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Cardiovascular Course

Conquer the Deteriorating Cardiac Patient Day 2

Cheryl Herrmann

1

Statement of Accuracy and Utility/Scope of Practice

- "Materials that are included in this course may include interventions and modalities that are beyond the authorized practice of mental health professionals.
- As a licensed professional, you are responsible for reviewing the scope of practice, including activities that are defined in law as beyond the boundaries of practice in accordance with and in compliance with your professions standards."

2

Limitations of the Research and Potential Risks

- Cardiology Guidelines were reviewed for this seminar. In each topic, the level of evidence and risks for the interventions will be listed.
- Culture, race, and other variables which are often not adequately represented in the literature will be listed.

3

Classification of Recommendations and Levels of Evidence

A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Although randomized trials are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as sex, age, history of diabetes, history of prior myocardial infarction, history of heart failure, and prior aspirin use.

†For comparative effectiveness recommendations (Class I and IIa; Level of Evidence A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

Helping Cardiovascular Professionals Learn. Advance. Heal.

American Heart Association.

4

Cutting Edge Assessment Skills & Treatments

Cardiomyopathy
"One Name - Many Causes"
&
Acute & Chronic Heart Failure
"Too Pooped to Pump"

Cheryl Herrmann, APRN, CCRN, CCNS-CSC-CMC
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Cardiomyopathy – Definition

Cardio	Myopathy
Heart	Myo– Greek μυο "muscle" + Pathos –pathy Greek "suffering"
	Muscle disease in which the muscle fibers do not function resulting in muscular weakness

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Cardiomyopathy leads to Heart Failure

- In the past, left ventricular dysfunction and cardiomyopathy were sometimes thought of as equivalent to HF
- But rather they are **structural or functional reasons for the development of HF**

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Cardiomyopathy vs Heart Failure

Cardiomyopathy	Heart Failure
Primary disorder of cardiac muscle causing abnormal myocardial performance ◦ Mechanical and/or electrical dysfunction ◦ Exhibits ventricular hypertrophy or dilation ◦ Leads to progressive heart failure	***Complex clinical syndrome*** ◦ Pathologic state ▪ The heart is unable to pump enough oxygenate blood to meet the metabolic needs of the body ◦ Develops from any cardiac disorder that impairs the ability of the ventricle to fill or eject adequately ▪ Ie Cardiomyopathy ▪ AMI ▪ Hypertension

8

Normal

9

Abnormal – what caused it?

Numerous causes

Normal and Cardiomyopathy hearts

Road goes to one lane– causing no problems -- yet	Cardiomyopathy– Low EF

10

Becomes Congested

Causes slow down and problems	Symptoms of HEART FAILURE

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Cardiomyopathies

- Primary disorder of cardiac muscle causing abnormal myocardial performance

Normal and Cardiomyopathy hearts

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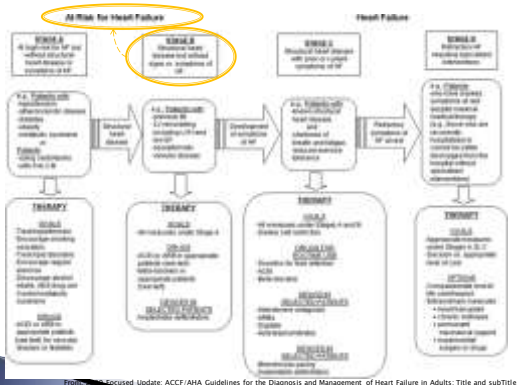

From the Focused Update: ACCF/AHA Guidelines for the Diagnosis and Management of Heart Failure in Adults: Title and subTitle BreakA Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines Developed in Collaboration with the International Society for Heart and Lung Transplantation

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Cardiomyopathy

- How can one name mean so many different things?
- One name – Many causes

- Work in a group and write down as many cardiomyopathies that you can think of.

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Cardiomyopathies

- Hypertrophic
- Stressed Induced (Takotsubo)
- Restrictive
- Dilated
  - Ischemic
  - Nonischemic
  - Idiopathic
  - Valvular
  - Genetic
  - Hypertensive
  - Myocarditis (Infection/Inflammatory)
  - Chemotherapy induced
  - Peripartum syndrome related to toxemia
  - Cardiotoxic induced (effects of drugs or alcohol)
  - Tachycardia induced

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Cardiomyopathies

- Hypertrophic
- Dilated (ischemic and nonischemic)
- Stressed Induced (Takotsubo)
- Restrictive

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Hypertrophic Cardiomyopathy (HCM)

- Myocardial hypertrophy
  - Without the presence of associated hemodynamic stress (no ↑ in afterload)
- Hypertrophy of the heart muscle
  - Includes the septum and ventricular free wall
- Previously called IHSS – idiopathic hypertrophic subaortic stenosis
- Leading cause of death in athletics < 35 y/o

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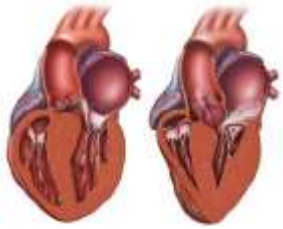
Hypertrophic Obstructive Cardiomyopathy (HOCM)

- May develop obstruction
- Once obstruction occurs it is called: Hypertrophic obstructive cardiomyopathy (HOCM)



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Hypertrophic Cardiomyopathy (HCM)

Pathophysiology

1. Hypertrophy of heart muscle including septum and ventricular free wall.
2. Rigid, noncompliant ventricles do not stretch
3. Causes diastolic dysfunction
4. ↓ preload and cardiac output
5. Left atrial dilatation from inability to empty LA
6. Mitral regurgitation occurs from papillary muscles and mitral valve pulled out of alignment

Hypertrophy of LV, septum and ventricular wall, LA enlargement, MR

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Hypertrophic Obstructive Cardiomyopathy (HOCM)

Pathophysiology

1. With severe hypertrophy, left ventricular outflow tract becomes obstructed --- especially with ↑ contractility from ↑ catecholamines (exercise)
2. Decrease in blood flow to coronary arteries (angina) and brain (syncope)
3. May result in sudden cardiac death

LV outflow tract obstructed – syncope, sudden death

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Hypertrophic Cardiomyopathy (HCM)

Causes

- Probably genetic
- May occur as early as the 1st year of life
- Develops most commonly during adolescence
- Hypertrophy manifests after age 20
- Diagnosis is usually made by age 25
- Persons with normal echo and EKG after 25 y/o are unlikely to develop HCM

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Hypertrophic Cardiomyopathy (HCM)

Clinical Presentation

- Often diagnosed incidentally as may be asymptomatic
- Dyspnea on exertion
- Chest pain on exertion – relieves with rest
- Syncope on exertion or rest
- Palpitations

- Jugular venous palpitation
  - Associated with prominent "a" wave secondary to ↓ RV compliance
- Heart Sounds
  - Harsh systolic murmur LSB
  - Murmur increases with movement
  - S4 from LVH
- EKG
  - Repolarization abnormalities
  - Atrial enlargement (large p waves)
  - Pathological Q waves – inferior leads

Sudden cardiac death often the first presentation

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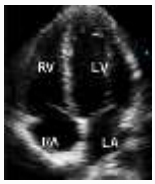
Normal Echocardiogram



Diastole

Systole

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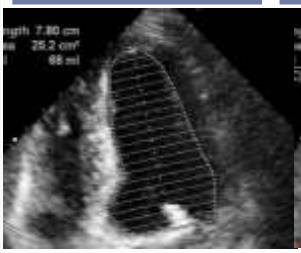
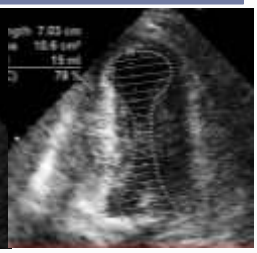
HOCM

Diastole



Systole



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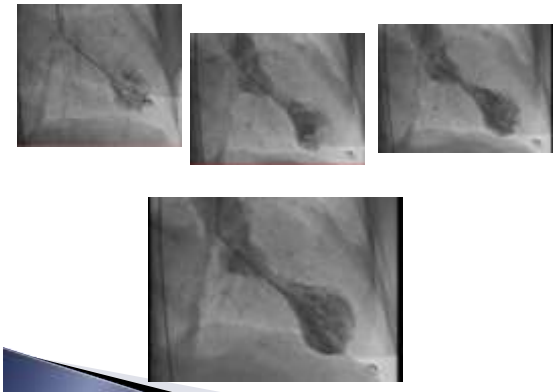

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Hypertrophic Cardiomyopathy (HCM)

Clinical Management

- Symptom relief
- Prevention of sudden cardiac death
- Beta blockers for chest pain and dyspnea with exertion in HOCM
- Disopyramide (Norpace and Rythmodan) – reduces obstruction by ↓ inotropic action
- Verapamil – used only for mild obstruction
- Atrial kick more essential than normal
- If symptoms persist
  - Ventricular Septal myectomy – removal of muscle from septum.
  - Percutaneous septal alcohol ablation – causes controlled septal MI
- ICD
  - History of cardiac arrest or sustained ventricular dysrhythmias
  - Multiple clinical risk factors
- Counseling & genetic testing
  - Restrict from intense competitive sports
- SBE prophylaxis for HOCM

Symptom relief & prevention of cardiac arrest

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Hypertrophic Cardiomyopathy (HCM)

Medications

- Beta blockers
  - 1st choice
  - Increase exercise tolerance
  - ↓ heart rate
  - Improves LV relaxation
  - Control of arrhythmias
- Disopyramide (Norpace and Rythmodan)
  - Negative inotrope (↓ contractility)
  - Used with BB to treat LV outflow track obstruction
  - ↓ SAM
  - Assists with HR control
  - Monitor QT – may causes arrhythmias
  - Class I antiarrhythmic
- Calcium Channel Blockers
  - Verapamil or diltiazem
  - Used only for mild obstruction
  - Use if BB ineffective
  - ↓ LV wall tension
  - Negative inotrope
  - ↓HR
- Antiarrhythmic medications
  - Treat A fib and/or vent arrhythmias
  - Amiodarone or sotolol

Disopyramide may cause uncomfortable anticholinergic side effects and may enhance the hypoglycemic effect of gliclazide, insulin, and metformin.

