Heart Failure

for St. Vincent's

Joanna M. Joly, MD | Assistant Professor of Medicine – Cardiovascular Disease

Advanced Heart Failure | Heart Transplantation | Mechanical Circulatory Support | Pulmonary Vascular Disease

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I have no conflicts of interest.

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Objectives

- Case: New diagnosis of HF
- Cornerstones of chronic HF management
- Epidemiology and Pathophysiology
- Case: Acute, decompensated HF
- Clinical management of ADHF
- When to refer for advanced therapies

Case of new HF: Mr. VF

Referred for increasing dyspnea with minimal exertion, fatigue at work.

- 59 M with PMH: HTN, CKD, GERD
- Social Hx: Non-smoker, occasional beer, works at a factory, lives in Oxford, AL
- Family Hx: Father-colon cancer, Mother-heart failure
- Medications: HCTZ, amlodipine, pantoprazole

Mr. VF

Referred for increasing dyspnea with minimal exertion, fatigue at work.

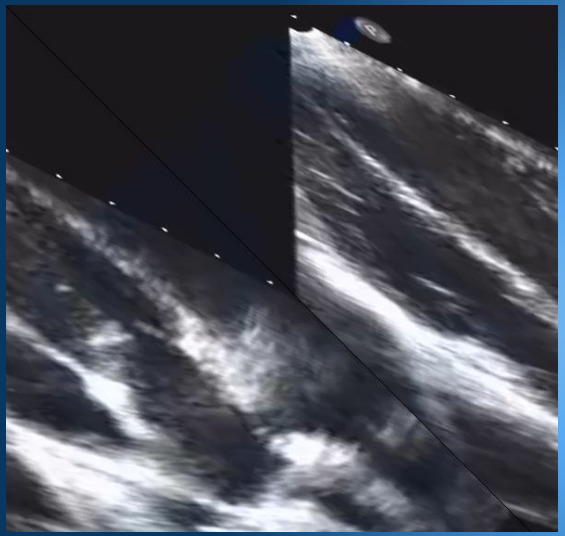
- Vitals: HR 90, BP 131/85, Afebrile, 97% RA
- Labs: CBC – Hgb 10, MCV 78), BMP – Cr 1.4 (stable), Na 130, K 3.5, BNP – 200
- Physical exam: Elevated JVP, regular rhythm, no murmurs or gallops, laterally displaced PMI, no ascites, mild ankle edema, all extremities warm and well-perfused

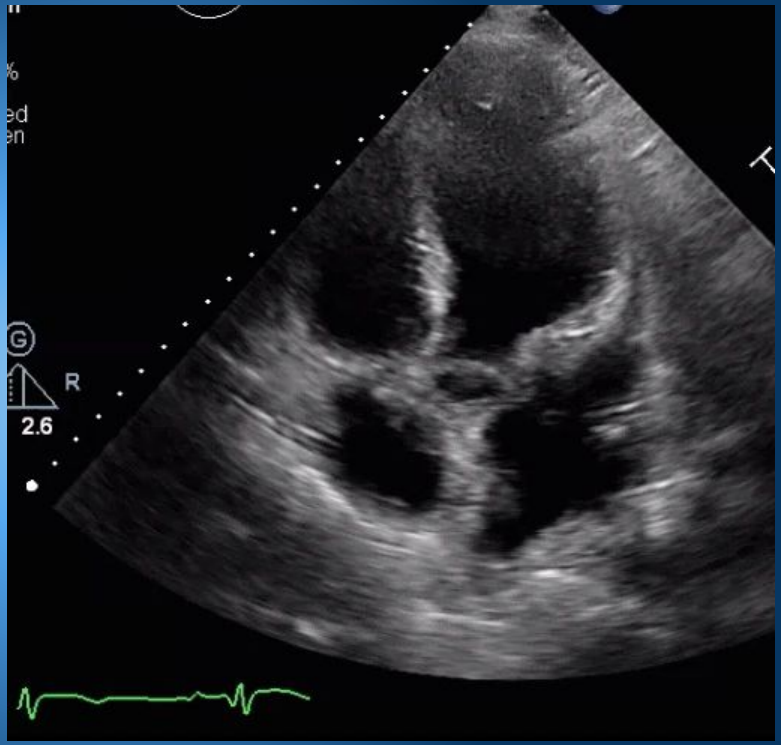
Mr. VF

Referred for increasing dyspnea with minimal exertion, fatigue at work.

- CXR: Mild cardiomegaly, no acute lung disease.
- ECG: Normal sinus rhythm, normal amplitude, no Q waves, no bundle branch block (*LBBB = CRT!)
- Echo:

Normal





Mr. VF ?

Cornerstone of Therapy

- Neurohormonal antagonists
  - Renin-angiotensin/ Neprilysin
    - » ARB, ARNI, ACEi
  - Sympathetic nervous system
    - » Bblockers
  - Aldosterone
    - » MRA
- Smooth muscle relaxation
  - » Hydralazine/ Nitrates

