Hospitalists at the HEART of Heart Failure Care

John Romond, MD

HEARTland Hospital Medicine Conference

8.2025

Disclosures / Conflicts of Interest

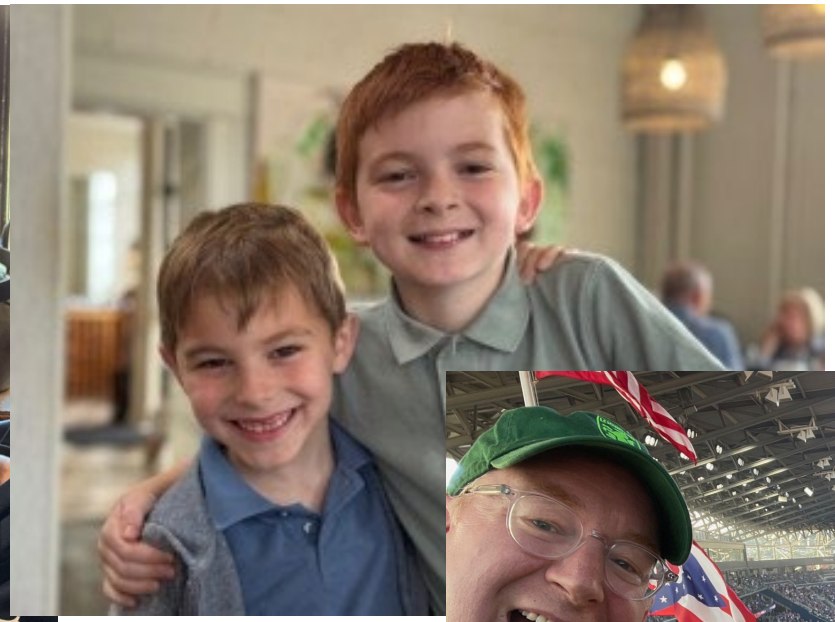
- None 😔

Meet Your Speaker

- Associate Professor of Hospital Medicine
- Home grown at University of Kentucky
- Clinician
- Teacher
- Unit Medical Director

- Not a cardiologist but, like you, see a lot of patients with heart failure

My heart → Dad



Objectives

By the end of this talk you will be able to:

1. Recognize key hemodynamic concepts for CHF exacerbations
2. Distinguish classes of GDMT therapy and use evidence-based approaches for initiation and titration
3. Describe treatment goals for diuretics
4. Identify cases that are appropriate for cardiology consultation
5. Optimize transitions of care from the hospital

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6. Put up with some of the internet's finest cardiology dad jokes

Agenda

- Introduction
- The HEART of the talk
  - Hemodynamics first
  - Evidence based therapies
  - Acute interventions
  - Risk stratification
  - Transition planning
- Conclusions

Why can’t you lie to a cardiologist?

Why can’t you lie to a cardiologist?

They can always spot afib!

We see a lot of heart failure!

UK Hospital Admissions with primary diagnosis of Heart Failure 7/1/24 – 6/30/25

Primary Care Team	HM Teams	CAR Teams	Rest of UKHC	Total
Measures				
Number of Hospital Admissions	1,642	1,635	1,650	**4,927**
Percentage of Population	33.3%	33.2%	33.5%	**100%**

Credit to Dr. Romil Chadha

Opportunity for improvement

Discharge Processes and 30-Day Readmission Rates of Patients Hospitalized for Heart Failure on General Medicine and Cardiology Services

Brian M. Salata, MD, MS[a], Madeline R. Sterling, MD, MPH[b], Ashley N. Beecy, MD[c], Ajayram V. Ullal, MD[a], Erica C. Jones, MD[c], Evelyn M. Horn, MD[c], and Parag Goyal, MD, MSc[b,c,*]

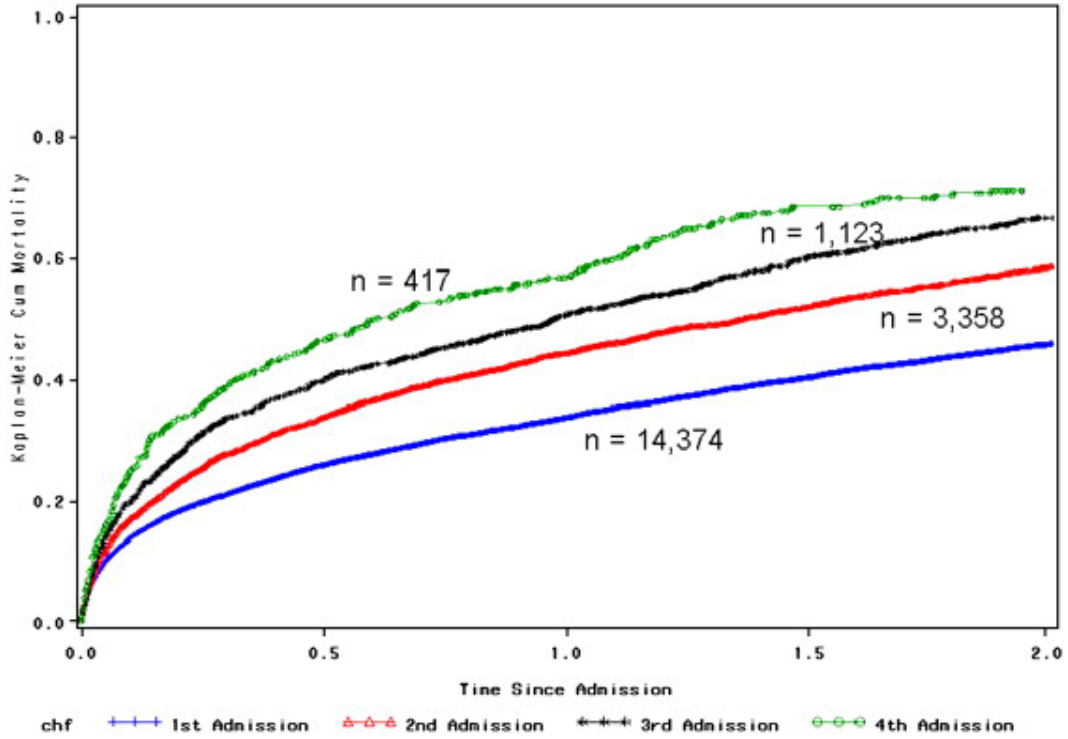
Opportunity for improvement

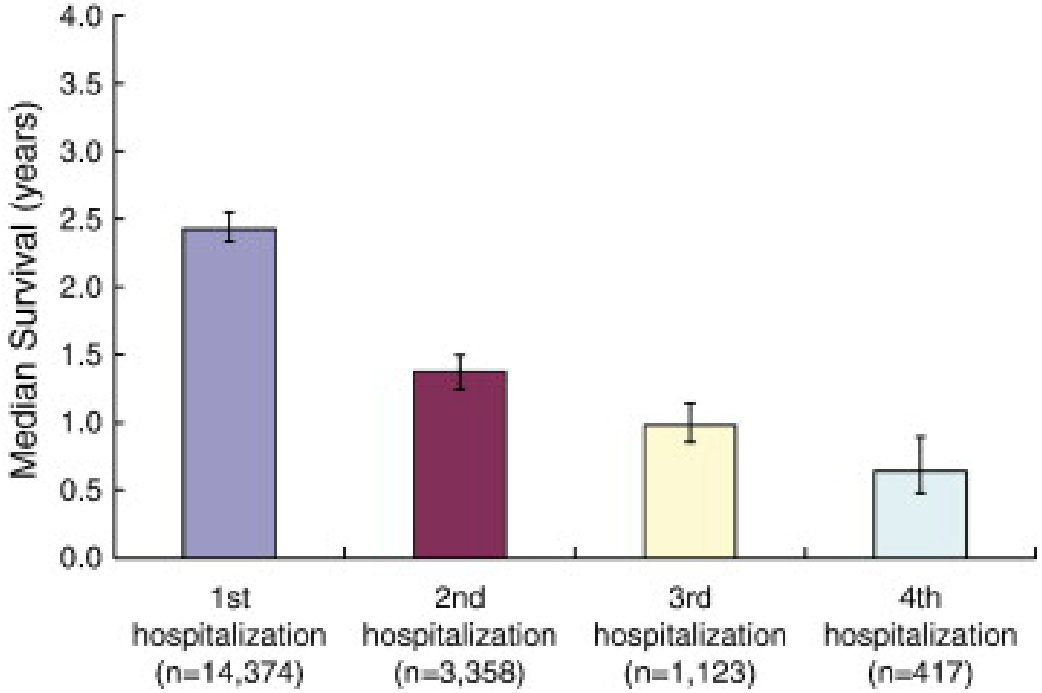
- **Population:** 926 patients hospitalized for heart failure
- 60% admitted to **Cardiology services**
- 40% admitted to **General Medicine (GM) services**

Opportunity for improvement

- **30-Day Readmission Rates**
  - **GM services:** 32% (15% related to CHF)
  - **Cardiology services:** 25% (12% related to CHF)
  - **Adjusted Odds Ratio:** 1.43 (95% CI: 1.05–1.96, $p = 0.02$)
- **Conclusion**
  - Cardiology service patients had **better outcomes**
  - Highlights need for **improved discharge processes** and **targeted interventions** for hospitalists managing CHF

CHF Hospitalizations predict mortality





Setoguchi, Soko, MD, DrPH; Stevenson, Lynne Warner, MD; Schneeweiss, Sebastian, MD, ScD. Published July 31, 2007. Volume 154, Issue 2. Pages 260-266. © 2007.

Universal Definition and Classification of Heart Failure (HF)

Definition

HF is a *clinical syndrome* with current or prior

- ***Symptoms and or signs caused by a structural and/or functional cardiac***

And corroborated by at least one of the following:

- ***Elevated natriuretic peptide levels***
- ***Objective evidence of cardiogenic pulmonary or systemic congestion***

Stages

AT RISK *(STAGE A)*	**Patients at risk for HF, but without current or prior symptoms or signs of HF and without structural cardiac changes or elevated biomarkers of heart disease**
PRE-HF *(STAGE B)*	**Patients without current or prior symptoms or signs of HF with evidence of <u>one</u> of the following:** • *Structural Heart Disease* • *Abnormal cardiac function* • *Elevated natriuretic peptide or cardiac troponin levels*
HF *(STAGE C)*	**Patients with current or prior symptoms and/or signs of HF caused by a structural and/or functional cardiac abnormality**
ADVANCED HF *(STAGE D)*	**Severe symptoms and/or signs of HF at rest, recurrent hospitalizations despite GDMT, refractory or intolerant to GDMT, requiring advanced therapies transplantation, mechanical circulatory support, or palliative care**

Classification By EF

HF with reduced EF (HFrEF)
- *HF with **LVEF < 40%***

HF with mildly reduced EF (HFmrEF)
- *HF with **LVEF 41-49%***

HF with preserved EF (HFpEF)
- *HF with **LVEF > 50%***

HF with improved EF (HFimpEF)
- *HF with a baseline LVEF of < 40%, a 10-point increase from baseline LVEF, and a second measurement of LVEF of > 40%*

Language matters! The new universal definition offers opportunities for *more precise communication* and description with terms including ***persistent HF*** instead of "stable HF," and ***HF in remission*** rather than "recovered HF."

Gibson, Blumer, Mentz, Lala. ACC

Agenda

Hemodynamics first

Evidence based therapies

Acute interventions

Risk stratification

Transition planning

Hemodynamics First

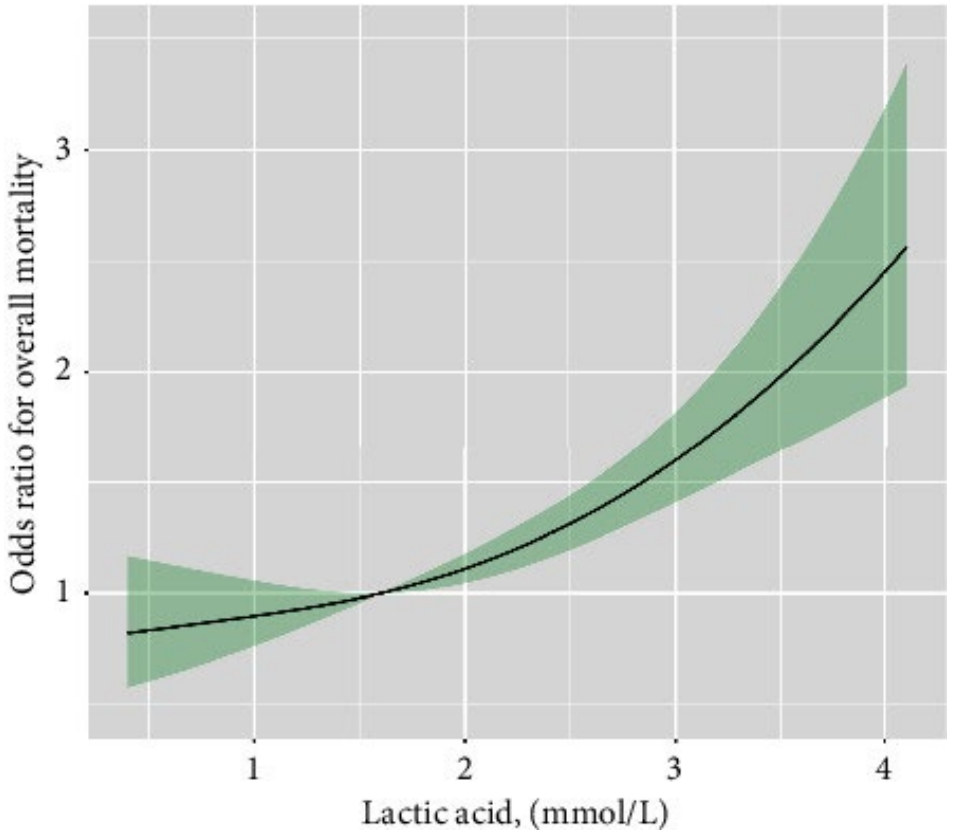
How to tell when you have the gift of time

Two Key Concepts in Hemodynamics:

