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Heart Failure Care in 2026: Case-Based Practical Approach to Inpatient Management

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DHM Weekly Meeting
February 19th, 2026

Outline

1. Scope of the problem with emphasis on Kentucky
2. Case-based conundrums:
    A. Cardio-renal syndrome: the tug-of-war
    B. Quadruple therapy in HFrEF: are we actually doing it?
    C. HFpEF: is it finally treatable?
    D. Atrial fibrillation and HF: new evidence, old pitfalls
    E. Advanced heart failure: what is it, and when is it time to refer?
3. Take Home Points

Heart Failure by the Numbers

An estimated 64.3 million people are living with HF worldwide

An estimated 6 million Americans ≥ 20 years of age have HF with ~1 million new cases occurring annually in people ≥55 years of age

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HF, heart failure.

1. GBD 2017 Disease and Injury Incidence and Prevalence Collaborators. *Lancet*. 2018;392(10159):1789-1858. 2. Groenewegen A, et al. *Eur J Heart Fail*. 2020;22(8):1342-1356. 3. Virani SS, et al. *Circulation*. 2021;143:e254-e743. 4. Heidenreich PA, et al. *Circ Heart Fail*. 2013;6:606-619.

Risk of HF is Increasing

Mean age of population is on the rise

Rates of obesity and severe obesity are on the rise

Rates of pre-diabetes and diabetes are on the rise

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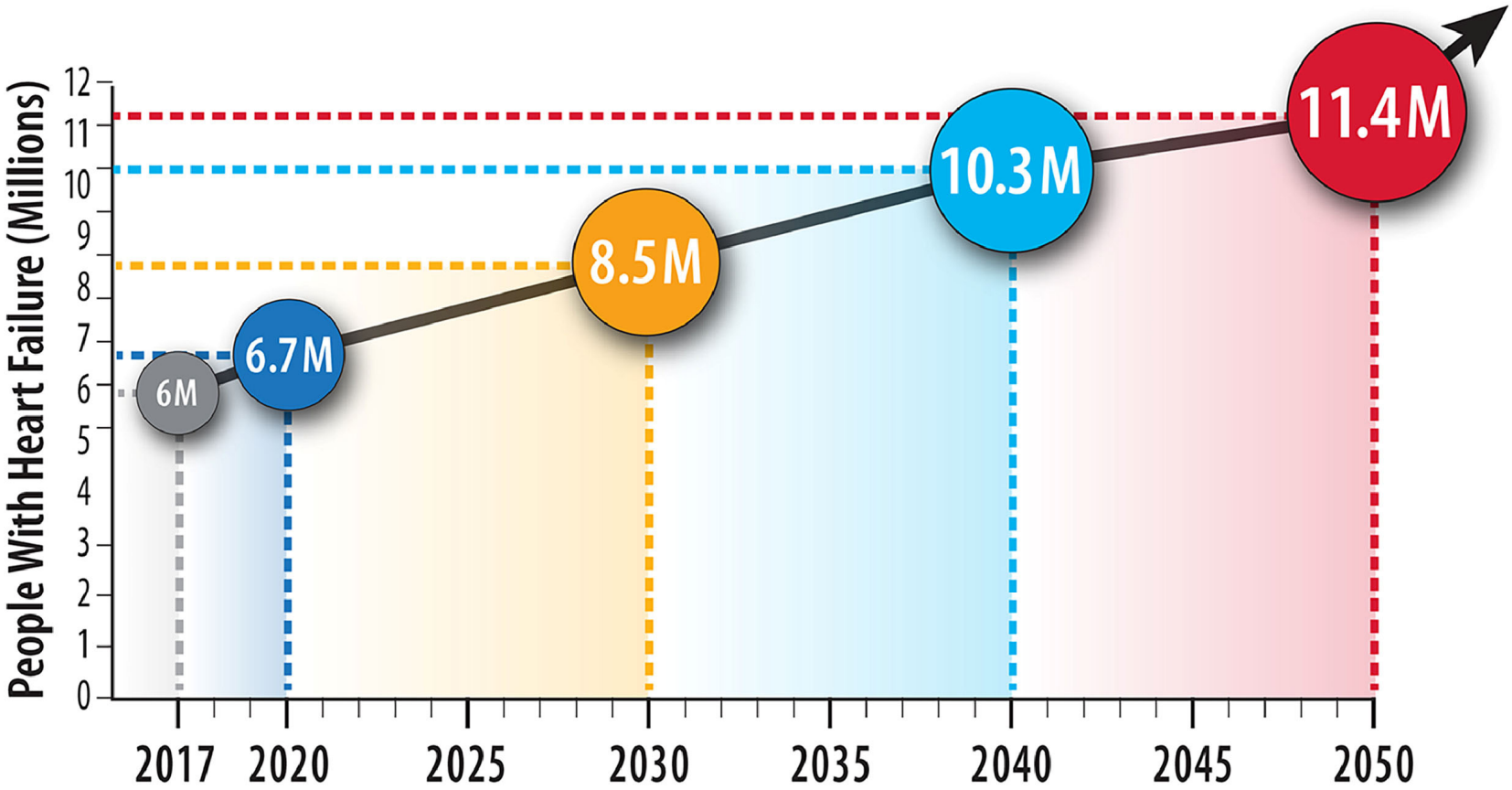
Ogden et al, Hirode et al, JAMA 2020

Scope of the Problem: Prevalence of HF



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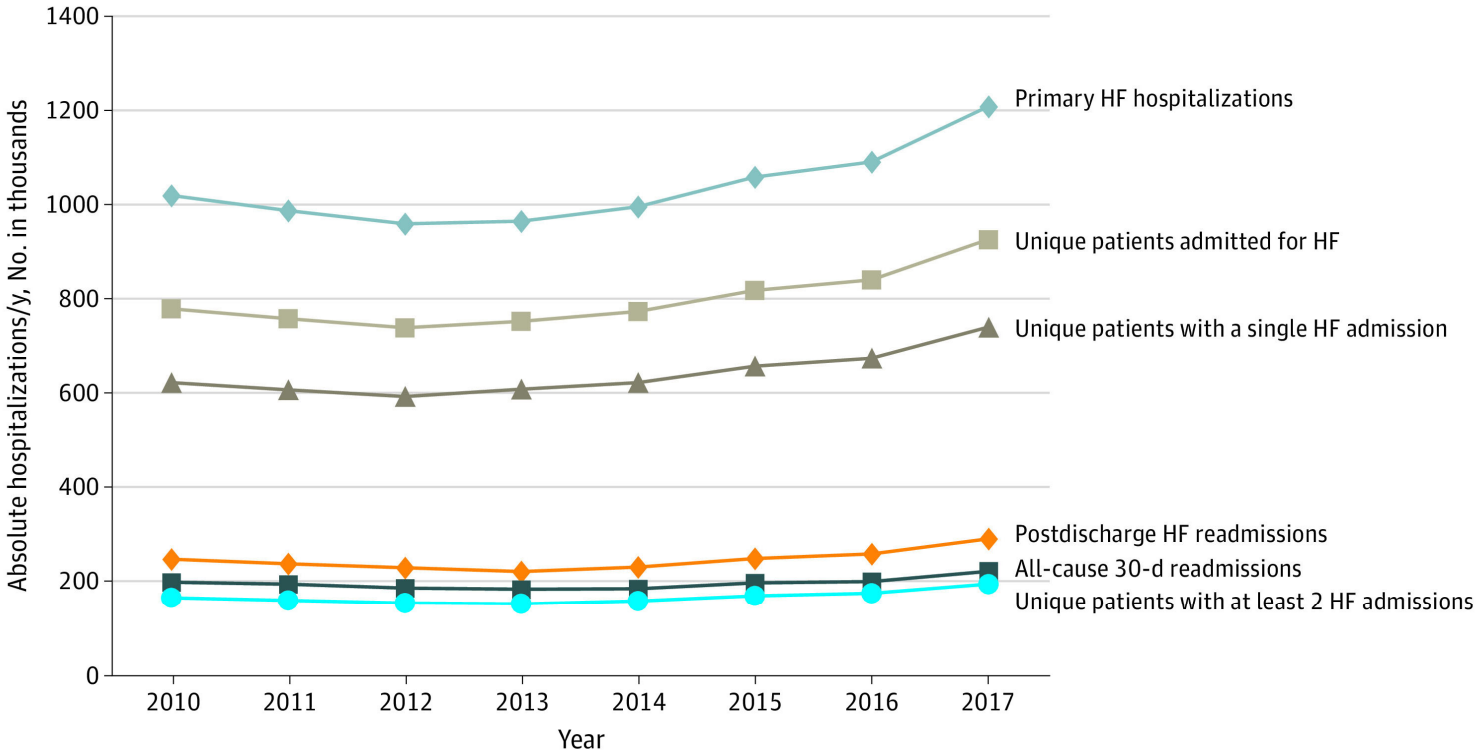
Bozkurt et al. HF STATS 2024: HF Epidemiology and Outcomes Statistics An Updated 2024 Report from the HFSA. JCF. 2024

Trends in HF Hospitalization



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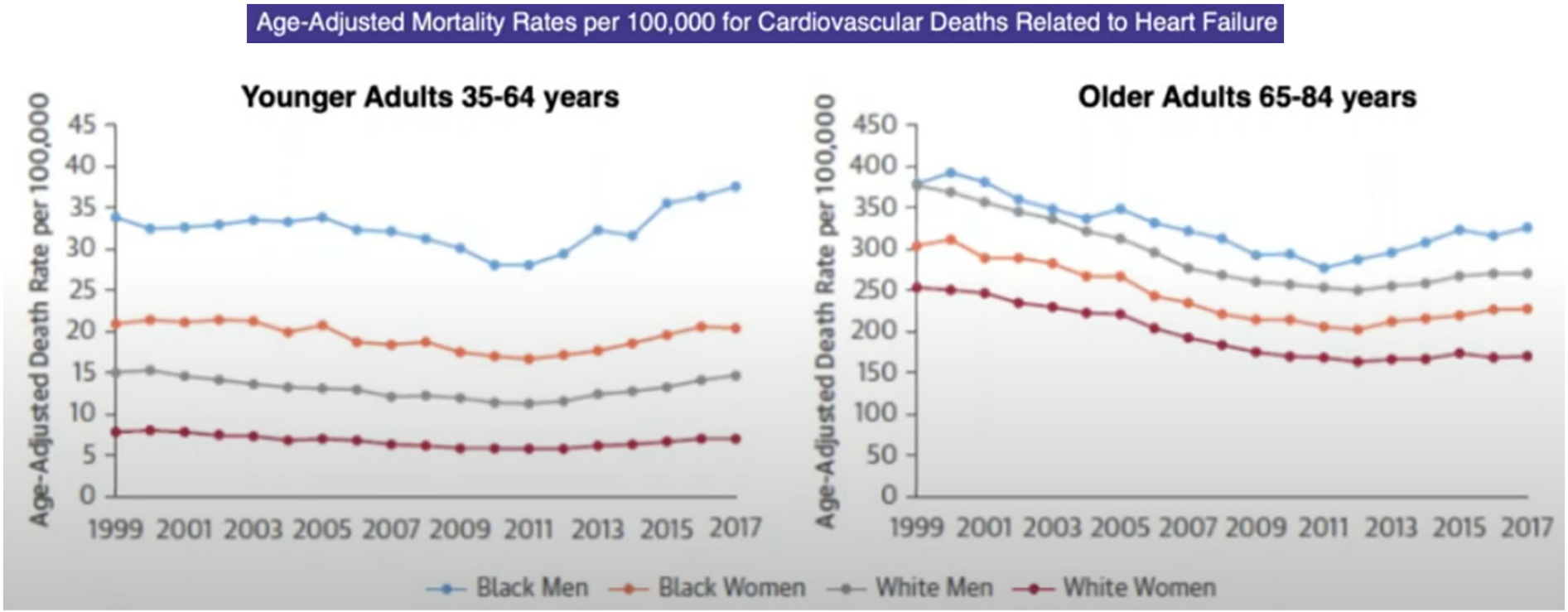
Agarwal et al. JAMA Cardiology 2021

Trends in HF Mortality in US



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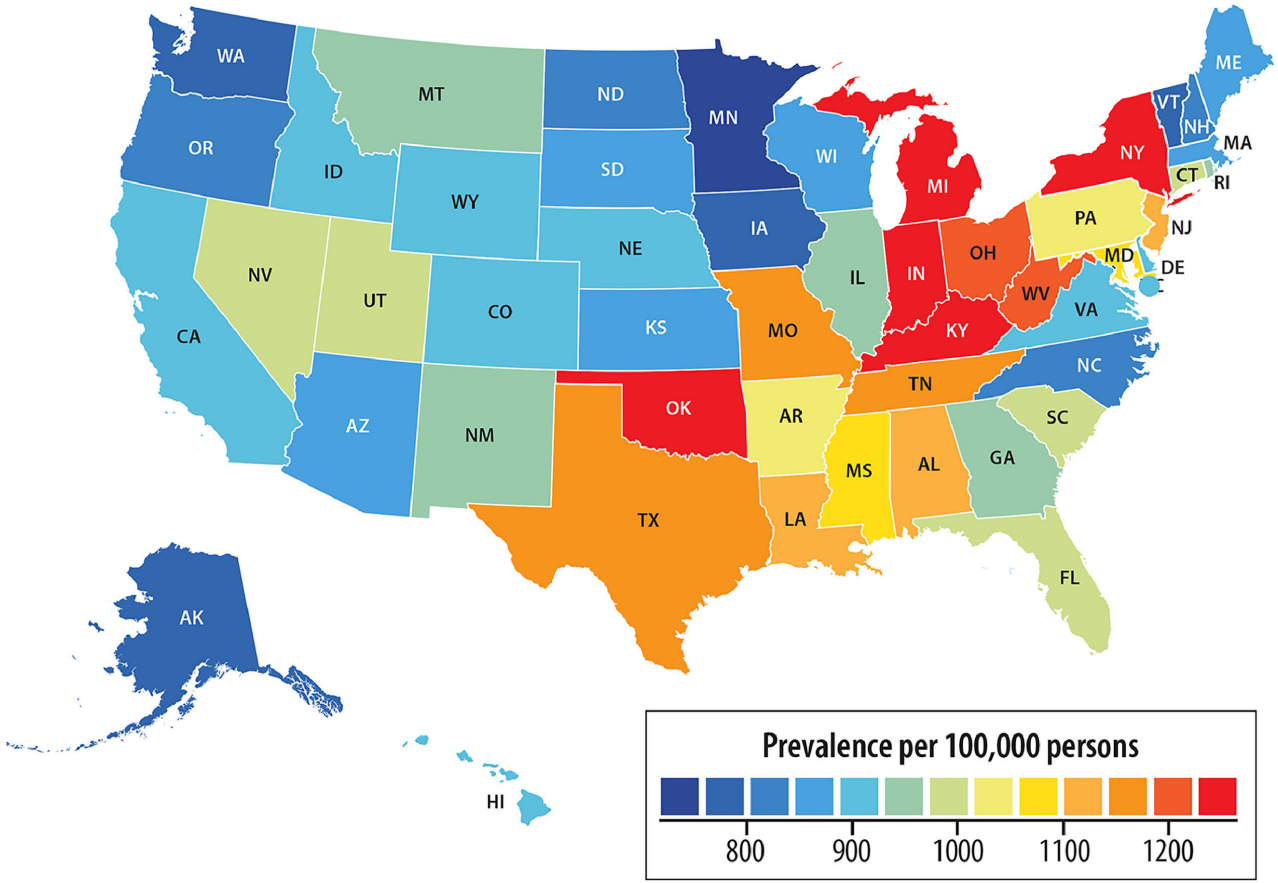
Geographic Variations in Prevalence of HF



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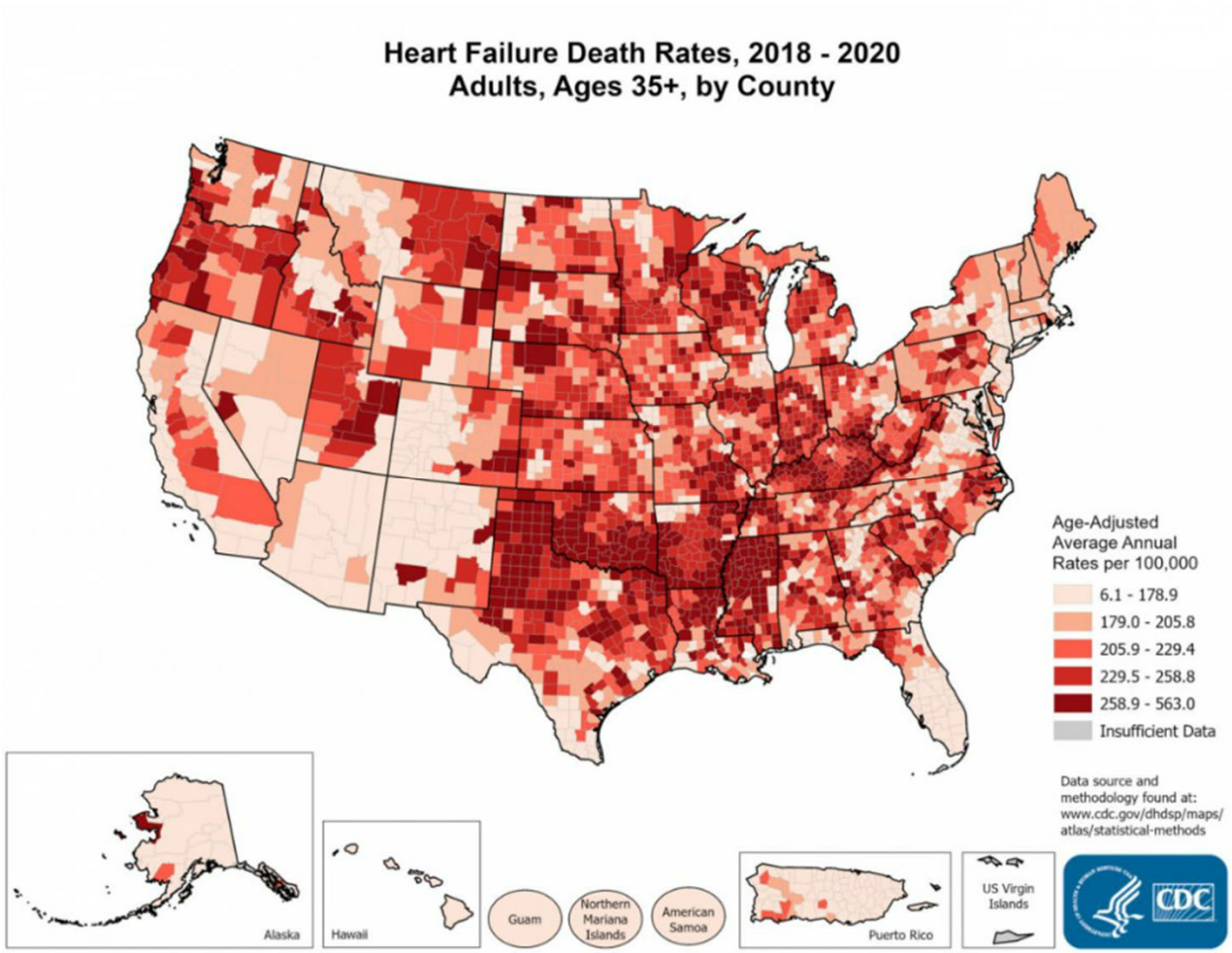
Heart Failure is a Public Health Emergency