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Hypertrophic Cardiomyopathy (HCM)

Medications CAUTION

- Diuretics
  - Give with caution as volume status is important

AVOID in HOCM

- Nitroglycerin
- Ace Inhibitors
- Positive inotropes
- Anything that ↑ contractility
- Nifedipine, amiodipine, felopine because of the vasodilatory effects

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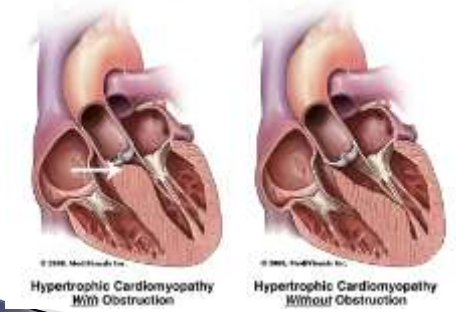
Diagnosis is Hypertrophic Obstructive Cardiomyopathy.



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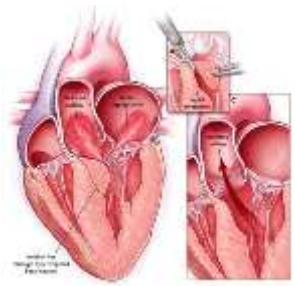
Septal Myomectomy



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Septal Myomectomy
Postop Management

- Watch for LBBB and CHB
  - 5-10% require permanent pacemaker
- Avoid hypovolemia
  - Immediate effects of hypovolemia are pronounced
  - Maintain stable HR and rhythm to maximize filling time
  - Avoid vasodilators to ↓ SVR
  - Use Volume and vasopressors
- Avoid inotropes

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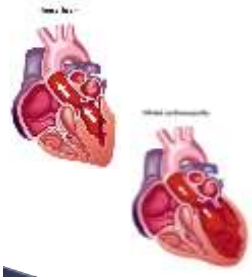
Dilated Cardiomyopathy



- Enlarged, dilated cardiac chamber
- Can affect one or all four chambers
- As chamber enlarges, its ability to contract becomes impaired, resulting in systolic dysfunction
- Most common cause of HF

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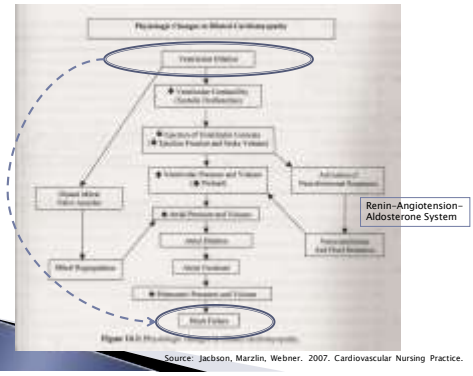
Physiologic Changes in Dilated Cardiomyopathy



Source: Jacbson, Marzlin, Webner. 2007. Cardiovascular Nursing Practice.

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Dilated Cardiomyopathy (DCM)

Clinical Presentation

- Directly related to the severity of the disease process
- Symptoms reflect inadequate CO and perfusion, fluid overload
- Left sided HF symptoms occur before right sided HF symptoms occur

- Hallmark signs
  - Weakness
  - Fatigue
  - Decreased activity tolerance
- Biventricular hypertrophy
- Biatrial enlargment
- Decreased EF
- S3, Systolic murmur

Symptoms develop slowly; patient adjusts ADLs without realizing it.

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Dilated Cardiomyopathy

Clinical Management

- Eliminate the cause
- Medical treatment same as for HF
- Symptom relief
  - Preload reduction
  - Afterload reduction
  - Increased contractility

- Five year survival = 50%

Eliminate the cause and symptom relief

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Dilated Cardiomyopathy

Causes

- Ischemic
- Nonischemic
- Idiopathic
- Valvular
- Genetic
- Hypertensive
- Myocarditis (Infection/Inflammatory)
- Chemotherapy induced
- Peripartum syndrome related to toxemia
- Cardiotoxic induced (effects of drugs or alcohol)
- Tachycardia induced

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Primary vs Secondary Dilated Cardiomyopathy

Primary	Secondary
Idiopathic Dilated Cardiomyopathy (IDC)	Ischemic Dilated Cardiomyopathy Hypertensive Dilated Cardiomyopathy Valvular Dilated Cardiomyopathy Anthracycline Dilated Cardiomyopathy Peripartium Dilated Cardiomyopathy Alcholol Dilated Cardiomyopathy

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Idiopathic Dilated Cardiomyopathy (IDC)

Causes	Treatment
50% of IDC is familial Suspected when other causes are excluded ◦ CAD ◦ Thyroid disease ◦ Valvular abnormalities ◦ Infiltrative causes ◦ Hypertension ◦ Alcohol	ACE inhibitors Beta blockers Anticoagulation is required due to the risk of thromboembolism Improvement of LV function is often better in IDC than in patients with IDM (ischemic dilated cardiomyopathy)

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Ischemic Dilated Cardiomyopathy (IDM)

Causes	Treatment
Most common type of dilated cardiomyopathy Occurs when CAD or ischemic heart disease causes remodeling of the LV with an associated reduction of EF Remodeling is the compensatory response of the ventricles to improve its function. ◦ Harms ventricular muscle ◦ Worsens stroke volume ◦ Develops ventricular dilation ◦ Decreases EF	ACE inhibitors Beta blockers Diuretics/spironolactone Anticoagulation is required due to the risk of thromboembolism ICD Amiodarone to prevent dysrhythmias Monitor electrolytes Prognosis is worse for IDM than nonischemic cardiomyopathy.

Treat ischemic disease – prevent remodeling with ACEI & BB

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Hypertensive Dilated Cardiomyopathy

Causes	Treatment
Diagnosed when systolic function remains depressed despite adequate treatment of hypertension Myocardial systolic function is depressed out of proportion to the increase in wall stress Prognosis is influenced by other comorbidities	Same as IDM (ischemic) Afterload reduction is the most important goal Antihypertensive vasodilators ◦ Amiodipine (Norvasc, besylate, mesylate or maleate) ◦ Hydralazine Alpha-blocking agents ◦ Alfuzosin (Uroxatral) ◦ Doxazosin (Cardura) ◦ Prazosin (Minipress) ◦ Terazosin ◦ Tamsulosin (Flomax)

Afterload reduction – Alpha-blocking agents

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Valvular Dilated Cardiomyopathy

Causes	Treatment
Myocardial systolic function is depressed out of proportion to the increase in wall stress secondary to valvular abnormalities Most caused by left sided valves ◦ MR & AR ◦ AS less common cause	Valve replacement or repair – improves wall stress but not depressed LVF ACEI & BB Aggressive afterload reduction ◦ Hydralazine ◦ Nitrates ▪ nitroglycerin, isosorbide dinitrate, isosorbide mononitrate. With AR- calcium channel blockers

Valve repair --Afterload reduction: hydralazine & nitrates

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Ischemic Cardiomyopathy

Case Study

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69-year-old female with an advanced cardiomyopathy and systolic heart failure, New York Heart Association class IIIB.

- The patient is short of breath on minimal exertion. She is maximally medicated with neurohormonal blockade, beta blocker, angiotensin receptor blocker, and spironolactone. Ejection fraction is still low.
- Biventricular AICD was implanted for resynchronization. In spite of it, she did not respond. Ejection fraction is still in the range of 20% to 25%.
- She is symptomatic with shortness of breath on minimal exertion. She comes in today to discuss different options managing her cardiomyopathy.
- Denies angina. Etiology of her cardiomyopathy is nonischemic. No coronary disease in her background.

- MEDICAL HISTORY: Significant for:
- Systolic heart failure.
- Biventricular AICD.
- Hypothyroidism, on replacement therapy.
- Hypertension.
- Hyperlipidemia.

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69-year-old lady with advanced heart failure, New York Heart Association class IV

- Debating whether or not she is a candidate for destination therapy or bridging to left ventricular assist device.
- At the age of 69, consider her not an ideal patient for heart replacement therapy
- Admitted for right heart study
  - Right atrial pressure was 4 mm
  - PA pressure mean of 20 mmHg
  - mixed venous saturation 73%.
  - Mean pulmonary capillary wedge pressure was 13
  - Cardiac output was 4.2
  - Pulmonary vascular resistance is under 2 Wood units which is excellent.
- The patient was admitted to the CVICU for initiation of intravenous milrinone at 0.375 mcg/kg/minute.