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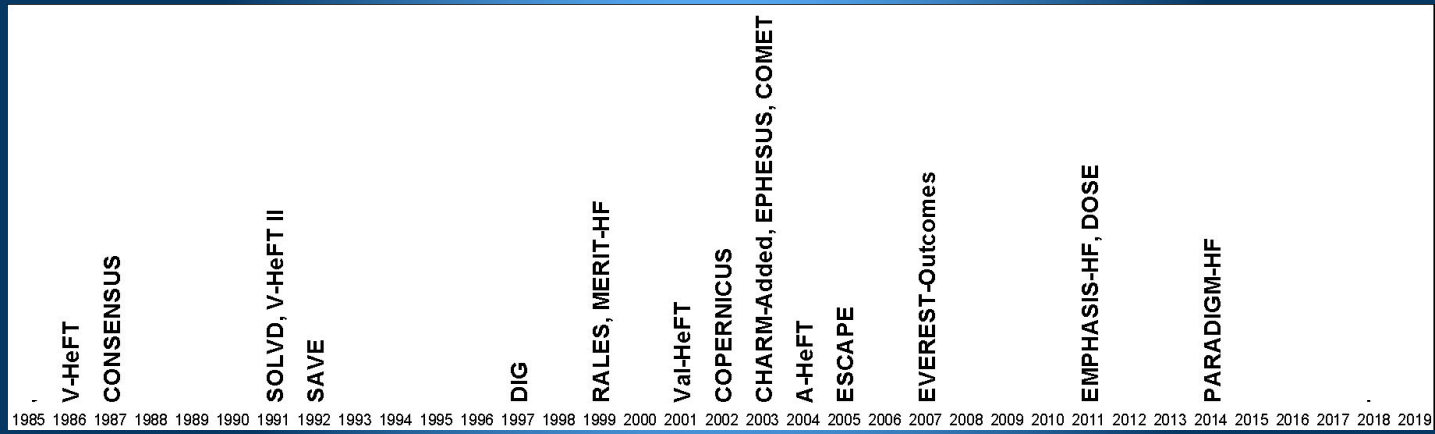
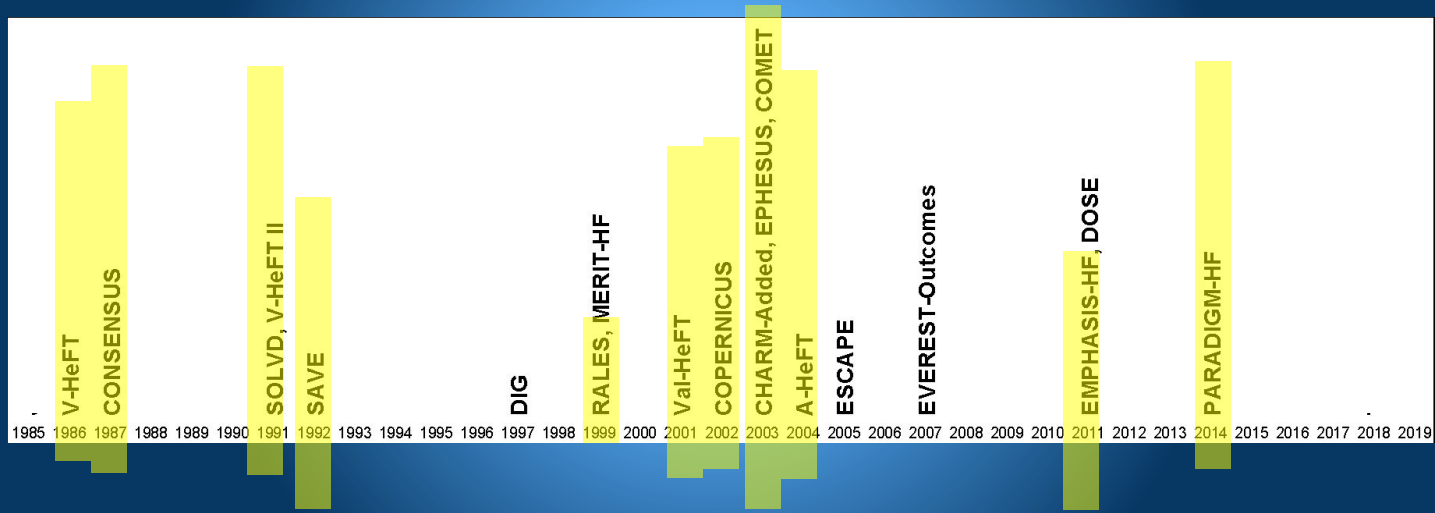
AFTERLOAD REDUCTION

Timeline


V-HeFT
CONSENSUS
SOLVD, V-HeFT II
SAVE
DIG
RALES, MERIT-HF
Val-HeFT
COPERNICUS
CHARM-Added, EPHESUS, COMET
A-HeFT
ESCAPE
EVEREST-Outcomes
EMPHASIS-HF, DOSE
PARADIGM-HF
1985 1986 1987 1988 1989 1990 1991 1992 1993 1994 1995 1996 1997 1998 1999 2000 2001 2002 2003 2004 2005 2006 2007 2008 2009 2010 2011 2012 2013 2014 2015 2016 2017 2018 2019

Timeline



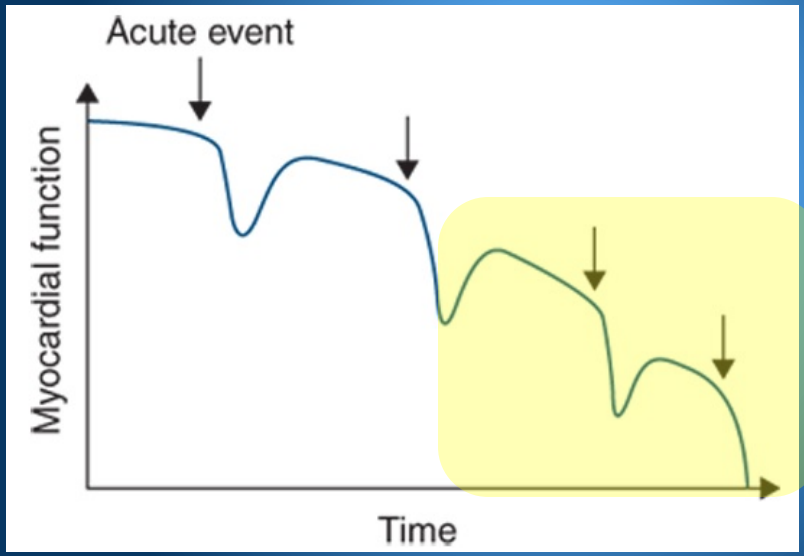
**All of these show benefit of vasodilation/afterload reduction in HF

Increasing utilization

	VMAC 2002	OPTIME 2002	ADHERE 2004	EURO-HF 2005	GWTG-HF 2011	GWTG-HF upon d/c
% on ACE-is	60%	70%	41%	62%	65.3%	92.9%
% on ARBs	10%	13%	12%	ND		
% on Bblockers	33%	22%	48%	37%	72.6%	94.6%
% on MRA	ND	ND	ND	21%	15.6%	32.2%
% on nitrates	35%	ND	26%	ND	ND	ND

The changing face of HF…

What don’t we know?