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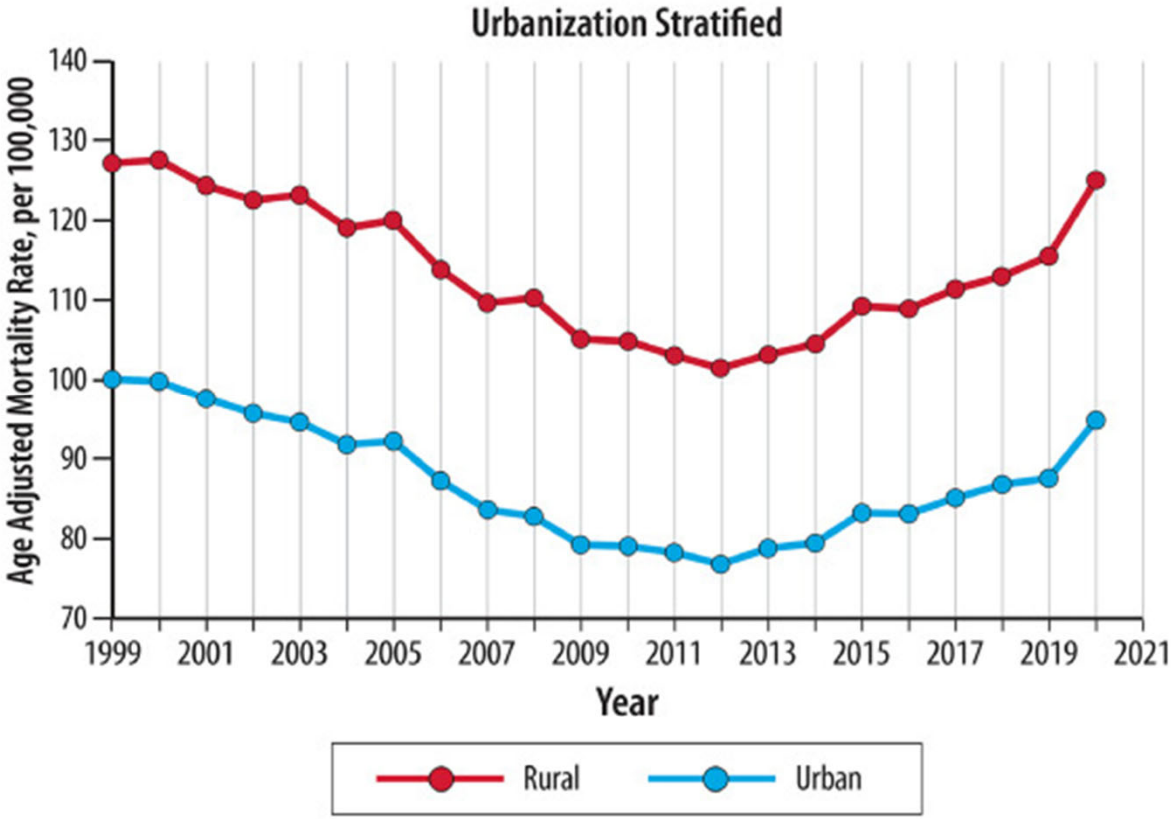
Higher Mortality Rates in Rural Areas



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Bozkurt et al. HF STATS 2024: HF Epidemiology and Outcomes Statistics An Updated 2024 Report from the HFSA. JCF. 2024

Mismatch in HF Care

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Case 1: Cardio-Renal Syndrome - The Tug-of-War

68 YO M with HFrEF (LVEF 25%), CAD, CKD stage 2 admitted with HF exacerbation.

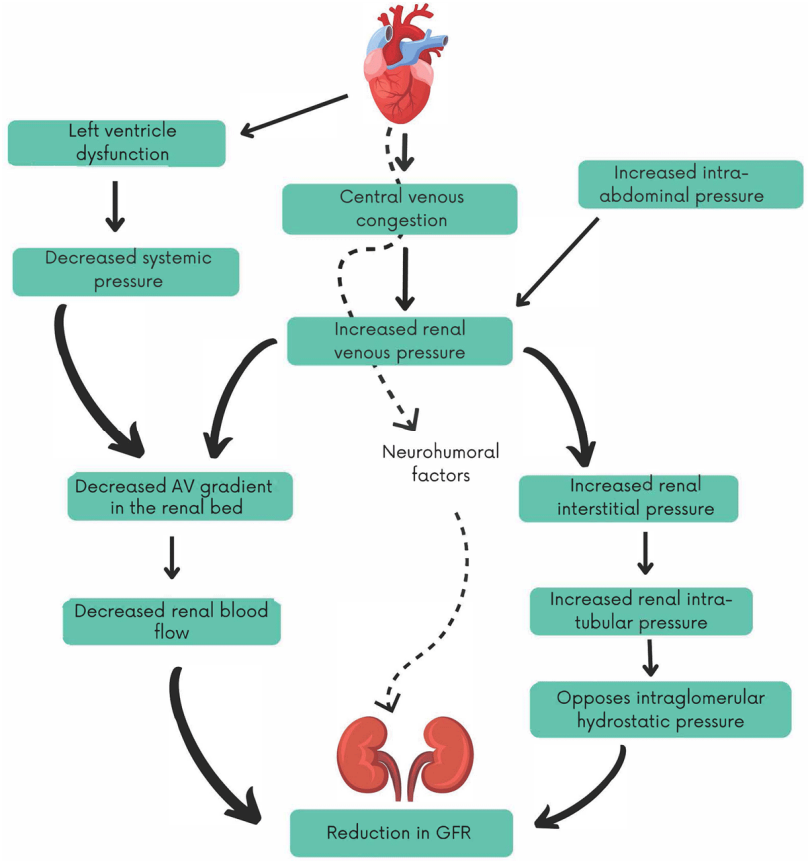
On admission, Cr was 1.9 (baseline of 1.7) and with initiating IV diuresis continues to rise to 2.6 in spite of modest UOP. He still appears edematous and has orthopnea and PND.

Question: What should you do?

(A) Keep pushing + add booster (thiazide)

(B) Slow down/hold diuretics

Rise in Cr with diuresis with Decongestion: What's Really Happening?



1. Removal of fluid shifts intravascular volume causing transient hemodynamic changes in glomerular filtration pressure

2. Venous pressure improvement enhances renal blood flow over time, but short-term creatinine may rise as plasma volume falls

3. Persistent high right-sided pressures and congestion, if *not relieved*, independently worsen renal function

4. **True Renal Injury**: rare with aggressive diuresis; studies using specific biomarkers show that creatinine fluctuations often represent functional filtration changes rather than structural kidney damage

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UK HFrEF Clinical Pathway

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Heart Failure Reduced Ejection Fraction (HFrEF) Clinical Pathway

Goal

Standardize the identification and inpatient treatment of patients presenting with heart failure reduced ejection fraction in order to reduce symptoms and optimize their short-term and long-term outcomes while simultaneously minimizing complications, reducing length of stay and reducing readmissions.

Patient Population

- Heart failure is a clinical syndrome with symptoms or signs caused by a structural and/or functional cardiac abnormality and corroborated by elevated natriuretic peptide levels and/or objective evidence of pulmonary or systemic congestion

Note: This Clinical Pathway is intended to serve as a provider driven standardized plan of care for HFrEF

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Diuretic Management Trajectory

Guidance statements

- Monitor strict I/O's with daily standing weights in the morning to follow this algorithm
- Obtain BMP and Mag twice daily; If potassium is above 3.5, you may discontinue checking twice daily unless on continuous IV diuretic therapy. Closely monitor for electrolyte replacement needs and schedule as needed.
- If urine output reaches above 4-5L, and/or patient becomes hypotensive, consider reducing dose or holding diuretic
- ***Consider consulting cardiology for: known diuretic resistance on presentation, significant history of diuretic resistance (previous clinical pathway failure), diuresis but continual fluid overload, congestion, worsening SCr that rises greater than 1.5x in 24hrs, or showing signs/symptoms of hemodynamic decline*
- ***Consult nephrology for EGFR<60, continual electrolyte imbalances*

Diuretic Naïve

Administer:

- 1mg to 2mg of bumetanide IV
  OR
- 40mg to 80mg furosemide IV

Pre-existing Diuretic Therapy

Administer:

- IV loop diuretic equivalent to 2x patient's home dose according to equivalency table

Reassess patient's urine output and response to diuretics in 4 to 8 hours

Urine Output > 100 ml/hr

Administer:

- Repeat previous diuretic IV dose until symptoms improve

Urine Output < 100 ml/hr

Administer:

- Double previous diuretic IV dose

Reassess patient's urine output and response to diuretics in 4 to 8 hours

Consider nephrology consult

Urine Output > 100 ml/hr

Urine Output < 100 ml/hr

Administer:

- Repeat previous diuretic IV dose
- Metolazone 2.5mg to 5mg PO

Reassess patient's urine output and response to diuretics in 4 to 8 hours

Urine Output > 100 ml/hr

Urine Output < 100 ml/hr (hours 24 - 48)

Administer:

- Continue daily metolazone PO
- Bolus and begin drip of same diuretic (bolus dose will be double hourly drip dose)
- Drip Dosing:
  - —Furosemide— Start drip at 20mg/hr and increase up to 40mg/hr if needed
  - —Bumetanide— Start drip at 1 mg/hr and increase up to 2mg/hr if needed

If patient has continued drip for 24 hours, reached maximum drip dose, and is still not meeting goal of 1.5 to 2 liters off in last 24 hours, consult cardiology or nephrology depending on primary organ dysfunction

Case 1 Revisited: Cardio-Renal

Care plan:

1. Increased IV diuretic dosing
2. Added daily Metolazone (thiazide-like diuretic)

		Jan 28 - Jan 29 0700 - 0659	Jan 29 - Jan 30 0700 - 0659	Jan 30 - Jan 31 0700 - 0659	Jan 31 - Feb 1 0700 - 0659	Feb 1 - Feb 2 0700 - 0659	Feb 2 - Feb 3 0700 - 0659	Feb 3 - Feb 4 0700 - 0659
In	P.O.	1,054	2,590	1,560	1,540	1,100		840
	I.V. (mL/kg)	172 (1.2)	134 (1)	95.4 (0.7)	71.1 (0.6)	30.5 (0.3)		532 (4.4)
	IV Piggyback		50					
	Total Intake (mL/kg)	**1,226** (8.4)	**2,774** (20.5)	**1,655.4** (13)	**1,611.1** (13.3)	**1,130.5** (9.4)		**1,372** (11.4)
Out	Urine (mL/kg/hr)	6,400	12,150 (3.7)	10,601 (3.5)	9,100 (3.1)	5,300 (1.8)	3,080 (1.1)	3,650 (1.3)
	Stool	1						
	Total Output	**6,401**	**12,150**	**10,601**	**9,100**	**5,300**	**3,080**	**3,650**
Net	**I/O**	**-5,175**	**-9,376**	**-8,945.6**	**-7,488.9**	**-4,169.5**	**-3,080**	**-2,278**

Weight
120 kg (264 lb 8.8 oz)
120 kg (264 lb 8.8 oz)
121 kg (266 lb 12.1 oz)
121 kg (265 lb 10.5 oz)
121 kg (266 lb 1.5 oz)
128 kg (281 lb 8.4 oz)
135 kg (298 lb 1 oz)
146 kg (322 lb 1.5 oz)
153 kg (336 lb 10.3 oz)

2.14
1.98
1.81
1.84
2.27
2.03
2.07
2.27
2.42
2.38
2.64
2.03
1.89

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Case 1: Cardio-Renal Pearls

1. ADHF admission: decongestion > creatinine for prognosis
2. Start IV loop diuretic at ≥1–2× home oral dose, reassess UOP in 6 hours to re-dose; target net ≥ 2L daily and add thiazides for synergy if needed
3. Mild-moderate transient rise in Cr is acceptable -> inadequate decongestion drives readmissions and mortality
4. Consult Cardiology/Nephrology (cardiorenal service and clinic) for help
5. Stop/reduce diuresis if:
   - Significant hypotension
   - Severe electrolyte derangements
   - True hypovolemia

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Case 2: Quadruple Therapy in HFrEF - Are We Actually Doing It?

57 YO F with poorly controlled HTN and DMII presents with orthopnea, PND, 20 lb weight gain and bilateral LE edema.

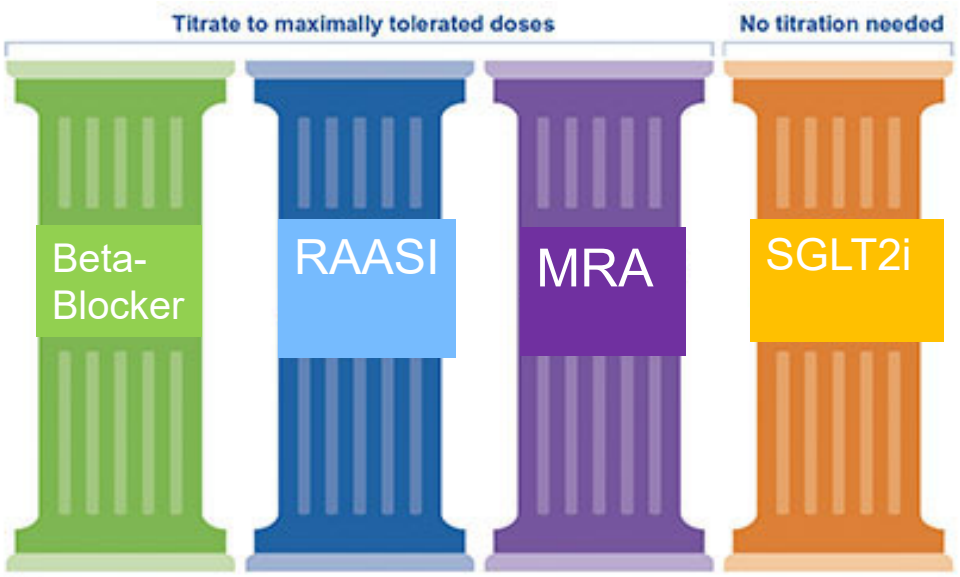
- Vital signs: BP 142/92 mmHg, HR 105 BPM, 93% O2 saturation
- Labs: NT-proBNP 5000, Cr 1.4 (baseline <1)
- TTE: Dilated LV cavity to 5.6 cm, LVEF ~25%, moderate MR

Question: How do you approach treating this patient?

2022 HF Guidelines - Initiate and Up-titrate GDMT According to LVEF

HF with reduced EF (HFrEF)

- *HF with **LVEF < 40%***



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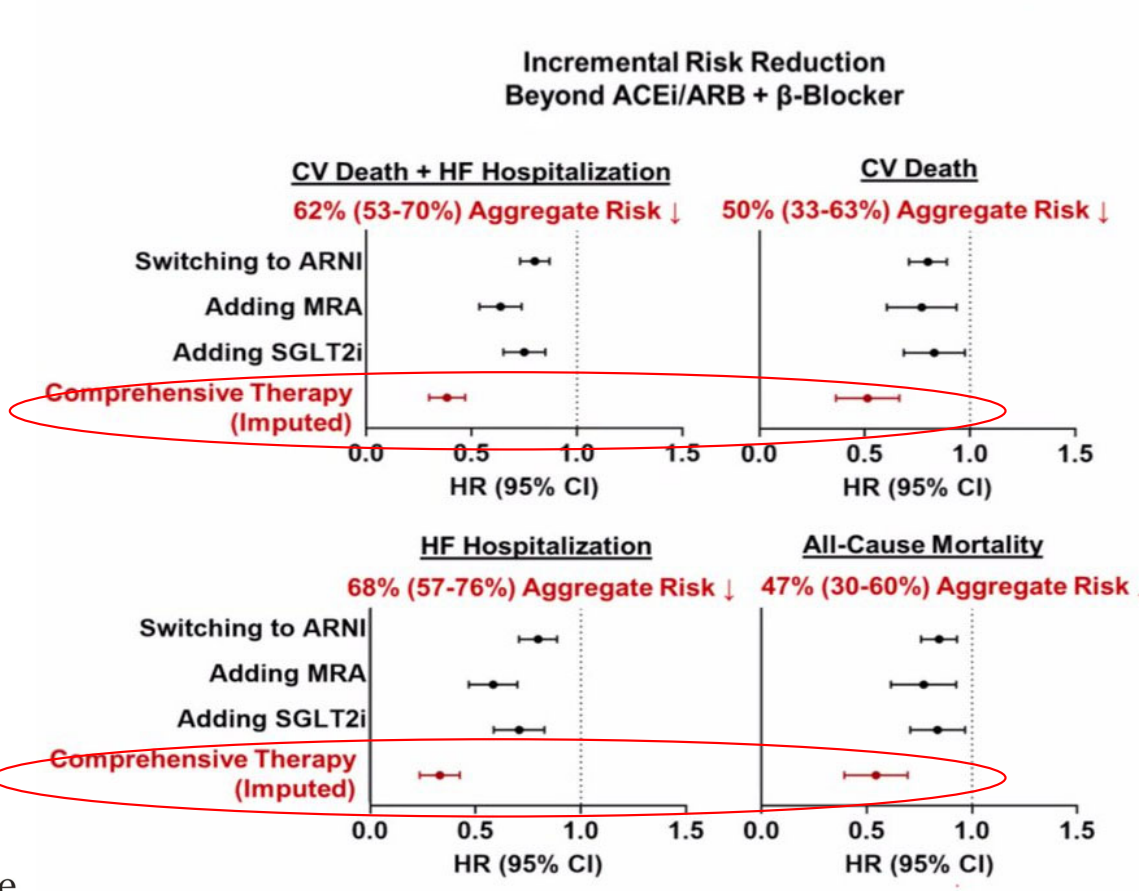
https://www.nhcs.com.sg/news/murmurs/for-physicians-management-of-heart-failure-2022

Expected Lifetime Benefit of Comprehensive Disease-Modifying Therapy in HFrEF



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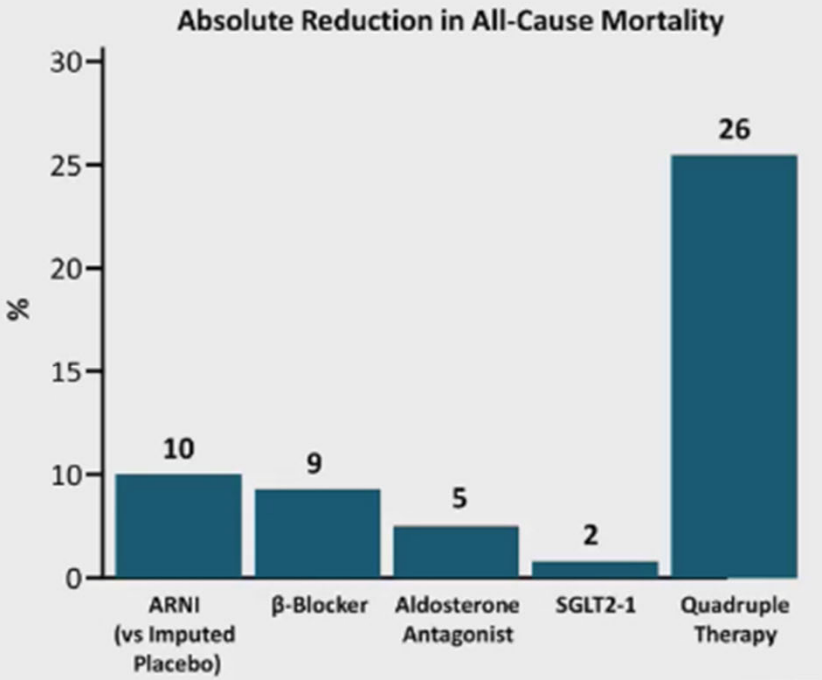
Vaduganathan M et al. *Lancet* 2020;396:121–128 (including supplementary appendix).

