Heart Failure with Preserved Ejection Fraction: A Clinician’s Perspective

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• We pronounce HFpEF: “huff-puff”
Take home messages

• Don’t miss the diagnosis of HFpEF
  » Many patients go undiagnosed for years
  » Always think of HFpEF in the dyspneic patient

• Your HFpEF patients need help!
  » High risk for hospitalization and death
  » Symptomatic, depressed, debilitated
  » Complex cases in need of multidisciplinary care
  » Several clinical trials are available
6 MYTHS AND MISCONCEPTIONS
Myth #1: Diastolic dysfunction, diastolic HF, and HFpEF are all the same

Fact #1: HFpEF is more than just diastolic dysfunction
DD vs. DHF vs. HFpEF

**DD**
Pathophysiologic condition: impaired relaxation, ↓compliance, ↑LV filling pressures

**DHF**
Normal LVEF plus sign/symptoms of HF due to DD

**HFpEF**
Normal LVEF plus signs/symptoms of HF (excluding severe valve disease, prior ↓LVEF, constriction)
DD vs. DHF vs. HFpEF
DD vs. DHF vs. HFpEF

Pure diastolic HF is actually a rare syndrome.

“pure” DHF
Diastolic HF study:
Started with 1,119 patients…
…after exclusions only
23 patients met
enrollment criteria!

Screened 2,054 Patients (age > 65) with a discharge diagnosis of CHF

1,119 patients with a LVEF > 50 %

<table>
<thead>
<tr>
<th>Reason for exclusion</th>
<th># of patients excluded</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial fibrillation at the time of study</td>
<td>313</td>
</tr>
<tr>
<td>Prior CABG</td>
<td>179</td>
</tr>
<tr>
<td>Incomplete records</td>
<td>111</td>
</tr>
<tr>
<td>Left bundle branch block or paced rhythm</td>
<td>90</td>
</tr>
<tr>
<td>No clear documentation of CHF</td>
<td>77</td>
</tr>
<tr>
<td>Active malignancy</td>
<td>55</td>
</tr>
<tr>
<td>Myocardial ischemia/infarction</td>
<td>53</td>
</tr>
<tr>
<td>Dementia/cognitive impairment</td>
<td>44</td>
</tr>
<tr>
<td>Deceased prior to enrollment evaluation</td>
<td>41</td>
</tr>
<tr>
<td>Dialysis dependant or creatinine ( &gt; 2.5 g/dl)</td>
<td>40</td>
</tr>
<tr>
<td>Severe COPD/pulmonary disease</td>
<td>39</td>
</tr>
<tr>
<td>Warfarin use</td>
<td>30</td>
</tr>
<tr>
<td>Moderate to severe valcular heart disease</td>
<td>13</td>
</tr>
<tr>
<td>History of organ transplantation</td>
<td>6</td>
</tr>
<tr>
<td>Ejection fraction unclear</td>
<td>5</td>
</tr>
<tr>
<td><strong>Total Patients Excluded</strong></td>
<td><strong>1096</strong></td>
</tr>
</tbody>
</table>

935 patients with a LVEF ≤ 50 % (excluded)

23 patients met criteria for enrollment

11 patients agreed to participate
4 men and 7 women
HFpEF: A debilitating syndrome

• TOPCAT trial (spironolactone vs. placebo) N=3445:
  » At baseline:
    – Activity level very low (9.3 MET-hr/week)
    – Poor QOL similar to ESRD
    – 27% with moderate or greater depression
  » Baseline echo:
    – Structural heart disease (LVH, LA enlargement)
    – Diastolic function “normal” in approximately 1/3

HFpEF survival: poor

Dismal 35% survival at 5 years after HF hospitalization, regardless of LVEF

Myth #2: Diagnosing HFpEF is difficult

Fact #2: Diagnosing HFpEF is easy (if you know what to look for)
How is HFpEF diagnosed?

*Keep it simple...*
Diagnosis of HFpEF

- **Step 1**: clinical symptoms/signs of HF
  - Low CO and/or ↑LV filling pressures at rest or with exertion

- **Step 2**: normal LVEF (> 50%)

- **Step 3**: objective evidence of cardiac structural and/or functional problem
  - LVH or LA enlargement or diastolic dysfunction or ↑PASP (in absence of PAH)
Diagnosis of HFpEF?