Gheorghiade M et al: Pathophysiologic targets in the early phase of acute heart failure syndromes. Am J Cardiol. 2005 Sep 19;96(6A):11G-17G.

Definition of acute decompensated HF

- New or worsening signs & symptoms of heart failure requiring unscheduled hospitalization and urgent therapy

Epidemiology of ADHF

- HF is a leading cause of hospitalization among adults and the elderly, with an estimated prevalence of >37.7 million individuals globally.

- Total medical costs in the US for patients with HF are expected to rise from$20.9 billion in 2012 to $53.1 billion by 2030.

Ziaeian B, Fonarow GC. Epidemiology and aetiology of heart failure. Nat Rev Cardiol. 2016 Jun;13(6):368-78.

Risk factors

Associated with post-discharge death or re-hospitalization:

- Renal insufficiency
- COPD
- HF hospitalization within the prior 6 mos.
- Nitrates, digoxin, or diuretic use at admission
- Mechanical ventilation during hospitalization
- History of CVA or TIA
- Liver disease

OPTIMIZE-HF Registry, 2008

Presentation

Registries: ADHERE (2003), EURO-HF (2003), OPTIMIZE-HF (2008) totaling ~210,000 patients

- 70-90% reported any dyspnea
- <1-<8% had SBP <90 mmHg
- Majority/many with >90-140/>140

Presentation

- Small % of ADHF is a new diagnosis
- Majority live with chronic HF and some amount of baseline symptoms
  - Mean age 75
  - Almost half have preserved EF
  - Comorbidities are common

Presentation

- Identify triggers or amplifiers
  - Ischemia: CAD, hypoxemia, excess O2 demand > supply
    - Hypoxemia: COPD, infection, pulmonary edema, PE
  - Arrhythmia: Afib/Aflutter, VT (can be 1° or 2°)
  - Systemic hypertension
  - Renal insufficiency

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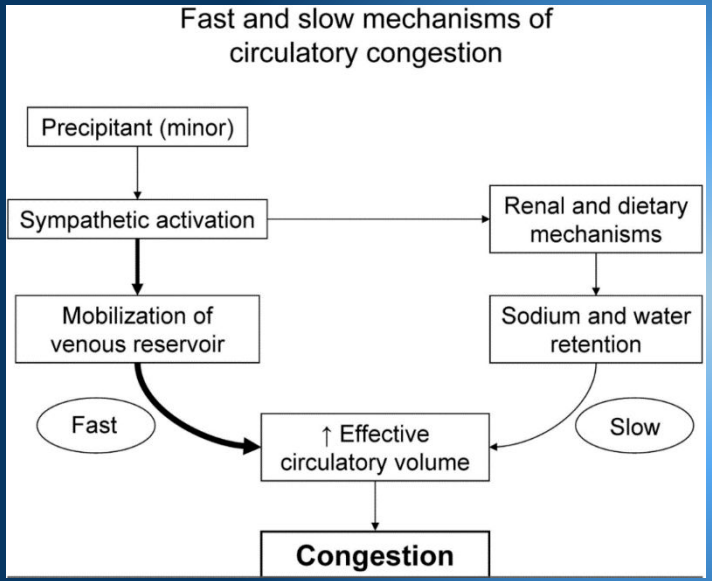
- Identify triggers or amplifiers
  - Ischemia: CAD, hypoxemia, excess O2 demand > supply
    - Hypoxemia: COPD, infection, pulmonary edema, PE
  - Arrhythmia: Afib/Aflutter, VT (can be 1° or 2°)
  - Systemic hypertension
  - Renal insufficiency
  - Volume overload: dietary indiscretion, Na/fluid retention, fluid mobilization from venous capacitance

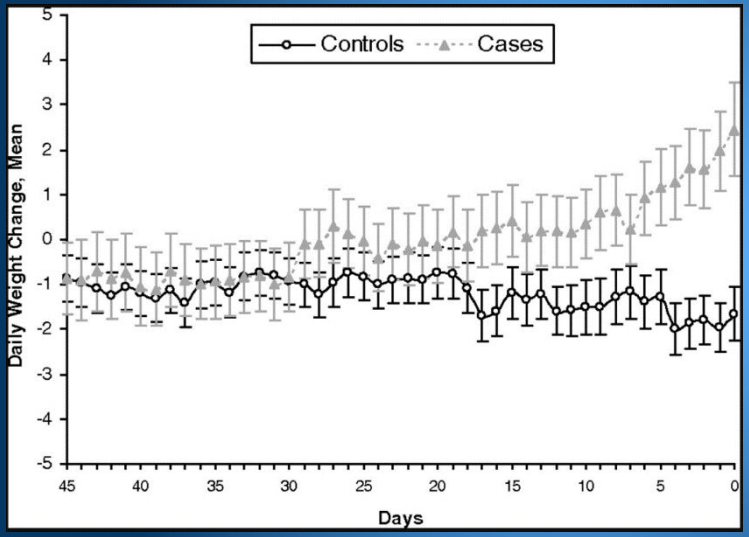
Venous capacitance culprit



Fallick C. Sympathetically mediated changes in capacitance: redistribution of the venous reservoir as a cause of decompensation. Circ Heart Fail, 2011;4:669-675.