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Hemodynamic values in CVICU prior to Milrinone

- 5/2 0800
- CVP 3
- CO
- CI 1.3
- SV 32
- SVR 2150
- Wt 104 kg
- Ht 69”

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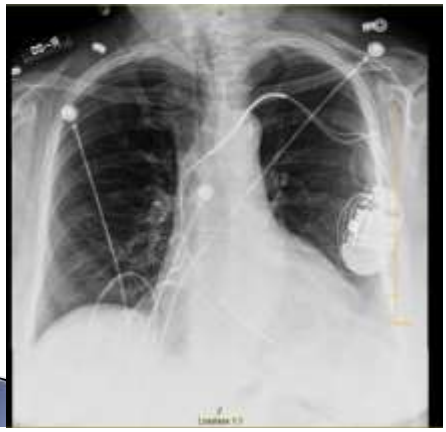

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	RHC	Prior to Milrinone	8 hours on Milrinone	24 hours on Milrinone
BP			73/40	94/65
HR			106	85
PAS/PAD			23/10	20/10
PAP mean	20		15	14
PAOP	13		3	0
CVP	4			
CO	4.2		3.6	5.4
CI		1.3	1.8	2.5
SVR		2150	1279	1084
SV		32		45
SVO2	73			
PVR	< 2 woods			
Milrinone		0.375 mcg/kg/min	↓ 0.25 mcg/kg/min	0.25 mcg/kg/min
			Fluids @ 50ml/hr	

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Peripartum Dilated Cardiomyopathy

Causes	Treatment
▸ Occurs when myocardial systolic dysfunction occurs during the last trimester of pregnancy or within 6 months of childbearing. ▸ Outcomes are better with peripartum than with other dilated cardiomyopathies	▸ Treatment is aggressive & consistent with IDC (idiopathic) ▸ 50% will recover completely ▸ Small minority will need transplant

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Peripartum Cardiomyopathy (PPCM)

Also called pregnancy associated cardiomyopathy

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Peripartum Cardiomyopathy (PPCM)

5th leading cause of mortality during the pregnancy period

Source: Moser & Riegel. 2008. *Cardiac Nursing.* And Tsang, W. Peripartum Cardiomyopathy:.Retrieved 2-9-15 from Up To Date

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Peripartum Cardiomyopathy (PPCM)

1. Cardiomyopathy in the last month or the first five months after pregnancy
2. Absence of another identifiable cause of HF
3. EF < 45%
   - LV may or may not be dilated

Source: Moser & Riegel. 2008. *Cardiac Nursing.* And Tsang, W. Peripartum Cardiomyopathy:.Retrieved 2-9-15 from Up To Date

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Peripartum Cardiomyopathy (PPCM)

- Incidence per live births
  - 1:4350 USA– 10 years ago
  - 1:2399 USA – 2011
    - ↑ maternal age, ↑ multifetal pg, ↑ recognition PPCM
  - 1:1000 South Africa
  - 1:300 Haiti
  - 1:100 Nigeria
- Cause – Unknown
- Usually occurs with first or second pregnancy

Source: Moser & Riegel. 2008. *Cardiac Nursing:* Tsang, W. Peripartum Cardiomyopathy:.Retrieved 2-9-15 from Up To Date: Sundin. C 2014. Peripartum Cardiomyopathy. *MCN* 39(4)

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Pregnancy

- High output state
- 30% decrease in systemic vascular resistance
- 30–40% increase in cardiac output by 2nd and 3rd trimester
- Changes may not resolve completely until 12 weeks postpartum

Source: Garg, J et al. 2015. Peripartum Cardiomyopathy. *Cardiology in Review;* 23(2).

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PPCM Risk Factors

- Advancing maternal age > 30 years
  - Extreme age (very young or advanced age)
- African descent
- Multi-fetal gestation
- History of preeclampsia, eclampsia, postpartum hypertension
- Long term (> 4 weeks) use of beta adrenergic agonists (terbutaline) for preterm labor suppression
  - Tocolytic agents used > 4 weeks have higher incidence of pulmonary edema (terbutaline, salbutamol, ritodrine, and magnesium sulfate)
- Maternal cocaine abuse

Source: Moser & Riegel. 2008. *Cardiac Nursing.* And Tsang, W. Peripartum Cardiomyopathy:.Retrieved 2-9-15 from Up To Date
Garg, J et al. 2015. Peripartum Cardiomyopathy. *Cardiology in Review.* 23(2).

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PPCM signs and symptoms

Similar to other forms of systolic HF

- Dyspnea – most common
- Tachycardia
  - Early sign
- Cough
- Orthopnea
- Paroxysmal nocturnal dyspnea (PND)
- Pedal edema
- Nonspecific fatigue
- Hemoptysis

High clinical suspicion

- Elevated jugular venous pressure
- Displaced apical impulse
- S3
- Murmur from tricuspid or mitral regurgitation

Source: Tsang,W. Peripartum Cardiomyopathy:. Retrieved 2-9-15 from Up To Date
Garg, J et al. 2015. Peripartum Cardiomyopathy. *Cardiology in Review*; 23(2).

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PPCM symptoms

- Often missed or delayed
- Similar signs and symptoms of normal pregnancy

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Comparison of Symptoms

Preeclampsia	Similar Symptoms	PPCM
Edema Proteinuria Headache Blurred vision Decreased renal perfusion Hyperactive DTRs Impaired hepatic function	Weight gain Tachycardia Adventitious breath sounds Hypertension Fatigue	Dyspnea Cough Chest pain Palpitations Third heart sound S3 Jugular venous distension Tachypnea Murmur

Sundin. C 2014. Peripartum Cardiomyopathy. *MCN* 39(4)

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Early Recognition is Key!

- For patients who develop
  - Dyspnea
  - Increasing blood pressure
  - Increased edema or edema that doesn't decrease
  - Chest pain
  - Tachycardia
  - Nonproductive cough
- Detailed, serial, frequent nursing assessments with accurate documentation
- Comprehensive and thorough communication with provider
- BNP, Echo
- Cardiology consult

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PCCM Diagnosis

Three Clinical criteria

1. Development of HF end of pregnancy or first 5 months post delivery
2. Absence of other identifiable cause of HF
3. EF < 45%

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PPCM Prognosis

- Related to the severity of cardiac dysfunction and return of ventricular function
- Outcomes are better with peripartum than with other dilated cardiomyopathies
- 50% will recover completely
- Small minority will need transplant

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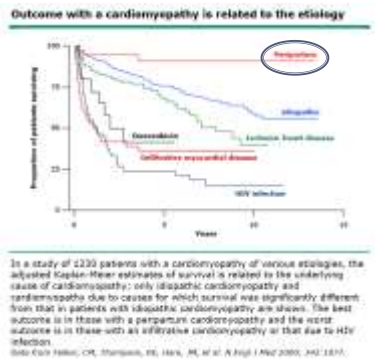

In a study of 1230 patients with a cardiomyopathy of various etiologies, the adjusted Kaplan-Meier estimates of survival is related to the underlying cause of cardiomyopathy; only idiopathic cardiomyopathy and cardiomyopathy due to causes for which survival was significantly different from that in patients with idiopathic cardiomyopathy are shown. The best outcome is in those with a peripartum cardiomyopathy and the worst outcome is in those with an infiltrative cardiomyopathy or that due to HIV infection.

Source: Tsang, W. Peripartum Cardiomyopathy: Treatment and Prognosis. Retrieved Feb 9, 2015 from Up To Date

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Alcohol-Related Dilated Cardiomyopathy

Causes	Treatment
▸ Diagnosed when there is a history of sustained and heavy alcohol consumption and other causes of dilated cardiomyopathy are excluded. ▸ Toxic effects of alcohol are thought to cause the nonspecific changes in the myocardium ▸ Thiamine deficiencies can compromise cardiac function	▸ Alcohol abstinence ▸ Same as for IDC (idiopathic) ▸ Prognosis is somewhat better than for IDC depending on the degree of myocardial impairment and alcohol abstinence

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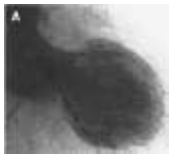
Stressed Induced Cardiomyopathy

- Broken Heart Syndrome
- Takotsubo Cardiomyopathy
- A specific syndrome of stress-related reversible cardiomyopathy
- Mimics acute myocardial infarction without obstructive disease



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Case Study

- 69 y/o female comes to ED with c/o of severe chest discomfort
- PMH: mild HTN and hyperlipidemia
- B/P 173/89, HR 91, RR 21
  SpO_2 98% on 2 l/np

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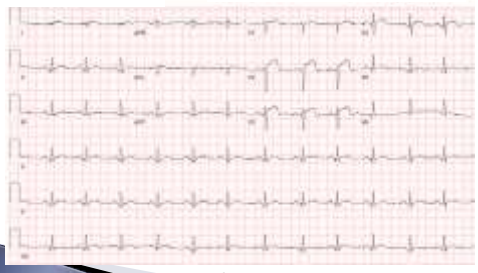
EKG on admission



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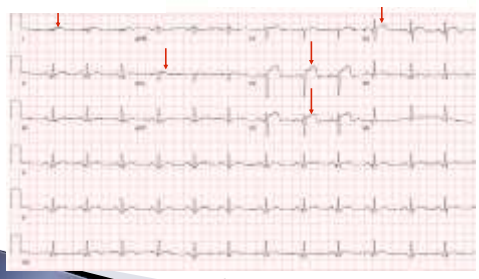
EKG on admission



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- Rural hospital with no cath lab
- NTG 0.4 mg SL x 3 in 30 minutes
- ASA 81 mg po
- Metoprolol 25 mg po
- Retavase

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More history….

