling $\rightarrow$ ↑ obstruction

Any factor that decreases venous return to the heart increases the murmur in HOCM

- Squatting increases venous return
- Standing decreases venous return

Aortic stenosis murmur becomes quieter during Valsalva’s maneuver
Hypertrophic Cardiomyopathy Treatment

- **Goals**
  - Relief of symptoms
  - Preventing complications
  - Preventing or reducing risk of sudden death
  - No evidence to support treatment of non-symptomatic patients

**Beta Blockers**
- 1st choice (with or without HOCM)
- Symptomatic benefit / improved exercise tolerance
- Decreases HR
- Improves LV relaxation
- Helps control arrhythmias

**Calcium Channel Blockers**
- If Beta Blocker not effective
- Decrease LV wall tension
- Decreases HR
- Diltiazem or Verapamil (no nifedipine D/T vasodilatation)

9/22/2017
## Hypertrophic Cardiomyopathy

### Treatment

#### Disopyramide
- Negative inotrope
- Class I antiarrhythmic
- Use with BB to treat LV outflow track obstruction
- Assists in HR control
- May cause ventricular

#### Anti arrhythmic Therapy
- Atrial Fibrillation
- Most common arrhythmia
- Poorly tolerated
- Anticoagulation
- Amiodarone or sotolol
- Obstructive or non-obstructive OK
- Ventricular or atrial arrhythmias

### Other Medications
- Diuretics
  - With caution
- ACE Inhibitors and NTG
  - Avoided in HOCM
- Positive Inotropes
  - Strictly avoid any medication that increases contractility in HOCM

### Pregnancy
- Not restricted in non-obstructive disease

### Endocarditis Prophylaxis
- NO LONGER INDICATED
  (was previously indicated in obstructive disease only)

### Non-Obstructive Disease Treatment
- More difficult to treat if no symptoms
- Ultimately evolves into dilated cardiomyopathy
Surgical Myectomy

- Marked outflow obstruction
- On maximum medical therapy
- NYHA Class III or IV
- MV Replacement or repair at same time (increases operative mortality)
- Improvement noted immediately and last 20-30 years
- Survival Rates 80% at 10 years
- May need pacemaker (2%)

Percutaneous Alcohol Septal Ablation

- Symptomatic with full therapy
- NYHA Class III or IV
- Not appropriate if MVR needed
- Cath Lab Procedure
- Catheter in septal perforator
- Ethyl alcohol injected
- Myocardial infarction occurs
- Enlarged septum eventually shrinks
- May need pacemaker (20%)
Risk for Sudden Death

• One or more 1st degree relative with an episode of SCD
• Left ventricular wall thickness greater than 35 mm
• Prolonged or repetitive non-sustained ventricular tachycardia on Holter monitor
• Hypotensive BP response to exercise
• Syncope or near syncope

Family Evaluation

• Screen 1st degree relatives
• Genetic testing best if available
• Screenings
  • Annually from age 12 -18 then every 5 years
  • Not necessary in relatives < 12 unless a particularly high risk family profile or a desire to play intense competitive sports.
• Screenings include:
  • Physical exam
  • 12 lead ECG
  • ECHO
Outcomes

Normal life span

Once diagnosed – routine follow up every 12 -18 months

SCD primary cause of shortened life span

Arrhythmogenic Cardiomyopathy
Arrhythmogenic Cardiomyopathy: ECG Signs

- ECG
  - T Wave inversion in leads V1-V6
  - Epsilon wave
  - VT with LBBB pattern
  - Conduction delays through right bundle
Arrhythmogenic Cardiomyopathy

Figure 10.16: Prolonged upstroke of the S wave.

Figure 10.17: Epsilon waves in a patient with ARVC.
Arrhythmogenic Cardiomyopathy

- Inherited muscle disorder
- Often referred to as Arrhythmogenic Right-Ventricular Dysplasia (ARVD)
- Manifest as an arrhythmia, heart failure, or sudden death
- Genetic characteristics include autosomal dominance inheritance (most common)
- Most frequently affects the right ventricle
- More often than thought also affects left ventricle
- More often males than females

Arrhythmogenic Cardiomyopathy

- Cardiomyocyte replaced with fibro fatty tissue
- Initially patchy infiltration
- Progressive loss of muscle leads to thinning of the ventricular wall, dilation and pump dysfunction
- Thinnest portions of the right ventricle affected first
- Triangle of dysplasia: Inflow, outflow, apical regions of RV
### Arrhythmogenic Cardiomyopathy