- Venous system accounts for 70% of TBV
- Veins contain >5x the density of adrenergic terminals found in arteries
- For a given sympathetic stimulus, veins respond >> arteries
- Decrease in capacitance of the venous reservoir → almost instantaneous increased preload in the stimulated state

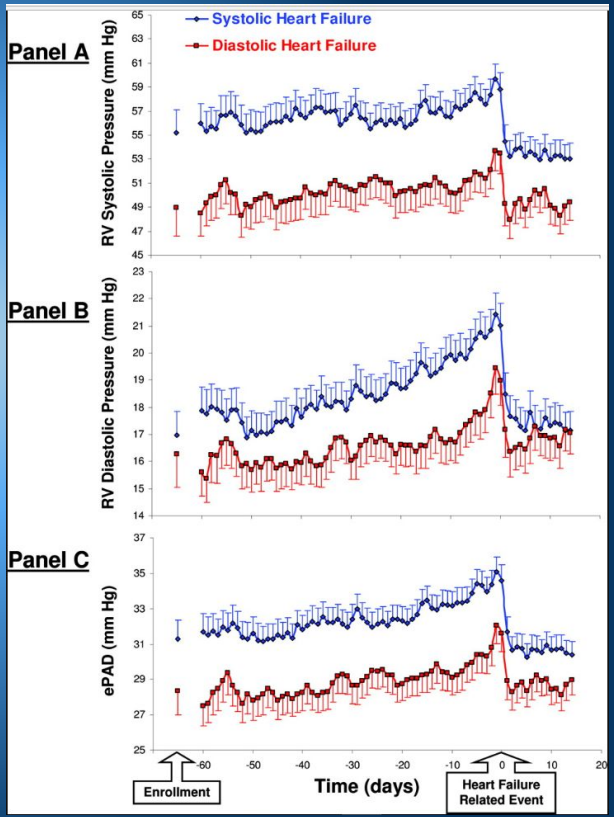
Venous capacitance culprit



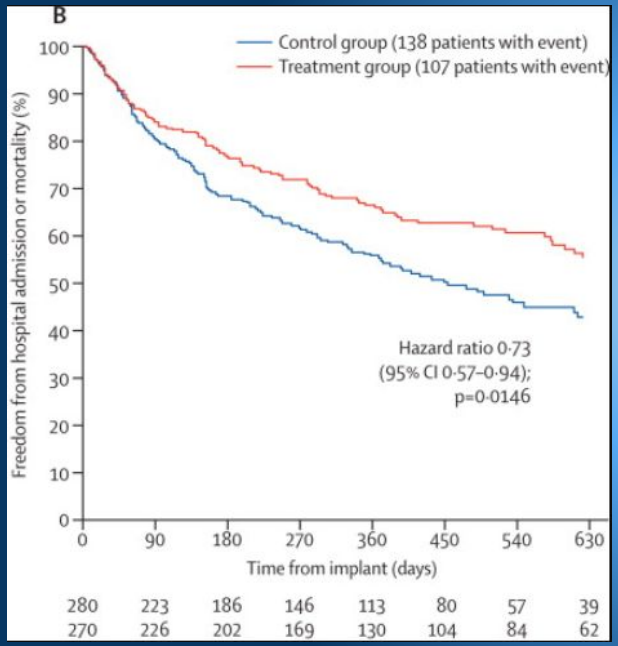
Chaudhry SI. Patterns of Weight Change Preceding Hospitalization for Heart Failure. Circulation. 2007; 116: 1549-1554.

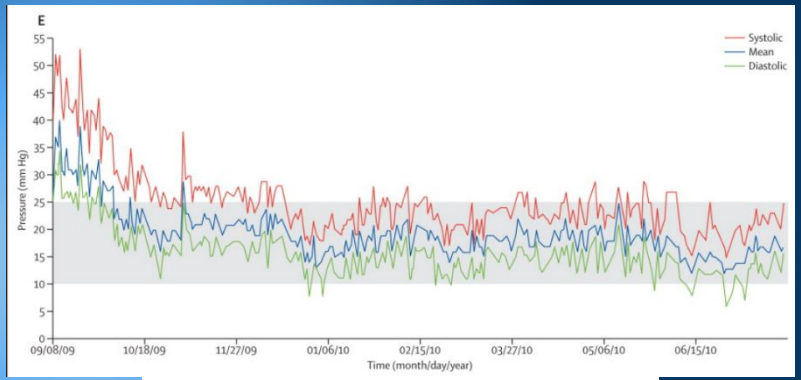
Venous capacitance culprit

Zile MR. Transition From Chronic Compensated to Acute Decompensated Heart Failure. Circulation. 2008;118:1433-1441.



Venous capacitance culprit



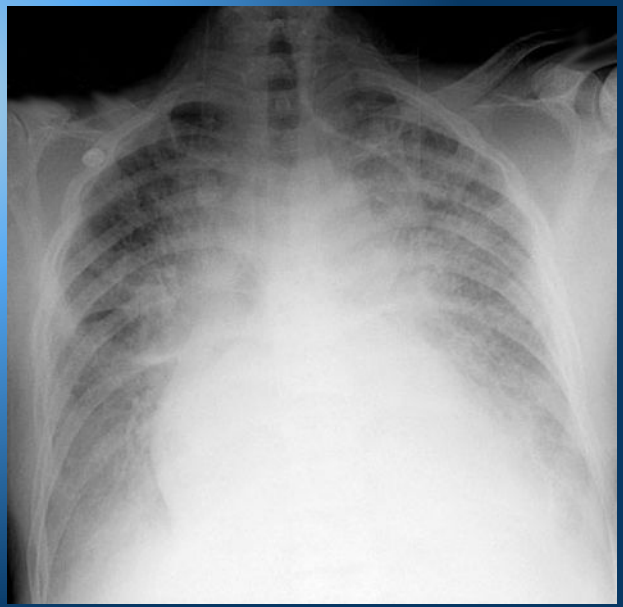


Abraham WT et al. Wireless pulmonary artery haemodynamic monitoring in chronic heart failure: a randomised controlled trial. Lancet. 2011 Feb 19;377(9766):658-66.