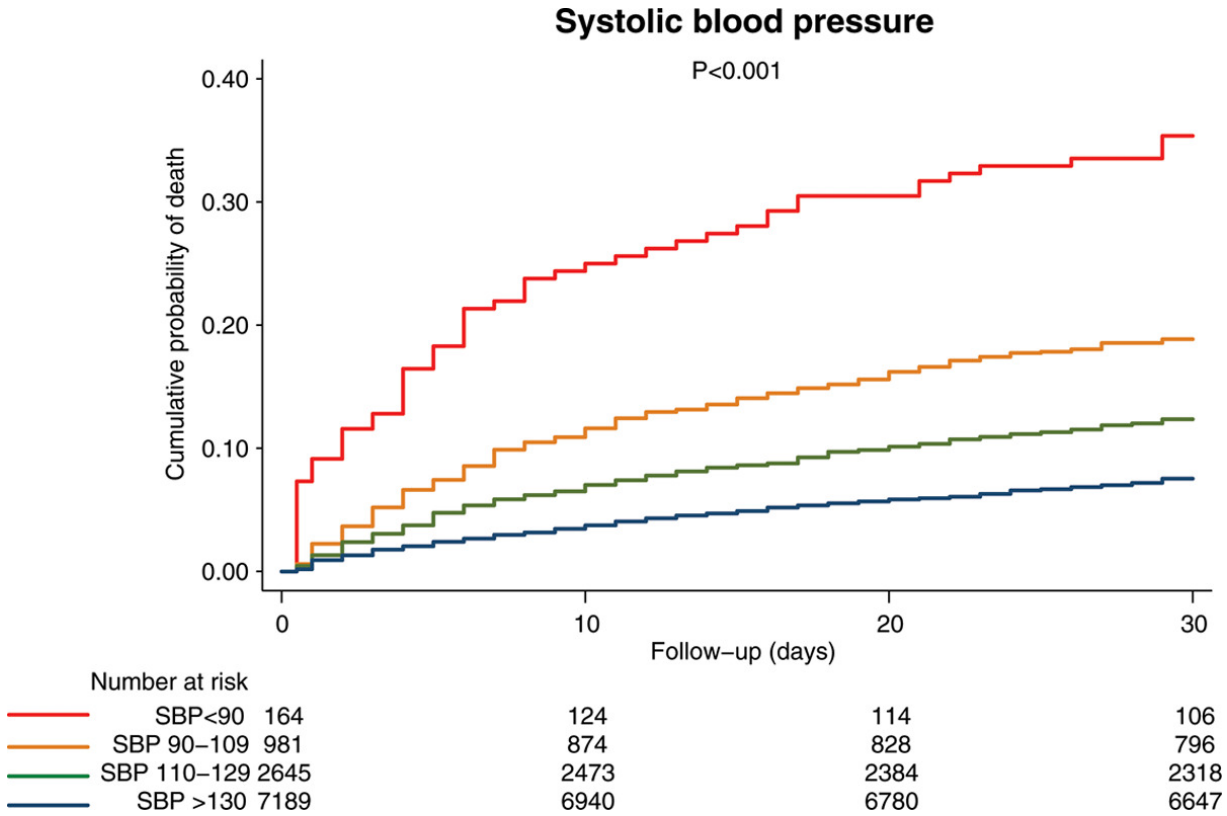
- **Perfusion**
  - Low perfusion signs: cool extremities, hypotension, narrow pulse pressure, altered mental status, rising creatinine.
  - Measured by: Cardiac Index (CI), blood pressure, clinical exam.

- **Congestion**
  - Congestion signs: elevated JVP, crackles, edema, hepatomegaly, orthopnea.
  - Measured by: PCWP, JVP, lung ultrasound, weight changes.

Perfusion is acutely important



"Predictive Value of Arterial Blood Lactic Acid Concentration on the Risk of in-Hospital All-Cause Death in Patients with Acute Heart Failure." *International journal of clinical practice* 2022.1 (2022)



"Synergistic impact of systolic blood pressure and perfusion status on mortality in acute heart failure." *Circulation: Heart Failure* 14.3 (2021)

Suspect Shock!

TABLE 1 SUSPECT CS: A Mnemonic to Aid in Confirming a Diagnosis of CS

Symptoms/**S**igns	Altered mental status, confusion, chest pain or pressure, cold and clammy extremities, rapid pulse, low pulse pressure (<25% of SBP), elevated jugular venous pressure, crackles, rales, orthopnea, paroxysmal nocturnal dyspnea, lower extremity edema
Urine output	Oliguria or anuria, <30 mL/h (<0.5 mL/[kg·h])
Sustained hypotension	SBP <90 mm Hg, MAP <65 mm Hg for >30 min or a >30-mm Hg decrease from baseline, or the need for pharmacological or mechanical support to maintain SBP >90 mm Hg
Perfusion	Evaluate markers of end-organ malperfusion, including lactic acid >2 mmol/L, ALT >200 U/L or >3× upper limit of normal, creatinine ≥2× upper limit of normal, pH <7.2, metabolic acidosis without another known cause
ECG/**E**chocardiogram	Evaluate acute ischemia, including ECG and sonographic evidence of STEMI (regional wall motion abnormalities); evidence of LV or RV dilation and systolic dysfunction; valvular pathology
Congestion	Presence or absence of congestion based on physical signs and hemodynamics; elucidation of ventricular involvement (LV vs RV vs BiV)
Triage	Appropriate triage/shock team activation or possible transfer to a higher level of care

"2025 Concise Clinical Guidance: an ACC expert consensus statement on the evaluation and management of cardiogenic shock: a report of the American College of Cardiology Solution Set Oversight Committee." *Journal of the American College of Cardiology* 85.16 (2025)

According to the ACC:

Fewer than 10%

of ED visits with ADHF have acute life-threatening illness

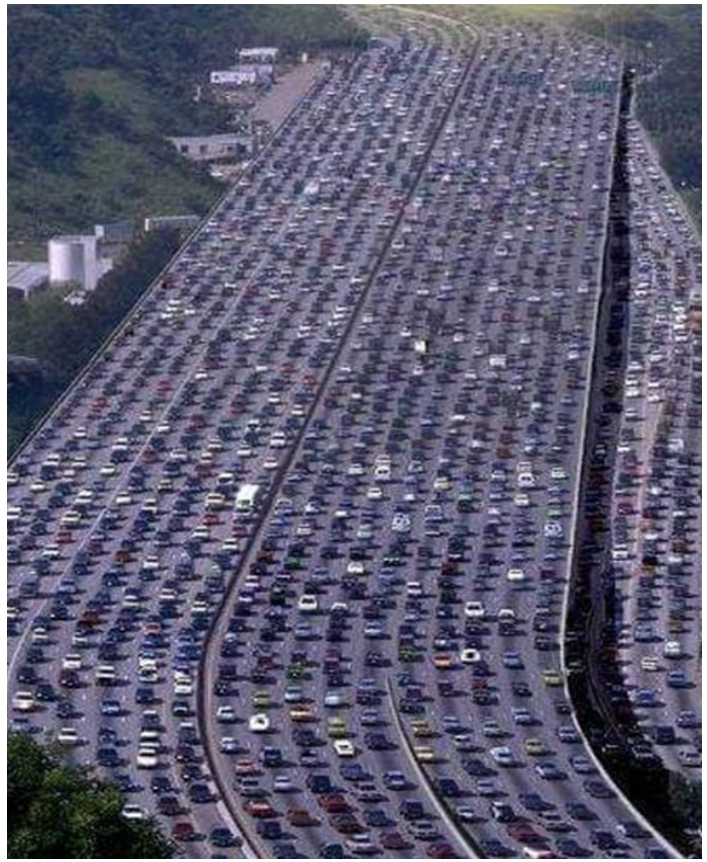
2019 ACC Expert Consensus Decision Pathway on Risk Assessment, Management, and Clinical Trajectory of Patients Hospitalized With Heart Failure

Congestion is the ballgame

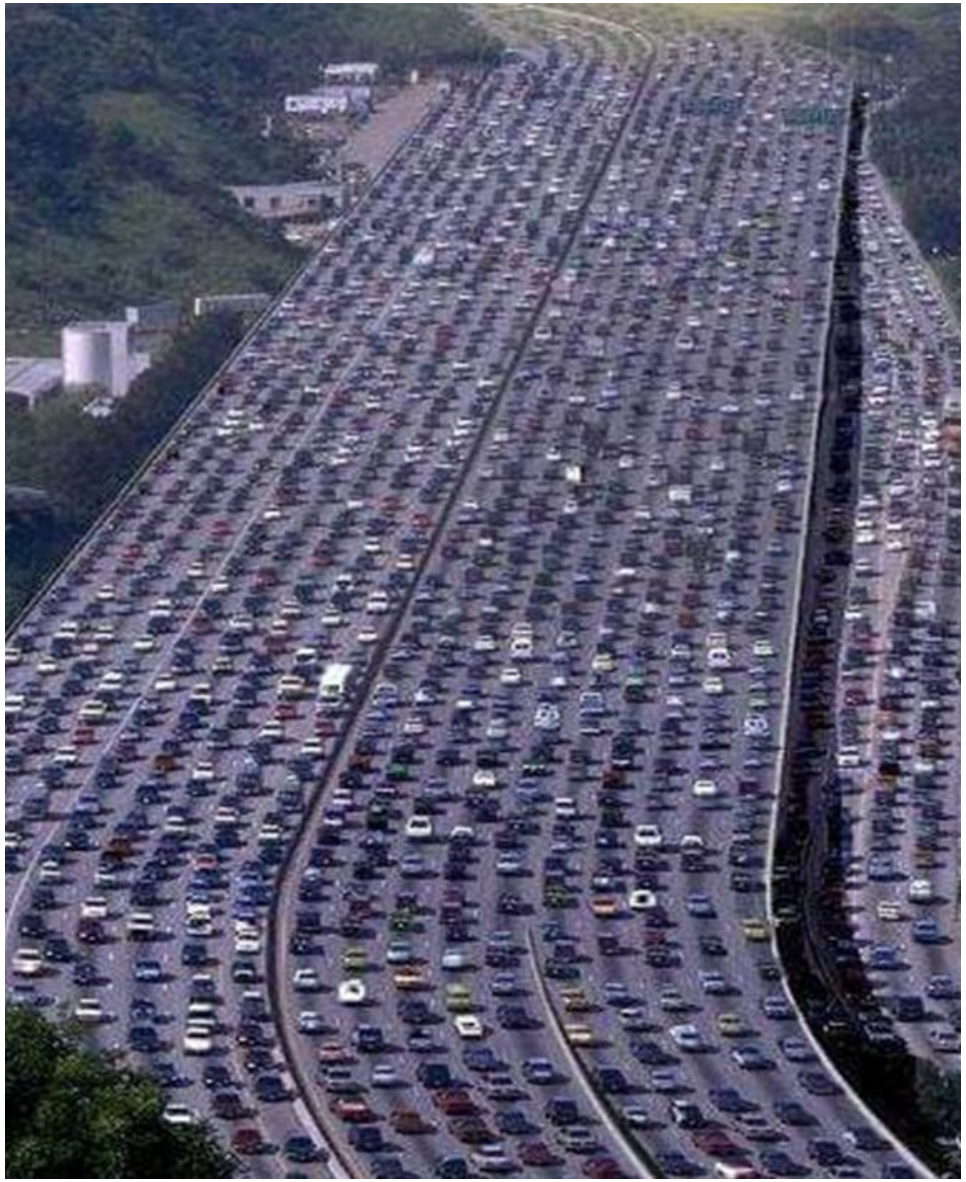
- **OPTIMIZE-HF**

- On ***admission***:

  Each 3-point increase in a 15-point congestion score was associated with higher 1-year mortality (adjusted HR 1.06, 95% CI 1.03–1.09).



Cooper, et al. Am J Card. 2019 Aug 15;124(4):545-553



Congestion is the ballgame

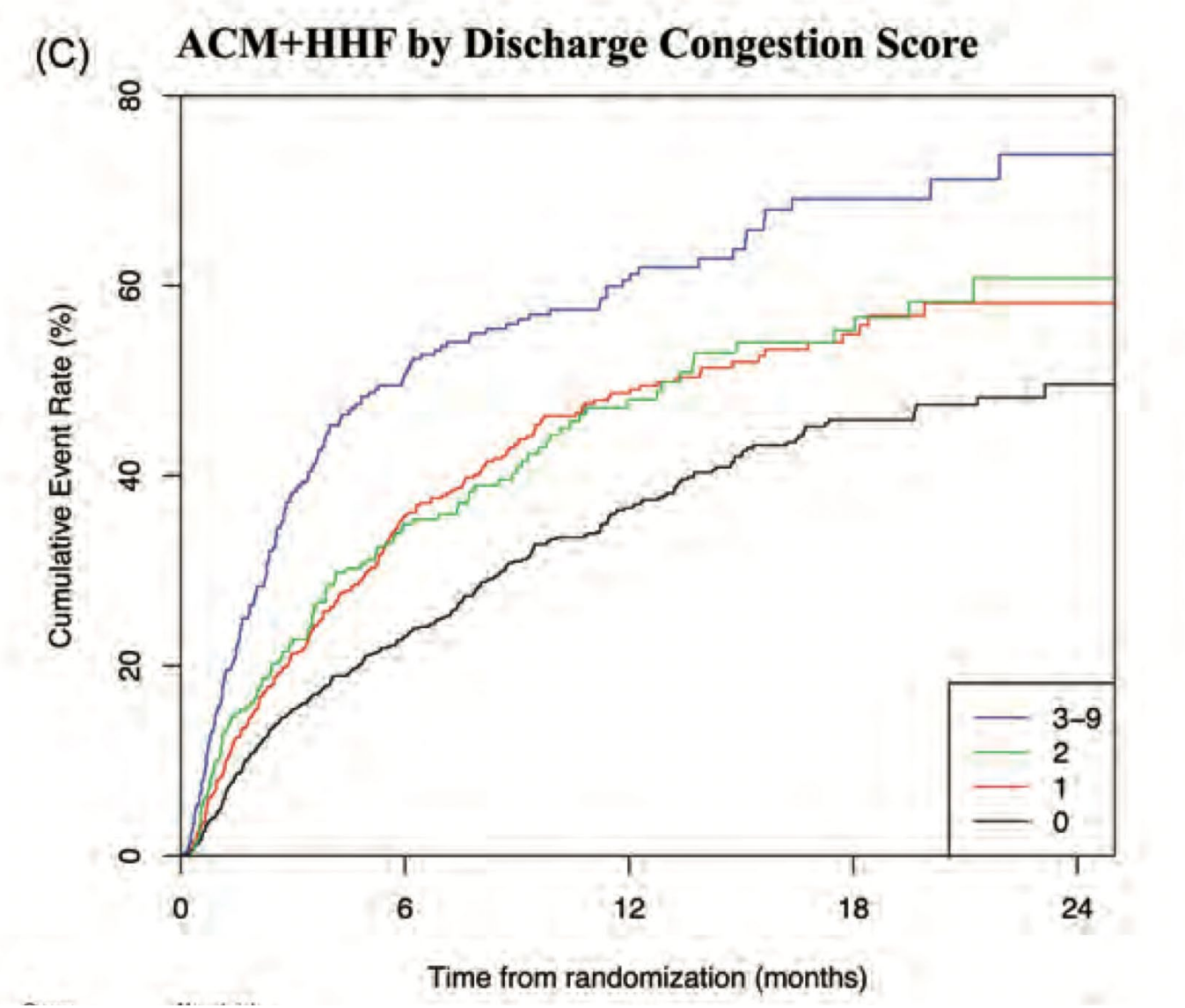
- EVEREST

- On ***Discharge:***

  each 1-point higher congestion score was associated with increased all-cause mortality at 30 days and over longer follow-up (HR per point 1.16, 95% CI 1.09–1.24).