- Diastolic dysfunction (DD) on echo:
  - Not required for the diagnosis
  - Often uninterpreted or misinterpreted
  - Grade 2 (moderate) or grade 3 (severe)
    DD helpful but not required for diagnosis
  - Patients can have HFpEF with “normal” diastolic function or “mild” DD
- When in doubt: do a right heart cath!
Respiratory variation in PCWP
Respiratory variation in PCWP

End-expiratory PCWP = 27 mmHg

Lung disease
Morbid obesity
Abnormal LA mechanics in HFrEF

- LA reservoir strain > 31.2%
- LA reservoir strain < 31.2%

LA strain is a better predictor of outcomes compared to LV or RV longitudinal strain

Abnormal LA mechanics in HFpEF

Chronic LA pressure and volume overload
- Pulmonary venous congestion
- Pulmonary vasoconstriction
- ↑RV afterload
- RV failure

LV diastolic dysfunction
LA fibrosis
Atrial fibrillation
Atrial myopathy

ABNORMAL LA MECHANICS

Reduced atrial emptying
Decreased LV filling
Decreased cardiac output
Decreased peak VO₂

EXERCISE INTOLERANCE
ADVERSE OUTCOMES
HFpEF: Global CV reserve dysfxn

HFpEF: evidence of impaired CV reserve at 20W exercise

Borlaug B, et al. JACC 2010
Effect of ↑ preload on LA strain

*HFpEF vs. non-cardiac dyspnea*
Diastolic stress echo

REST

L-wave

Rest
E/E' = 13.5

STRESS

Stress
E/E' = 16.7
Myth #3:
A normal BNP rules out HFpEF as a diagnosis

Fact #3:
Up to 1/3rd of patients with confirmed HFpEF have a normal BNP
A typical HFpEF patient...

- 63-year-old man
- Morbid obesity, HTN, DM
- Admitted with SOB, DOE, leg swelling
- ?JVP “thick neck”, lungs clear, severe LE edema
- BNP 42 pg/ml, Cr 1.2 mg/dl
- IV diuresis, negative 3L by hospital day #3
- Echo: normal EF, ?filling pressures, ?DD grade
- Hospital day #4: HCO₃ 42, Cr 1.6 mg/dl... stop diuretics??
A typical HFpEF patient...

- Cardiology consult team:
  - Stop diuretics, give fluids, swelling all lymphedema “he's dry”
A typical HFpEF patient...

- Cardiology consult team:
  - Stop diuretics, give fluids, swelling all lymphedema “he's dry”
- STOP! Do a cardiac catheterization
  - RA 18, PA 64/28, PCWP 28, LVEDP 28
  - Lasix gtt started, diuresed 20L further
A typical HFpEF patient...

- Cardiology consult team:
  » Stop diuretics, give fluids, swelling all lymphedema “he's dry”
- STOP! Do a cardiac catheterization
  » RA 18, PA 64/28, PCWP 28, LVEDP 28
  » Lasix gtt started, diuresed 20L further
- Normal BNP does not rule out HFpEF
- ~30% of HFpEF with PCWP: normal BNP

Anjan V...Shah SJ. Am J Cardiol 2012
Myth #4: HFpEF is a single disease

Fact #4: HFpEF is a heterogeneous syndrome
The many faces of HFpEF
The many faces of HFpEF

HFpEF: not 1 single "disease"
Pathophysiologic contributors to HFpEF

- Diastolic dysfunction
- Longitudinal systolic dysfunction
- Chronotropic incompetence
- Extra-cardiac causes of volume overload
- Arterial stiffness
- Inflammation
- Pulmonary hypertension / RV failure
- Abnormal V-A coupling
- Endothelial dysfunction
- Autonomic dysfunction
- Skeletal muscle abnormalities

Clinical categories of HFpEF

1. “Garden-variety” HFpEF (HTN, DM, obesity, CKD)
2. CAD-HFpEF
3. Right heart failure-HFpEF
4. A-fib predominant HFpEF
5. HCM-like HFpEF
6. High-output HFpEF
7. Valvular HFpEF (multiple 2+ lesions)
8. Rare causes of HFpEF (“zebras”)