Quadruple Therapy Saves Lives!

Evidence-Based Therapy	Relative Risk Reduction in All-Cause Mortality, %
None	N/A
ARNI (vs imputed placebo)	28
β-blocker	35
Aldosterone antagonist	30
SGLT2i	17
Quadruple therapy	73



Bassi. JAMA Cardiol. 2020;5:948.

Slide credit: clinicaloptions.com

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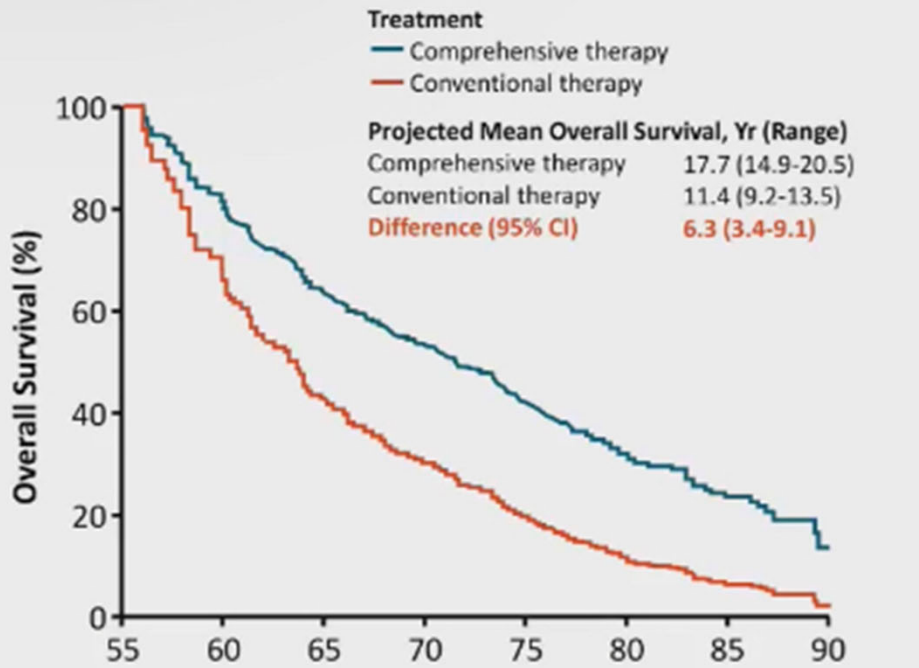
Quadruple Therapy Saves Lives!

- Crosstrial analysis
  - EMPHASIS-HF
  - PARADIGM-HF
  - DAPA-HF
- ARNI/β-blocker/MRA/SGLT2i vs ACEI/β-blocker

"A new therapeutic standard"



Vaduganathan. Lancet. 2020;396:P121.

Slide credit: clinicaloptions.com

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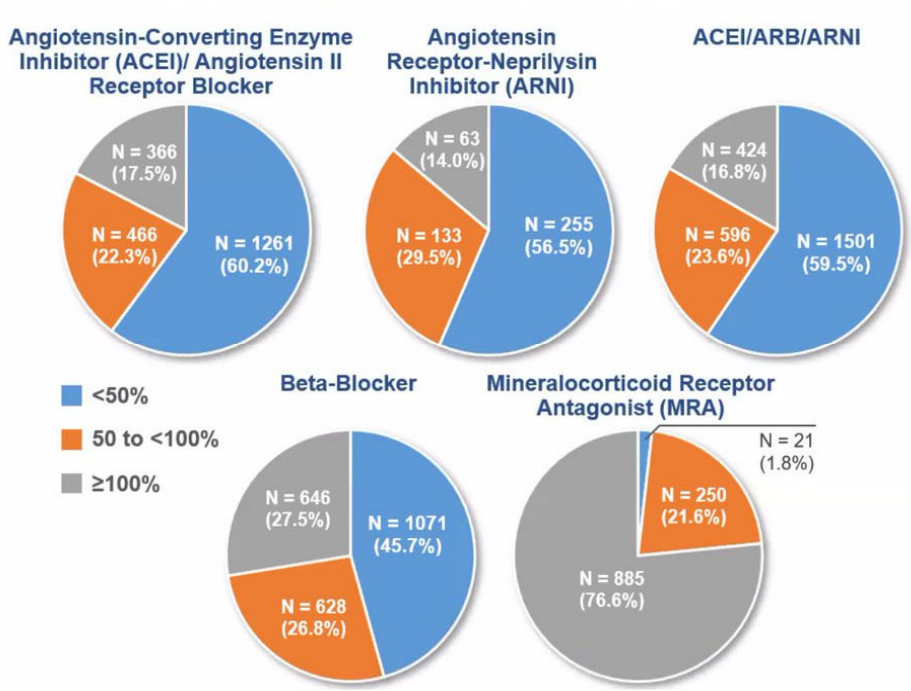
CHAMP-HF Results: The Grim Reality of GDMT



- <25% received all three GDMT classes (ACE/ARB/ARNI, BB, MRA).
- When prescribed, the largest % of doses were <50% target.
- Only 1% received target doses of all three GDMT classes.
- As in other studies a risk-therapy mismatch was present.

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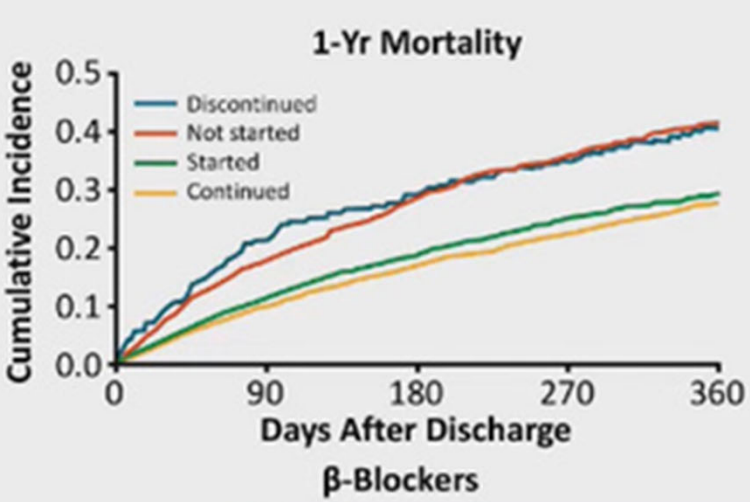
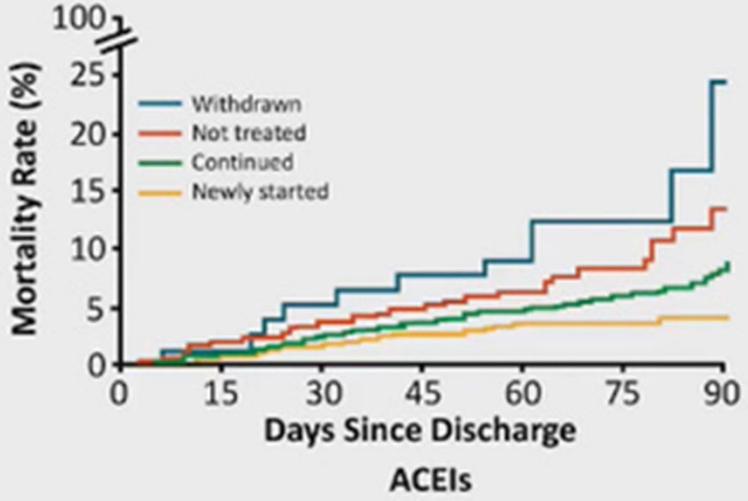
Inpatient GDMT is Important





Risks Associated With Failure to Continue/Initiate/Switch GDMT During Hospitalization

- ↑ risk of readmission and short-, intermediate-, and long-term mortality
- ↓ medication adherence and ↓ medication persistence
- Substantially ↑ likelihood of never being initiated or switched to GDMT as outpatient
- Missing out on teachable moment during hospitalization

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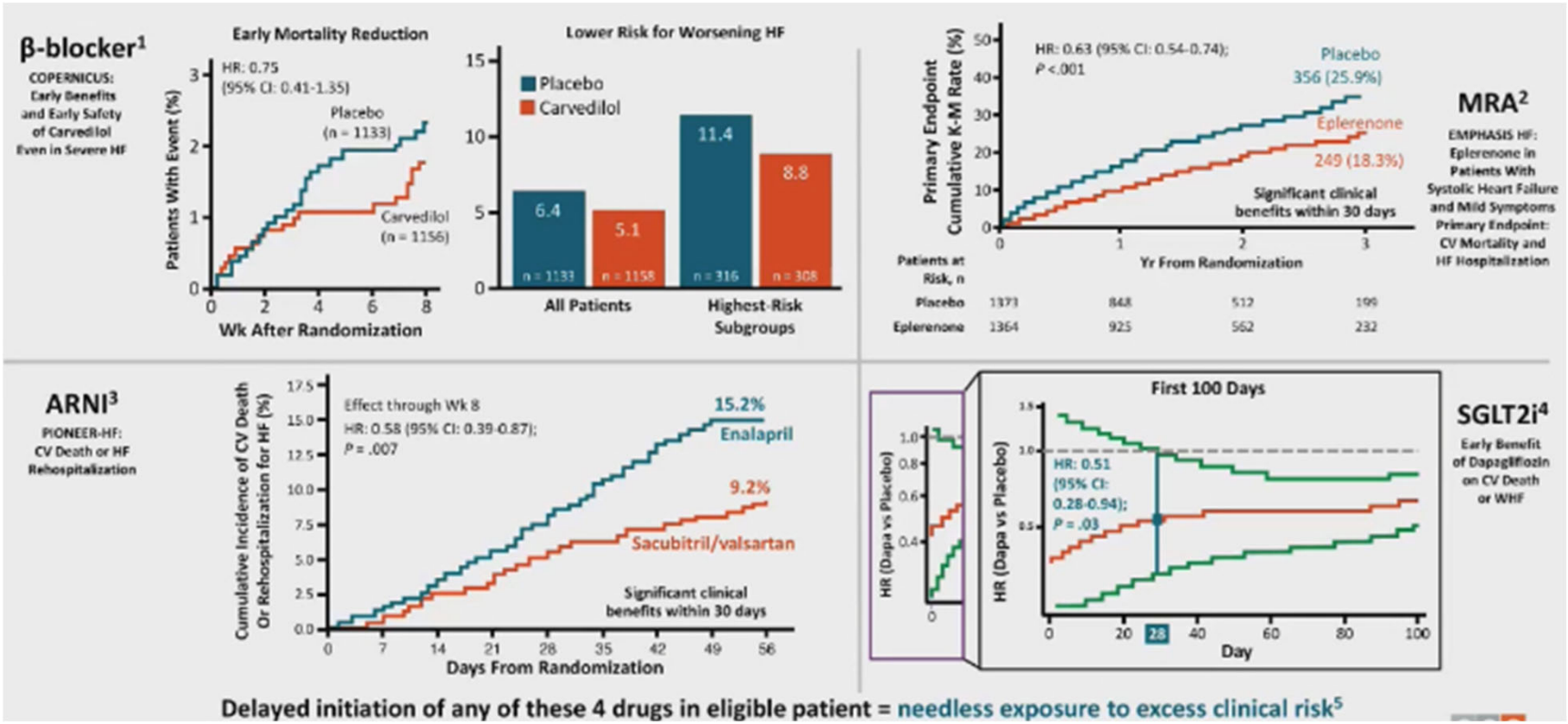
Fonarow. J Am Coll Cardiol. 2008;52:190. Gilstrap. J Am Heart Assoc. 2017;6:e004675.

Timing Matters - Clinical Benefits Appear Within Days to Weeks of GDMT Initiation



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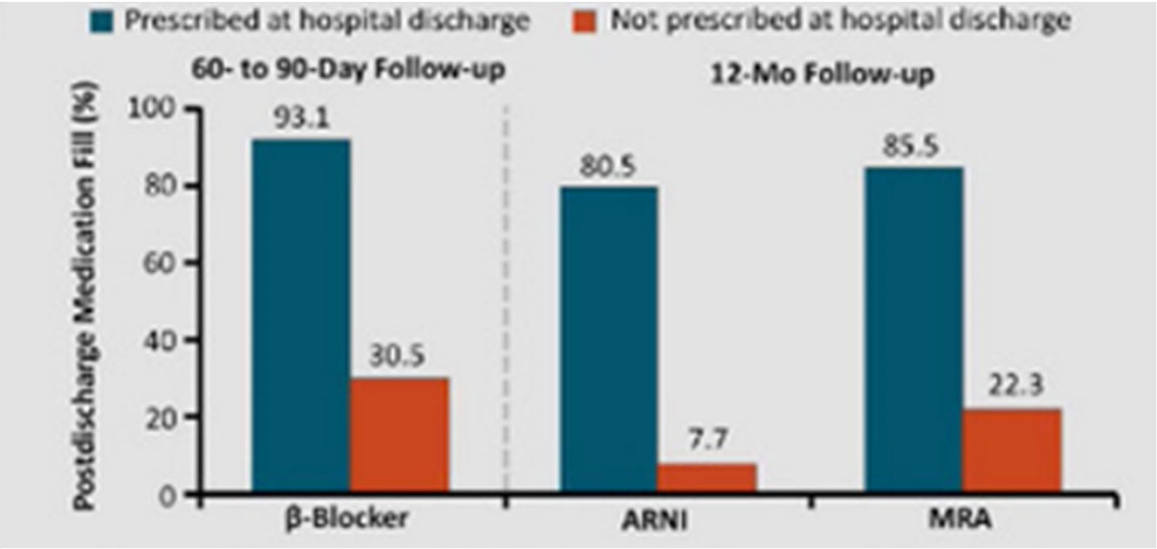
1. Krum. JAMA. 2003;289:712. 2. Zannad. NEJM. 2011;364:11. 3. Morrow. Circulation. 2019;139:2285. 4. Berg. JAMA Cardiol. 2021;6:499. 5. Greene. JAMA Cardiol. 2021;6:743.

Deferring Initiation Leads to No Initiation or Substantial Delay



Failure to initiate GDMT during admission = 75% chance medications will not be started in the next calendar year

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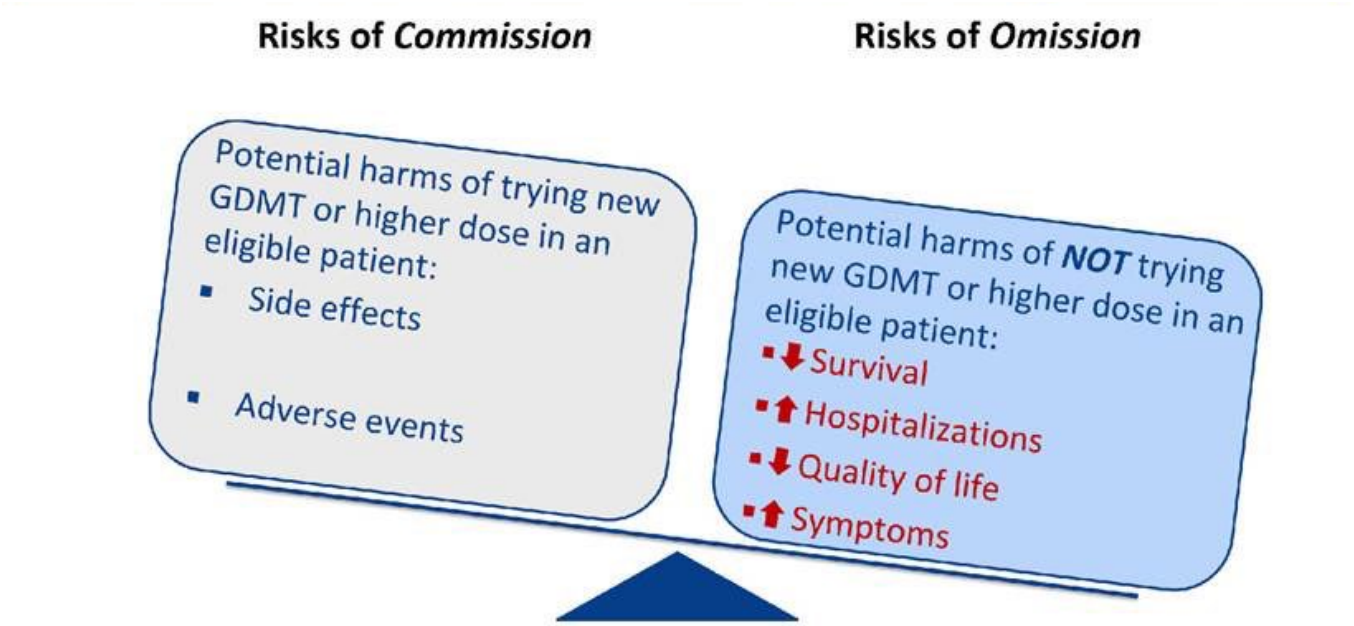
The Risks of Omitting GDMT in HFrEF



Every visit/every setting is an opportunity to initiate and escalate GDMTs, as tolerated

- New-onset heart failure ≠ "low risk"
- "Stable" outpatient heart failure ≠ "low risk"
- Hospitalized heart failure ≠ "low risk"