Ambrosy, et al. European Heart Journal (2013) 34, 835–843

More congestion = Higher mortality and readmissions



Ambrosy, et al. European Heart Journal (2013) 34, 835–843

Clinical Evidence of Congestion	
Symptoms	**Signs†**
• Orthopnea • Dyspnea on minimal exertion • Paroxysmal nocturnal dyspnea • Nocturnal cough* • Bendopnea • Abdominal swelling • Early satiety • Anorexia, nausea • Right upper quadrant pain • Peripheral swelling • Rapid weight gain	• Elevated jugular venous pressure • Rales‡ • Pleural effusion‡ • Increased intensity of pulmonary component of second sound • Third heart sound • Murmurs of mitral and/or tricuspid regurgitation • Pulsatile hepatomegaly • Ascites§ • Pre-sacral, scrotal, or perineal edema • Peripheral edema

* Often when supine; † JVP is the most sensitive sign. Rales may not always be present; ‡ Not common in chronic HF;
§ May be difficult to distinguish from central adiposity

ACUTE CHF EXACERBATION MANAGEMENT

	DRY (No congestion)	**WET** (Congestion present)
WARM (Good perfusion)	**WARM/DRY** Compensated, no urgent treatment needed	**WARM/WET** Decongest with diuretics ± vasodilators
COLD (Poor perfusion)	**COLD/DRY** Optimize preload, avoid over-diuresis, consider inotropes if needed	**COLD/WET** Cautious diuresis + inotropes if hypoperfused

Why did no one like the EKG technician’s jokes?

Why did no one like the EKG technician’s jokes?

They were too tachy!

Agenda

Hemodynamics first

Evidence based therapies

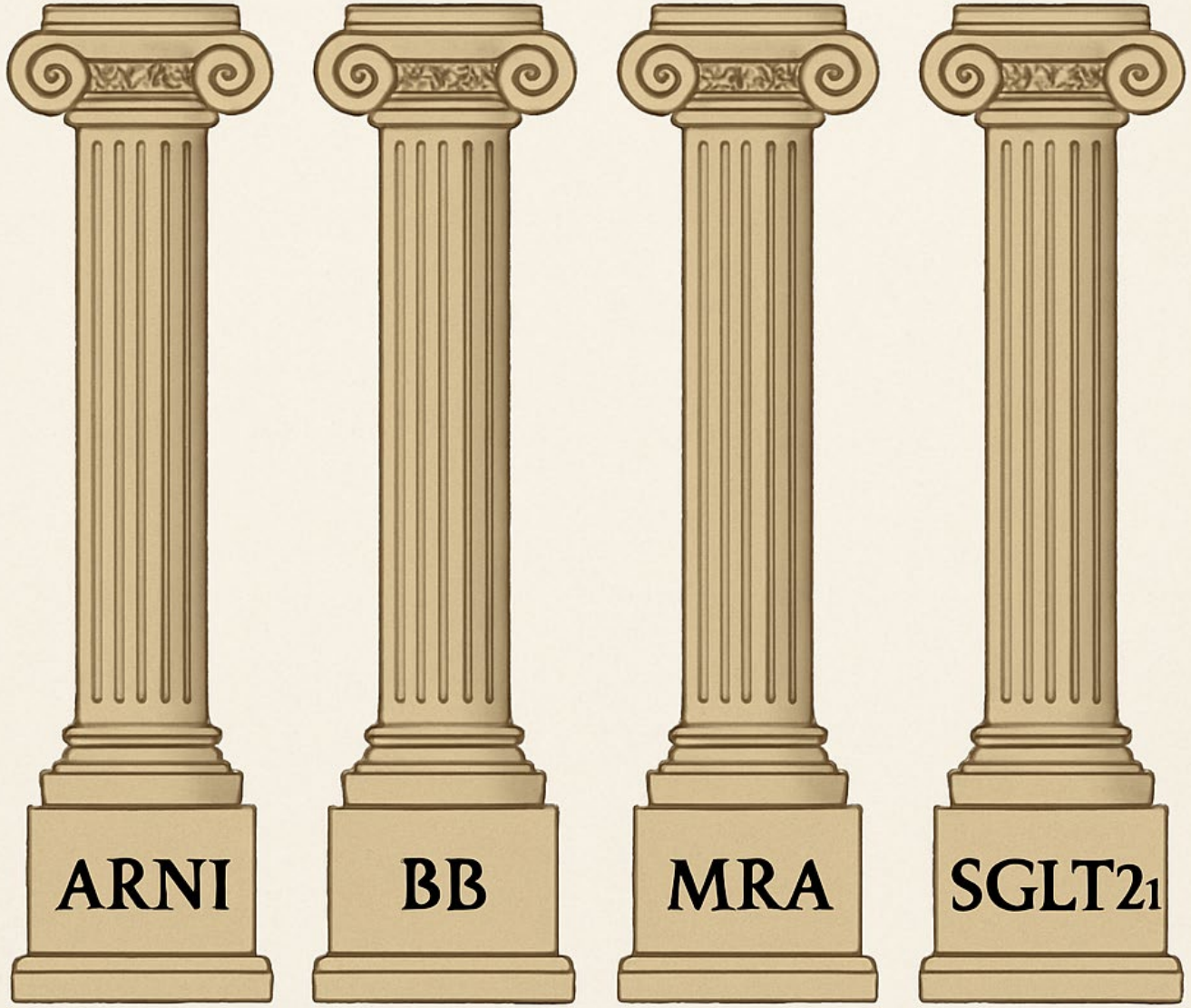
Acute interventions

Risk stratification

Transition planning

GDMT

THE FOUR PILLARS OF TREATMENT



ACE

- **Mechanism:** block the enzyme which is responsible for converting angiotensin I to angiotensin II.

- **Effect:** Reduce levels of angiotensin II, leading to:
  - **Vasodilation:** Blood vessels relax, lowering blood pressure.
  - **Reduced aldosterone secretion:** reduce fluid overload.
  - **Reduced sympathetic nerve activity:** The sympathetic nervous system is activated in heart failure, and ACE inhibitors help dampen this response.
  - **Increased bradykinin levels:** ACE also degrades bradykinin, a vasodilator, so inhibiting ACE leads to another mechanism of improving vasodilation

ACE of Hearts

	Clinical trial	No. of patients	Follow-up, mo	End point	Event rate, %		HR (95% CI)	*P* value
					Study drug	Control		
ACE inhibitors								
Captopril	SAVE[34]	2231	42	All-cause mortality	20.4	24.6	0.81 (0.68-0.97)	.02
				CV mortality	16.8	20.9	0.79 (0.65-0.95)	.01
Ramipril	AIRE[35]	2006	15	All-cause mortality	16.7	22.4	0.73 (0.60-0.89)	.002
Enalapril	SOLVD[36]	2569	41.4	All-cause mortality	35.2	39.7	0.84 (0.74-0.95)	.003
				Deaths from HF	16.3	19.5	0.78 (0.65-0.94)	

Heart Failure with Reduced Ejection Fraction: A Review. JAMA.2020;324(5):488-504

ARNI

- **Mechanism:** ARB + neprilysin inhibitor
  - **ARB component:** Blocks the effects of angiotensin II at the receptor level
  - **Neprilysin inhibitor component:** Neprilysin degrades peptides like ANP and BNP, which promote vasodilation and diuresis. By inhibiting neprilysin, ARNIs increase the levels of these beneficial peptides.

- **Effect:**
  - **Reduce the harmful effects of angiotensin II:** Similar to ACE inhibitors and ARBs.
  - **Enhance the beneficial effects of natriuretic peptides:** promoting vasodilation, diuresis, and natriuresis (increased sodium excretion in urine).

ARNI you glad to learn about CHF?

Pioneer Trial

NEJM 2014

- **Angiotensin–Neprilysin vs Enalapril**

- **Hazard ratio 0.79 – 0.84 for**
  - **Death from any cause**
  - **Death from CV cause**
  - **Hospitalization for CHF**

ACE / ARB / ARNI
To Allow or to Abstain

Indications

- *Symptomatic* HFrEF, HFmrEF, HFpEF
- ARNI (sacubitril/valsartan) is preferred over ACE/ARB when tolerated

Contraindications

- History of angioedema with ACEi/ARB/ARNI
- Pregnancy
- bilateral renal artery stenosis
- symptomatic hypotension
- severe hyperkalemia ($K^+ > 5.5$)
- ARNIs should not be given within 36 hours of last ACEi dose

Beta Blockers

- **Block Sympathetic Activation**
- **Reduce Heart Rate and Blood Pressure**
- **Improve diastolic filling**
- **Decrease contractility (less work)**
- **Decrease Cardiac output**
- **Cardioprotective – less apoptosis**

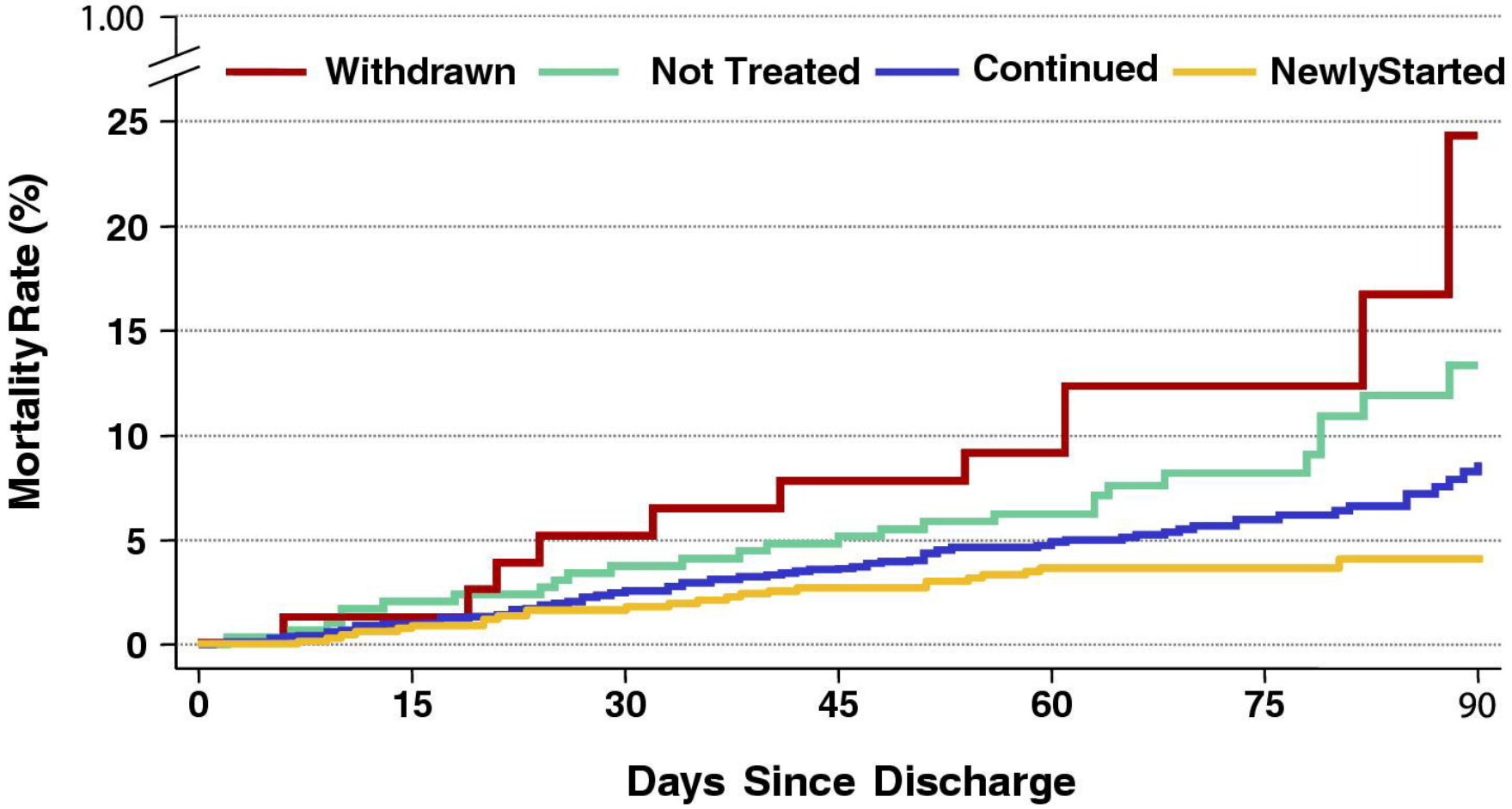
You Beta (blocker) Believe It!