Oktay AA, Shah SJ. *Curr Cardiol Rev* 2014
3 types of HFpEF presentation

ENVIRONMENT, DIET \[\rightarrow\] COMORBIDITIES \[\rightarrow\] GENETIC SUSCEPTIBILITY \[\rightarrow\] VULNERABLE HEART, VULNERABLE PATIENT \[\rightarrow\] HFpEF

- EXERCISE-INDUCED \[\uparrow\text{LA PRESSURE}\]
- VOLUME OVERLOAD
- PULMONARY HTN, RV FAILURE

Shah SJ. JACC 2013
Risk profile, BNP vary by type of HFpEF presentation

Clinical course

EXERCISE-INDUCED DIASTOLIC DYSFUNCTION
VOLUME OVERLOAD
PULMONARY HYPERTENSION RV FAILURE

Shah SJ. JACC 2013
Myth #5: There are no proven treatments for HFpEF
State-of-the-art in 2016: Treatment of HFpEF
HFpEF: “no treatments”
Myth #5: There are no proven treatments for HFpEF

Fact #5: HFpEF is treatable, but we need to change the treatment paradigm.
Why have treatments failed?

• Multiple potential risk factors
• “Difficult diagnosis”
• Poor recognition of presence/prognosis
• Heterogeneity of HFpEF syndrome
  » Several pathophysiologic mechanisms
• Care by multiple different providers
• Comorbidity burden is high
  » Cause of death often not related to progressive heart failure
Rx Step #1: Prevent HFpEF before it even occurs
HFpEF can be prevented...

**HYVET trial**
indapamide resulted in
64% reduction
in HF hosp.
compared to
placebo

Beckett NS, et al. NEJM 2008
HFpEF can be prevented...

**ALLHAT-HFpEF**: chlorthalidone best for HFpEF prevention

![Graph showing cumulative HF rate over years to HF for different treatments.](image)

- **Chlorthalidone**
- **Amlodipine, lisinopril**

Rx Step #2: Before treating, remember the zebras
HFpEF: Know your zebras

• Assessment of HFpEF: a diagnostic mystery until proven otherwise
• Careful history, physical examination
• Clues to zebras:
  » Kussmaul’s sign: ↑JVP with inspiration
  » ↓Voltage ECG with ↑LV wall thickness
  » Careful evaluation of echo is essential

Oktay AA, Shah SJ. Curr Cardiol Rev 2014
50-year-old woman with SOB

Low voltage, pseudoinfarct pattern
50-year-old woman with SOB

Thick LV, “texture” of myocardium consistent with infiltrative cardiomyopathy
50-year-old woman with SOB

High E velocity, elevated E/A ratio, reduced E', ↓E deceleration time

Grade III (severe) LV diastolic dysfunction due to cardiac amyloidosis
44-year-old man with chronic ascites
44-year-old man with chronic ascites
44-year-old man with chronic ascites

Thickened, enhancing pericardium
HFpEF pearls: Etiology of HF?

31-year-old woman presents with dyspnea, lower extremity edema, BNP 166 pg/ml (while preparing for marathon)
31-year-old woman presents with dyspnea, lower extremity edema, BNP 166 pg/ml (while preparing for marathon)

Normal LV diastolic function

\[ e' = 15 \text{ cm/s}; \quad E/e' = 6 \]
HFpEF pearls: Etiology of HF?

31-year-old woman presents with dyspnea, lower extremity edema, BNP 166 pg/ml (while preparing for marathon)

Stroke volume = 100 ml / beat

\[ VTI_{LVOT} = 30 \text{ cm} \]
HFpEF pearls: High-output HF

31-year-old woman presents with dyspnea and lower extremity edema (while preparing for marathon)

**BASELINE**
LV EDVI = 85 ml/m²
LA volume index = 50 ml/m²

**12 DAYS LATER**
LV EDVI = 75 ml/m²
LA volume index = 37 ml/m²
31-year-old woman presents with dyspnea and lower extremity edema (while preparing for marathon)