- A few hours earlier in the same ED, her husband came in full arrest and was not able to be resuscitated

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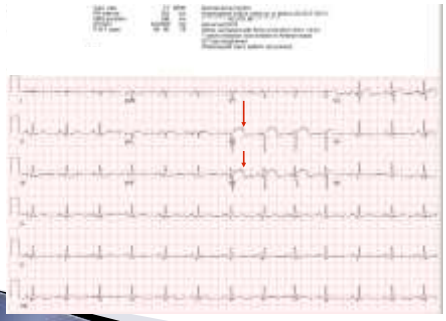
No relief of symptoms… Repeat EKG
No improvement



Transported via helicopter to hospital with cardiac cath

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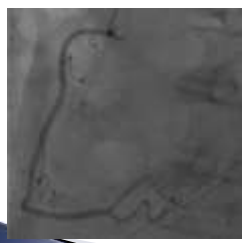
Labs on admission

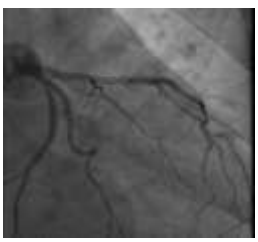
- CK = 156
- CKMB = 10.7 ↑
- Myoglobin = 298 ↑
- Troponin I = 2.91 ↑
- BNP = 35

70

Cardiac Cath findings

Normal coronary anatomy – No CAD





71

Cardiac Cath findings

- Markedly depressed LV function with ejection fraction = 5 – 10%
- Severe hypokinesis to akinesis of the distal 2/3 anterolateral, apical, and inferior walls.
- The basal segments contract vigorously giving it very Japanese amphora shape suggestive of Takotsubo cardiomyopathy

72

Management

- Transferred to CVICU
- No IABP due to hemodynamically stable and recent Retavase
- Diagnosis: Broken Heart Syndrome or Takatsubo cardiomyopathy

73

Discharged the next day so she could attend her husband's funeral

- Discharge medications
- Aldactone 25 mg every day
- Alprazolam 0.5 mg prn
- Altace 2.5 mg every day
- ASA 81 mg every day
- Coreg 6.35 mg every 12 hours
- Coumadin 5 mg po every day
- Lasix 20 mg every other day
- Lipitor 40 mg po at hs

74

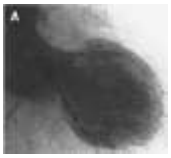
6 weeks later

- EF 60%
- Patient doing well

75

Stressed Induced Cardiomyopathy

- Broken Heart Syndrome
- Takotsubo Cardiomyopathy
- A specific syndrome of stress-related reversible cardiomyopathy
- Mimics acute myocardial infarction without obstructive disease



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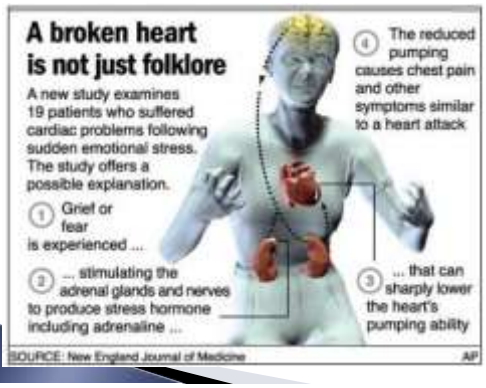

77

Precipitating factors

Marked psychosocial or physical stress

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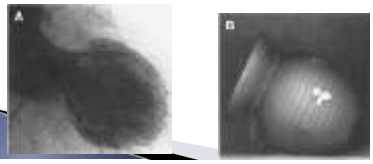
Original name given "Takotsubo Cardiomyopathy"

- Takotsubo is the narrow-necked bulging container used by Japanese fisherman to trap octopus
- The shape of the takotsubo pot resembles the distorted ballooning ventricle.
- Also called: Transient Left Ventricular Apical Ballooning



79

Etiology

- Unclear etiology
- 1 – 2% of patients who have S/S AMI have apical ballooning (Japan & USA)
- 6-9 times more common in women
- 6% of women with AMI have apical ballooning
- Most often in postmenopausal women

80

Takotsubo Cardiomyopathy

Pathophysiology

- Marked systolic ballooning of the ventricular apex
- Hypercontractility of the base of the heart
- Now thought to be related to stunning of the myocardium related to excessive catecholamines
- Since preceded by increased psychosocial or physical stress suggest an association with ↑ SNS activity
- Catecholamines have a toxic effect on the myocardium

Catecholamine levels reported to be 7 – 34 times as high as the normal 2 – 3 elevation in classic AMI patients

81

Takotsubo Cardiomyopathy

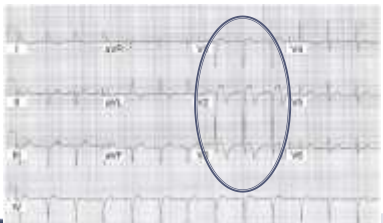
Clinical Presentation

- Chest pain
- ST segment changes
- Cardiac biomarkers
  - Only moderately elevated
  - Do not follow the typical rise-fall-pattern seen with AMI

- 12 Lead EKG Variable findings
  - ST segment elevation or depression usually in the precordial leads (V2 – V5)
  - Reciprocal changes in the inferior leads may not occur
  - Q waves usually do not develop or Q waves V3 – V6
  - Deeply inverted T waves are common in the recovery period
  - Markedly prolonged QT interval

82

A 12-lead electrocardiogram showing ST-segment elevations and T-wave inversions in the right precordial leads, which is a typical pattern observed in Takotsubo cardiomyopathy



Metzl MD *et al.* (2006) A case of Takotsubo cardiomyopathy mimicking an acute coronary syndrome *Nat Clin Pract Cardiovasc Med* **3:** 53–56 doi:10.1038/ncpcardio0414

83

Takotsubo Cardiomyopathy

Diagnosis

Cardiac Cath/Echo

- Systolic ballooning of the ventricle, akinetic or dyskinetic left ventricle
- Ejection fraction markedly decreased in the acute phase – as low as 14 – 40%
- No significant coronary artery disease to account for the marked left ventricular dysfunction

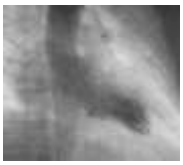
- Nuclear stress testing
  - Evidence of **reversible** myocardial injury

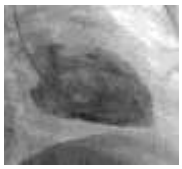
Low EF – no CAD – precipitating stressor

84

Normal LV on angiogram

- Systole
- Diastole

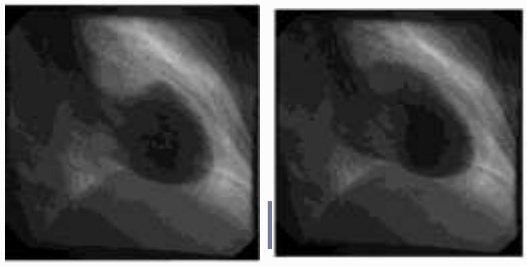




85

Left ventriculogram in systole (3a) and diastole (3b) to illustrate the ballooning

- Systole
- Diastole



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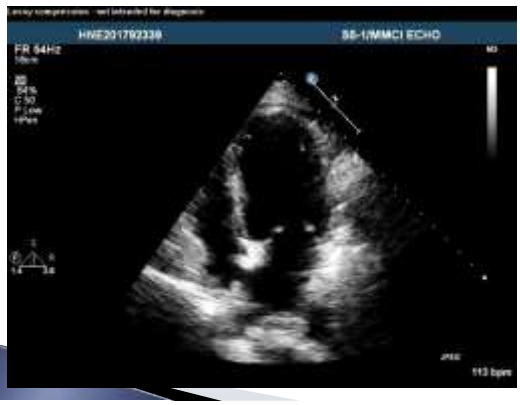

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Takotsubo Cardiomyopathy

Diagnosis

- Immediately difficult to differentiate between STEMI caused by thrombosis
- Suspect Takotsubo Cardiomyopathy when obstructive CAD is not present to explain the LV dysfunction
- Confirmation of diagnosis: typical octopus morphology of LV
- Stressor considered supportive evidence
- Complete resolution of LV dysfunction weeks after the event

Low EF – no CAD – precipitating stressor – octopus morphology

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Takotsubo Cardiomyopathy

Clinical Management

- Prompt recognition of apical ballooning prevents unnecessary administration of fibrinolytics with the ST segment elevation
- Specific guidelines do not exist -- Mostly managed per NSTEMI and STEMI guidelines
- Proceed with STEMI treatment & emergent cardiac cath
- Management of cardiogenic shock
  - Vasopressors
  - Pacemaker
  - Intraaortic balloon pump (IABP)
  - Support until LV recovers
- Supportive management
  - Arrhythmias → antiarrhythmic drugs
  - Diuretics → pulmonary congestion
  - B Blockers, vasodilators, ACEI, vasocontrictors, IABP → left sided HF
  - Short term anticoagulant → prevent LV thrombus

No fibrinolytics --- Supportive management

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Takotsubo Cardiomyopathy

Prognosis

- Left ventricular function improves rapidly
- Often within 7 – 30 days
- EKG changes may be slower to resolve
- Generally favorable prognosis
- Mortality of 0 – 8%

LV function recovers

90

Restrictive Cardiomyopathy (RC)

- Restrictive filling and reduced diastolic volume of either or both ventricles
- Least common cardiomyopathy (5%)

91

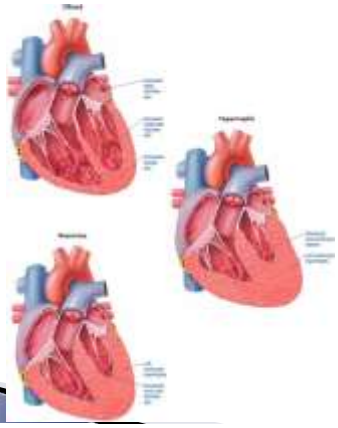
Restrictive Cardiomyopathy (RC)

Pathophysiology

1. Disease process causes noncompliant ventricles
2. Noncompliant ventricles resist ventricular filling
3. ↓ diastolic filling = ↓ blood volume = ↓ SV
4. Results in ↑ blood volume and pressure in atria
5. Blood backs up into the lungs (left sided failure) and then in the venous circulation (right sided failure)
6. Disease process affects both ventricles
7. Ventricular size usually normal or slightly decreased
8. Systolic function is not usually affected

Heart becomes noncompliant and cannot stretch and fill

92



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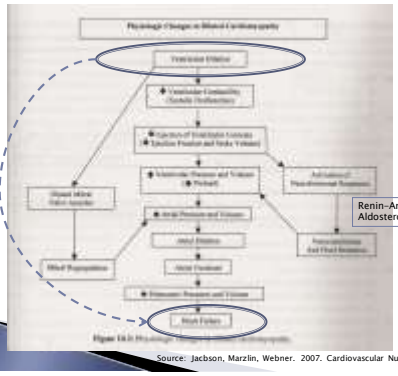
Physiologic Changes in Dilated Cardiomyopathy



Source: Jacbson, Marzlin, Webner. 2007. Cardiovascular Nursing Practice.