- **Bisoprolol**: The CIBIS-II trial showed a 32% reduction in all-cause mortality (HR 0.66, 95% CI 0.54–0.81) in NYHA class III–IV patients.

- **Carvedilol**: The COPERNICUS trial found a 35% reduction in all-cause mortality (HR 0.65, 95% CI 0.52–0.81), and pooled analyses of carvedilol trials showed up to a 65% reduction in mortality.

- **Metoprolol succinate (CR/XL)**: The MERIT-HF trial demonstrated a 32% reduction in all-cause mortality, hospitalization or ER visit for worsening CHF (HR 0.66, 95% CI 0.53–0.81).

Don't Stop the Block!



Patients at risk:							
Withdrawn	79	77	73	68	66	26	10
Not Treated	303	275	269	262	242	114	51
Continued	1350	1303	1268	1236	1123	536	224
NewlyStarted	632	609	591	575	531	274	110

Fonarow GC, Abraham WT, et al. OPTIMIZE-HF program. J Am Coll Cardiol. 2008 Jul 15;52(3):190-9

Beta Blockers
To Bequeath or to Ban

Indications

- All patients with stable HFrEF, unless contraindicated

Contraindications

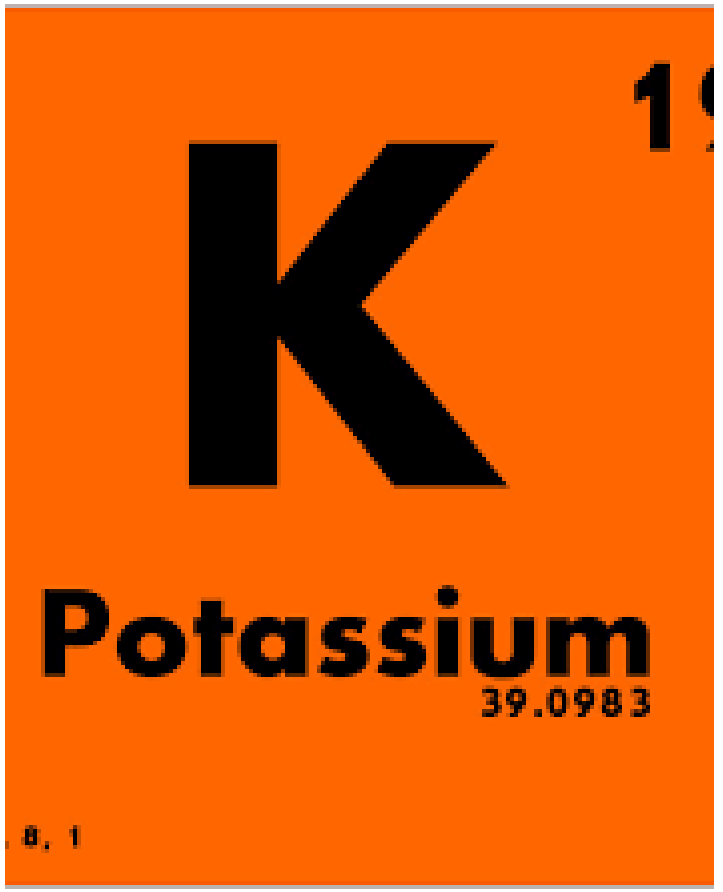
- Severe bradycardia
- Second- or third-degree heart block without a pacemaker
- Severe asthma
- Acute decompensated HF with evidence of hypoperfusion or requiring inotropes

Mineralocorticoid Antagonists

In CHF, RAAS becomes overactive and increased Aldosterone causes increased salt and water retention. Aldosterone can also increase inflammation and cardiac fibrosis.

Mineralocorticoid Receptor Antagonists:

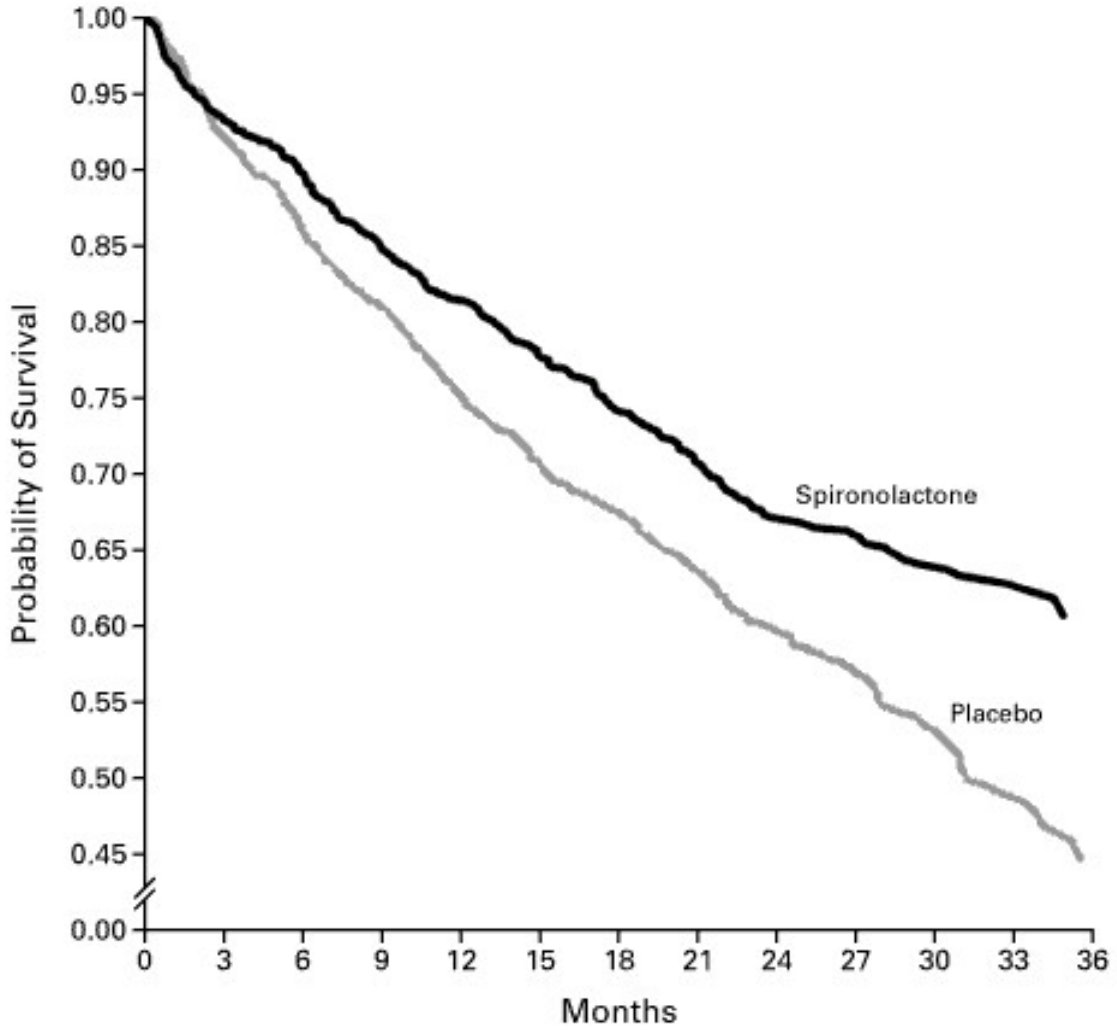
Decrease inflammation and cardiac remodeling / fibrosis	Decrease sodium and water retention	Decrease BP



MRA Magic

Spironolactone:

30% Reduction in mortality (RR 0.70 CI 0.59-0.82)



No. at Risk													
Placebo	841	775	723	678	628	592	565	483	379	280	179	92	36
Spironolactone	822	766	739	698	669	639	608	526	419	316	193	122	43

‘The effect of spironolactone on morbidity and mortality in patients with severe heart failure." *New England Journal of Medicine* 341.10 (1999): 709-717

MRAs
To Mete or to Muzzle

Indications

- NYHA class II–IV HFrEF

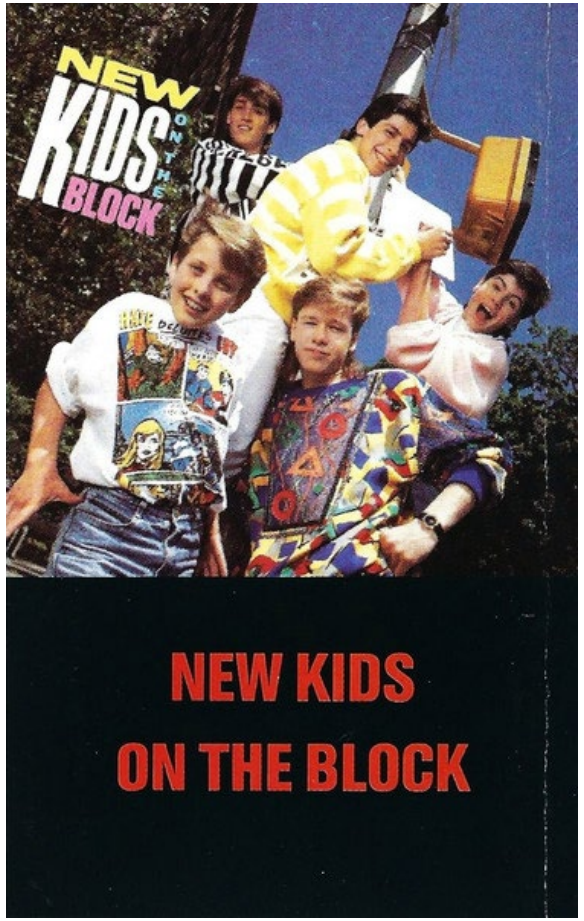
Contraindications

- eGFR <30
- $K^+ > 5.0$
- History of hyperkalemia

SGLT2i - No Dozin’ with Flozins

Not just for diabetics!

Dapagliflozin (Farxiga)
Canagliflozin (Invokana)
Empagliflozin (Jardiance)



SGLT2i

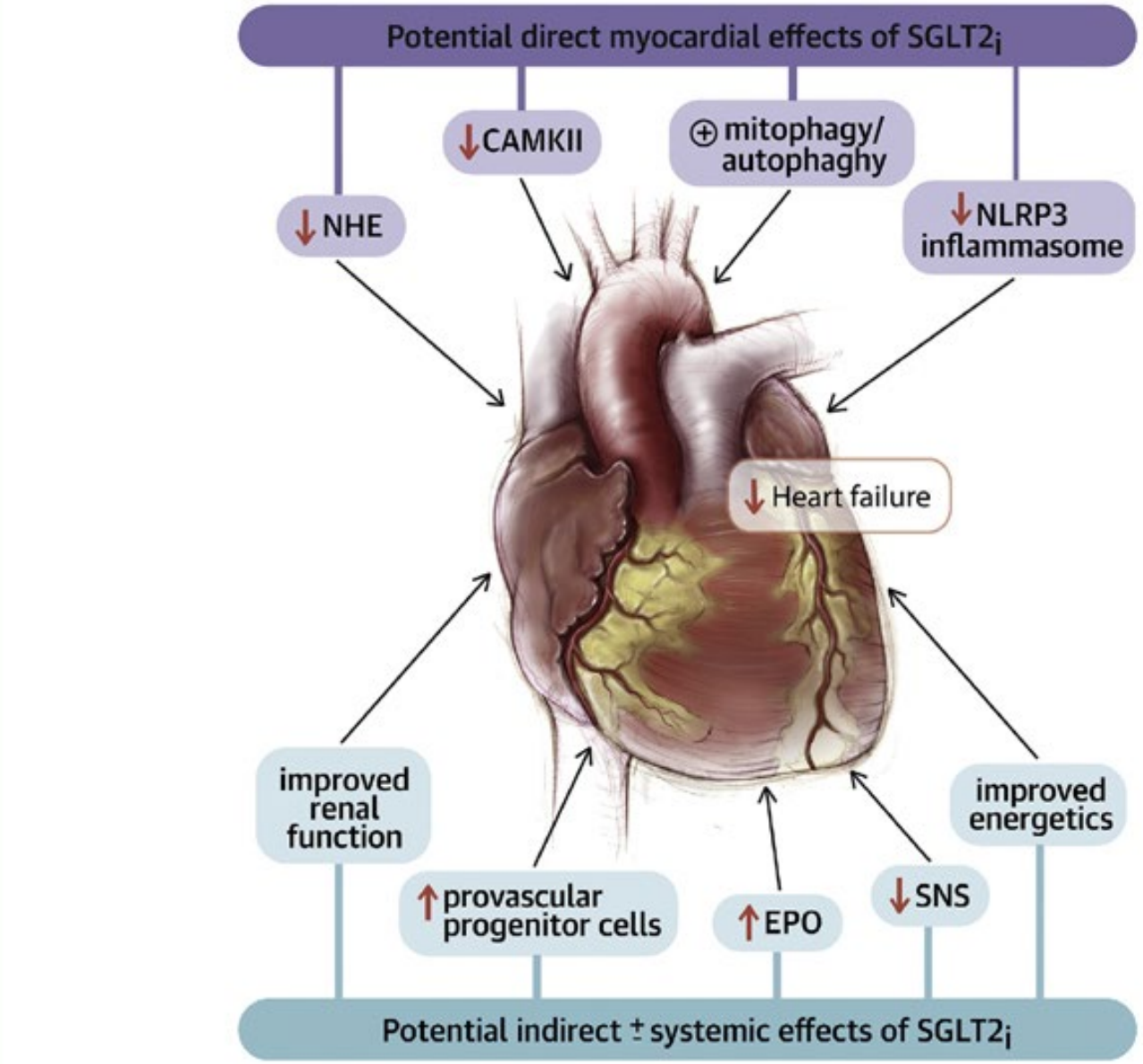
- Sodium-Glucose Cotransporter 2
  - Proximal tubules
  - Resorbs the majority of glucose in the tubule
  - Resorbs sodium

- Blocking this Chanel
  - Increases glucosuria / natriuresis

But…..