**Baseline**
- LV EDVI = 85 ml/m²
- LA volume index = 50 ml/m²

**12 Days Later**
- LV EDVI = 75 ml/m²
- LA volume index = 37 ml/m²

**Thiamine supplementation**
HFpEF: Know your zebras

- **Restrictive cardiomyopathy:**
  - Sparkling myocardium
  - Severely decreased tissue Doppler s' or e'
  - Preserved radial function, reduced longitudinal function

- **Constrictive pericarditis:**
  - Diastolic septal bounce
  - Preserved e' velocity
  - Respiratory variation in mitral inflow

Oktay AA, Shah SJ. *Curr Cardiol Rev* 2014
## HFpEF: Know your zebras

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Constriction</th>
<th>Restriction</th>
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<tbody>
<tr>
<td>↑↑E velocity, ↑E/A E/A</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Short E decel time</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mitral inflow</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>respiratory variation</td>
<td></td>
<td></td>
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<tr>
<td>Tissue Doppler e' velocity</td>
<td>Normal or increased</td>
<td>Severely reduced</td>
</tr>
<tr>
<td>Hepatic vein imaging</td>
<td>Flow reversal during expiration</td>
<td>Flow reversal during inspiration</td>
</tr>
<tr>
<td>Simultaneous LV/RV tracings</td>
<td>Discordant</td>
<td>Concordant</td>
</tr>
</tbody>
</table>

Oktay AA, Shah SJ. *Curr Cardiol Rev* 2014
Rx Step #3: Treat comorbidities, BP, fluid overload
HFpEF treatment algorithm

• Diagnose HFpEF accurately
  » Remember that HFpEF is extremely common
  » Make sure you’re not dealing with a “zebra”
  » Low threshold for cardiac cath, CAD eval
• Treat the underlying cause of HFPEF
• Treat BP, fluid overload
• Treat comorbidities aggressively
• CHF education, chronic dz. management
Carvedilol
Bumetanide
Chlorthalidone
Lisinopril
Spironolactone

HFpEF “poly-pill”
Step #4: Tailor treatment to the type of HFpEF
Step #4: Tailor treatment to the type of HFpEF
EXERCISE-INDUCED ↑LA PRESSURE

- Exercise training
- Structure diet/weight loss
- Nitrates/nitrites?
- Ivabradine?
- Late Na+ current inhibitors (e.g., ranolazine)?
Interatrial shunt device for HFpEF

Creation of L-to-R shunt = ↓↓LAp at rest/exercise = ↓↓symptoms in HFpEF

InterAtrial Shunt Device: Concept

Transcatheter implant to create a small permanent interatrial shunt (Qp:Qs ratio 1.2-1.3)

Implant 19mm OD
8 mm ASD

Animal explant

Courtesy of Finn Gustafsson, MD, PhD, DMSci
• Elevated Cr during diuresis? Consider hemoconcentration
• Spironolactone
• Hemodynamic monitoring for tailored diuretic therapy
• Neprilysin inhibition? (PARAGON-HF trial)
• sGC stimulator therapy? (SOCRATES trial)
• Serelaxin for acute HF? (RELAX-AHF2 trial)
Spironolactone

- Great for volume overload, RV failure
- ALDO-DHF and RAAM-PEF:
  » Mineralocorticoid receptor antagonists probably don’t work in exercise-induced DD
- TOPCAT (N=3445):
  » Spironolactone vs. placebo for HFpEF
  » More volume overloaded than ALDO-DHF
  » ↓ hospitalization but missed 1° endpoint
  » *In Americas, spironolactone = better outcomes*
1° Outcome
(CV Death, HF Hosp, or Resuscitated Cardiac Arrest)

Spironolactone vs Placebo

HR = 0.89 (0.77 – 1.04)

p=0.138

351/1723 (20.4%)
320/1722 (18.6%)