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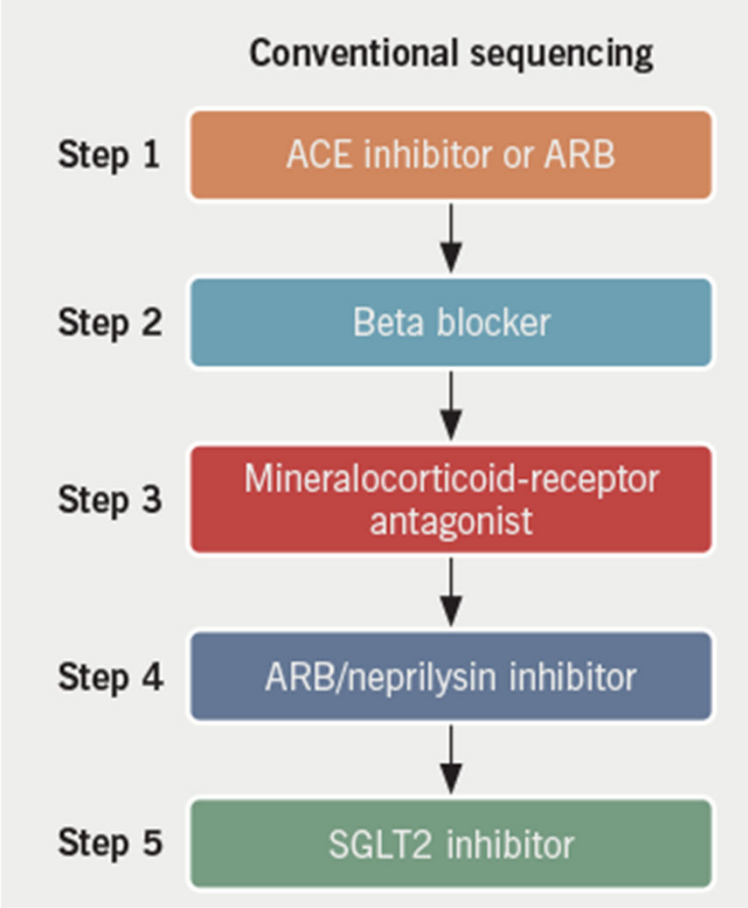
Conventional Sequencing of GDMT: Too Little, Too Late



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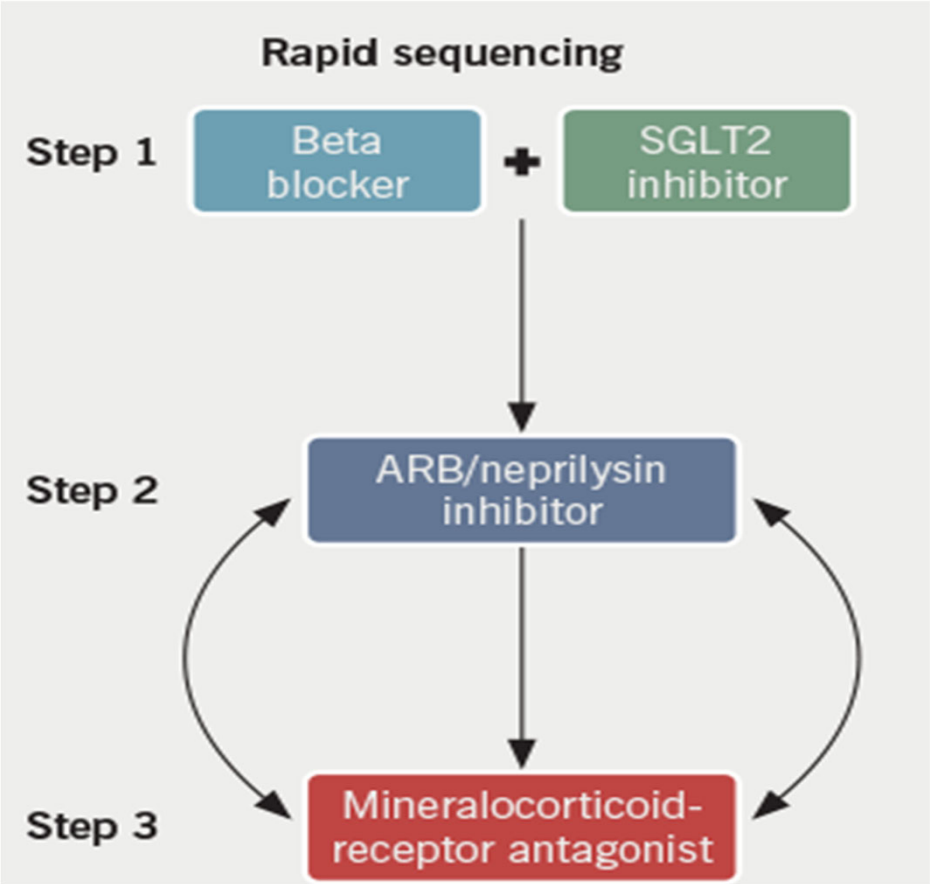
Packer et al. Rapid evidence-based sequencing of foundational drugs for heart failure and a reduced ejection fraction. Eur J Heart Fail. 2021

Rapid Sequencing of GDMT



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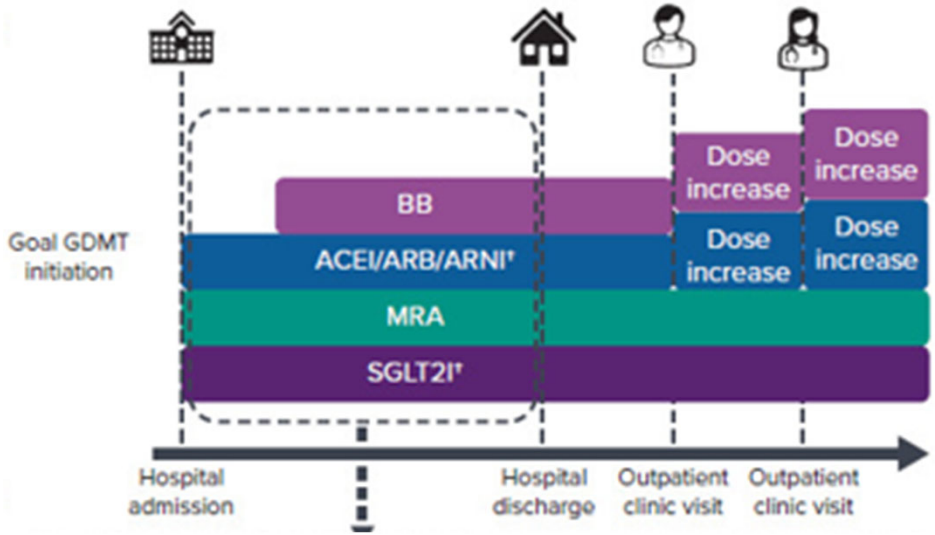
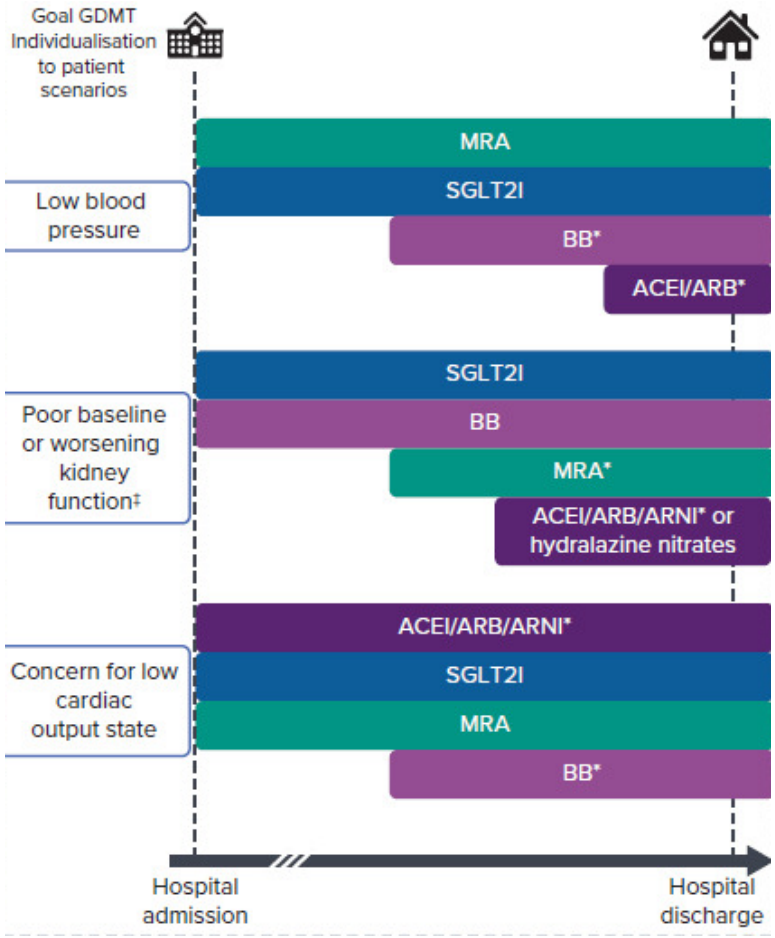
Packer et al. Rapid evidence-based sequencing of foundational drugs for heart failure and a reduced ejection fraction. Eur J Heart Fail. 2021

How to Initiate GDMT?





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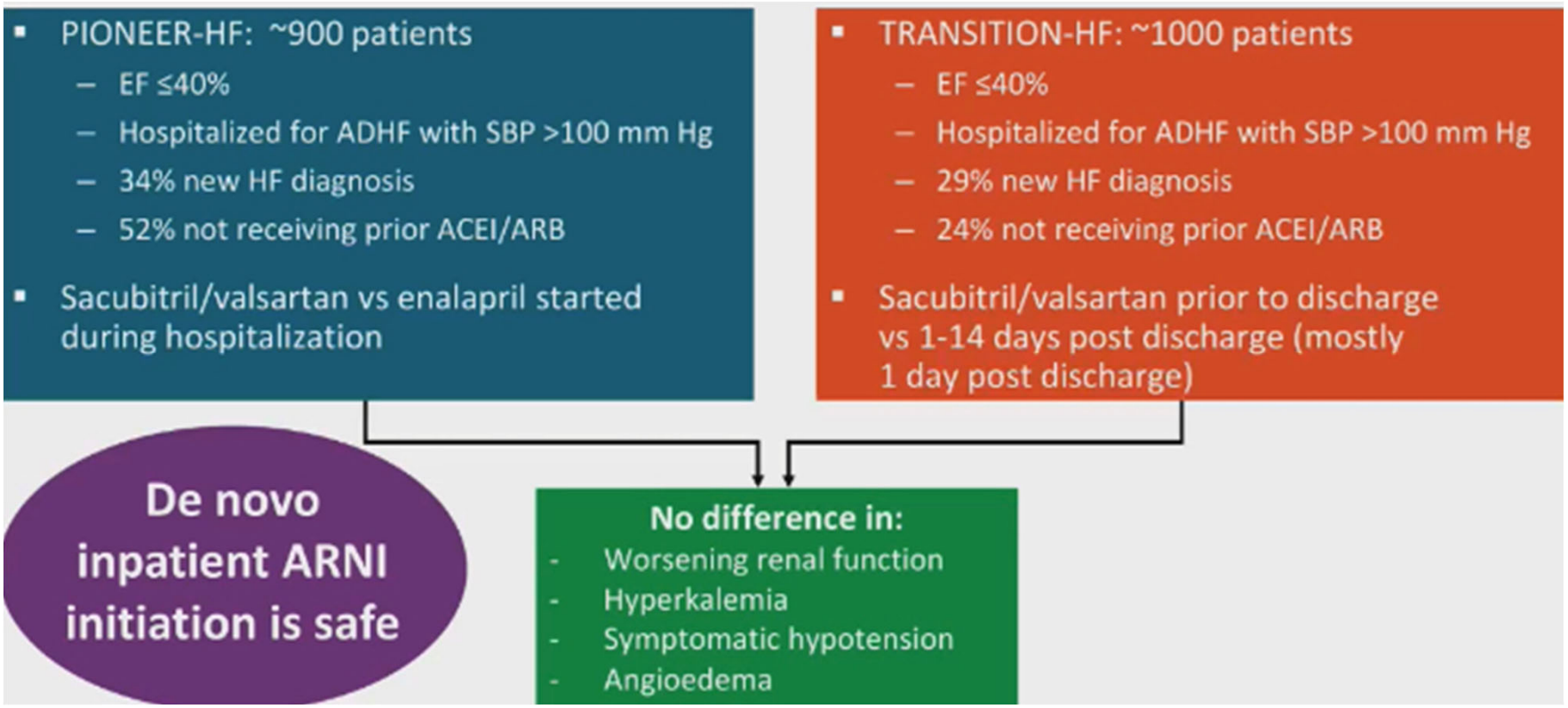
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9253962/

ARNI in Hospitalized Patients



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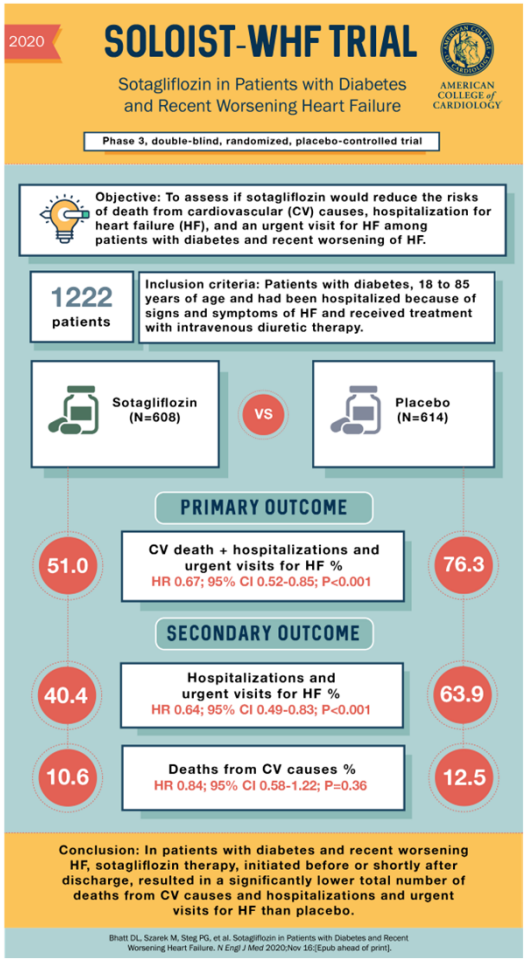
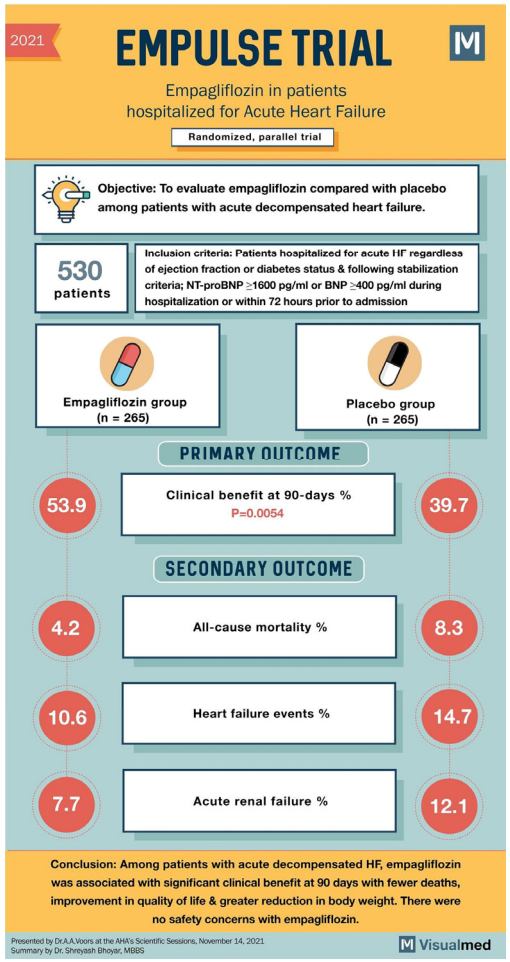
Velazquez. NEJM. 2019;380:539. Wachter. Eur Heart J. 2019;21:998.

SGLT2i in Hospitalized Patients





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What are the Barriers to Initiating/Optimizing GDMT?

Patient-Level Barriers

- Knowledge gap
- Difficulty in recognizing treatment benefits and importance (i.e.feeling better after hospitalization)
- Concerns about the safety of rapid and virtual uptitration
- Cognitive limitations
- Polypharmacy and multimorbidity
- Travel/time to visits
- Caregiver burden

Health System Barriers

- Limited bandwidth of clinic volumes
- Limited access (i.e. rural vs urban, veterans)
- Financial burden
  - Cost of brand name medications
  - Travel time
  - Lost work days

Clinician-Level Barriers

- Knowledge gap
- Myth of stable patient
- Multiple concurrent conditions
- Rapid uptitration concerns
- Monitoring tolerance

Medication Titration Clinics

MAP UK Specialty Pharmacy

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Case 2 Revisited: Inpatient GDMT Titration

Inpatient treatment plan:

1. Diuresed with IV Bumex until weight back to baseline and no further signs of congestion on physical examination
2. Day 1-2: Initiated Sacubitril-Valsartan 24-26 mg BID and Spironolactone 25 mg daily
3. Day 3-4: Started Dapagliflozin 10 mg daily
4. Day prior to discharge when approaching euvolemia: Started Metoprolol succinate XL 50 mg QHS and transitioned to PO diuretics

Followed up in HF clinic within 7 days of discharge:

1. NYHA Class II symptoms, weight stable, labs improving (Cr back to baseline and NTproBNP down to 765)
2. Increased Sacubitril-Valsartan to 49-51 mg BID
3. Referred to medication titration clinic

At 3 month follow up:

1. NYHA Class I symptoms, weight stable, labs at baseline
2. Now on Sacubitril-Valsartan 97-103 mg BID, Spironolactone 50 mg daily, Dapagliflozin 10 mg daily and Carvedilol 12.5 mg BID
3. Repeat TTE shows LVEDD <5 cm, LVEF up to 45% and MR now trace-mild

Case 2: GDMT Inpatient Titration Pearls

1. Goal: Get all 4 classes started – not maximized
2. SGLT2i have minimal BP effect and are safe and protective with modest renal dysfunction
3. MRAs are underused due to exaggerated fear of hyperkalemia (and we now have hyperK management to mitigate this)
4. Add beta blocker once decongested and closer to euvolemic
5. **Remember** – discharge with a plan beats deferral

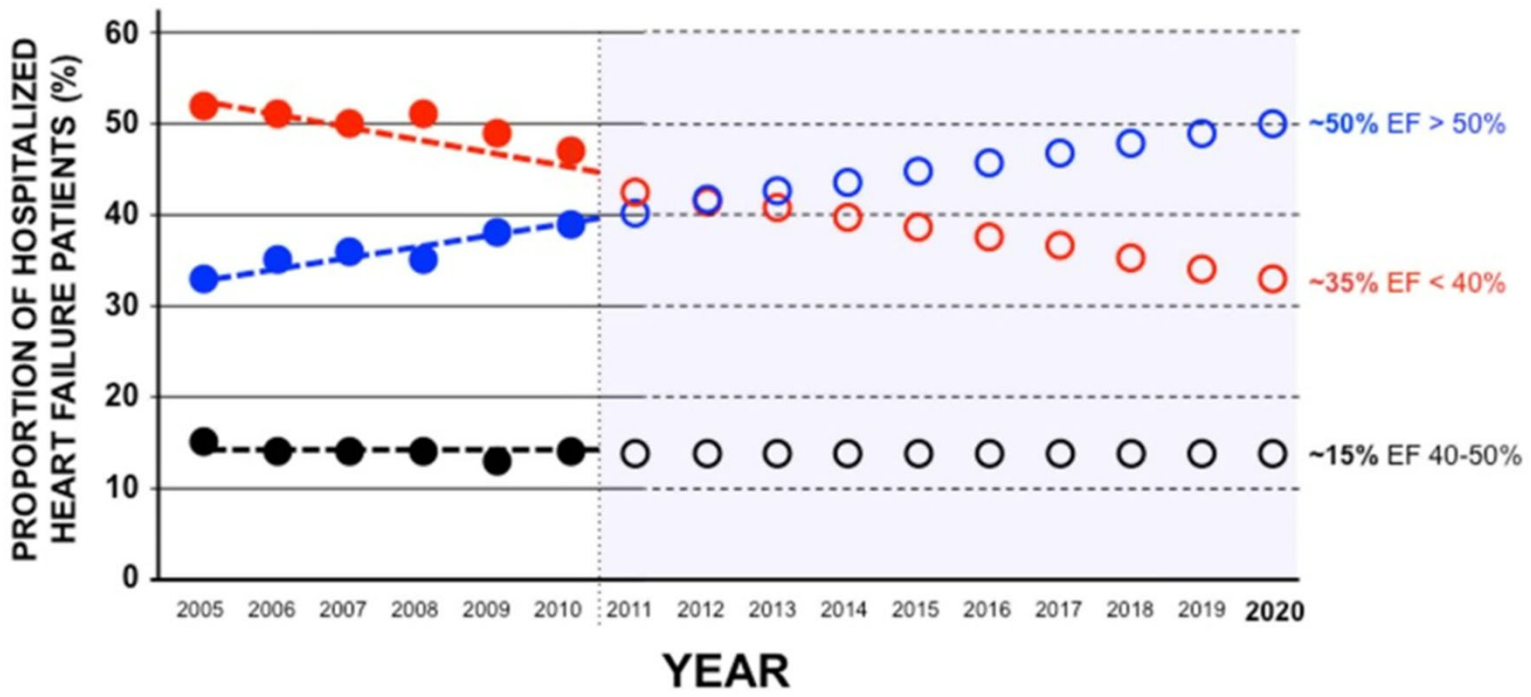
Case 3: HFpEF - Is It Finally Treatable?