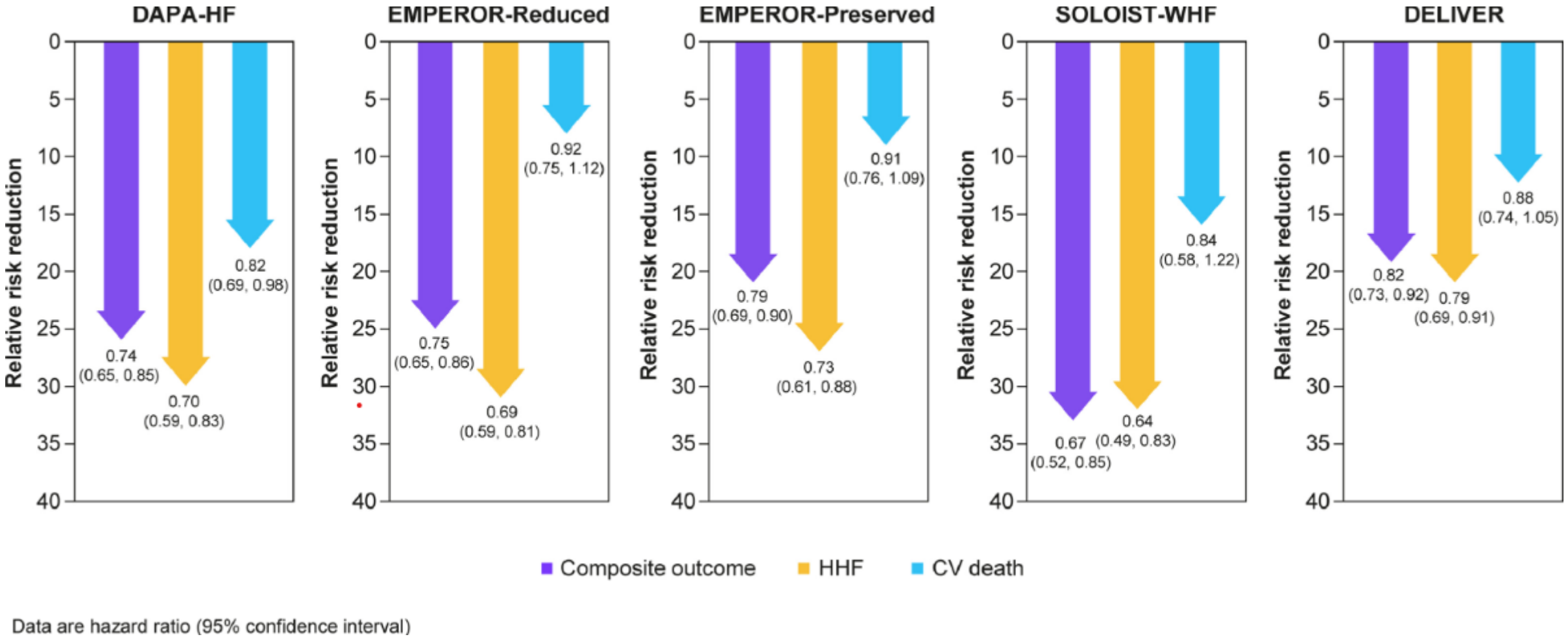
SGLT2i – Magic Beyond the sugars

CENTRAL ILLUSTRATION: Potential Direct Myocardial and Indirect ± Systemic Effects of SGLT2$_i$



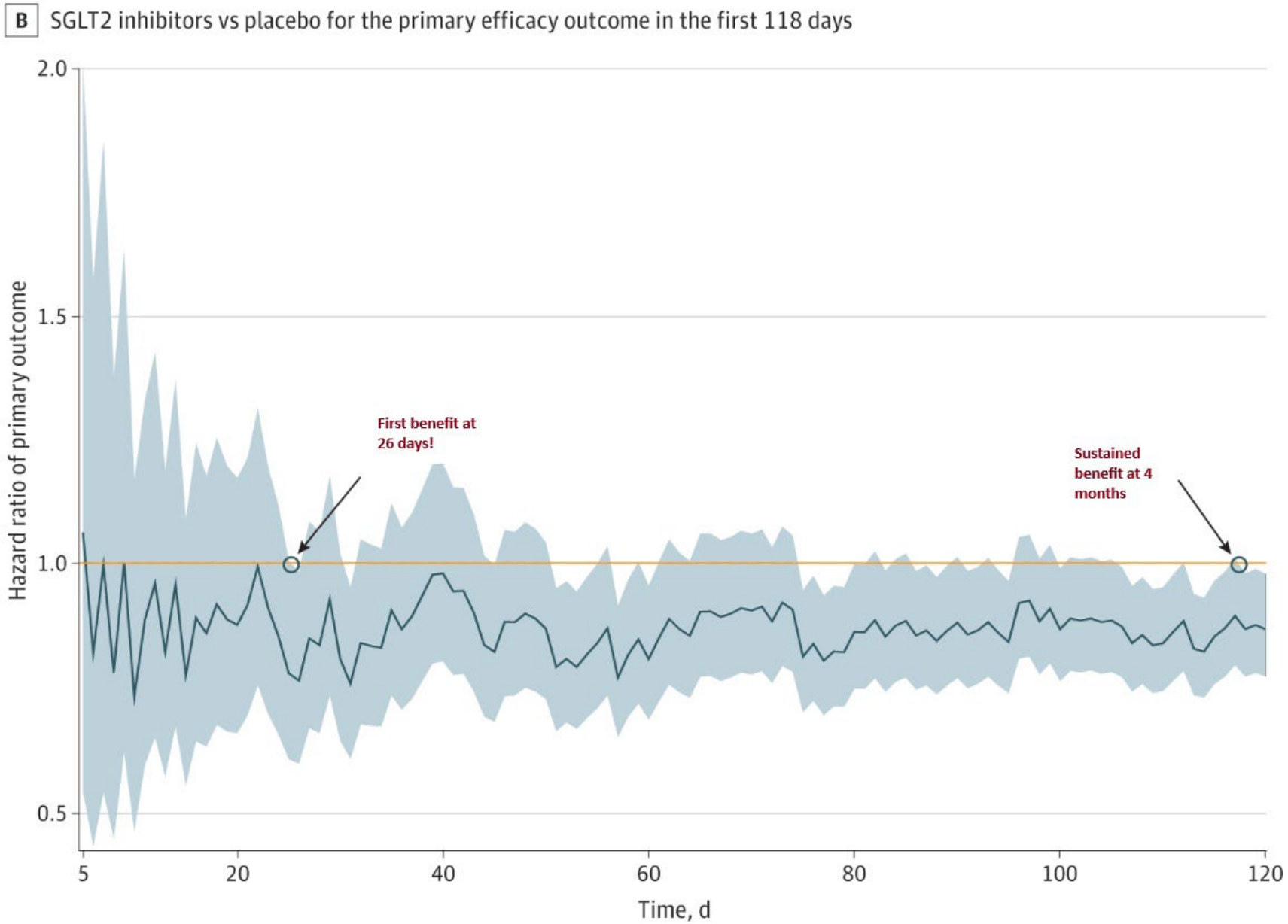
Lopaschuk, G.D. et al. J Am Coll Cardiol Basic Trans Science. 2020;5(6):632-44.

The Power of the Flozins



Data are hazard ratio (95% confidence interval)

Stephen J. Greene MD, Javed Butler MD, MPH, MBA and Mikhail N. Kosiborod MD. Chapter 3: Clinical Trials of Sodium-Glucose Co-Transporter-2 Inhibitors for Treatment of Heart Failure. The American Journal of Medicine, 2024-02-01, Volume 137, Issue 2, Pages S25-S34.



Rapid benefit from starting SGLT2s

Chen, Kangyu, et al. "Time to benefit of sodium-glucose cotransporter-2 inhibitors among patients with heart failure." *JAMA network open* 6.8 (2023):

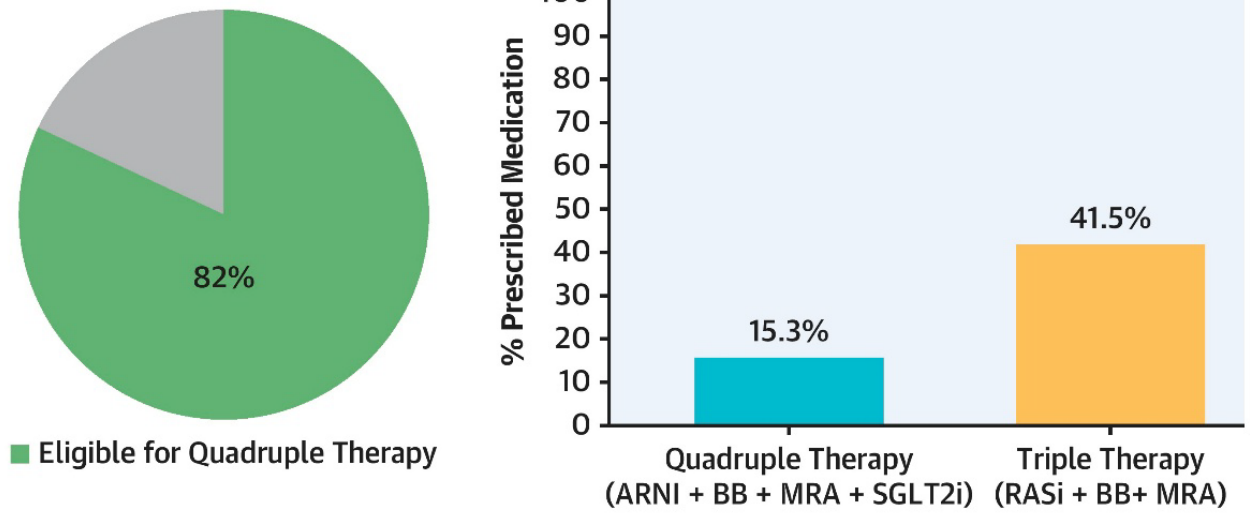
SGLT2i
To Supply or to Stop

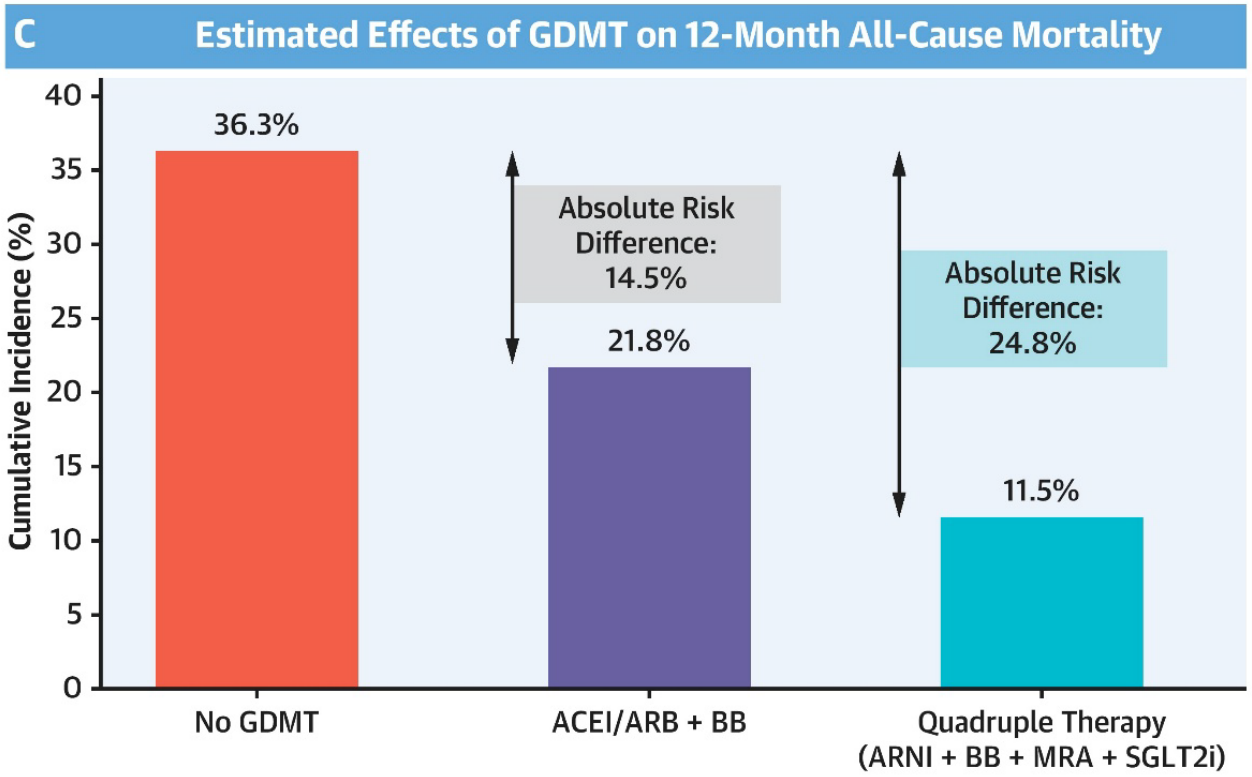
Indications

- Symptomatic HFrEF (LVEF ≤40%), regardless of diabetes status
- eGFR above agent-specific thresholds
  - empagliflozin ≥20
  - dapagliflozin ≥30

Contraindications

- Type 1 diabetes
- History of diabetic ketoacidosis
- Severe hypersensitivity
- eGFR below agent-specific threshold