94

Heart Failure

- Syndrome preceded by an initiating cardiovascular event (MI, hypertension, etc)
- On the cardiac continuum HF is an end event – represents the most severe manifestation of cardiovascular disease

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Heart Failure

Characteristics

1. Arises from alterations in systolic and diastolic dysfunction
   - Systolic Dysfunction
   - Diastolic Dysfunction
2. Systolic and Diastolic Dysfunction are progressive syndromes that develop over the course of many years
3. Heart Failure preferred term
   - Not all HF patients (especially those with diastolic dysfunction) exhibits symptoms of congestion

HF is a progressive syndrome --- develops over many years

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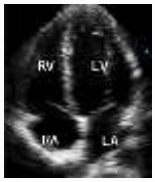
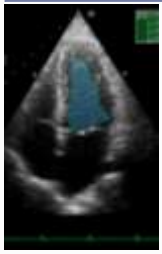
Normal Echocardiogram



Diastole

Systole



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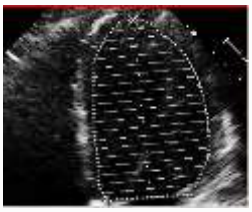
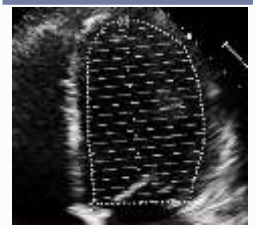
EF 5- 10%

Diastole



Systole



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Acute Exacerbation vs Chronic HF

Acute HF	Chronic HF
• Also called: Decompensated HF • New or worsening signs and symptoms of the HF syndrome • Frequently leads to ED visits or hospitalization • May also be: Sudden onset of HF signs and symptoms that occur in patients with no previous HF history	• Denotes the slow progression and continuance of the HF syndrome • Chronic HF patients frequently experience "*exacerbations of HF*" also known as acute HF or decompensated HF

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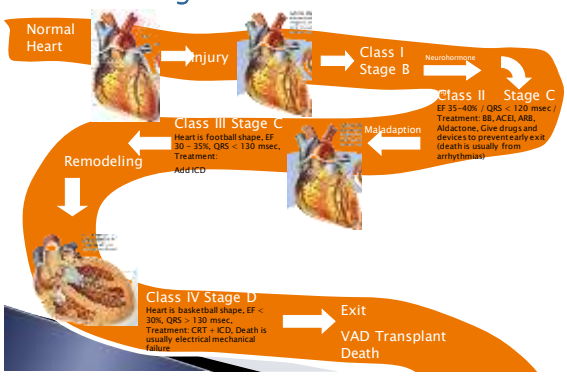
Clinical Progression of Heart Failure



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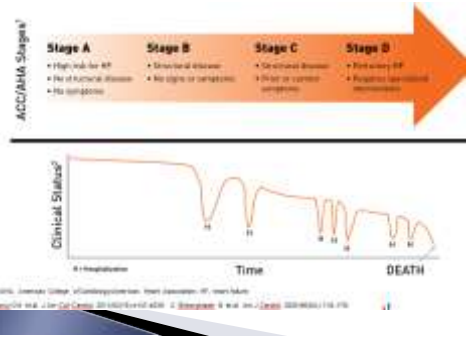
HF progression

~50% mortality within 5 Years of Diagnosis



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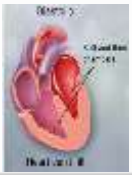
Systolic vs Diastolic HF

Systolic HF – HFrEF (reduced)	Diastolic HF-HFpEF (preserved)
• Inability of LV to contract against a load and eject blood into the aorta • Hallmark signs: ◦ **Reduced ejection fraction (EF)** ◦ Reduced stroke volume	• Abnormalities of diastolic filling or relaxation of the LV • Hallmarks signs ◦ **Normal EF** ◦ Abnormal diastolic function



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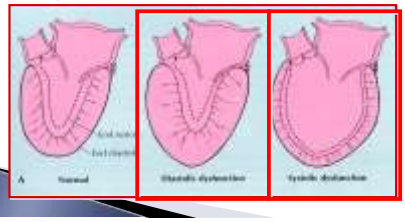
Types of heart failure

Diastolic Dysfunction
- Heart failure symptoms with EF ≥ 40%
- Hypertrophic CMP, HTN, Ischemia, Age
- Imbalance in volume/ pressure relationship

Systolic Dysfunction
- Depressed contractility EF ≤ 40%
- CAD, Valve disease, Ischemic and Idiopathic CMP



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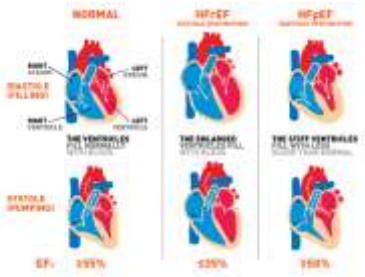
HFrEF vs HFpEF



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Systolic Dysfunction (HFrEF)

Pathophysiology

1. LV wall thins and the cavity dilates (eccentric hypertrophy)
2. Thin, dilated ventricle unable to contract effectively
3. EF decreases
4. Leads to ↓ CO, ↑ LVEDV, ↑ preload → pulmonary congestion
5. Dilated cardiomyopathy common cause of systolic HF
6. Cardiomyopathy and systolic dysfunction should NOT be used interchangeably

EF < 40% systolic dysfunction

105

Diastolic Dysfunction (HFpEF)

Pathophysiology

1. Ventricular muscle thickens (concentric hypertrophy)
2. Ventricular cavity size may remain normal or become smaller
3. Noncompliant ventricle unable to relax, impairing filling
4. To ↑ filling, left atrial pressure ↑; leading to pulmonary congestion

EF normal in diastolic dysfunction

106

Diastolic Dysfunction

Clinical Presentation

- Symptomatic with exertion and ↑ HR
  - Faster HR ↓ filling time & ↓ CO
  - Exercise → ↑ catecholamines → ↑ HR → worsens diastolic function
  - Flash pulmonary edema can develop during periods of ischemia

Elevated heart rate worsens diastolic function

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Diastolic Dysfunction

Diagnosis

- Three conditions required:
  1. Signs and symptoms of HF
  2. Normal or only slightly decreased EF
  3. Increased diastolic filling pressures and abnormal relaxation of LF
- Diagnosis made in patients presenting with the clinical syndrome of HF with no evidence of systolic dysfunction

Commonly associated with chronic hypertension or ischemic heart disease

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New York Heart Association (NYHA) Classification of Heart Failure

Class	Patient Symptoms
Class I (Mild)	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, rapid/irregular heartbeat (palpitation) or shortness of breath (dyspnea).
Class II (Mild)	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, rapid/irregular heartbeat (palpitation) or shortness of breath (dyspnea).
Class III (Moderate)	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, rapid/irregular heartbeat (palpitation) or shortness of breath (dyspnea).
Class IV (Severe)	Unable to carry out any physical activity without discomfort. Symptoms of fatigue, rapid/irregular heartbeat (palpitation) or shortness of breath (dyspnea) are present at rest. If any physical activity is undertaken, discomfort increases.

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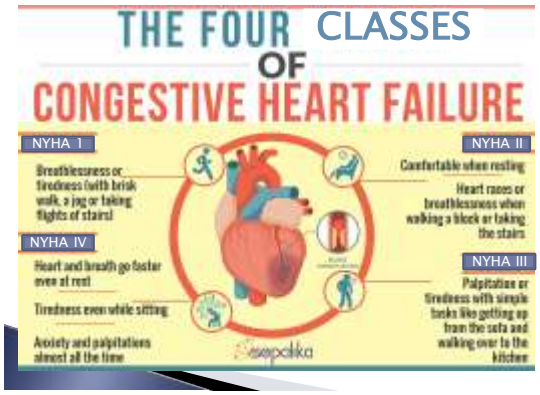

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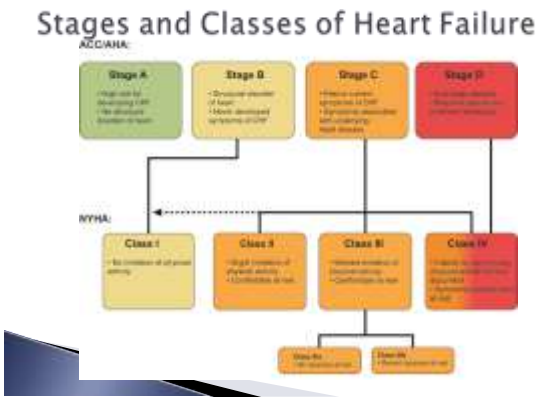