76 YO F with long-standing HTN, DMII, PAF and obesity (BMI 41) presents with 1-week of dyspnea, fatigue, abdominal distension and 10 lb weight gain.

- Vital signs: BP 155/94 mmHg, HR 94 BPM and irregular, 96% O2 saturation
- Labs: NT-proBNP 2400, Cr 1.3 (baseline normal)
- TTE: Normal LV size, LVEF 60%, LAE, G2DD and estimatd PASP of ~50 mmHg

Question: How do you approach treating this patient?

HFpEF Hospitalizations on the Rise



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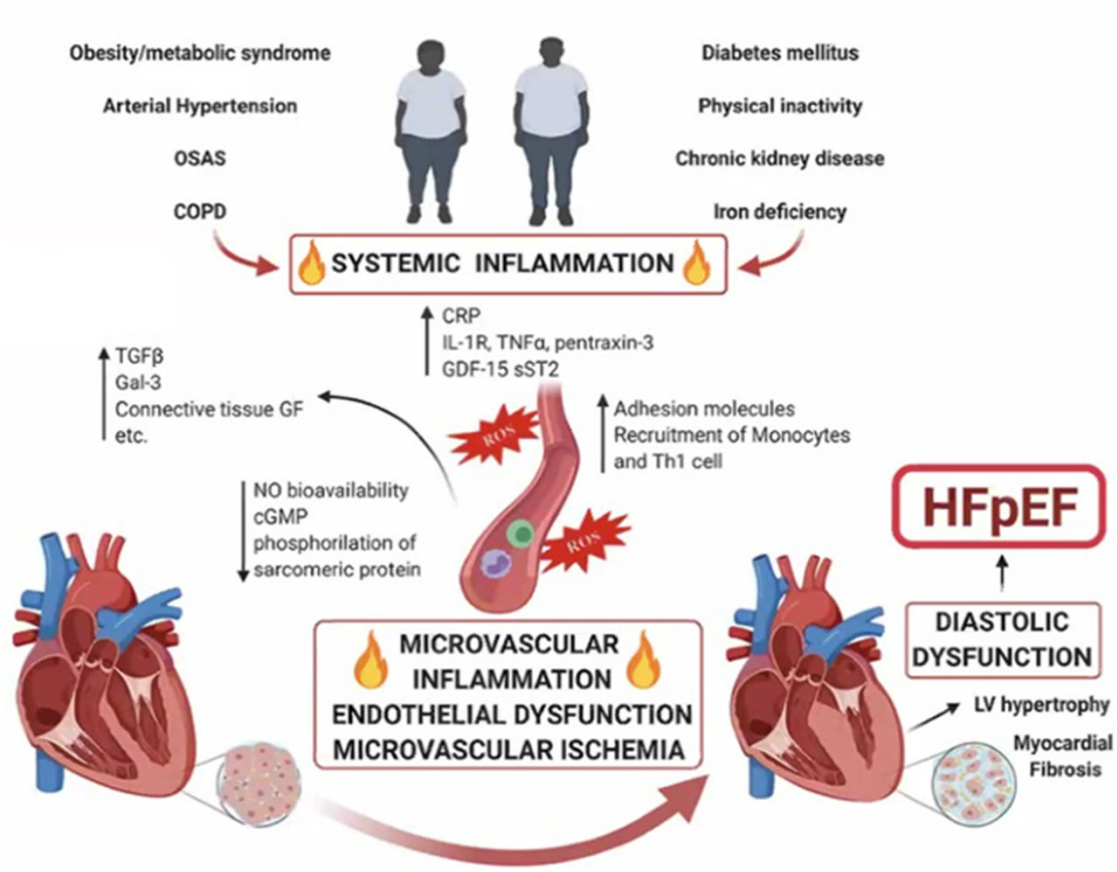
Oktay AA, et al. Curr Heart Fail Rep 2013

Complex and Varied Pathophysiology of HFpEF



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Shah et al, Circulation 2016; Graziani et al, Front CV Medicine 2021;
Del buono et al, Prog Cardiovascular 2020, Goyal et al, Cardiol Clinics 2022;

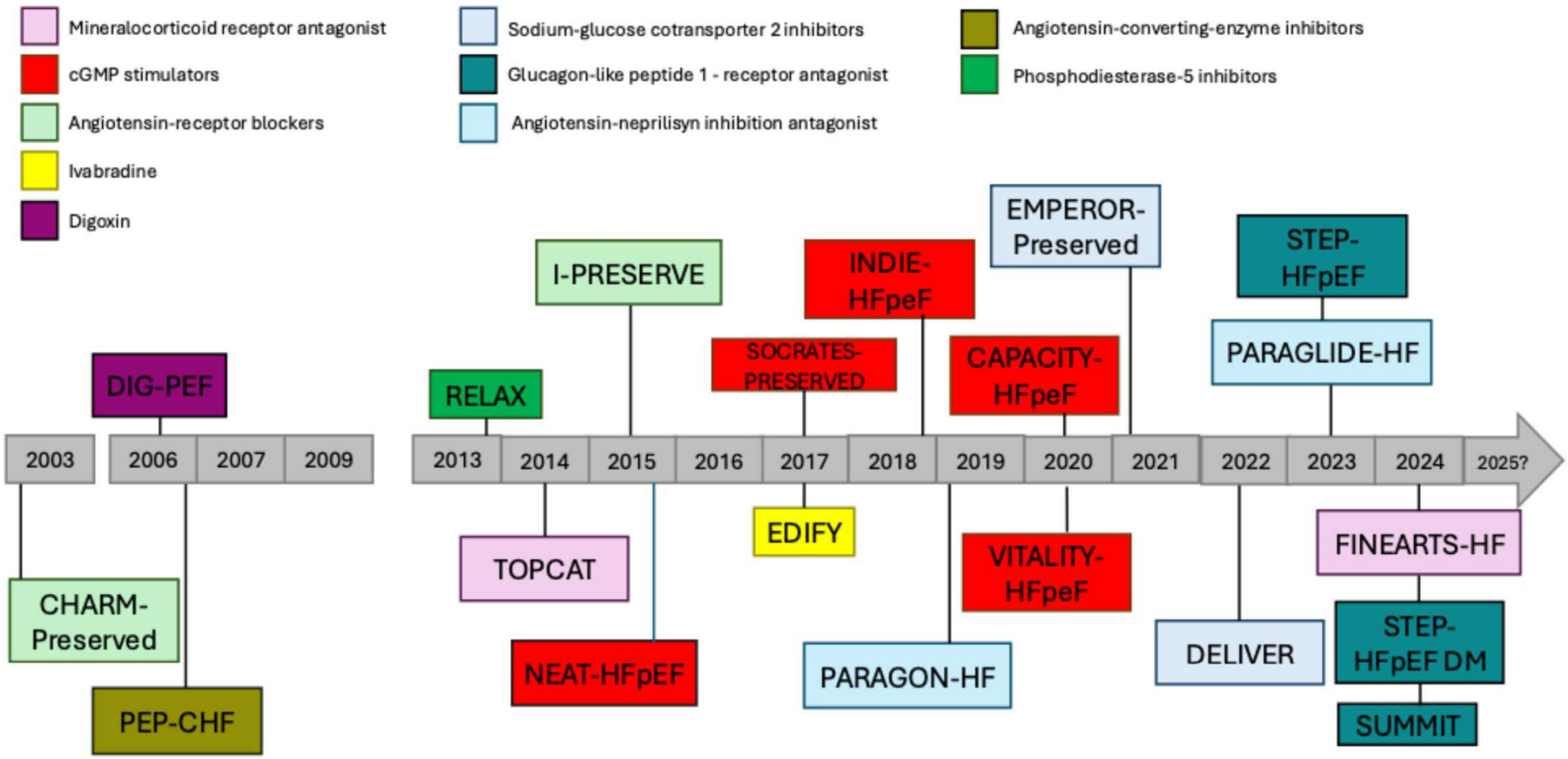
HFpEF: Why Phenotyping Matters

HFpEF is not one disease. It's a **syndrome of impaired diastolic reserve** driven by different upstream pathobiology:

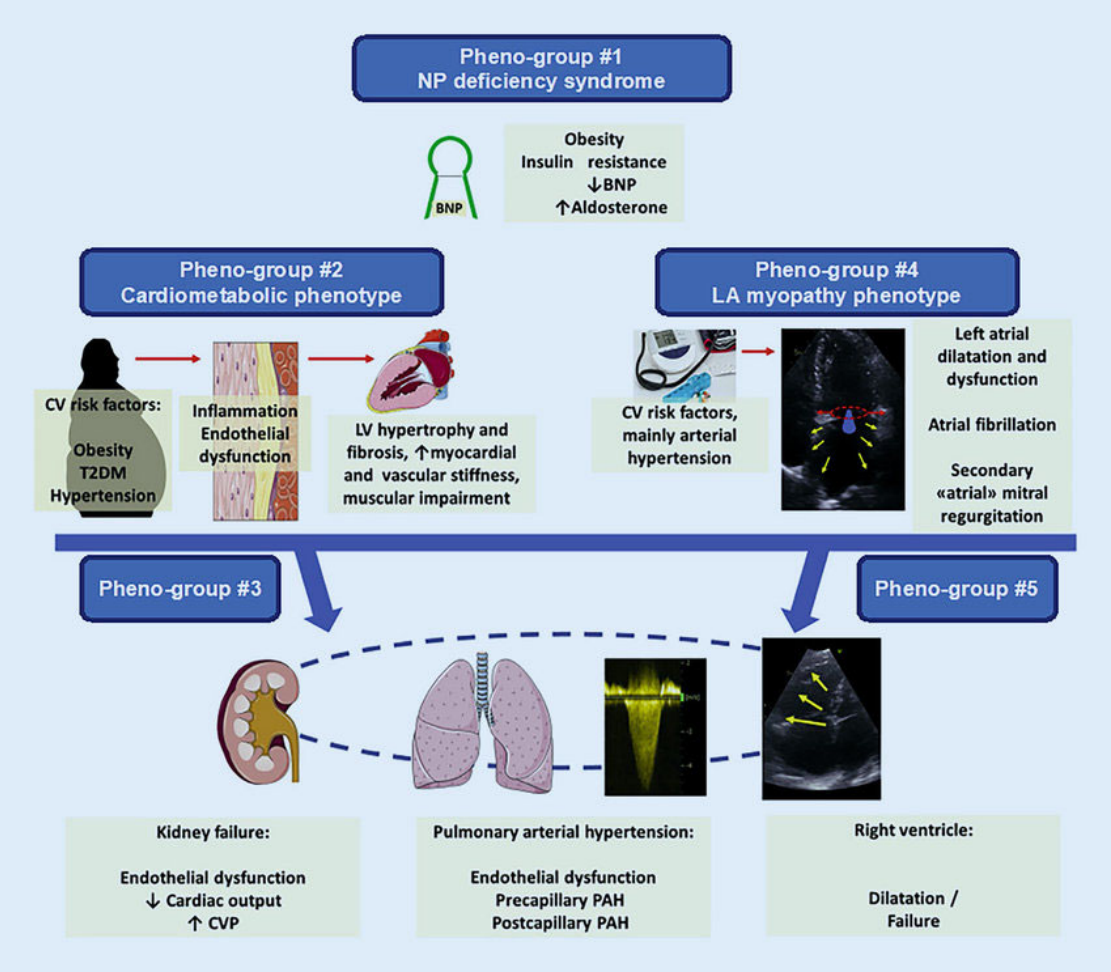
- Metabolic/inflammatory
- Infiltrative
- Ischemic
- Valvular
- Pulmonary vascular
- Chronotropic incompetence
- High-output states

This explains why many therapies historically "failed" in broad HFpEF trials.

HFpEF Major Trials



HFpEF Phenotypes: No One Size Fits All



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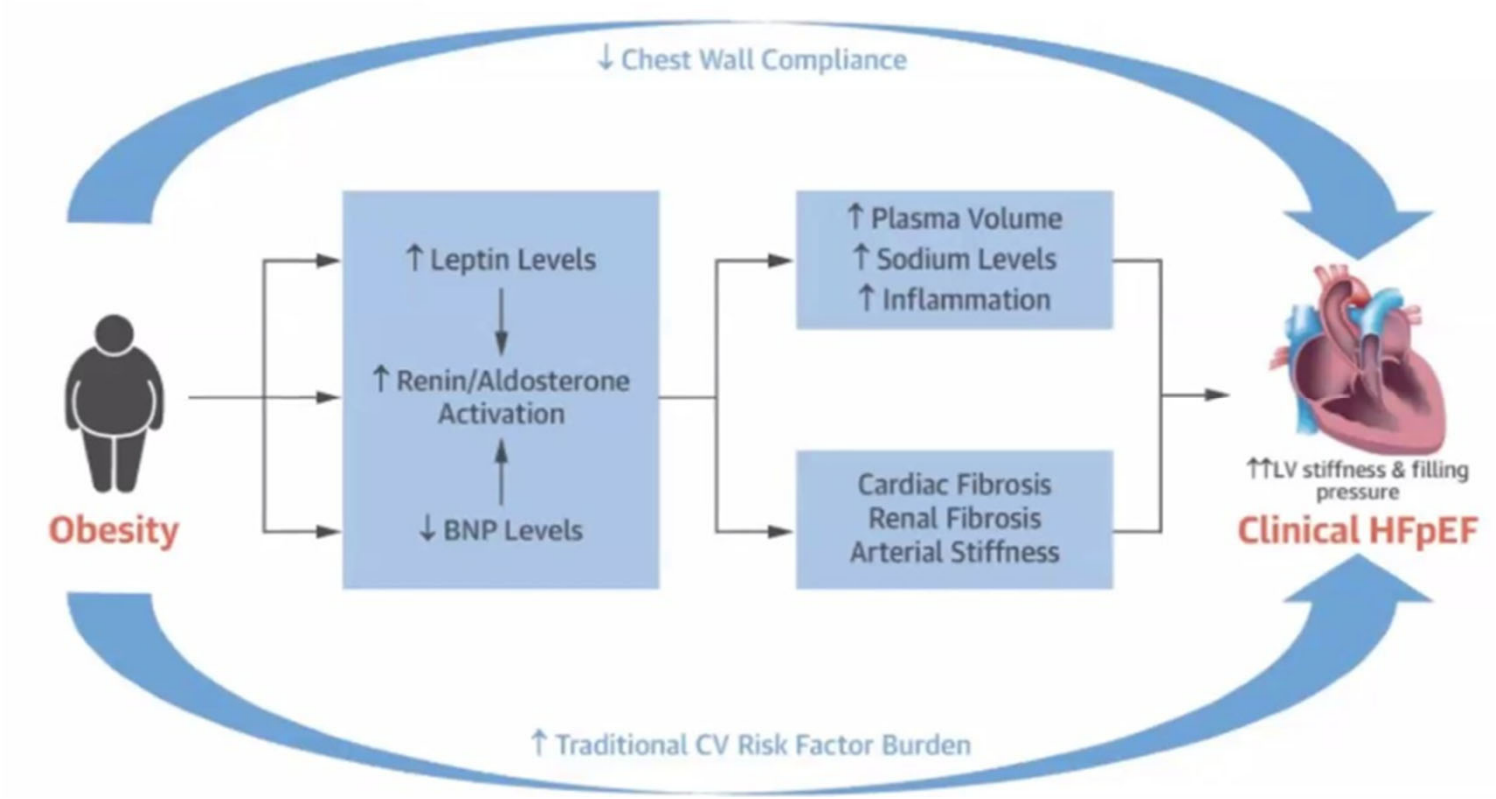
HFpEF Obesity Phenotype