Case of ADHF: Ms. AF

74 F presents with worsening dyspnea. Your exam suggests ADHF.

BP 100/85 (90) HR 100



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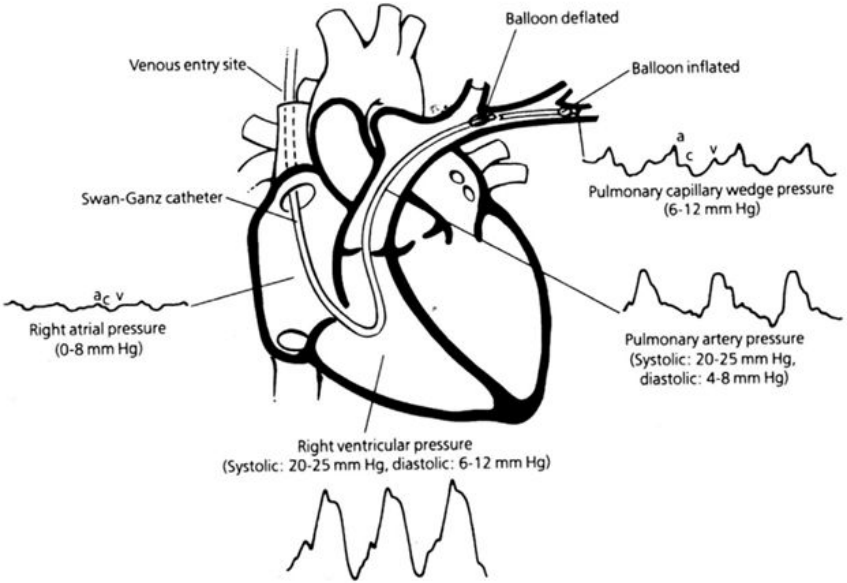


Fig. 3. Hemodynamic waveforms in the RA, RV, PA, and wedge (PCWP) positions of the PAC. (*From* Disease-a-month. The Swan-Ganz Catheter. Dis Mon 1991;37(8):509–43; with permission.)

Ms. AF

74 F presents with worsening dyspnea. Your exam suggests ADHF.

BP 100/85 (90) HR 100

RA 12 PA 60/30/40 PCWP 30

CO 3.0 CI 1.5

SVR 2080

PVR 400

Ms. AF

74 F presents with worsening dyspnea. Your exam suggests ADHF.

BP 100/85 (90)
HR 100
RA 12
PA 60/30/40
PCWP 30
CO 3.0
CI 1.5

SVR 2080
PVR 400

Ms. AF

74 F presents with worsening dyspnea. Your exam suggests acutely decompensated heart failure.

BP 100/85 (90) HR 100

RA 12 PA 60/30/40 PCWP 30

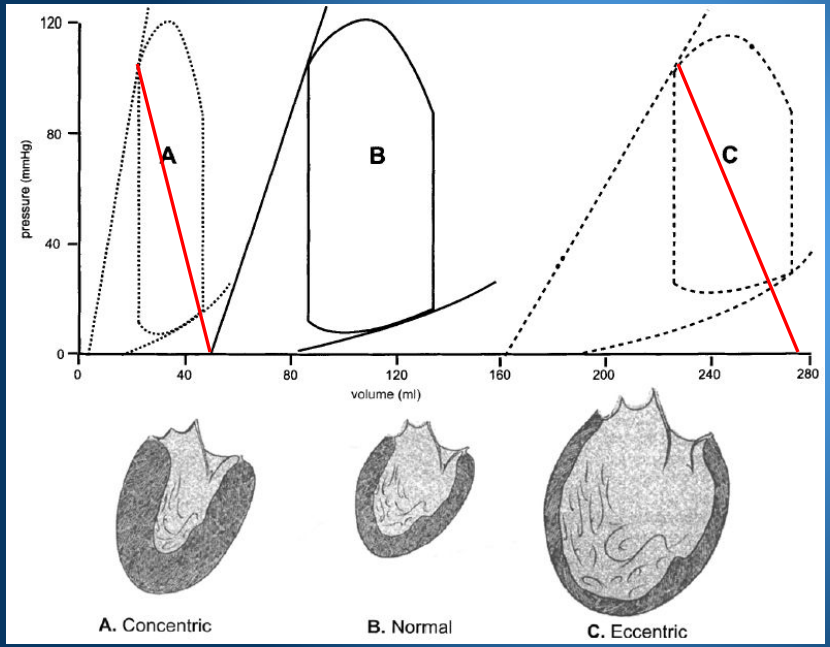
CO 3.0 CI 1.5

SVR 2080

PVR 400

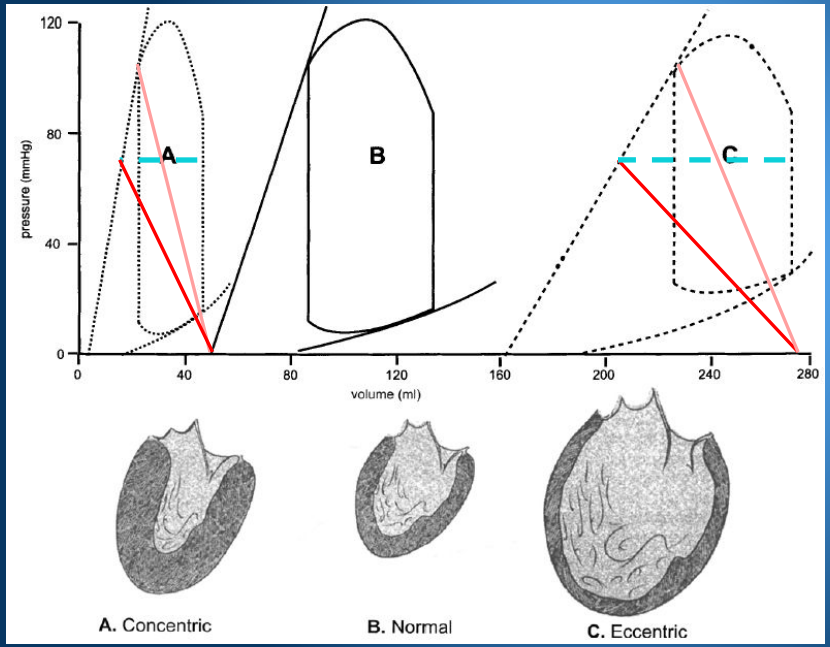
$$\frac{90 - 12}{3.0} = \frac{78}{3.0} \times 80 = 2080 \text{ d/s}$$

Afterload reduction



M Katz. Physiology of The Heart, 3rd ed.