**Disease Progression**

<table>
<thead>
<tr>
<th>Early / Concealed phase</th>
<th>Overt Phase</th>
<th>Impaired contractility and right-sided failure</th>
<th>Bi-ventricular failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Subtle structural changes</td>
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<tr>
<td>- Often asymptomatic</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>- Noticeable structural and functional changes</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>- Palpitations, pre-syncope, syncope, arrhythmias</td>
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<td></td>
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<tr>
<td>- Right ventricular dilation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Decreased contractility</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>- Signs of right sided heart failure</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>- Disease spreads to left ventricle</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Signs of biventricular failure</td>
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</tbody>
</table>
Arrhythmogenic Cardiomyopathy

Presentation

- Palpitations
- Presyncope
- Syncope
- Often episode of sudden cardiac death is first presentation
- Signs of heart failure are late sign

Diagnosis

<table>
<thead>
<tr>
<th>ECG</th>
<th>Echo</th>
<th>Endomyocardial Biopsy</th>
<th>MRI / CT</th>
</tr>
</thead>
<tbody>
<tr>
<td>T Wave inversion in leads V1-V6</td>
<td>RV enlargement and dysfunction</td>
<td>Unreliable</td>
<td>Detect fatty infiltrate</td>
</tr>
<tr>
<td>Epsilon wave</td>
<td>VT with LBBB pattern</td>
<td></td>
<td></td>
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<tr>
<td>Conduction delays through right bundle</td>
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</table>
Arrhythmogenic Cardiomyopathy

Treatment

- No cure
- Goal: Manage arrhythmias
- Antiarrhythmics: Amiodarone and beta blockers
- Implantable Cardiovertor Defibrillator
- Radiofrequency catheter ablation if unsuccessful in treating VT with antiarrhythmics
- Refrain from competitive / intense sports
- Screening of family members
  - 1st and 2nd degree relatives
ICD Implantation

- Recommended for the prevention of SCD in patients with ARVC and documented sustain VT or VF who are optimal medical therapy and have a reasonable expectation of good survival for more than one year (Class IB recommendation).
- ICD implantation can be effective for the prevention of SCD in patients with ARVC with extensive disease, including those with LV involvement, 1 or more affected family member with SCD, or undiagnosed syncope when VT or VF has not been excluded as the cause of syncope, who are receiving chronic optimal medical therapy, and who have reasonable expectation of survival with a good functional status for more than 1 year (Class IIa recommendation). (Zipes et al., 2006).

Heart Transplant

- Represents less than 1% of the heart transplant population
- Viable option for those with refractory or terminal heart failure
- Disease process does not rule out heart transplantation
- Outcomes after heart transplant demonstrate a survival similar to HCM, DCM, & others while significantly better than restrictive & ischemic disorders
Outcomes

Progressive disease

Long term prognosis continues to be evaluated
Population is small
Not in the literature 30 years ago
Median survival – 60 years.
SCD primary cause of mortality

Tako-Tsubo Cardiomyopathy
Tako-Tsubo Cardiomyopathy

- Transient left ventricular apical ballooning
- Abrupt onset of ballooning or dilatation of left ventricle
- Post menopausal women
- Occurs after psychosocial or physical stressors
- Also referred to as Stress Cardiomyopathy
- Cause unknown
  - Related to excessive catecholamines

Tako-Tsubo Cardiomyopathy

- Chest Pain mimicking acute MI
- ST-segment changes similar to anterior MI
- Elevated cardiac biomarkers
- Dyspnea
- Hypotension
- Signs of left ventricular failure
Tako Tsubo Cardiomyopathy Diagnosis

<table>
<thead>
<tr>
<th>ECG</th>
<th>Cardiac Biomarkers</th>
<th>Cardiac Cath</th>
<th>Echo</th>
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<tbody>
<tr>
<td>• ST elevation mimicking AMI</td>
<td>• Mildly elevated</td>
<td>• No significant coronary artery disease</td>
<td>• LV Dysfunction with decreased ejection fraction</td>
</tr>
<tr>
<td>• Prolonged QT interval</td>
<td>• Do not follow same rise and fall as AMI</td>
<td>Visualize ballooning of LV</td>
<td>• Visualize ballooning of LV</td>
</tr>
</tbody>
</table>

Modified Proposed Mayo Clinic Criteria for Apical Ballooning Syndrome

1. Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always present.

1. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.