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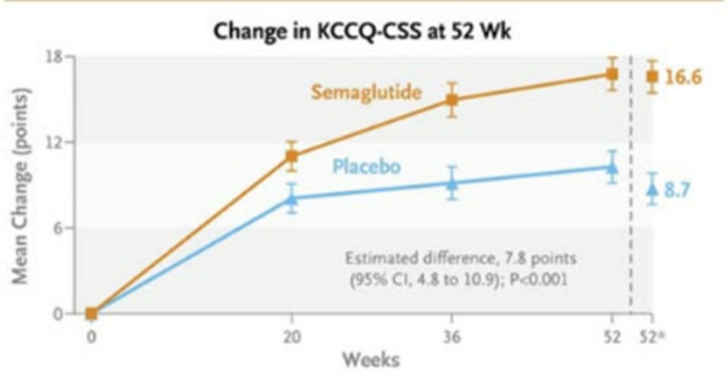
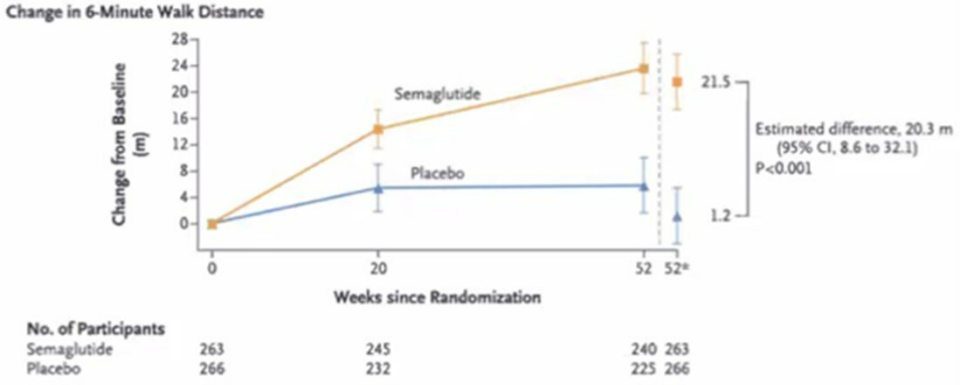
Pandey et al, JACC HF 2018; Kosiborod et al JACC HF 2023; Kosiborod et al NEJM 2023

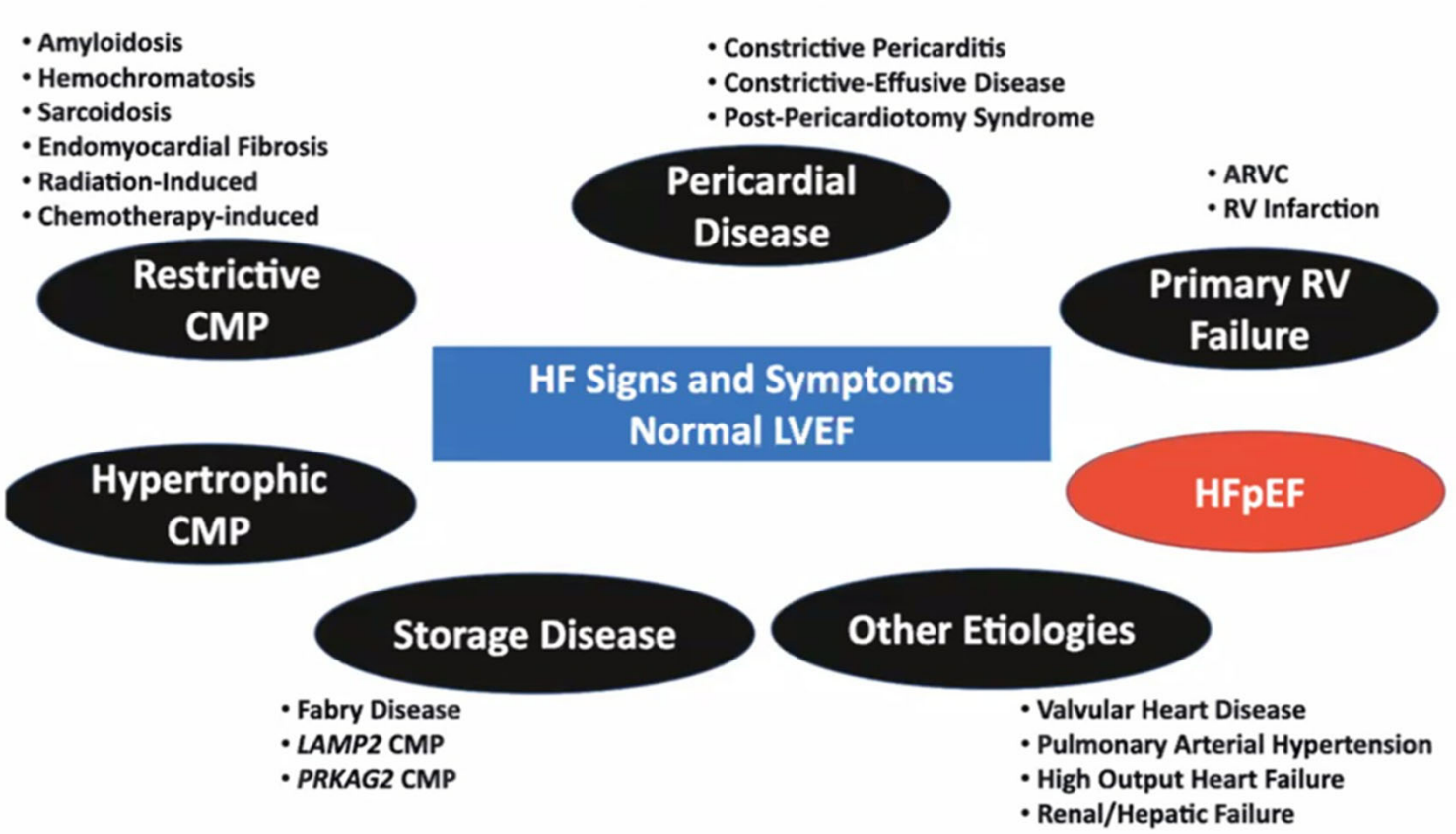
HFpEF Obesity Phenotype: GLP1 as Treatment – STEP HFpEF Trial





Kosiborod et al 2023 NEJM

HFpEF – Rule out "Mimickers"



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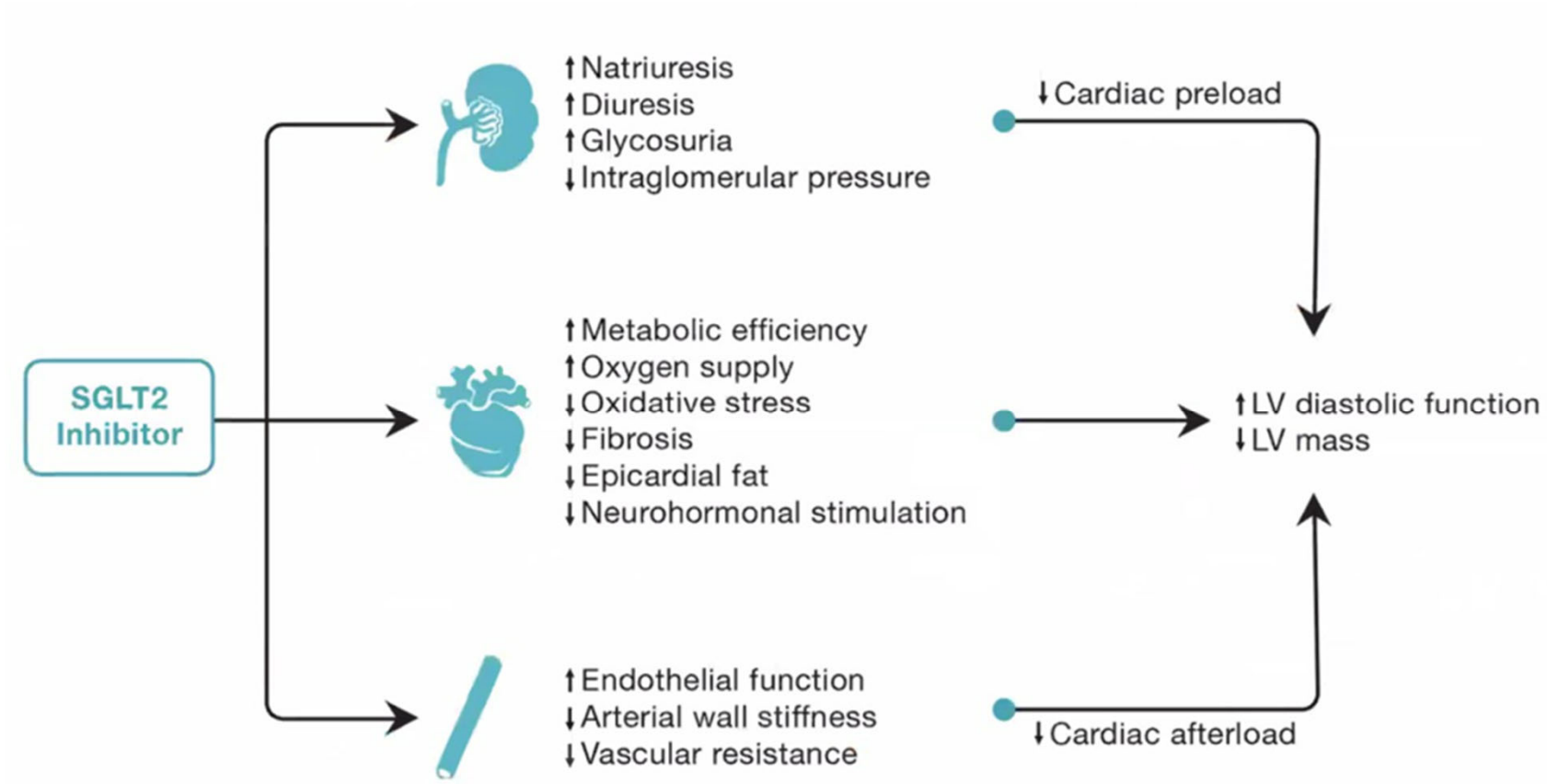
Desai et al JACC HF 2023

HFpEF Management Requires Total Substrate Modification

1. Aggressive treatment of obesity:
   - Empower patients through education-nutritional facts
   - Lifestyle modifications, exercise prescription, GLP1 agonists, bariatric surgery
2. Tight control of BP
   - Rule out secondary causes
3. Recognition & Treatment of OSA
   - STOP-BANG screening tool, refer for sleep study
4. Optimization of HF: diuretics, SGLT2i, ARNI, MRA
5. Early and intentional management of AF
   - Rhythm superior to rate control strategy

SGLT2i Mechanisms for Preventing and Treating HF



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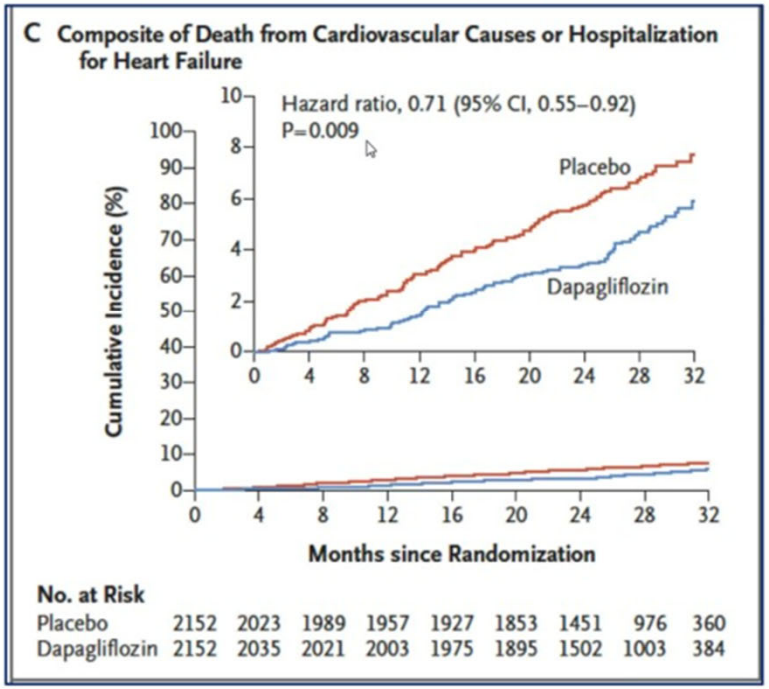
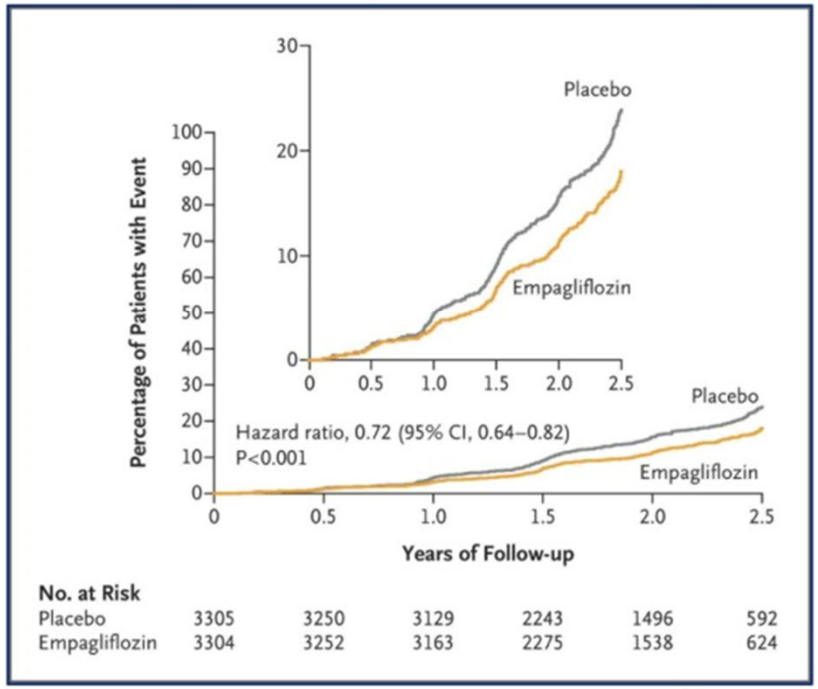
Lan et al ESC HF 2019

SGLT2i as Treatment in HFpEF with Co-Morbidities: CKD, DMII etc

DAPA-CKD Secondary Outcome



EMPA-KIDNEY Primary Outcome



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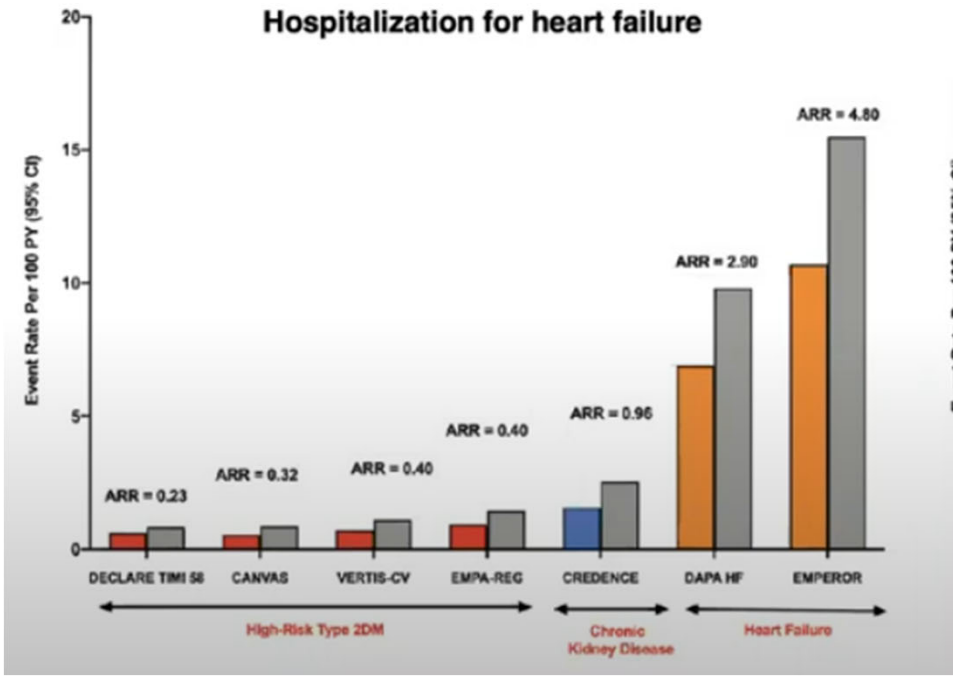
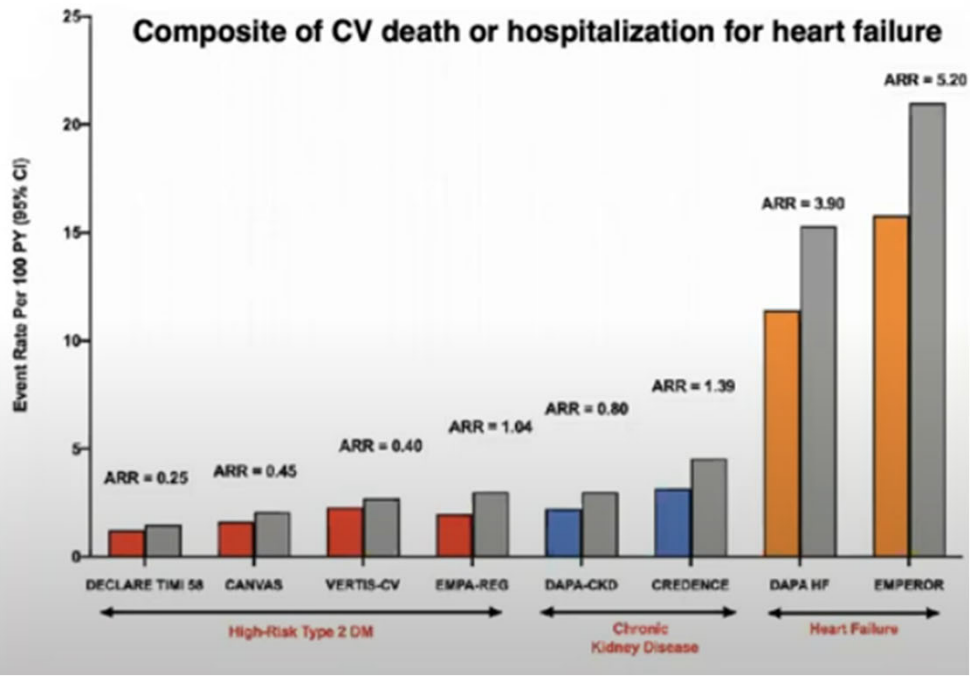
Heerspink H, et al. NEJM 2020; The EMPA-KIDNEY Collaborative Group, et al. NEJM 2023.

SGLT2i: Statins of the 21st Century?