Afterload reduction



M Katz. Physiology of The Heart, 3rd ed.

Afterload Reduction

May be less commonly indicated in era of better use/adherence to GDMT

- Nonrandomized, n=97 vs. 78, CI ≤ 2
- Titrated to MAP 65-70
- Pts treated with sodium nitroprusside = greater improvement in HD measurements during hospitalization, higher rates of PO vasodilator Rx at d/c, lower rates of all-cause mortality, without increase in rehospitalization rates

Mullens W et al. Sodium nitroprusside for advanced low-output heart failure. J Am Coll Cardiol. 2008 Jul 15;52(3):200-7.

Afterload Reduction

Method

- Once MAP was achieved, nitroprusside weaned while uptitrating ISDN/hydral
- Discretion of treating MD
- Duration of infusion varied, typically between 24-72 hrs

Table 1 Standard Medication Protocols for the Cleveland Clinic Heart Failure Intensive Care Unit

Sodium nitroprusside
Begin at 10 to 40 μg/min (without bolus)
↑ as tolerated to achieve desired hemodynamic goals, targeting MAP 65 to 70 mm Hg
Do not ↑ dose beyond 400 μg/min
To wean off: ↓ infusion gradually as tolerated while maintaining MAP goals and initiating/increasing oral vasodilators
Captopril
Incremental ↑ 6.25→12.5→25→50 mg
Begin at 6.25 to 12.5 mg orally
After 2 h, if initial dose tolerated, ↑ incrementally to next dose
After 2 h, if previous dose tolerated, ↑ incrementally to next dose
After 6 h, if previous dose tolerated, then 50 mg orally TID*
Isosorbide dinitrate
Begin 10 mg orally
After 2 h, if initial dose tolerated, ↑ to 20 mg
After 8 h, if 20 mg tolerated, ↑ to 40 mg
After 8 h, if 40 mg tolerated, ↑ to 60 mg
After 8 h, if 60 mg tolerated, then 60 mg orally TID*
Hydralazine
Begin 25 mg orally (or 10 mg if MAP is low or patient is in labile condition)
After 2 h, if initial dose tolerated, ↑ to 50 mg
After 6 h, if 50 mg tolerated, ↑ to 75 mg
After 6 h, if 75 mg tolerated, ↑ to 100 mg
After 6 h, if 100 mg tolerated, then 100 mg QID*

*If previous dose is not tolerated, administer highest dose tolerated TID or QID.
MAP = mean arterial pressure; QID = 4 times daily; TID = 3 times daily.

Mullens W et al. Sodium nitroprusside for advanced low-output heart failure. J Am Coll Cardiol. 2008 Jul 15;52(3):200-7.

PRACTICE GUIDELINE

2013 ACCF/AHA Guideline for the Management of Heart Failure: Executive Summary

A Report of the American College of Cardiology Foundation/
American Heart Association Task Force on Practice Guidelines

Developed in Collaboration With the American College of Chest Physicians, Heart Rhythm Society, and International Society for Heart and Lung Transplantation

Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation

HF Classification

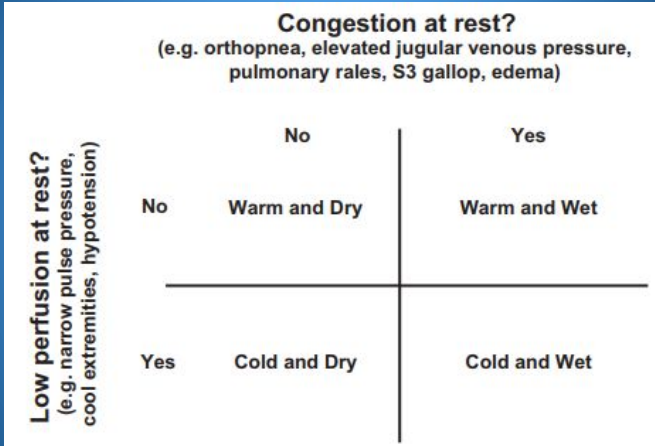


Figure 4. Classification of patients presenting with acutely decompensated heart failure. Adapted with permission from Nohria et al. (306).

Therapy - Diuretics

- Diurese promptly in those with volume overload
- Use an IV dose equivalent to or greater than chronic PO dose
- Intermittent boluses or continuous infusion
- Serial assess volume, adjust accordingly

Therapy - Diuretics

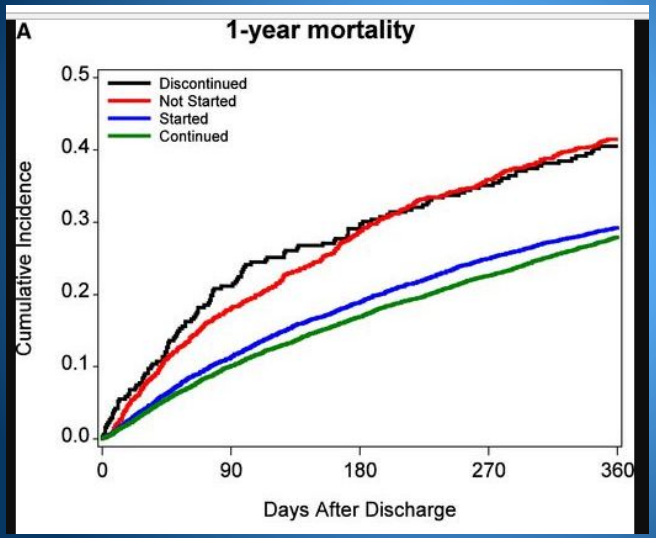
- If desired effect is not attained, intensify dose; add thiazide