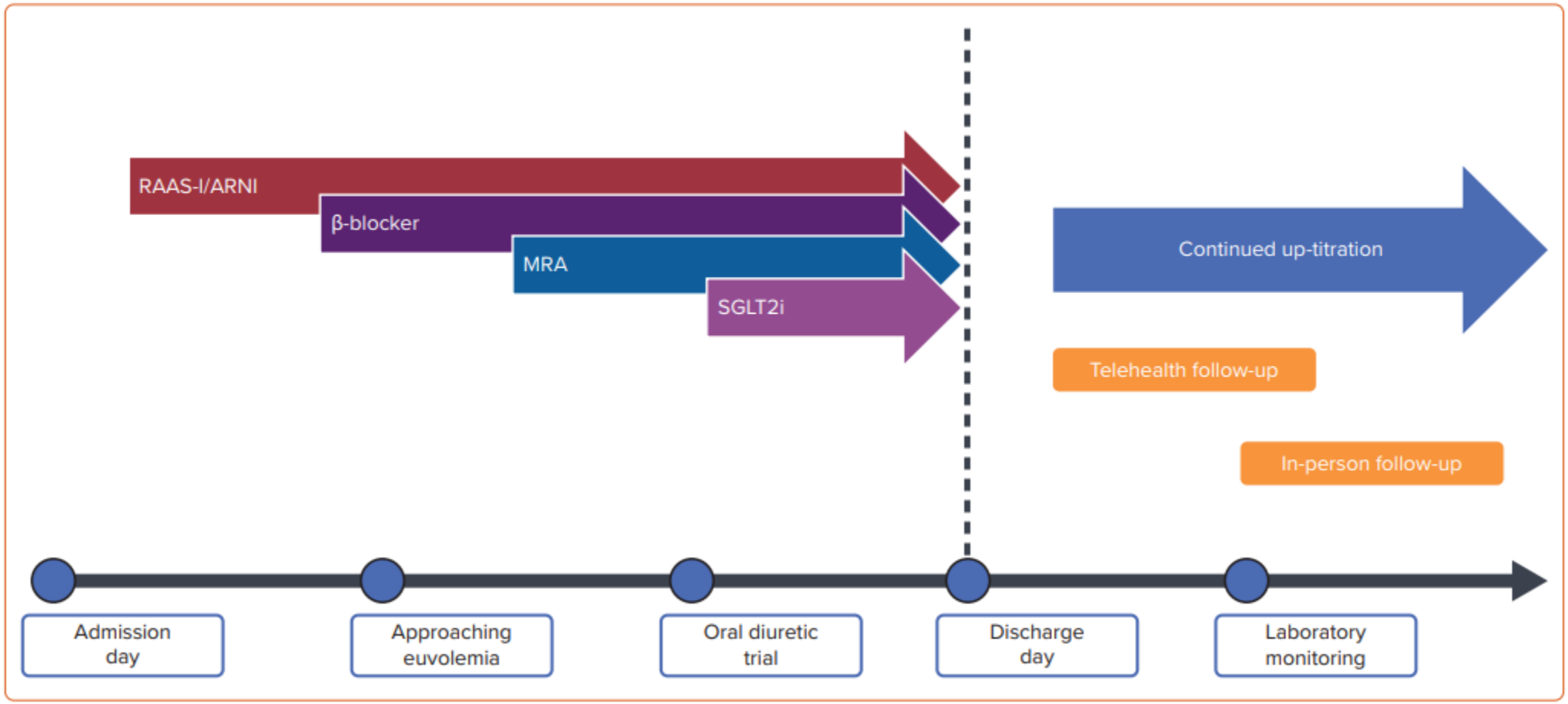


Greene SJ, et al. J Am Coll Cardiol HF. 2024;12(8):1365-1377.

Opportunity knocks!

Timing

Figure 1: Shifting the Paradigm of Guideline-directed Medical Therapy Initiation



A suggested timeline of initiating guideline-directed medical therapy (GDMT) for patients admitted with heart failure with reduced ejection fraction during their hospitalization. ACEi = angiotensin converting enzyme inhibitor; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor–neprilysin inhibitor; MRA = mineralocorticoid receptor antagonist; RAAS-I = renin-angiotensin-aldosterone system inhibitor; SGLT2i = sodium–glucose cotransporter-2 inhibitor.

Optimizing Guideline-directed Medical Therapies for Heart Failure with Reduced Ejection Fraction During Hospitalization. US Cardiol. 2021 Apr 23;15:

Class	Agent	Starting Dose	Target Dose
ARNI	Sacubitril / valsartan	24/26 mg or 49/51 mg BID	97/103 mg BID
ACE Inhibitors	Enalapril	2.5 mg BID	10–20 mg BID
Beta-blockers	Bisoprolol	1.25 mg daily	10 mg daily
	Carvedilol	3.125 mg BID	25–50 mg BID
	Metoprolol succinate	12.5–25 mg daily	200 mg daily
Mineralocorticoid Receptor Antagonists (MRA)	Spironolactone	12.5–25 mg daily	25–50 mg daily
	Eplerenone	25 mg daily	50 mg daily
SGLT2 Inhibitors	Dapagliflozin or empagliflozin	10 mg daily	No titration needed

OK to Uptitrate

STRONG-HF: Rapid Up-Titration of GDMT in Acute Heart Failure

Design: Multinational, open-label, randomized trial
Population: 1078 patients **hospitalized** for acute HF

Intervention:

High-intensity care: Rapid up-titration of GDMT to full doses within 2 weeks + 4 follow-up visits

Usual care: Local standard practice

Primary Endpoint: 180-day all-cause death or HF readmission

OK to Uptitrate

STRONG-HF: Rapid Up-Titration of GDMT in Acute Heart Failure

- **Medication uptitration to full dose by day 90 (high-intensity vs usual care):**

- Renin-angiotensin blockers: **55% vs 2%**
- β-blockers: **49% vs 4%**
- MRAs: **84% vs 46%**

OK to Uptitrate

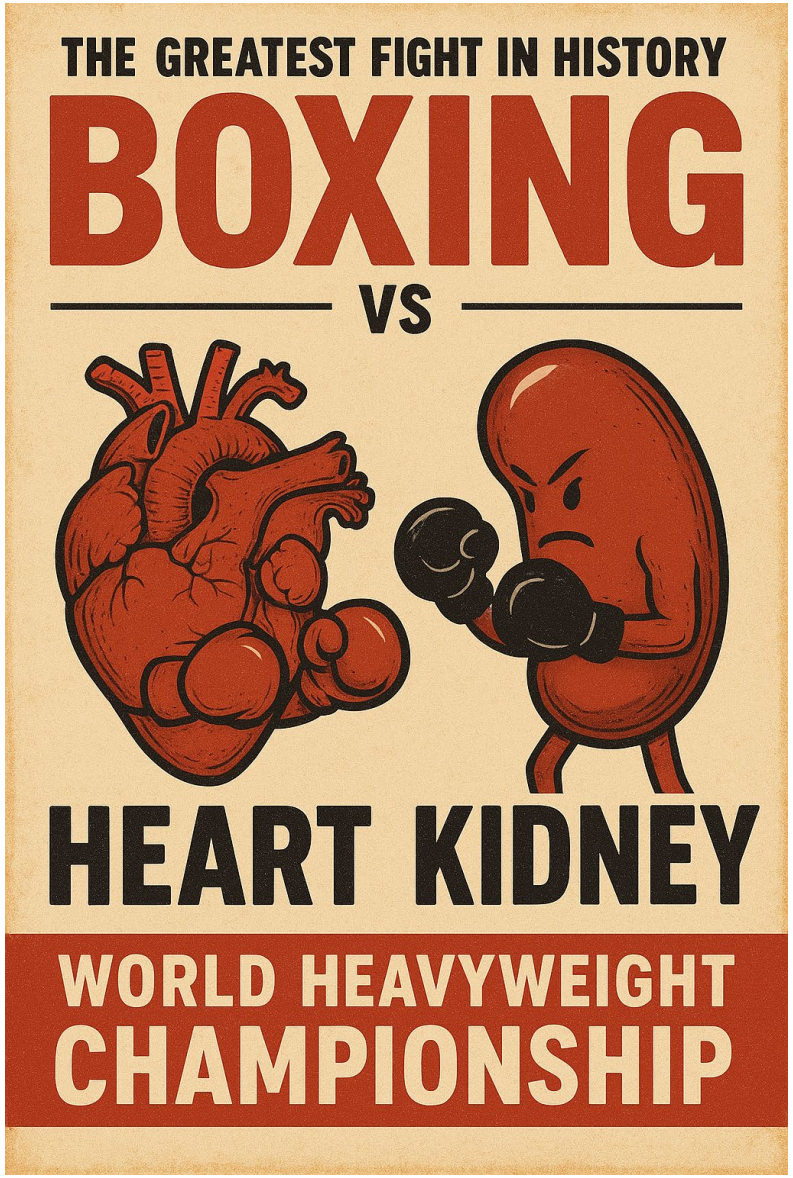
STRONG-HF: Rapid Up-Titration of GDMT in Acute Heart Failure

- Greater reductions in **NT-proBNP**, **bodyweight**, **NYHA class**, and **congestion signs**

- **Primary endpoint** (all cause mortality +6 mo CHF admission) occurred in **15.2%** (high-intensity) vs **23.3%** (usual care)
  - **Risk ratio:** 0.66 (95% CI: 0.50–0.86), **p = 0.0021**

What about…
AKI

- **ACE / ARB / ARNI** – dose reduce or hold if >30% decrease in eGFR or hyperkalemia
- **Beta blockers** - hold if concern for pulmonary congestion refractory to diuretics
- **MRA** – ok to continue if eGFR is >30 and K <5
- **SGLT2i** - ok to continue if eGFR is at least 20



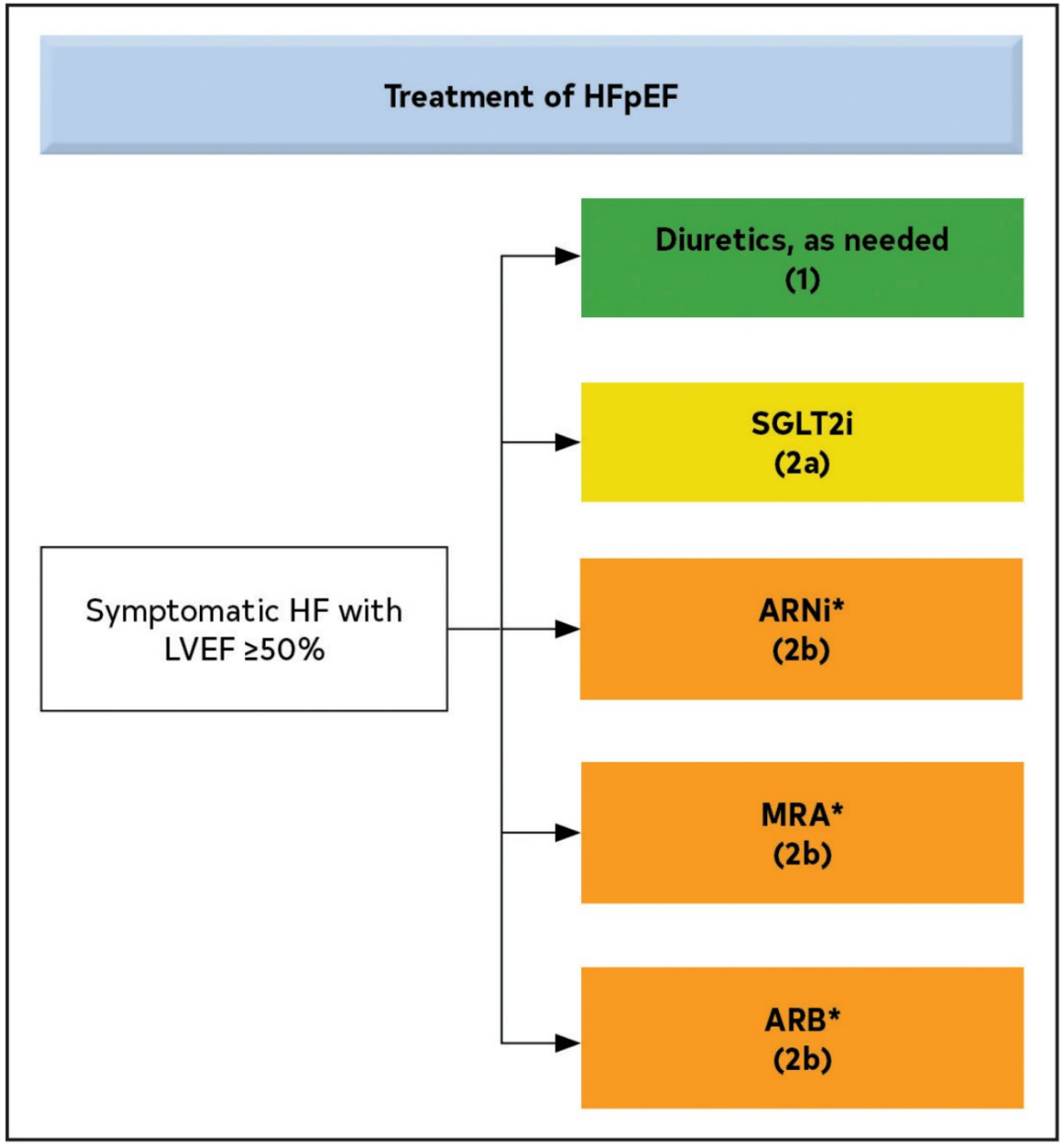
"Heart failure with reduced ejection fraction: a review." *Jama* 324.5 (2020): 488-504.

What about…

HFpEF

EF > 50%

Class (Strength) of recommendation	
Class 1a (Strong)	Benefit >>> Risk
Class 2a (Moderate)	Benefit >> Risk
Class 2b (Weak)	Benefit ≥ Risk



AHA/ACC/HFSA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *JACC.* 2022 May, 79 (17)

What about…

HFmrEF
EF 41-49%

Class (Strength) of recommendation	
Class 1a (Strong)	Benefit >>> Risk
Class 2a (Moderate)	Benefit >> Risk
Class 2b (Weak)	Benefit ≥ Risk

Recommendations for HF With Mildly Reduced Ejection Fraction
Referenced studies that support the recommendations are summarized in the Online Data Supplements.