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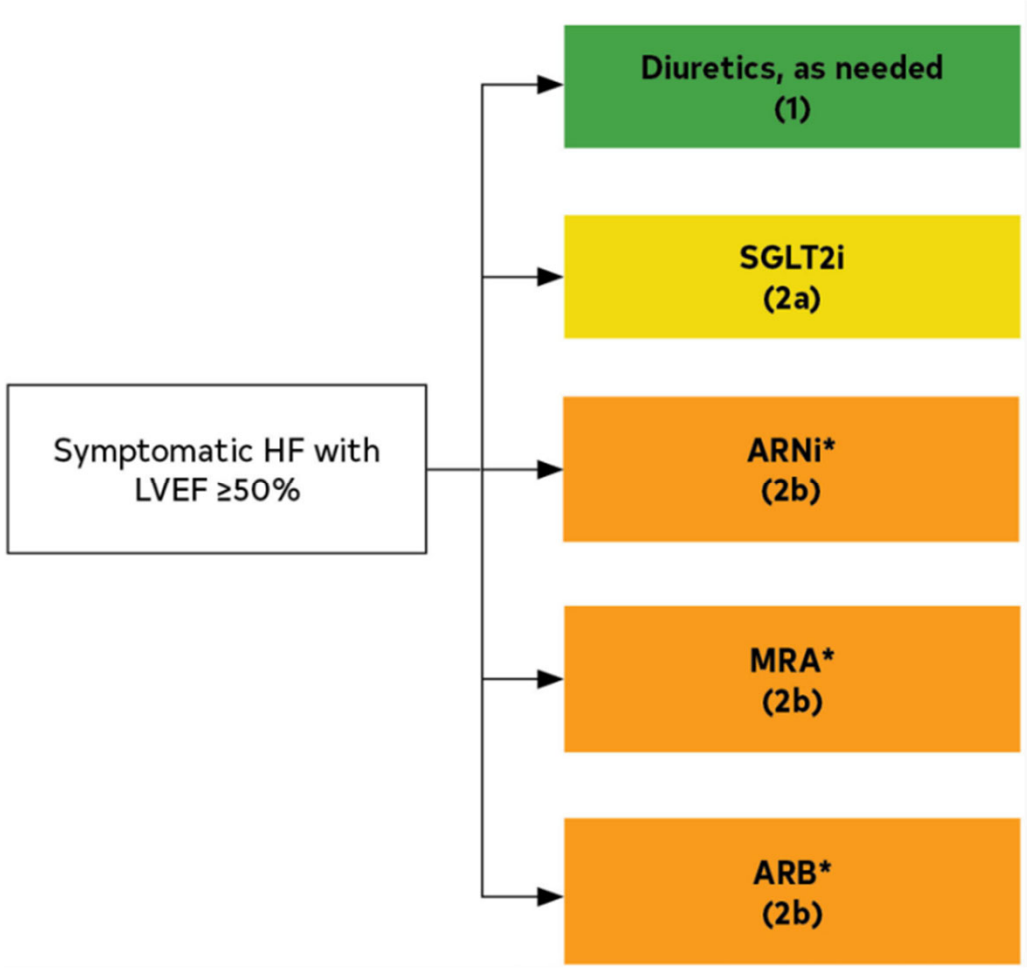
Bhatia et al. Eur J HF 2021

Specific Treatment for HFpEF



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Case 3 Revisited: HFpEF

Inpatient treatment plan:

1. Day 1-3: Diuresed with IV Bumex, optimized BP control by initiating Sacubitril-Valsartan 24-26 mg BID and Spironolactone 25 mg daily
3. Day 3-4: Started Dapagliflozin 10 mg daily, repleted iron stores and engaged nutrition consult

Followed up in Cardiology clinic within 7-10 days of discharge:

1. NYHA Class II symptoms, weight improved, labs back to baseline
2. Increased Sacubitril-Valsartan to 49-51 mg BID
3. Referred for sleep study, to EP for AF ablation consideration and GLP1 initiated

At 3 month follow up:

1. NYHA Class I symptoms, BMI down to 33, labs at baseline. Underwent AF ablation and remains in NSR
2. Repeat TTE shows preserved LV function with improved diastolic dysfunction and reduced PASP

Case 3 Pearls: HFpEF

1. Rule out HFpEF mimickers including amyloidosis
2. SGLT2i are foundational therapy
3. Volume status drives symptoms more than LVEF
4. HFpEF is heterogenous – treat comorbidities (CAD, HTN, obesity, AF, PH etc) aggressively
5. Exercise training improves peak VO2 max – cardiac rehabilitation
6. Avoid beta blockers unless alternate indication

Case 4: AF and HF – New Evidence, Old Pitfalls

46 YO M with HTN and obesity (BMI 31) with no prior cardiac history presents with 3 weeks of palpitations, exercise intolerance and dyspnea.

- Vital signs: BP 118/72 mmHg, HR 133 BPM and irregular, 98% O2 saturation
- Labs: NT-proBNP 1200, normal Cr
- TTE: Mild LV dilation, LVEF 35-40%, mild MR

Question: How do you approach treating this patient?

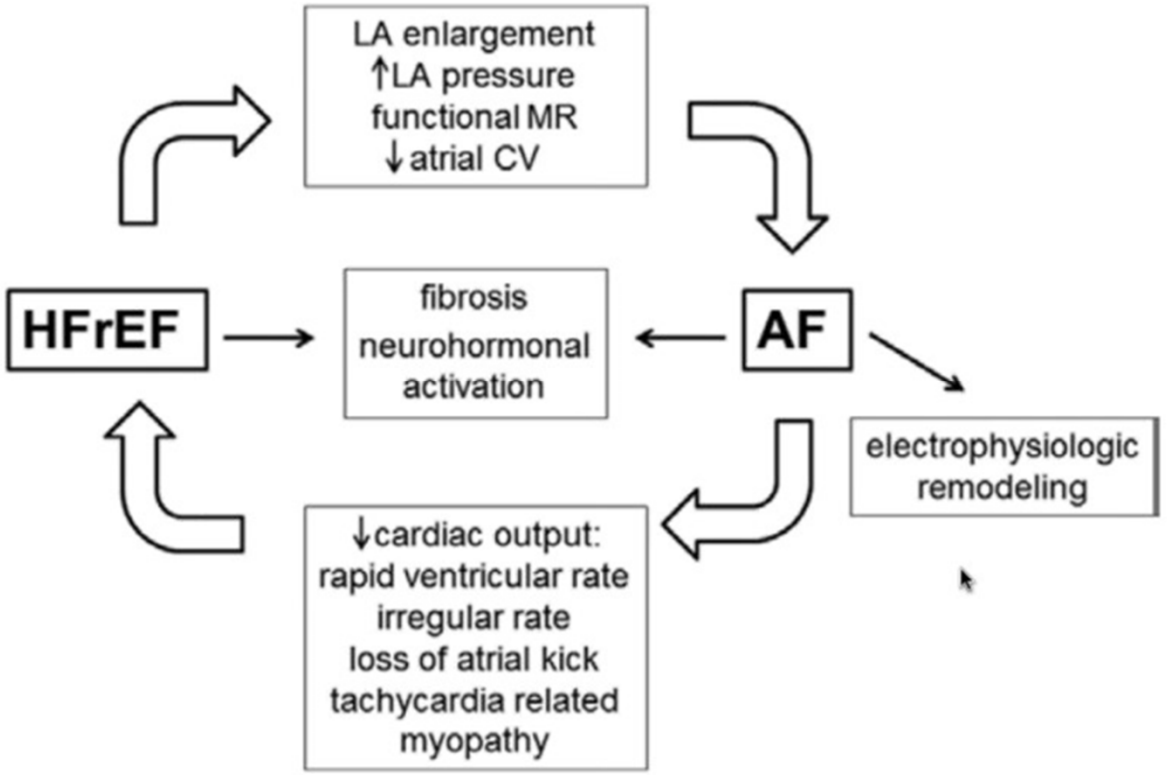
Case 4: AF and HF – New Evidence, Old Pitfalls

Historical context: AFFIRM trial (2002) showed no difference in outcomes between rhythm and rate controlling strategies, and even had higher AAD toxicity and hospitalizations.

Pitfalls:

1. Older AADs
2. Excluded ablation as first line therapy
3. Allowed discontinuation of AC in NSR in spite of risk profile

HF & AF: A Vicious Cycle



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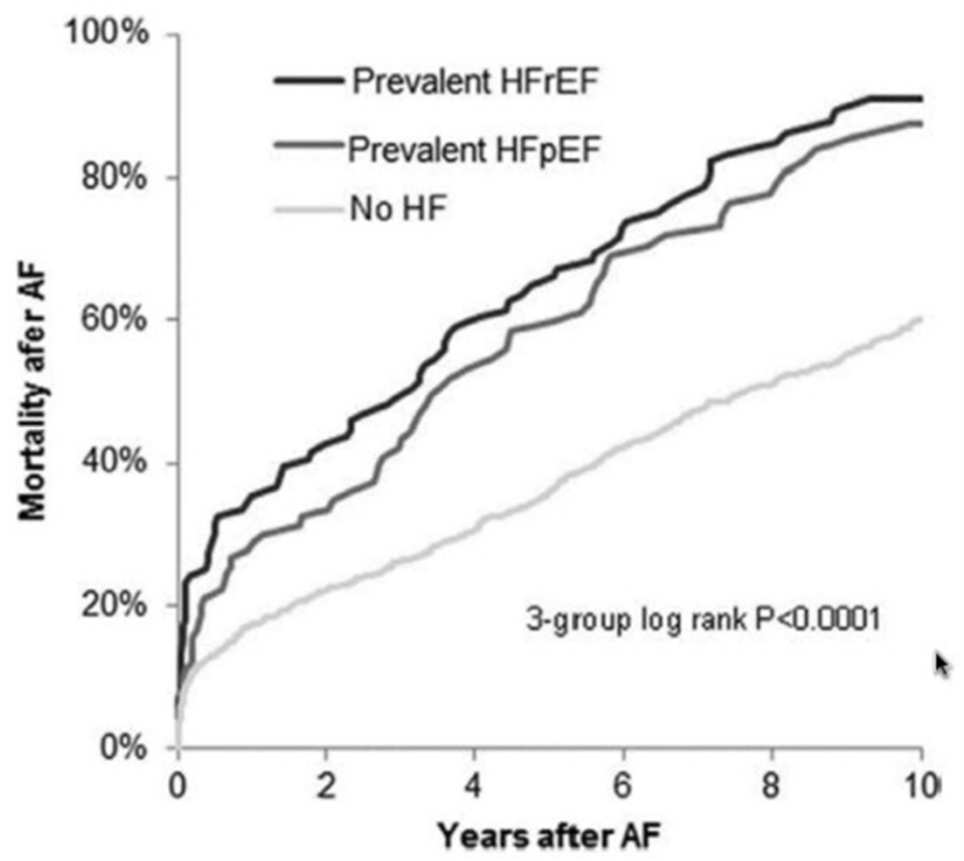
Verma A., et al. *Circulation* 2017;135:1

HF Nearly Doubles Mortality in AF



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Santhanakrishnan R, et al. *Circulation*. 2016;133:484-492

Rhythm vs Rate Control: Paradigm Shift

CABANA Trial (2019)

Catheter ablation vs AAD therapy

- Outcome: death and CV hospitalization lower with ablation
- AF recurrence significantly reduced with ablation

EAST-AFNET 4 Trial (2024)

- Early rhythm control with AADs, CV or ablation vs usual care

- Outcome: early rhythm control improves hard CV outcomes compared to usual rate control strategies

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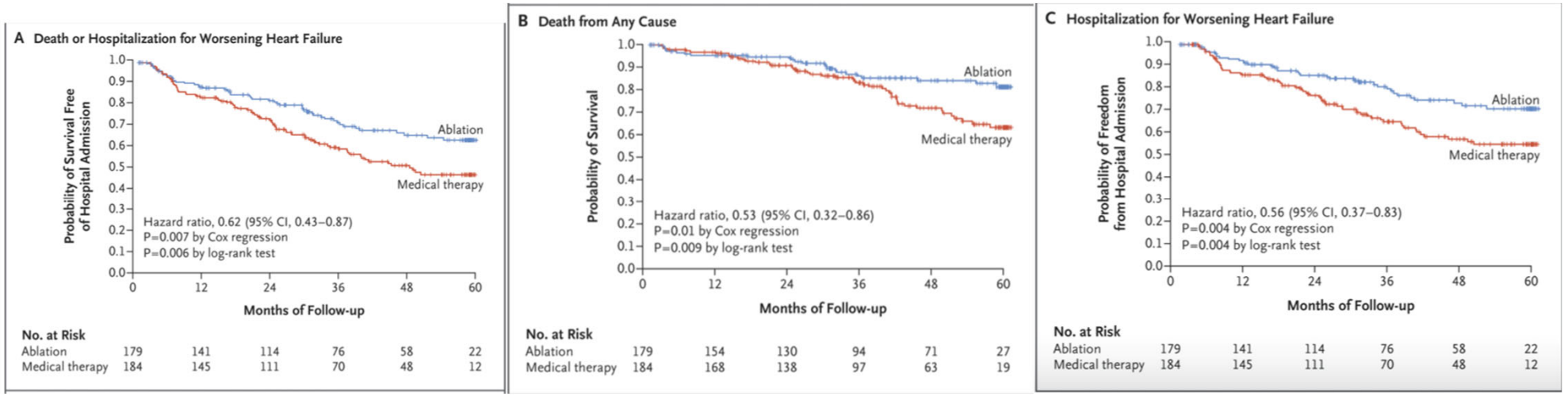
Rhythm vs Rate Control in HF: Paradigm Shift

Catheter Ablation for Atrial Fibrillation with Heart Failure

AATAC and CASTLE-AF



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Case 4 Revisited: AF and HF

Inpatient treatment plan:

1. Day 1-2: Diuresed with IV Bumex, initiated beta blocker and DOAC (CHADSVASC score of 3)
2. Day 3-4: HR improved to ~100s but remained in AF and ongoing NYHA Class 2-3 symptoms
3. Due to de-novo AF, newly reduced LVEF, no prior structural heart disease and concern for tachycardia-mediated CMP, underwent TEE/DCCV and was discharged in NSR

Followed up in Cardiology clinic within 7-10 days of discharge:

1. Back in AF with CVR and feeling poorly
2. Referred to EP and underwent AF ablation

At 3 month follow up:

1. NYHA Class I symptoms, remains in NSR
2. Repeat TTE shows normalization in LV cavity size and LVEF up to 55-60%

Case 4 Pearls: AF and HF

1. Rhythm controlling strategy is superior to rate controlling strategy
2. Timing matters! Early rhythm control improves outcomes
3. AF ablation improves outcomes in HFrEF and prevents development of de-novo HF
4. Anticoagulation strategy remains important to prevent secondary thromboembolic complications

Case 5: Advanced HF - What Is It & When To Refer?

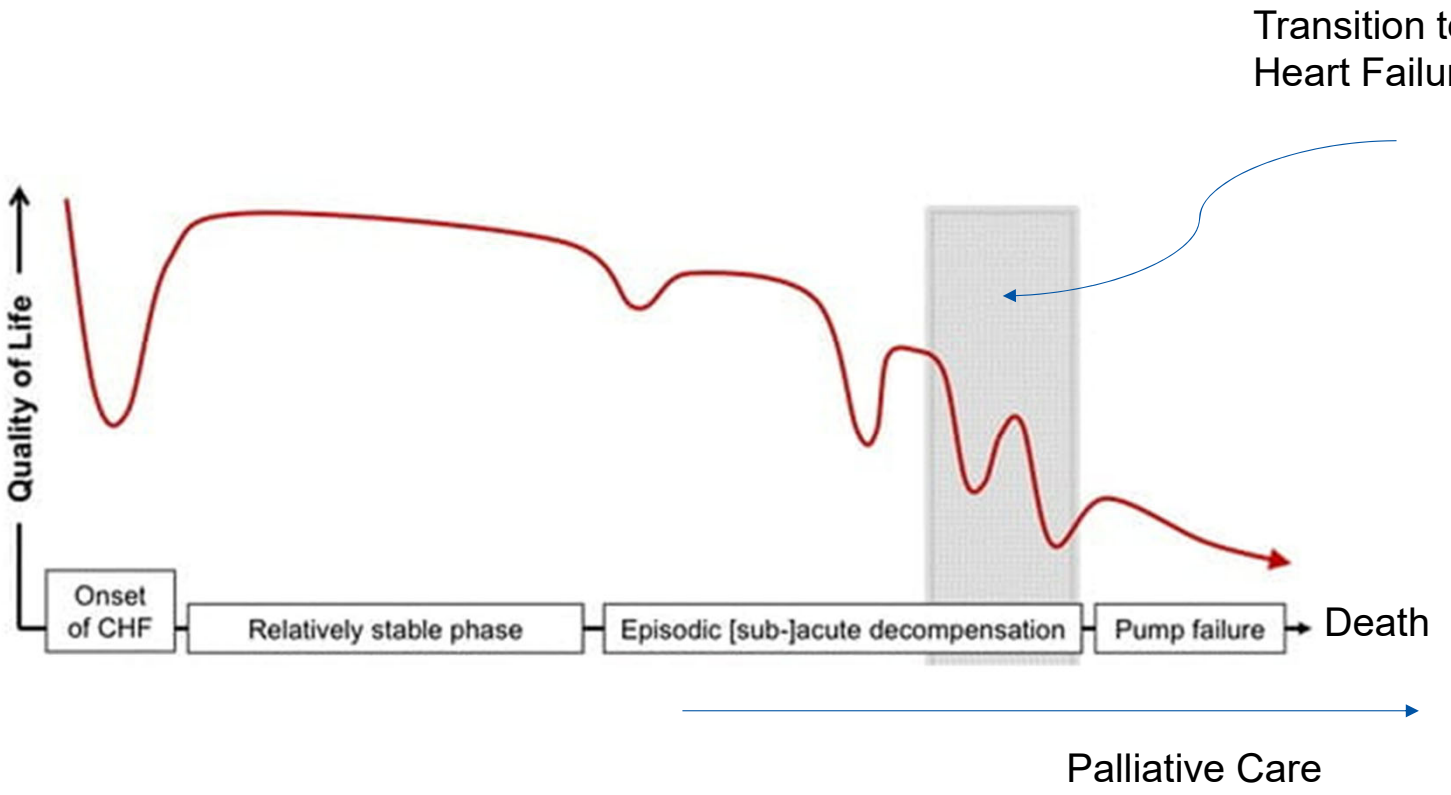
63 YO M with HFrEF due to NICM, difficult to control AF, ICD, CKD stage 2, DMII with 3 admissions for HF exacerbation, the current one which required Dobutamine-assisted diuresis and down-titration of GDMT due to hypotension. Transferred from OSH to UK HF service due to inability to wean inotrope and episode of VT requiring ICD shock.

On admission, reports poor appetite with minimal PO intake, weight gain, pre-syncopal symptoms.

- Vital signs: BP 89/68 mmHg, HR 129 BPM
- Labs: NT-proBNP 6500, Na 127, Cr 1.4, Bilirubin 2.1 and AST/ALT mildly elevated
- TTE: LVEDD 8 cm, LVEF <20%, RV dilated and hypokinetic, mod MR

Question: How do you approach treating this patient?