1. New electrocardiographic abnormalities (either ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin.

1. Absence of: Pheochromocytoma or Myocarditis

From Prasad, Lerman, & Rihal, 2009
Treatment

- Goals: Similar to patients with Acute MI
- Treat Left Ventricular Failure
  - Cardiogenic Shock
  - IABP
  - Arrhythmias
  - Hypotension
  - Avoid inotropes
  - Cardiac Rehabilitation
  - Stress Reduction
Immediate Care After ROSC
(Return of Spontaneous Circulation)

• Optimize ventilation and oxygenation
  – Maintain oxygen saturation ≥ 94%
  – Do not hyperventilate
    • Target PETCO2 of 35-40 mm Hg

• Treat hypotension

• Coronary Reperfusion
  – If STEMI or high suspicion of acute MI

• TTM – if does not follow commands
Coronary Angiography
Post Cardiac Arrest

• Coronary angiography should be performed emergently for OHCA patients with suspected cardiac etiology of arrest and ST elevation on the ECG. (COR I, LOE B)

• Emergency coronary angiography is reasonable for select (eg, electrically or hemodynamically unstable) adults who are comatose after OHCA of suspected cardiac origin but without ST elevation on the ECG. (COR IIa,B-NR)

• Coronary angiography is reasonable in post-cardiac arrest patients for whom coronary angiography is indicated, regardless of whether the patient is comatose or awake. (COR IIa, LOE C-LD)

Targeted Temperature Management

• Recommend that comatose (ie, lacking meaningful response to verbal commands) adult patients with ROSC after cardiac arrest should have TTM.
  – For VF/VT Out of Hospital Arrest (COR I, LOE B-R)
  – For non-VF/VT and in-hospital cardiac arrest (COR IIb, LOE C-EO)

• Recommend selecting and maintaining a constant target temperature between 32°C and 36°C (COR I, LOE B-R)

• It is reasonable that TTM be maintained for at least 24 hours after achieving target temperature (Class IIa, LOE C-EO).

• It may be reasonable to actively prevent fever in comatose patients after TTM (Class IIb, LOE C-LD)

• Routine prehospital cooling of patients with rapid infusion of cold IV fluids after ROSC is not recommended. (COR III: No benefit, LOE A)
Temperature

• Elevated temperature worsens outcomes in experimental models of cerebral ischemic and brain trauma
  – It is associated with increased levels of excitotoxins and \( \text{O}_2 \) radicals, destabilization of cell membranes and increased number of abnormal electrical depolarizations

Even a 0.5°C increase in temperature causes an increase in the zone of injury and neuronal loss

Hyperthermia

• Increased temperature is associated with an increase in morbidity, mortality and ICU/hospital length of stay

• Fever 2\textsuperscript{nd} most powerful predictor of hospital LOS
  – Twice as powerful as severity of illness
  – Mortality rates double with a moderate fever and triple in patients with the highest temps
  – Hospital disposition worsened as temp rose
Key Phases of TTM

- **Cooling**
  - Rapid 4-6 hours of arrest
  - Various methods but none stand out as superior
  - Target 32-36°C
- **Maintenance**
  - 24 hours from achievement of target temperature
- **Rewarming**
  - 0.25 to 0.33°C per hour – slower is perceived to be better
- **Post rewarming**
  - 48 hours
  - Avoid hyperthermia
- **Neurologic Prognostication**
  - Avoid for minimum of 72 hours post rewarming

Targeted Temperature Management at 33°C versus 36°C after Cardiac Arrest

- 950 unconscious adults after out of hospital cardiac arrest
- Randomized to 33 or 36°C
- Primary outcome: all cause mortality
- Secondary Outcome: poor neurologic function or death at 180 days, as evaluated with the Cerebral Performance Category (CPC) scale and the modified Rankin scale.

Nielsen et al NEJM 2013
Results

- At the end of the trial
  - 50% of the patients in the 33°C group (235 of 473 patients) had died, as compared with 48% of the patients in the 36°C group (225 of 466 patients) (hazard ratio with a temperature of 33°C, 1.06; 95% confidence interval [CI], 0.89 to 1.28; P = 0.51).
- At the 180-day follow-up
  - 54% of the patients in the 33°C group had died or had poor neurologic function according to the CPC, as compared with 52% of patients in the 36°C group (risk ratio, 1.02; 95% CI, 0.88 to 1.16; P = 0.78).

Editorial Comments

- 89% of patients were witnessed with 73% bystander CPR – may be higher than many communities experience?
- 36°C arm had 81% (to 79%) shockable rhythm and 77% (to 74%) V fib as rhythm/
- 36°C arm had 79% (to 75%%) pupillary reflexes at start and 66% (to 65%) corneal reflexes at start
- 33°C arm was rewarmed at 0.5 ° per hour with some patients being rewarmed more quickly than recommended
- 6 patients in 33 ° arm rewarmed before protocol completed
- 33 shown to be safe
- 36 should be standard of care in all post cardiac arrest patients that previously would not have been “cooled”
Our Vision:

Practice with Joy. Positively impact every patient and family on their journey; provide safe passage, meet them where they are, connect with them in a meaningful way, and delivering care with wisdom and intention.

- Cindy and Karen

BE THE BEST THAT YOU CAN BE EVERY DAY. YOUR PATIENTS ARE COUNTING ON IT!

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THANK YOU!!!