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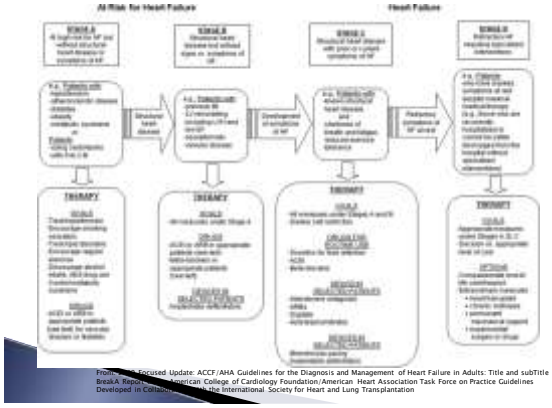

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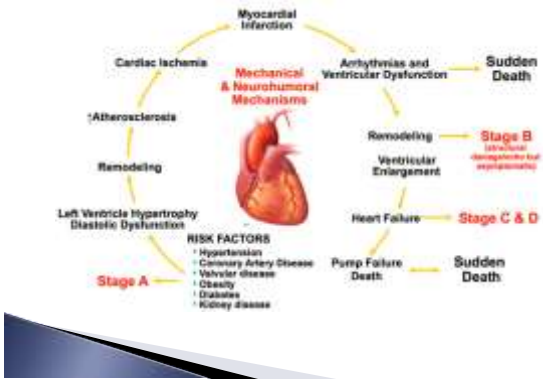

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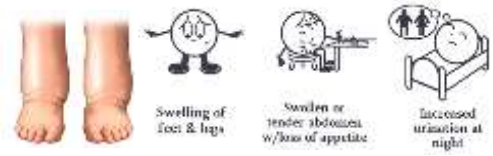
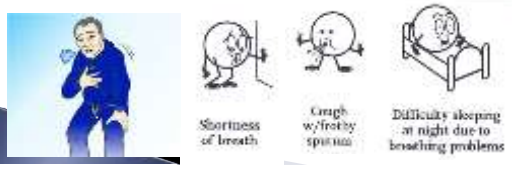
Right sided symptoms – think Circulation



Left sided symptoms – think Lungs



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Assessment Tips

Source: https://www.emergencynp.com/

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Right Ventricular Failure

Causes

- Usually results from prolonged LV failure
- Right ventricular MI or inferior wall MI
- Primary pulmonary hypertension
- Acute or chronic lung disease
- Chronic severe tricuspid regurgitation

117

Your patient has an

S 3

118

Neurohormonal Responses in HF

1. **Activation of Sympathetic Nervous System (SNS)**
2. **Renin–Angiotensin–Aldosterone System (RAAS) Kicks in…**

119

Chronic Compensation for Decreased Cardiac Output worsens HF

120

SNS Activation

↓ Cardiac Output
↓
Activates SNS
↓

Compensates by releasing neurohormones (Norepinephrine, Aldostrone, Vasopression)
↓
Sodium & Water Retention
↓
Congestion

↓

Vasoconstriction
↑ Afterload

Tachycardia
↓
↑ Myocardial Oxygen Time
↓ Diastolic Filling Time

121

SNS Activation

↓ Cardiac Output
↓
Activates SNS
↓

Block SNS
Vasodilate, ↓ HR, ↓arrhythmias
EBP HF Beta Blockers
- Carvedilol, Metoprolol Succinate, Bisoprolol

Compensates by releasing neurohormones (Norepinephrine, Aldosterone, Vasopression)
↓
Sodium & Water Retention
↓
Congestion

↓

Vasoconstriction
↑ Afterload

Tachycardia
↓
↑ Myocardial Oxygen Time
↓ Diastolic Filling Time

Diuresis and ↑ K
Anti-mineralocorticoid
Mineral Receptor Antagonists
Aldosterone Antagonists
Spironolactone

122

Renin-Angiotensin-Aldosterone System (RAAS)

Low Cardiac Output/Hypotension/Hypovolemia
Decreased Renal perfusion
↓
Afferent Arteriole (baroreceptors)
↓
Release Renin (a messenger)
↓
Go to Liver to stimulate Angiotensin I production
↓
Angiotensin I goes to the Lung
↓
Angiotension Converting Enzyme (ACE) located in the pulmonary vascular membrane
↓
Converts Angiotensin I to Angiotensin II
↓
Angiotensin II

↓ Growth Factor
↓ Potent Vasoconstrictor
↓ Increases B/P
↓ Increases SVR

↓ Adrenal Cortex
↓ Aldosterone
↓ Distal Renal Tubule
↓ Increases H2O & Na++ Reabsorption
↓ Excretes K+ for Na+

123

Renin-Angiotensin-Aldosterone System (RAAS)

Low Cardiac Output/Hypotension/Hypovolemia
Decreased Renal perfusion
↓
Afferent Arteriole (baroreceptors)
↓
Release Renin (a messenger)
↓
Go to Liver to stimulate Angiotensin I production
↓
Angiotensin I goes to the Lung
↓
Angiotension Converting Enzyme (ACE) located in the pulmonary vascular membrane
↓
Converts Angiotensin I to Angiotensin II
X
↓
Angiotensin II
X

↓ Growth Factor
↓ Potent Vasoconstrictor
↓ Increases B/P
↓ Increases SVR

↓ Adrenal Cortex
↓ Aldosterone
X
↓ Distal Renal Tubule
↓ Increases H2O & Na++ Reabsorption
↓ Excretes K+ for Na+

Diuresis and ↑ K
Anti-mineralocorticoid
Mineral Receptor Antagonist
Aldosterone Antagonist
Spironolactone

Dilatation & Diuresis
ACE Inhibitors
"Prils"
Angiotensin II receptor Blockers
"ARBs" "Sartans"

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Effect of Neurohormonal Response

SNS Activation
+
RASS Activation
→
Ventricular Remodeling

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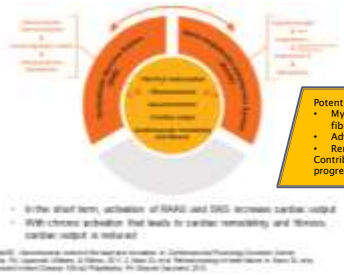
SNS and RASS Chronically Activated in HF

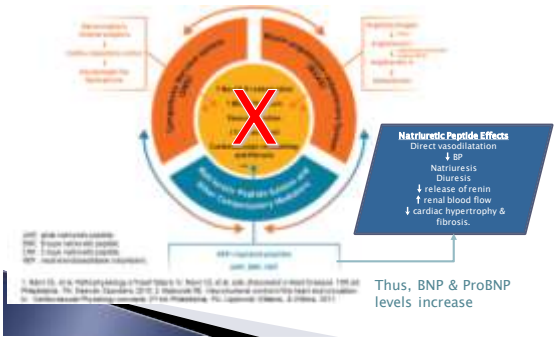


Potent stimulants for
- Myocardial hypertrophy & fibrosis
- Adverse vascular remodeling
- Renal parenchymal fibrosis

Contribute to heart failure disease progression.

126

Natriuretic Peptides Provide a Counter-Regulatory Neurohormonal Balance



Thus, BNP & ProBNP levels increase

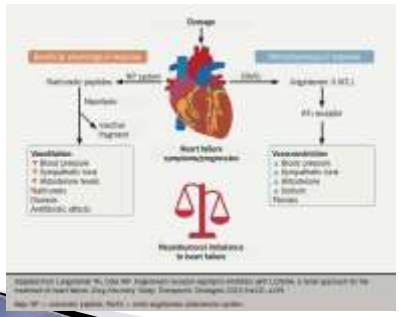
127

Overtime....
Blunted Natriuretic Peptide Response Causes a Neurohormonal Imbalance

Neprilysin, an enzyme, degrades and blunts natriuretic peptides further increasing the imbalance

128

Heart Failure
A State of Neurohormonal Imbalance



Source: https://bjcardio.co.uk/2016/06/neurohumoral-activation-in-heart-failure-and-the-implications-for-treatment/

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- BNP responds to counteract RASS
- Neprilysin blocks BNP
- ARNI blocks Neprilysin and lets BNP do it's job

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ARNI
Angiotensin Receptor Neprilysin Inhibitor

NEW KIDS ON THE BLOCK

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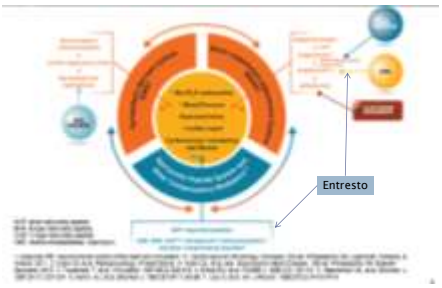
Neurohormonal Blockade Treatment for HF



Heart Failure
A state of neurohormonal imbalance

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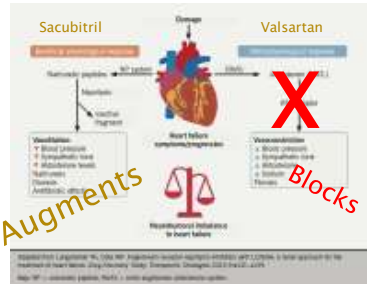
Angiotensin-receptor-neprilysin inhibitors (ARNIs)

Enteresto

ARB (Valsartan)
+
Neprilysin Inhibitor (Sacubitril)



Source: https://bjcardio.co.uk/2016/06/neurohumoral-activation-in-heart-failure-and-the-implications-for-treatment/

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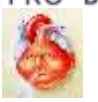
NT proBNP (N-TERMINAL PRO-B)



- Elevates with Heart Failure
- Not interchangeable with BNP
- proBNP is measured before it is metabolized into BNP (Hence proBNP)
- 5:1 ratio to BNP
- proBNP divided by 5 gives a number similar to the BNP values
  - For example: pro BNP of 500/5 = 100
- proBNP < 450, probably not Heart Failure
- If age over 50, need to look at specific ranges
  - 50 – 75 years proBNP > 900pg/mL consistent with HF
  - > 75 years proBNP > 1800pg/mL consistent with HF

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Heart Failure

Pharmacological Treatment

- Three primary goals of medical treatment
1. Reduce preload
2. Reduce afterload
3. Increase contractility

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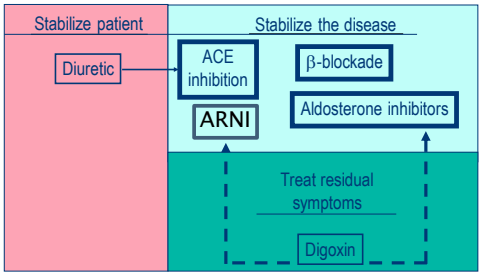
Paradigm for Outpatient Management of Heart Failure



Adapted from Packer M, Cohn JN, eds. Am J Cardiol. 1999;83(Suppl 2A).