Maintaining GDMT

CLASS I

1. In patients with HF*r*EF experiencing a symptomatic exacerbation of HF requiring hospitalization during chronic maintenance treatment with GDMT, it is recommended that GDMT be continued in the absence of hemodynamic instability or contraindications (307–309). *(Level of Evidence: B)*

Discontinuing ACEi/ARB

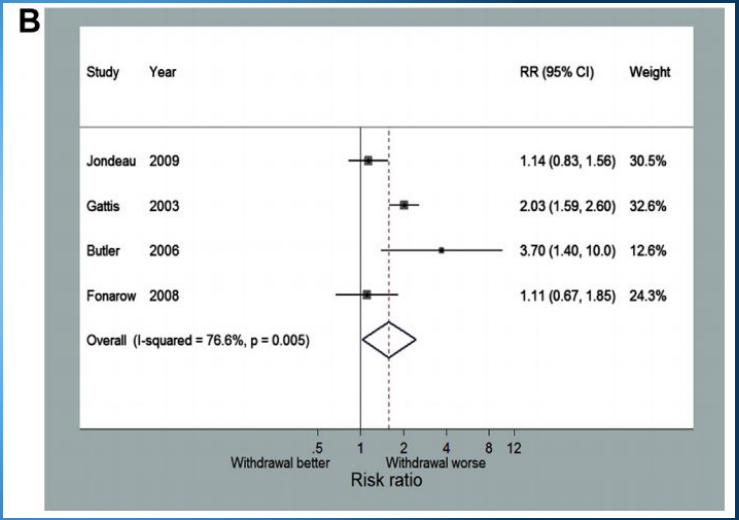


Gilstrap LG. Initiation, Continuation, or W/drawal of ACEi/ARB and Outcomes in Patients Hospitalized With HFrEF. J Am Heart Assoc. 2017 Feb 11;6(2).

Maintaining GDMT

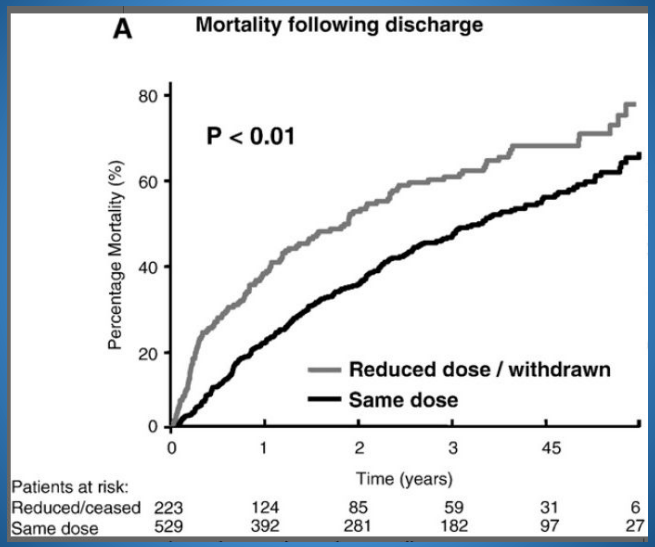
2. Initiation of beta-blocker therapy is recommended after optimization of volume status and successful discontinuation of intravenous diuretics, vasodilators, and inotropic agents. Beta-blocker therapy should be initiated at a low dose and only in stable patients. Caution should be used when initiating beta blockers in patients who have required inotropes during their hospital course (307–309). *(Level of Evidence: B)*

Discontinuing Bblocker



Prins KW. Effects of Beta-Blocker Withdrawal in ADHF: A Systematic Review and Meta-Analysis. JACC Heart Fail. 2015 Aug;3(8):647-53.

Discontinuing Bblocker



Metra M. Should beta-blocker therapy be reduced or withdrawn after an episode of ADHF? Results from COMET. Eur J Heart Fail. 2007 Sep;9(9):901-9.

Therapy - Vasodilators

- IV nitroprusside, nitroglycerin, or nesiritide may be considered for relief of dyspnea (level of evidence A)

2016 ACC/AHA/HFSA Focused Update on New Pharmacological Therapy for Heart Failure: An Update of the 2013 ACCF/AHA Guideline for the Management of Heart Failure

A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Failure Society of America

CLINICAL PRACTICE GUIDELINE: FOCUSED UPDATE

2017 ACC/AHA/HFSA Focused Update of the 2013 ACCF/AHA Guideline for the Management of Heart Failure

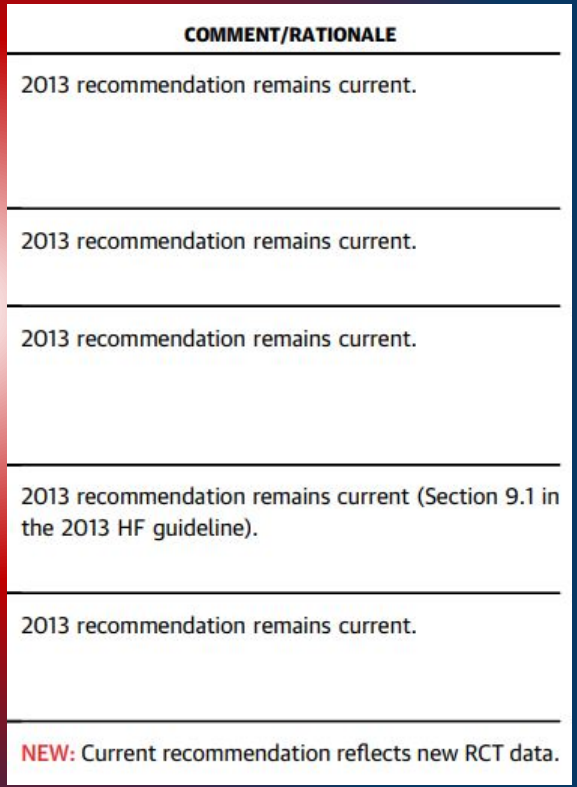
A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Failure Society of America

Developed in Collaboration with the American Academy of Family Physicians, American College of Chest Physicians, and International Society for Heart and Lung Transplantation

What About HFpEF…

From 2013 Guidelines:

- Control BP
- Relieve symptoms of congestion with diuretics
- CAD? Afib?