Clinical Course of Heart Failure



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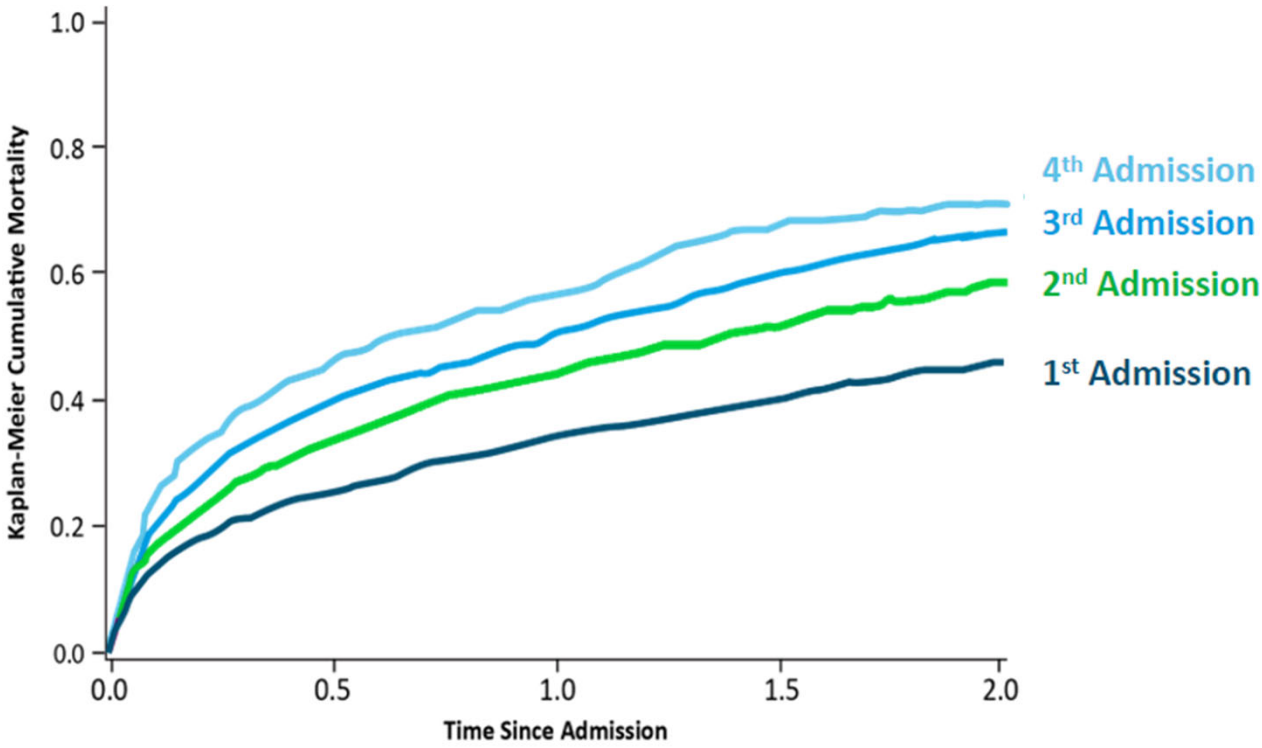
Greenhalgh et al. BMC Cardiovascular Disorders (2017)

Clinical Markers: HF Hospitalization is Ominous



MORTALITY RISK
INCREASES
AFTER EACH
HEART FAILURE
HOSPITALIZATION

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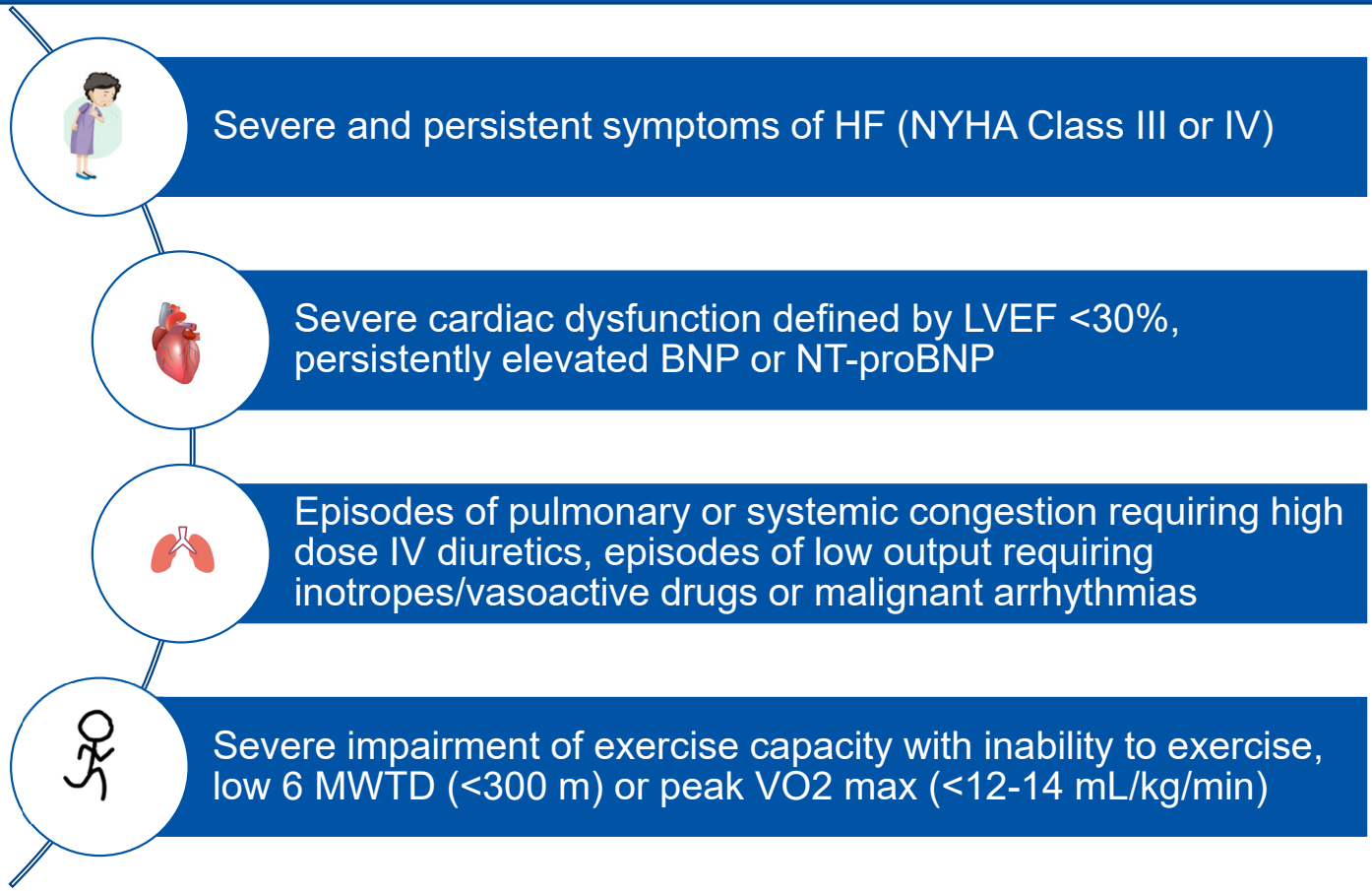
Setoguchi et al. AHJ (2007)

What is Advanced Heart Failure?



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Crespo-Leiro et al. EJHF (2018)

What Does Advanced Heart Failure Look Like?

(A) Clinical markers:

- High diuretic requirement aka diuretic resistance (cardio-renal)
- Recurrent admissions despite adherence to medications
- Medication intolerance/hypotension
- Exercise intolerance
- Weight loss/sarcopenia/cardiac cachexia

(B) Biochemical/laboratory markers:

- End organ dysfunction: rising BUN/Cr, elevation in bilirubin and liver enzymes
- Hyponatremia
- Elevations in BNP and NT-proBNP

(C) Imaging markers:

- Dilation in LV cavity size, severely reduced LVEF
- Pulmonary hypertension and RV involvement/dysfunction
- Development of secondary valvular abnormalities: MR and TR

“I NEED HELP” Algorithm

I	Need for **inotropes**
N	**New** York Heart Association Class IV
E	Worsening **end-organ** dysfunction
E	**Ejection fraction** <20%
D	**Defibrillator** shocks for ventricular arrhythmias
H	Recurrent **HF** hospitalizations
E	**Escalating** diuretic dose & **Elevated** NT-proBNP
L	**Low blood pressure**
P	**Progressive** intolerance of GDMT

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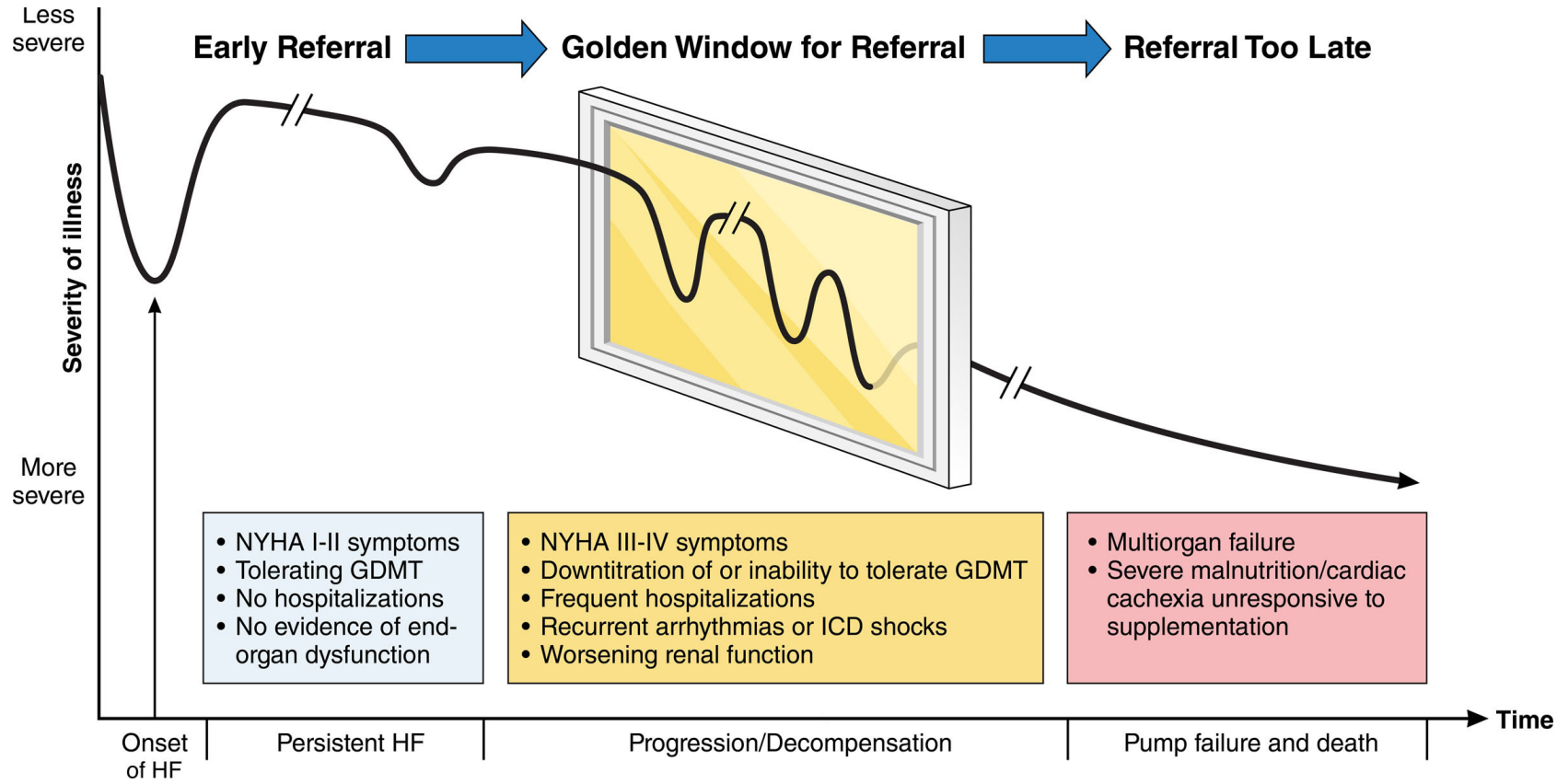
Baumwol. JHLT (2017)

Golden Window for Referral



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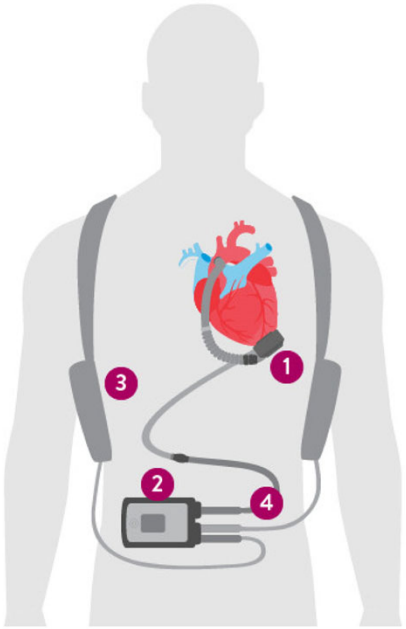
Morris et al. Circulation (2021)

Advanced Heart Failure Therapies



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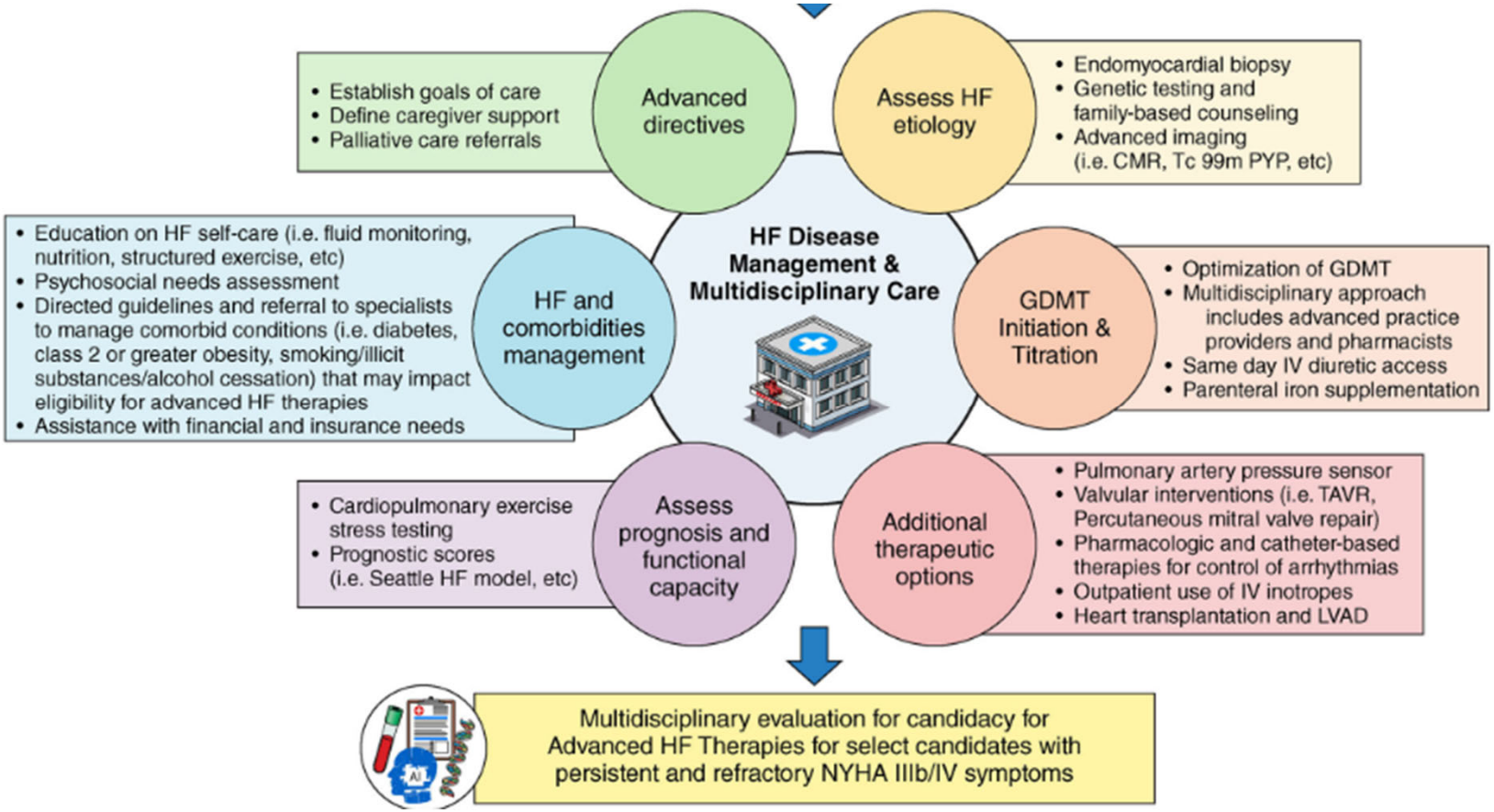
Scope of Advanced Heart Failure Centre



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Morris et al. Circulation (2021)

Case 5 Revisited: Advanced HF

Inpatient treatment plan:

Diuresed, underwent RHC which confirmed elevated filling pressures and low CI. Dobutamine transitioned to Milrinone gtt and initiated advanced HF therapies evaluation. Improved clinically and discharged home with PICC and inotrope with goal for nutritional optimization and physical strengthening

Followed up in HF clinic within 7 days of discharge:

1. Discussed at multidisciplinary meeting and approved for transplant listing
2. Initially improved but re-hospitalized for worsening low output HF
3. Implanted axillary IABP and listed status 2 for OHT, transplanted 3 days later

At 3 year follow up:

Doing amazingly! Able to see his daughter get married and birth of first grandchild

Case 5: Recognizing Advanced HF

- Identifying advanced/end-stage heart failure is hard!
- Use clinical, laboratory and imaging findings to your advantage to help understand where a patient is in their timeline
- When in doubt or uncertain, refer! Remember – it's never too early, but can certainly be too late

Why Should as Hospital Medicine Care?

1. HF is everywhere!
   - Leading cause of hospitalization in adults >65 YO
2. You control the most impactful window of care
3. Cardio-renal syndrome is a daily inpatient challenge
   - 40% of admission HF patients have CKD
4. HFpEF is treatable and on the rise
5. AF and HF is a high-risk combination
   - AF present in up to 50% of HF patients; increases CVA, mortality and drives readmissions
6. Advanced HF is often missed
7. **Bottom line – hospitalists are not just stabilizing HF; you determine trajectory, survival and drive outcomes**
8. Every admission is an opportunity to:
   - initiate lifesaving therapy
   - optimize volume status
   - risk stratify
   - improve long term outcomes

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Final Take Home Points

1. HF hospitalization is a therapeutic inflection point
2. Decongest aggressively
3. Creatinine rise during decongestion ≠ worsening renal condition
4. Initiate GDMT safely while inpatient
5. SGLT2i are your friend
6. HFpEF is now a treatable condition
7. Rhythm control improves outcomes in both AF and HF
8. Refer to advanced heart failure early
9. Hospitalists change HF trajectories – inpatient decisions shape long-term outcomes!
10. Partnership with Cardiology is crucial

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Questions?

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