COR	LOE	Recommendations
2a	B-R	1. In patients with HFmrEF, SGLT2i can be beneficial in decreasing HF hospitalizations and cardiovascular mortality.[1]
2b	B-NR	2. Among patients with current or previous symptomatic HFmrEF (LVEF, 41%–49%), use of evidence-based beta blockers for HFrEF, ARNi, ACEi, or ARB, and MRAs may be considered to reduce the risk of HF hospitalization and cardiovascular mortality, particularly among patients with LVEF on the lower end of this spectrum.[2–9]

AHA/ACC/HFSA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *JACC.* 2022 May, 79 (17)

What about…
for African Americans

Class (Strength) of recommendation	
Class 1a (Strong)	Benefit >>> Risk
Class 2a (Moderate)	Benefit >> Risk
Class 2b (Weak)	Benefit ≥ Risk

Recommendations for Hydralazine and Isosorbide Dinitrate
Referenced studies that support the recommendations are summarized in the Online Data Supplements.

COR	LOE	Recommendations
1	A	1. For patients self-identified as African American with NYHA class III-IV HFrEF who are receiving optimal medical therapy, the combination of hydralazine and isosorbide dinitrate is recommended to improve symptoms and reduce morbidity and mortality.[1,2]

AHA/ACC/HFSA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *JACC.* 2022 May, 79 (17)

What is the heart’s least favorite party?

What is the heart’s least favorite party?

A block party!

Agenda

Hemodynamics first

Evidence based therapies

Acute interventions

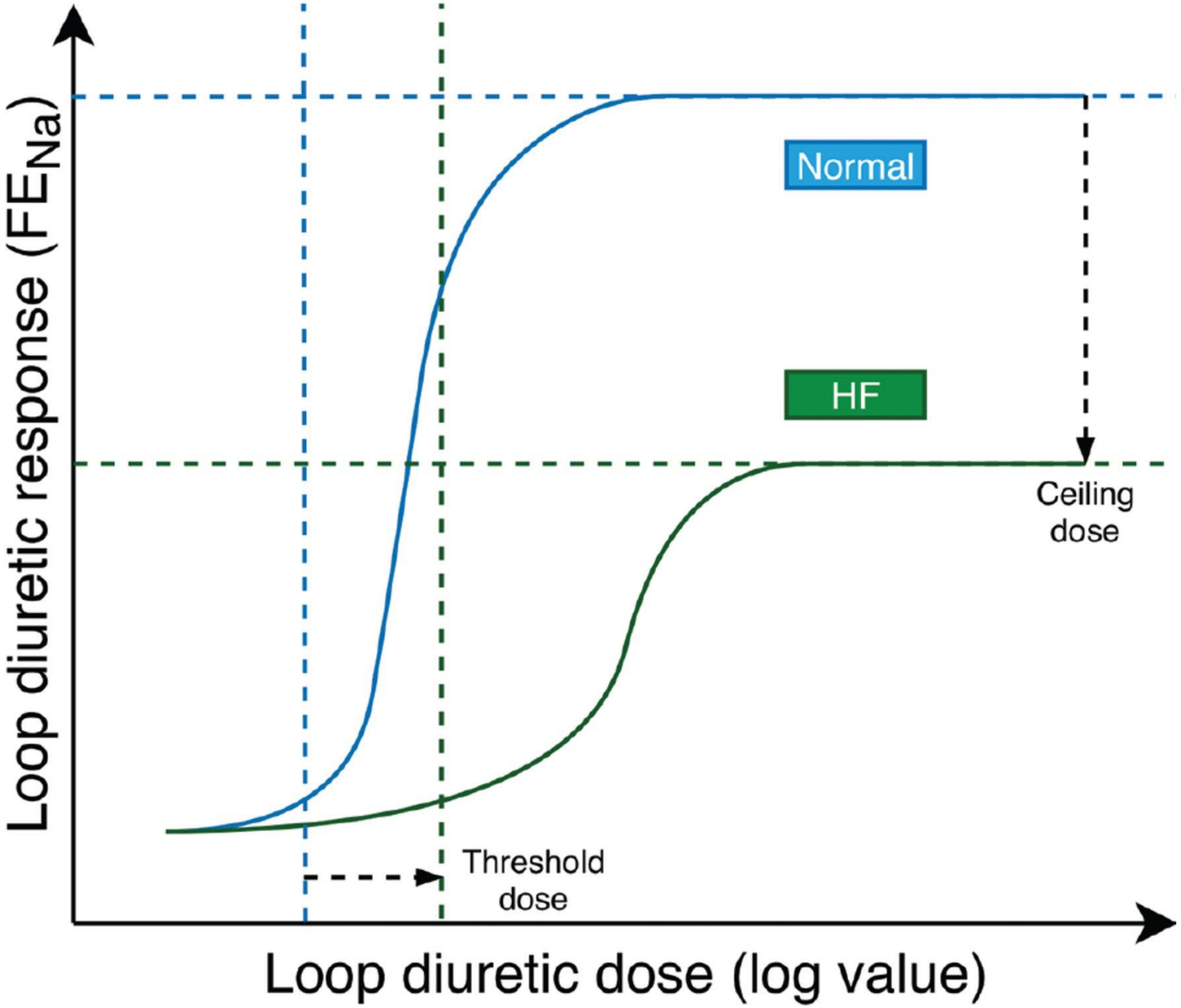
Risk stratification

Transition planning

Loop Diuretics

ACC:

- Start IV
- Doesn’t matter which loop you choose
- If lasix isn’t working, try bumex



Keep in mind..

Need ***higher*** doses

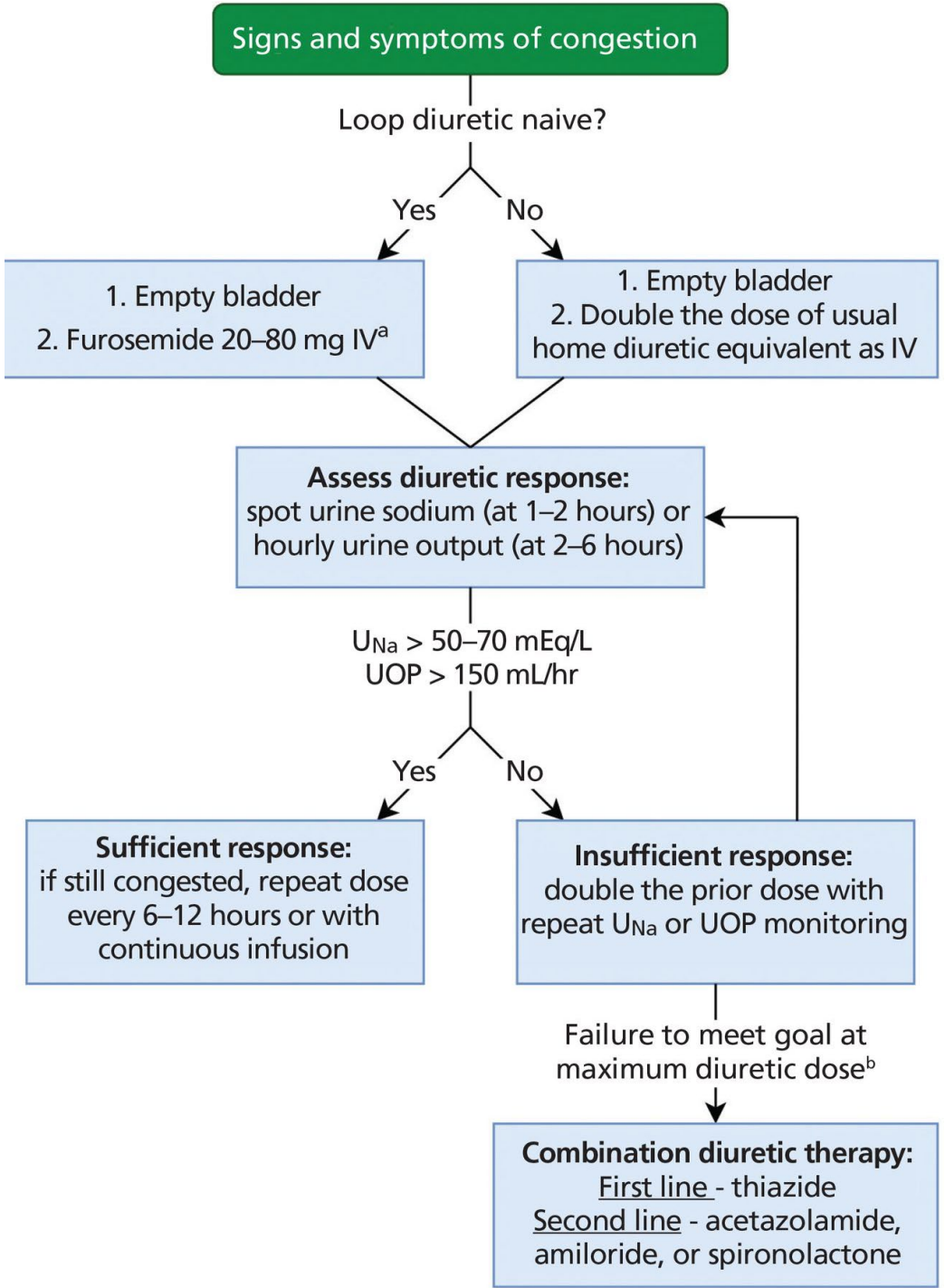
For a ***Lower*** total response

Dosing is a **LOG** scale
- based on doubling the dose

Cleveland Clinic journal of medicine 89.10 (2022): 561

The one-two punch with Thiazides

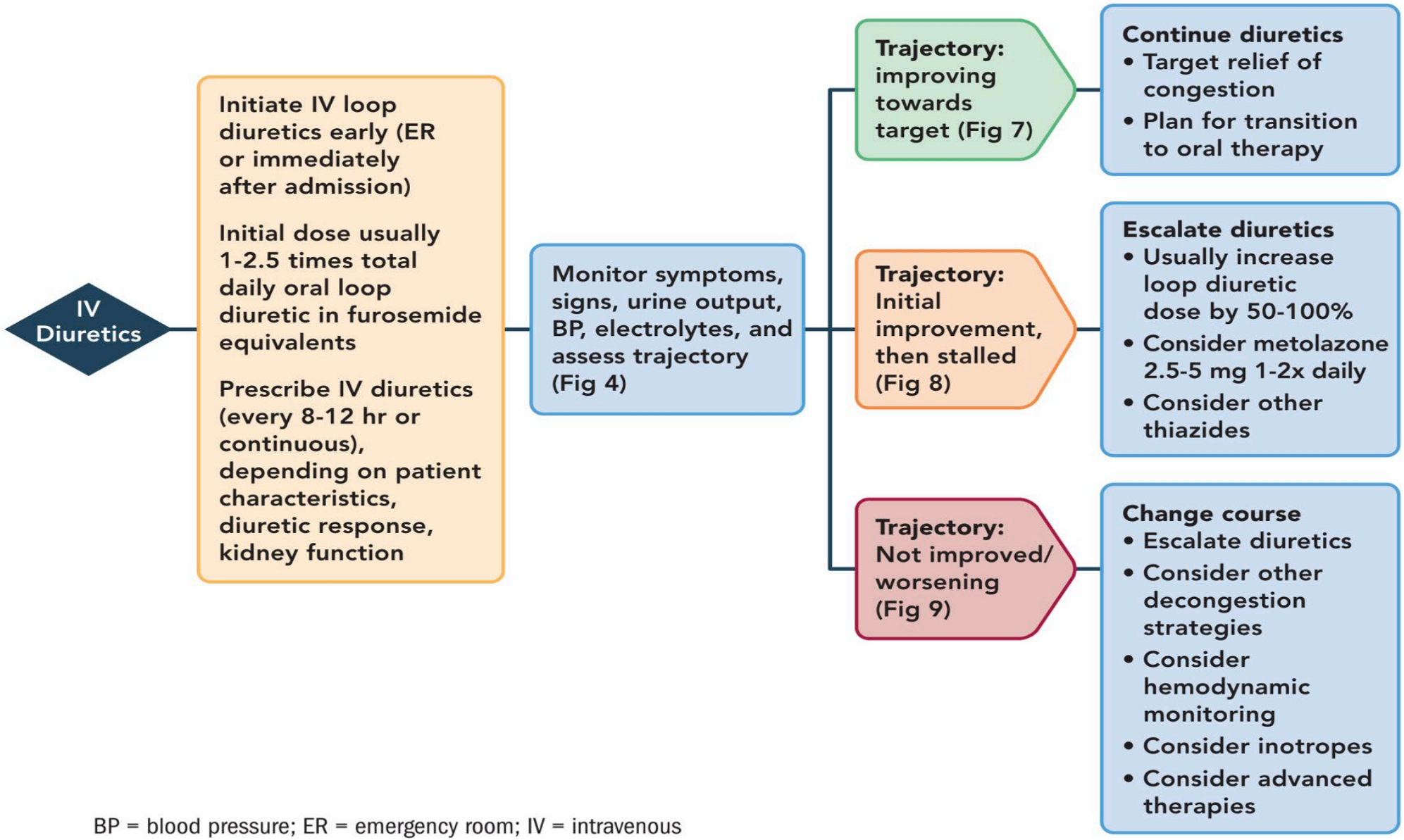
- Thiazides block the ability to resorb sodium in the distal tubule
- Maximize loop effect
- Synergistic effect