(AHA 2013, NEJM 2014)
Placebo Rates: Primary Outcome, by region

**Americas N=1767 (51%)**
- US: N=1151
- Canada: N=326
- Brazil: N=167
- Argentina: N=123

- Placebo: 280/881 (31.8%)
- 12.6 per 100 pt-yr

**Russia/Georgia N=1678 (49%)**
- Russia: N=1066
- Rep Georgia: N=612

- Placebo: 71/842 (8.4%)
- 2.3 per 100 pt-yr

AHA 2013
Systolic Blood Pressure Change by Region

Average SBP Change (Spiro-Placebo)  
-4.2 mmHg (p<0.001)  
-0.6 mmHg (NS)

SBP delta differed by region (p<0.001), adjusted
Potassium Change by Region

![Graph showing potassium levels by region and treatment group over months.](image)

- **Russia/Georgia**
  - Placebo: Blue solid line
  - Spironolactone: Blue dashed line

- **Americas**
  - Placebo: Red solid line
  - Spironolactone: Red dashed line

*Interaction p < 0.001*
Placebo vs. Spiro by Region

HR = 0.82 (0.69-0.98)

US, Canada, Argentina, Brazil

Interaction p = 0.122

Russia, Rep Georgia
HR = 1.10 (0.79-1.51)

Placebo:
280/881 (31.8%)
71/842 (8.4%)

Spiro:
242/886 (27.3%)
78/836 (9.3%)
CHAMPION TRIAL

LA pressure = improved outcomes in HFrEF

Adamson PB, et al. Circulation Heart Failure 2014
PULMONARY HYPERTENSION
RV FAILURE

- Aggressive diuresis, ultrafiltration often needed
- May need to discontinue systemic vasodilators
- Midodrine for low BP during diuresis (if not contraindicated)
- Digoxin to RV inotropy
- PDE5 inhibition if PADP-PCWP gradient > 5 mmHg
- Hemodynamic sensor for careful titration of diuretics
Treatment of PH-HFpEF

Treatment targets: LA, PA, RV

RV therapies
- Digoxin?
- Ranolazine?
- Istaroxime?
- Myosin activators?

Pulmonary vasodilators
- sGC stimulators?
- PDE5 inhibitors?
- ERAs?
- Prostacyclins?

LA assist device?
Treatment of PH-HFpEF

Treatment targets: LA, PA, RV

- RV therapies
  - Digoxin?
  - Ranolazine?
  - Istaroxime?
  - Myosin activators?

- Pulmonary vasodilators
  - sGC stimulators?
  - PDE5 inhibitors?
  - ERAs?
  - Prostacyclinls?

- LA assist device?
Renal venous congestion in PH-HFpEF

Many PH-HFpEF patients have RV failure:

\[
\text{RA pressure} = \text{renal venous pressure} + \text{CO} = \text{systemic BP} \downarrow \text{renal blood flow} = \text{transrenal pressure gradient}
\]
Renal venous congestion in PH-HFpEF

Many PH-HFpEF patients have RV failure:

†RA pressure = †renal venous pressure

+ ‡CO = ‡systemic BP

‡renal blood flow

= ‡transrenal pressure gradient

• Diuretics
• Stop anti-HTN meds
• Midodrine
• Pulmonary vasodilators?
HFpEF treatment pearls

1. “Garden-variety”-HFpEF: Rx BP, DM, obesity, refer for clinical trial; If AF → trial of cardioversion
2. CAD-HFpEF: Rx like HF w/reduced EF (BB, ACE-I/ARB, revasc)
3. Right heart failure-HFpEF: diuresis / ultrafiltration, digoxin, sildenafil?
4. HCM-HFpEF: verapamil, diltiazem, long-acting metoprolol
5. High-output HFpEF: Rx underlying cause; diuretics/UF
6. Valvular HFpEF: Rx valve disease if possible
7. Rare causes of HFpEF: clinical trial, Rx underlying cause
Myth #6: HFpEF clinical trials are doomed

Fact #6: The future is bright for HFpEF clinical trials
HFpEF survival: poor

Dismal 35% survival at 5 years after HF hospitalization, regardless of LVEF

HFpEF survival: poor

HFpEF survival: comparable to T4 non-small cell lung cancer, stage 3B or worse

Current HFpEF clinical trials

- PARAGON-HF: neprilysin inhibition
- SOCRATES-Preserved: sGC stimulator
- ATTR-ACT: transthyretin stabilizer
- ENDEAVOUR: transthyretin RNAi
- LIBERTY-HCM: late $I_{Na^+}$ inhibitor
- REDUCE-LAP: interatrial shunt device
- (INDIE-HF): inhaled nitrites
- (KNO₃CK-OUT): oral nitrites
ARNIs: Angiotensin Receptor / Neprilysin Inhibitors