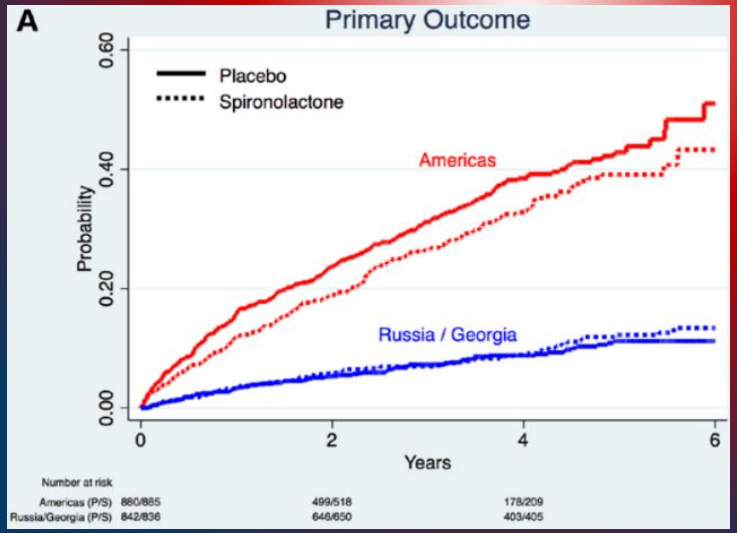


COMMENT/RATIONALE
2013 recommendation remains current.
2013 recommendation remains current.
2013 recommendation remains current.
2013 recommendation remains current (Section 9.1 in the 2013 HF guideline).
2013 recommendation remains current.
NEW: Current recommendation reflects new RCT data.

What About HFpEF…

From 2017 Guidelines:

- MRA (i.e. spironolactone) may be considered in HFpEF patients with elevated BNP or HF hospitalization within the last year, GFR >30, Cr <2.5, K <5



Pfeffer MA, et al. Regional variation in patients and outcomes in the Treatment of Preserved Cardiac Function Heart Failure With an Aldosterone Antagonist (TOPCAT) trial. Circulation. 2015 Jan 6;131(1):34-42.

Inotropes

Table 6. Adverse Events and Mortality*

Adverse Event, No. (%)	Placebo (n = 472)	Milrinone (n = 477)	*P* Value
Treatment failure cause at 48 hours	43/466 (9.2)	97/470 (20.6)	<.001
Progression of heart failure	6.8	7.9	.54
Adverse event	2.1	12.6	<.001
Events during index hospitalization			
Myocardial infarction	2 (0.4)	7 (1.5)	.18
New atrial fibrillation or flutter	7 (1.5)	22 (4.6)	.004
Ventricular tachycardia or fibrillation†	7 (1.5)	16 (3.4)	.06
Sustained hypotension‡	15 (3.2)	51 (10.7)	<.001
Death	11 (2.3)	18 (3.8)	.19
Events within 60 days			
Myocardial infarction	5/448 (1.1)	10/462 (2.2)	.21
New atrial fibrillation or flutter	16/446 (3.6)	26/462 (5.6)	.14
Ventricular tachycardia or fibrillation	20/446 (4.5)	23/461 (5.0)	.72
Death	41/463 (8.9)	49/474 (10.3)	.41

*Total number of patients listed only when it varies from number randomized as shown.
†Reported by the investigator.
‡Defined as a systolic blood pressure below 80 mm Hg for more than 30 minutes, requiring intervention.

Cuffe MS, et al; (OPTIME-CHF) Investigators. Short-term intravenous milrinone for acute exacerbation of chronic HF: an RCT. JAMA. 2002 Mar 27;287(12):1541-7.

Transition/ Discharge

- Optimal GDMT
- Volume status, blood pressure
- Symptoms, functional status
- Causes of HF/ barriers to care
- Renal function, electrolytes
- Co-morbid conditions
- Education, self-care
- Palliative care

Transition/ Discharge

- Multidisciplinary disease management
- Follow-up visit in 1-2 wks, telephone call

Mr. VF

Referred for increasing dyspnea with minimal exertion, fatigue at work.

- 59 M with PMH: Optimize treatment of comordibities
- Social Hx: Avoid EtOH
- Family Hx: Consider genetic testing
- Medications: Bblocker, ARB/ARNI/ACEi, MRA

Mr. VF

Referred for increasing dyspnea with minimal exertion, fatigue at work.

- Vitals: Aim for goal SBP 100-120, HR 60-80
- Labs: Treat anemia, watch electrolytes, serial BNP
- Physical exam: Volume status and perfusion (cardiac output)

Mr. VF

New diagnosis of HF warrants

- Ischemia work-up: noninvasive stress test + imaging, vs. "gold standard" coronary angiogram + RHC
- Search for reversible etiology!
- HF education: daily weight, diet/Na intake, fluid restriction, exercise regimen (cardiac rehab)

Mr. VF

New diagnosis of HF warrants

- Close follow-up for uptitration of GDMT
- Repeat evaluation of LVEF, to determine need for ICD
- If Mr. VF's symptoms do not improve to NYHA class I-II w/ GMT, regardless of EF, consider risk stratification!

When to refer for advanced therapies?

- Functional decline, worsening symptoms
- ICD shocks or syncope
- Dose reduction/ discontinuation of GDMT
- End-organ dysfunction
- Refractory angina
- Refractory, life-threatening arrhythmia
- Inotrope-dependence
- Change in hospitalization pattern

May threaten candidacy

- Tobacco, marijuana, or other substance abuse
- Obesity, BMI >35
- Significant comorbidities (DM, PVD, CKD, COPD, malnutrition)
- Lack of social support or inability to cope

Thank you for your attention.

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