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Reduce Preload

- Diuretics
- Venous Vasodilators
  - ACE (Angiotension-converting enzyme) Inhibitor
    - Blocks the RAAS effect of reaborption of sodium and water and thus decreases volume overload
  - Aldosterone antagonists
    - Spironolactone & eplerenone
    - Added to increase diuretic effect if symptoms at rest
  - Nitrates
    - Dilates veins, allowing more blood to remain in the vascular system and sending less to the heart

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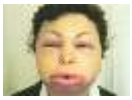
Reduce Afterload

- Arterial Vasodilators
  - ACE (Angiotensin-converting enzyme) Inhibitor
    - Blocks the RAAS effect of vasoconstriction
    - Enhances the action of kinins, which promotes a positive vasodilatory effect
    - Slows disease progression
  - Angiotensin Receptor Blockers
    - Directly blocks angiotensin II – results vasodilatory effect
    - Used if patient cannot tolerate ACEI due to cough or angioedema
  - ARNI (Angiotensin-Receptor-Neprilysin Inhibitor)
  - Hydralazine and Nitrate Combination
    - Hydralazine – arterial vasodilator
      - Recommended in African Americans with systolic dysfunction
    - Oral nitrates are venous vasodilators (preload reduction)
- Milrinone (Primacor) Phosphodiesterase inhibitor
  - Causes vasodilation to ↓ preload and afterload



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Beta Blockers

- Blocks the neurohormonal response of chronic SNS stimulation
- Slows heart rate for better diastolic ventricular filling
- Not initiated when fluid overload or in a decompensated state
- Initiate after fluid status optimized (no longer needing IV diuretics or IV vasodilators)
- Reduces arrhythmias
- Slows disease progression
- Carvedilol (Coreg), Metoprolol Succinate (Toprol XL), and Bisoprolol (Zebeta)

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Increase Contractility

- Increase contractility by first ↓ afterload
- Digoxin
  - Oral medicine of choice to assist with contractility
  - Enhances inotropy of cardiac muscle
  - Reduces activation of SNS and RAAS
  - Most effective in patients with low EF
- Dobutamine
  - ↑ contractility by stimulating beta receptors
- Milrinone (Primacor) Phosphodiesterase inhibitor
  - Increases calcium ion uptake.
  - Has positive inotropic effect

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ß-Blockers

Limit the donkey's speed, thus saving energy

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Beta Blocker "Olols"

Beta Blockade of the Sympathetic Nervous System

- Decrease oxygen demand
  - ↓ HR & contractility
  - Vasodilate
  - ↓ Afterload
  - ↓ O_2 wasteage
- Antiarrhythmic effect
- Increase oxygen supply
  - Increased diastolic perfusion
  - Less exercise vasocontriction

Side effect: May promote spasm in vasospastic angina

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Beta Blockers "Olols"

•Acebutolol	Sectral
•Atenolol	Tenormin
•Betaxolol	Kerlone
•Bisoprolol	Zebeta
•Carvedilol	Coreg
•Metoprolol tartrate	Lopressor
•Metoprolol succinate	Toprol XL
•Nadolol	Corgard
•Pindolol	Visken
•Propanolol	Inderal
•Timolol	Blocadren

EBP HF Beta Blockers
- Carvedilol, Metoprolol Succinate, Bisoprolol

- Toprol XL has less effect on BP and better for lungs than Coreg.
- Use Coreg if BP elevated, works better for hypertension than Toprol XL
- Okay to use Toprol XL if BP < 120

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Beta Blockers

Side Effects	Contraindications
• Bradycardia • Hypotension • Erectile Dysfunction • Fatigue	• Signs of heart failure • Low output state • Risk of cardiogenic shock • Hypotension • Bradycardia • PR interval >0.24 • Second or third-degree heart block without a pacemaker • Active asthma or reactive airway disease

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ACE Inhibitors/ARBs

Reduce the number of sacks on the wagon

RAAS Blockers Dilation & Diuresis

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ACE Inhibitors "Prils"

- •Benazepril Lotensin
- •Captopril Capoten
- •Lisinopril Zestril
  Prinivil
- •Quinapril Accupril
- •Ramipril Altace

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ARBs "Sartans"

- •Candesartan Atacand
- •Irbesartan Avapro
- •Losartan Cozaar
- •Valsartan Diovan
- •Telmisartan Micardis
- •Eprosartan Teveten

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ACEI & ARB Side Effects

- Hypotension
- Increase potassium
- Affect kidneys
  - Increase Creatinine
  - Decrease GFR
- Cough (ACEI)
- Angioedema

Contraindications

- Bilateral renal artery stenosis
- Creatinine > 3 mg /dL in CKD
- AKI (until resolved)
- Potassium > 5.0 mEq/L
- Systolic BP < 80 mmHg

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Starting and Target Doses of Select Guideline-Directed Medical Therapy for HF

Start LOW, Go SLOW To titrate to EBP TARGET

Source: 2017 ACC Expert Consensus Decision Pathway for Optimization of Heart Failure Treatment: Answers to 10 Pivotal Issues About Heart Failure With Reduced Ejection Fraction Journal of the American College of Cardiology Volume 71, Issue 2, January 2018

	Starting dose	Target dose
Beta Blockers		
Bisoprolol	1.25 mg once daily	10 mg once daily
Carvedilol	3.125 mg twice daily	25 mg twice daily for weight <85 kg and 50 mg twice daily for weight ≥85 kg
Metoprolol succinate	12.5–25 mg/d	200 mg daily
ARNI		
Sacubitril/valsartan	24/26 mg–49/51 mg twice daily	97/103 mg twice daily
ACEI		
Captopril	6.25 mg 3× daily	50 mg 3x daily
Enalapril	2.5 mg twice daily	10–20 mg twice daily
Lisinopril	2.5–5 mg daily	20–40 mg daily
Ramipril	1.25 mg daily	10 mg daily
ARB		
Candesartan	4–8 mg daily	32 mg daily
Losartan	25–50 mg daily	150 mg daily
Valsartan	40 mg twice daily	160 mg twice daily
Aldosterone antagonists		
Eplerenone	25 mg daily	50 mg daily
Spironolactone	12.5–25 mg daily	25–50 mg daily
Vasodilators		
Hydralazine	25 mg 3× daily	75 mg 3× daily
Isosorbide dinitrate	20 mg 3× daily	40 mg 3× daily
Fixed-dose combination isosorbide dinitrate/hydralazine†	20 mg/37.5 mg (one tab) 3× daily	2 tabs 3× daily
Ivabradine		
Ivabradine	2.5–5 mg twice daily	Titrate to heart rate 50–60 bpm. Maximum dose 7.5 mg twice daily

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Indications other HF medications

- **Vasodilators**
  - Hydralazine
  - Isosorbide dinitrate
  - Use in patients who cannot be on ACEI/ARB/ARNI and/or AA patient use nitrates and hydralazine as combination therapy
- **Angiotensin receptor-Neprilysin inhibitor (ARNI)**
  - Sacubitril/Valsartan – Entresto
  - Indication to reduce death and hospitalization in patients with chronic HF (NYHA II – IV) and reduced EF.
  - Used in place of ACEI/ARB, Needs 36 hour washout after ACEI to prevent angioedema
- HCN Channel Inhibitor
  - Ivabradine (Corlanor)
  - Indication to reduce the risk of hospitalization for worsening HF in patients with stable, symptomatic chronic HF with LVEf $\leq$ 35% and HR > 70 in NSR, on max tolerated BB. SA nodal blocking agent. Increased prevalence of afib

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Diuretics

Drug	Dose	Adverse Effects
Loop Diuretics		• Electrolyte abnormalities – hypokalemia, hypomagnesemia, hyponatremia, • Use with caution in true "sulfa" allergy • Constipation
Bumetanide (Bumex)	0.5 – 1 mg or bid	
Furosemide (Lasix) po	20 – 120 mg daily or bid	
Furosemide (Lasix) IV	Continuous 5 – 20 mg/hour; 20 – 80 mg BID or every 8 hours	
Torsemide (Demadex)	10–40 mg daily (may be divided)	
Thiazide Diuretics		
Metolazone (Zaroxolyn)	2.5 – 5 mg daily (30 minutes prior to loop diuretic)	
Potassium Sparing Diuretics		Hyperkalemia Gynecomastia
Spironolactone (Aldactone)	12.5 – 25 mg daily	
All three drug classes		• Renal dysfunction • Hypotension • Orthostatic hypotension • Gout

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Loop Diuretics Strategies

- Equivalents
  - Bumetanide 1 mg
  - Torsemide 20 mg
  - Furosemide 40 mg
- Dosing strategies
  - Increase current loop dose
  - Change loop to equivalent dose
- Goals
  - Increase urine output
  - Decrease weight
  - Symptom resolution
- In hospital exacerbation
  - Convert home oral dose to IV at $\geq$ home dose

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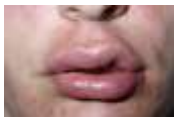
Angioedema Treatment



- Pepcid
- Benadryl 50 mg IV x
- Solu-Medrol 125mg
- Lisinopril was discontinued
- Norvasc added for HTN
- Angioedema improved
- Patient discharged with as needed Benadryl and oral prednisone burst
- Education on NOT to take ACEI/ARBs or Entresto

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Type II Diabetic and Heart Failure/CVD

Medications to consider to slow heart disease

- **FARXIGA** (dapgliflozin)
  - Used along with diet and exercise to improve blood sugar control
  - Used to **reduce the risk of hospitalization** for heart failure in adults with type II diabetes and known cardiovascular disease or multiple cardiovascular risk factors
- **JARDIANCE (empagliflozin)**
  - Used along with diet and exercise to lower blood sugar in adults with type 2 diabetes
  - Used to **reduce the risk of cardiovascular death** in adults with type 2 diabetes who have known cardiovascular disease.