Physiological response

NP system

RAS

Pathophysiological response

NPs

Vasodilation
- BP
- Sympathetic tone
- Aldosterone
- Fibrosis
- Hypertrophy
- Natriuresis

Vasoconstriction
- BP
- Sympathetic tone
- Aldosterone
- Fibrosis
- Hypertrophy

HF symptoms/progression

Ang II

- AT₁ receptor

Inactive fragments

Neprilysin

ARNI

Inactive fragments†
Hypothesis

Dr. A. Sauer, Dr. S. Shah, Northwestern

Soluble guanylate cyclase stimulators will:

» Increase cGMP → improve calcium handling

» Decrease heterogeneity of APD-CaT delay
Hypothesis

Dr. A. Sauer, Dr. S. Shah, Northwestern

- Soluble guanylate cyclase stimulators will:
  - Increase cGMP → improve calcium handling
  - Decrease heterogeneity of APD-CaT delay

PDE5 inhibitors

Endothelial Dysfunction

Oxidative Stress

Inflammation

sGC Insufficiency

sGC Stimulators

eNOS

GTP

cGMP

NO

Endothelial NO Synthase

cGMP

cyclic Guanosine Monophosphate

Myocardial Dysfunction

Impaired Relaxation, Diastolic Stiffening, Energy Wastage

Vascular Dysfunction

Disturbed Endothelium-Dependent Vasotone Regulation
Nitrites are very different than nitrates

Endothelial dysfunction plays a central role in HFpEF

Nitrites improve endothelial function

Nitrates may actually worsen endothelial function via increased ROS

Unlike nitrates, there is strong preliminary data for nitrites in HFpEF (both oral and inhaled forms)
**ISMN and Nitrite are very different**

<table>
<thead>
<tr>
<th></th>
<th>Isosorbide mononitrate (ISMN)</th>
<th>Nitrite</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Activation</strong></td>
<td>P450 enzymes in the endoplasmic reticulum</td>
<td>Heme-containing proteins, XO, others</td>
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<tr>
<td><strong>NO elaboration</strong></td>
<td>Tonic - Throughout the day</td>
<td>Intermittent - Coupled to tissue hypoxia, exercise</td>
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<tr>
<td><strong>Tolerance</strong></td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td><strong>Endothelial Dysfunction</strong></td>
<td>Yes</td>
<td>No</td>
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<tr>
<td><strong>↑ROS</strong></td>
<td>Yes</td>
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“Matchmaking” for optimizing HFpEF clinical trials

EXERCISE-INDUCED DIASTOLIC DYSFUNCTION ↔ VOLUME OVERLOAD ↔ PULMONARY HYPERTENSION RV FAILURE

THEORETICAL “MATCHED” THERAPIES

- Ivabradine
- Aldosterone blocker
- Neprilysin inhibitor
- PDE5 inhibitor
- Exercise training
- Hemodynamic sensor

Shah SJ. JACC 2013
“Matchmaking” for optimizing HFpEF clinical trials

THEORETICAL “MATCHED” THERAPIES

- Ivabradine
- Neprilysin inhibitor
- PDE5 inhibitor
- Inorganic nitrates
- sGC stimulator
- Hemodynamic sensor
- Interatrial shunt device
- Neprilysin inhibitor
- Aldosterone blocker
- sGC stimulator
- Hemodynamic sensor
- Ivabradine
- Neprilysin inhibitor
- PDE5 inhibitor
- Inorganic nitrates
- sGC stimulator
- Hemodynamic sensor

Shah SJ. JACC 2013
STOP!

STOP!
STOP!

1. Make sure you didn’t miss dx of HFpEF

2. Don’t forget the zebras

3. Categorize by type of HFpEF presentation and tailor treatment

4. There are treatment options for HFpEF!

5. Enroll in HFpEF clinical trials
thank you!