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Medications to Avoid in HF

- NSAIDS (non-sterodial anti-inflammatory drugs)
  - ↑ risk of fluid retention and renal failure
  - May diminish the efficacy of diuretics and ACE inhibitors
- Most antiarrhythmics
  - Poorly tolerated due to proarrhythmic & cardiodepressant effects
  - Amiodarone – does not adversely effect HF survival
- Calcium channel blockers
  - Diltiazem and Verapamil are very strong negative inotropic agents; Avoid in HFrEF
  - Amlodipine is okay
- Avandia (Rosiglitasone Maleate)
  - Can cause fluid retention and exacerbate HF

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Complimentary and Alternative Medicines (CAM) & HF Medications

Table 7. CAMs With Significant Interactions With Cardiovascular Medications Used in Patients With HF[59]

CAM Product	Digoxin	ACE-I/ARBs	β-Blockers	CCB	Amiodarone	Warfarin
St. John's wort	x	x	x	x	x	x
Grapefruit juice		x	x	x	x	x
Ginseng						x
Hawthorn	x					
Ephedra						x
Black cohosh		x	x		x	
Green tea						x

ACE-I, indicates angiotensin-converting enzyme inhibitors; ARBs, angiotensin receptor blockers; CAM, complementary and alternative medicine; CCB, calcium channel blockers; and HF, heart failure.

Reference: AHA Scientific Statement: *Drugs that may Cause or Exacerbate Heart Failure*. Circulation 2016; e32 – e 69. :

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Nonpharmacological Treatments of HF

- Diet
- Daily weights
- Exercise
- Teach back

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- ¼ teaspoon salt = 600 mg sodium
- ½ teaspoon salt = 1200 mg sodium
- ¾ teaspoon salt = 1800 mg sodium
- 1 teaspoon salt = 2300 mg sodium

Low Sodium Diet

Teach to read labels

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Sodium content

Soy Sauce 1 tbsp	> 800 mg
Salad Dressing 2 tbsp	500 – 800 mg
Chili Beans1/2 cup	500 – 800 mg
Cottage Cheese low fat ½ cup	350 – 500 mg
Tuna canned 3 oz	175 – 300 mg
Yogurt 1 cup	65 – 120 mg
Macaroni and Cheese 1 cup	> 800 mg
Tomato juice ¾ cup canned	350 – 500 mg
1 cup canned soup	> 800 mg
Oatmeal, regular cooked, not instant	< 10 mg
Vegetable, fresh or frozen	< 10 mg
Dill Pickle 1 large	> 800 mg
Pancake 1, 6 inch	350 – 500 mg

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Exercise

Exercise such as walking is important but don't overdo it. Set realistic goals and don't "push". Stop exercising immediately if you feel tired, have chest pain, or are very short of breath. Using your activity in the hospital as a guide start with that amount of exercise and gradually increase.

- Remember these tips as well:
  - Do things at a slow to moderate pace – don't rush.
  - Space out activities throughout the day taking 20-30 minute rest periods. Do "easy" activities alternating with harder ones.
  - Don't exercise for at least one hour after meals.
  - Avoid extremely hot or cold temperatures.
  - Avoid heavy exercise and weight lifting. Ask the doctor about returning to work and doing active recreational activities.
  - Keep the general guidelines above and use positions easy for you when you feel able to resume your sexual activity.

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Daily Weights

- Weigh same time every day
- Same clothes
- Empty bladder

- Have scales to give patients
- Telehealth – Remote Patient Monitoring

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Educating Patients About Daily Weights

- The vascular bed can hold 10 pounds of fluid before it starts to seep out into the tissues
  - 2 pounds = 1 quart of water extra in the circulation
- Keep track – standard is to notify MD of a 2-3 pound weight gain overnight or 5 pound gain in one week

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ACCURATE
I & O
ACCURATE
Weights

You will save more lives by being *METICULOUS* than by being BRILLIANT

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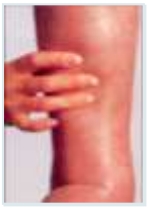
Assessment of Fluid Volume Status

- Teaching points for patients/families:
  - Ankle swelling (shoes too tight, socks make marks on leg)
  - Abdominal swelling ("bloated", belt too tight)
  - Orthopnea
  - Paroxysmal nocturnal dyspnea
  - Weight gain
  - Shortness of Breath with walking



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Heart failure teach-back questions

Four days of teach back questions

Day One questions:

1. What is the name of your water pill?
a. Your water pill will help your body to get rid of extra water and sodium that may collect in your lungs or settle in your feet and ankles.

2. What weight gain should you call your doctor about? Do you have a scale at home?
a. Weigh same time each AM, with same scale on hard flat surface, after urinating and before eating breakfast, in same attire.
b. Record date and weigh
c. Notify doctor if weight gain of > 3 lbs. in 1 day with normal eating. Patterns or steady increase in weight over 1 week

3. What foods should you avoid when you have heart failure?
a. It is important to lower the amount of sodium (salt) in your diet.
b. Sodium causes extra fluid to build up in your body—making your heart work harder.
c. A low sodium diet has 1500 mg of sodium per day.
d. Common foods high in sodium include canned soups, deli meats, ham, processed foods and fast foods.
e. Read labels

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Heart failure teach-back questions

4. **What are the symptoms of your heart failure?**
   a. Identify patient specific symptoms!
5. **What do you think triggered your admission to the hospital?**
   a. Document triggers in notes section of day 1 Questions.

- Interventions:
  - Give scale / weight documented on card.
  - Give and document heart failure packet.
  - Document triggers in the notes section of Day 1 questions
  - Document all education in computer

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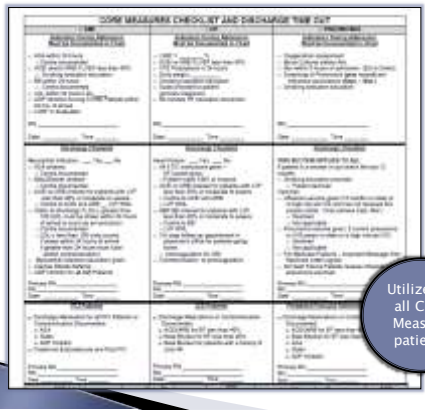

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HF RN Care Navigator

- Identify HF patients in hospital
- Round on patient in hospital
- Calls patient at least weekly for 30 days
- Patients have Navigators phone number to call anytime with any changes or questions
- HF Follow up appointment within one week post discharge with APP and HF navigator
- APP is available to HF navigator to give orders if needed for 30 days
- Heart Failure Support/Education Group

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	HCM	HOCM	Dilated	
Definition	Hypertrophy in < 25	Obstruction from hypertrophy	Enlarged dilated of one or all four chambers	
Symptoms	Syncope with exercise Harsh systolic murmur	Sudden cardiac death	Weakness Fatigue Decreased activity intolerance Systolic murmur S3	
LV Fx			Decreased EF Systolic dysfunction	
Treatment	Negative inotropes No aggressive exercise BB	Negative intropes No aggressive exercise BB SBE prophylaxis Myomectomy	Treat cause	

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	Idiopathic	Ischemic	Hypertensive	Valvular
Definition	No underlying cause	CAD Most commone	From high BP	MR/AI
Symptoms	Same	Same		
LV Fx	Decreased EF Systolic dysfunction	Decreased EF Systolic dysfunction	Systolic dysfunction	Systolic dysfunction
Treatment	Same Anticoagulation	Same	Alpha blocking agents Vasodilators	Valve repair/replacement Nitrates Hydralazine

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	Peripartum	Alcohol Related	Restrictive	Takotsubo
Definition	Last trimester or up to 6 months after delivery	From Alcohol	Decreased filling from noncompliant heart	Stressed induced Appears as AMI without CAD
Symptoms			May have no or minimal symptoms Affects both ventricles Right and Left sided failure	EF very low and recovers within 6-9 weeks
LV Fx	Systolic dysfunction	Systolic dysfunction	Normal EF	Ballooning of ventricle
Treatment	50% recover completely	Remove alcohol	Similar to diastolic	No fibrinolytics

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	Systolic HF	Diastolic HF		
Definition	Inability to contract	Inability to fill		
Symptoms				
LV F	Decreased EF	EF normal		
Treatment	Treat HF symptoms ACEI, BB	Treat HF symptoms ACEI, BB		

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