Goal UOP: 150 mL/hr

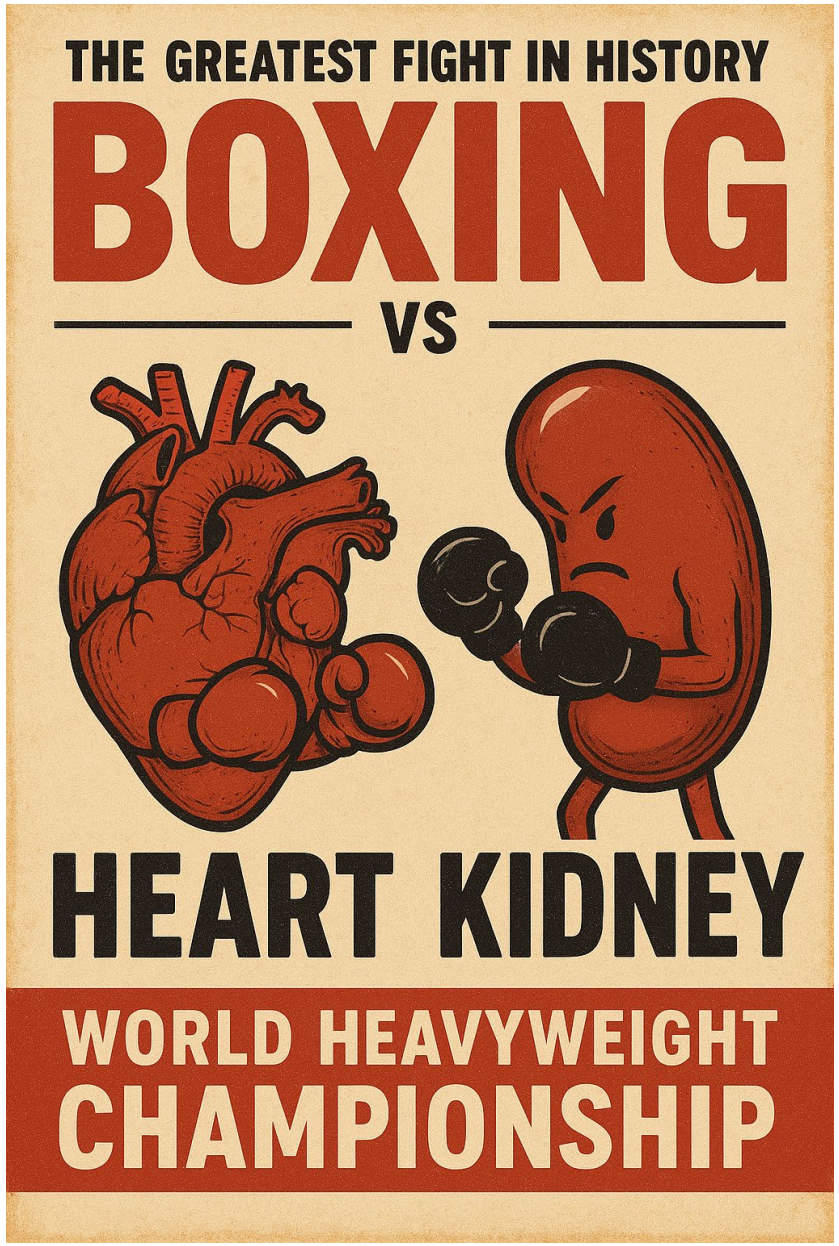
Cleveland Clinic journal of medicine 89.10 (2022): 561

Guidance on Diuretic Therapy



BP = blood pressure; ER = emergency room; IV = intravenous

2019 ACC Heart Failure Hospitalization Pathway Toolkit

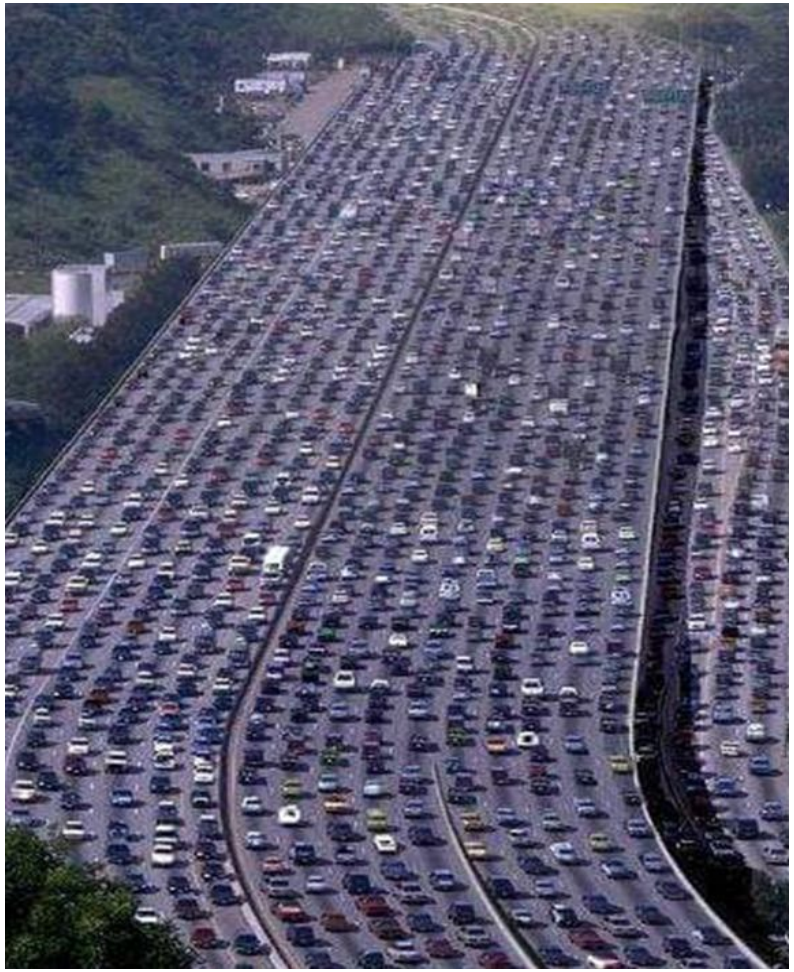


Again

What about…

AKI

Remember…



=

When giving diuretics…

Some decreased GFR is ok *if* there continues to be a good response

Table 2 **Differences in event rates between patients with good[a] or poor diuretic response in patients with worsening renal function**

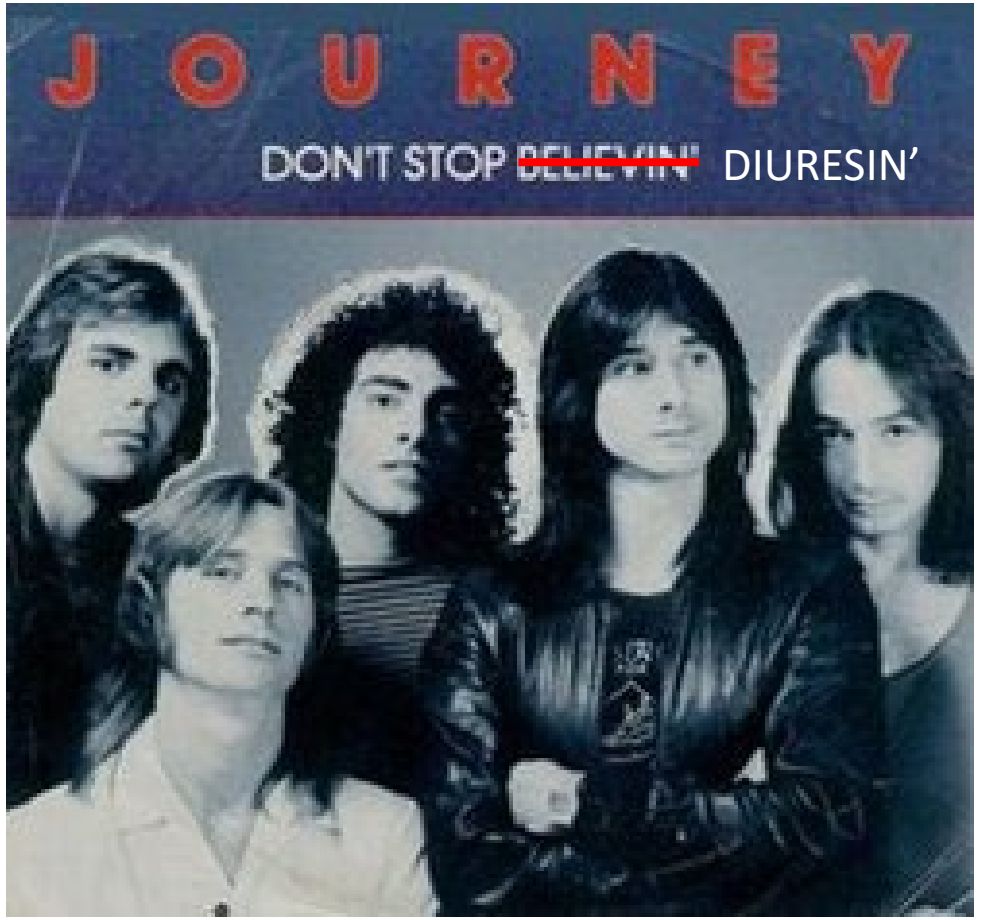
Outcome, *n* (%)	PROTECT		
	Poor diuretic response (*n* = 153)	Good diuretic response (*n* = 113)	*p*-value
180-day death	41 (27)	6 (5)	**<0.001**
60-day death or cardiovascular or renal hospitalization	65 (42)	23 (20)	**<0.001**
Death	22 (14)	1 (<0.1)	**<0.001**
Cardiovascular or renal hospitalization	44 (29)	22 (19)	0.112
Outcome, *n* (%)	**RELAX-AHF-2**		
	Poor diuretic response (*n* = 470)	Good diuretic response (*n* = 502)	*p*-value
180-day death	76 (16)	50 (10)	**0.005**
180-day cardiovascular death or heart/renal failure hospitalization	157 (33)	94 (19)	**<0.001**
Cardiovascular death	55 (12)	37 (7)	**0.028**
Heart/renal failure hospitalization	112 (24)	57 (11)	**<0.001**

[a]Defined as >Δ −0.35 kg/40 mg furosemide equivalent between baseline and day 4.

Don’t stop the diuretic journey!

Can consider using different cutoff for true renal dysfunction in diuresis for acute CHF exacerbation

- **doubling of creatinine**
- **Increase greater than 1 mg/dL** (instead of > 0.3 mg/dL)



"How do we maximize diuresis in acute decompensated heart failure?." *Cleveland Clinic journal of medicine* 89.10 (2022): 561

Agenda

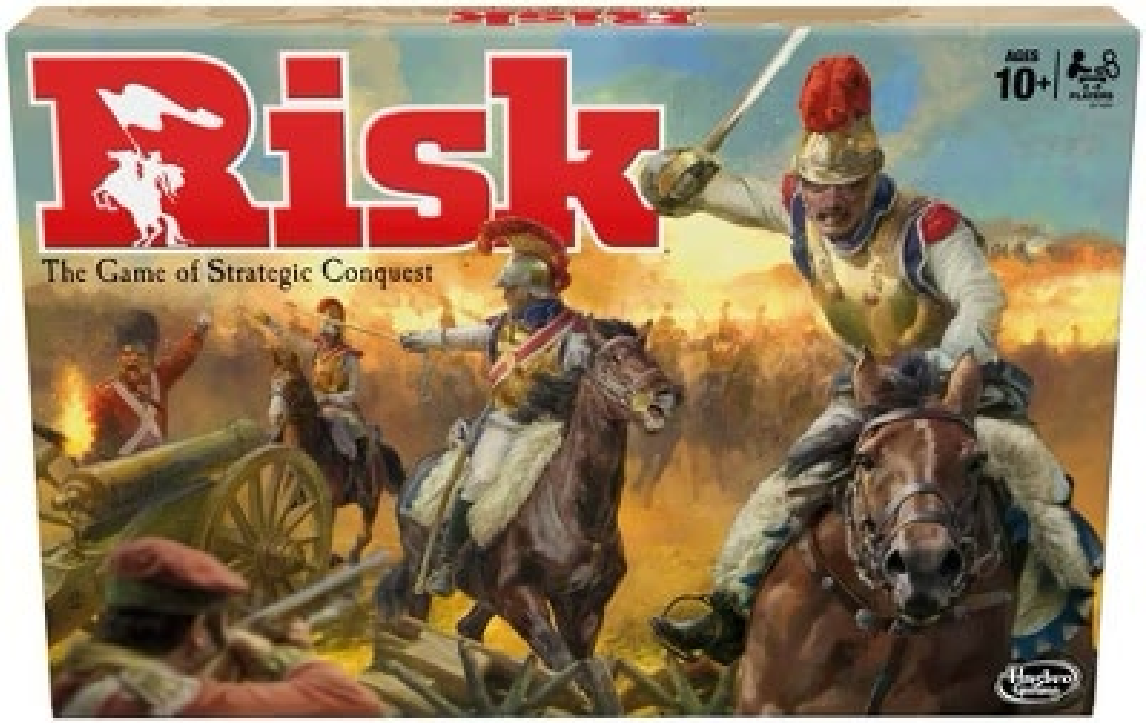
Hemodynamics first

Evidence based therapies

Acute interventions

Risk stratification

Transition planning



Risk Stratification

Immediate assessment

Warning signs for poor outcomes

When to call a cardiologist

Immediate Assessment

	DRY (No congestion)	WET (Congestion present)
WARM (Good perfusion)	**WARM/DRY** Compensated, no urgent treatment needed	**WARM/WET** Decongest with diuretics ± vasodilators
COLD (Poor perfusion)	**COLD/DRY** Optimize preload, avoid over-diuresis, consider inotropes if needed	**COLD/WET** Cautious diuresis + inotropes if hypoperfused

When to worry

Table 2. Precipitating Factors and Multivariate Risk-Adjusted In-Hospital Clinical Outcomes

Factor	No. of Patients	Adjusted Length of Stay Ratio	*P* Value	In-Hospital Mortality	
				Adjusted Odds Ratio (95% Confidence Interval)	*P* Value
Ischemia/acute coronary syndrome	7155	0.99	.22	1.20 (1.03-1.40)	.02
Arrhythmia	6552	1.04	<.001	0.85 (0.71-1.01)	.07
Nonadherence to diet	2504	0.96	.01	0.69 (0.48-1.00)	.05
Uncontrolled hypertension	5220	0.96	<.001	0.74 (0.55-0.99)	.04
Nonadherence to medications	4309	0.96	<.001	0.88 (0.67-1.17)	.39
Pneumonia/respiratory process	7426	1.08	<.001	1.60 (1.38-1.85)	<.001
Worsening renal function	3304	1.09	<.001	1.48 (1.23-1.79)	<.001
Other	6171	0.99	.23	1.15 (0.97-1.36)	.10

Arch Intern Med. 2008;168(8):847-854

When to call a Cardiologist

- New diagnosis
- Recurrent hospitalizations
- Arrhythmias
- Stage D symptoms (symptoms at rest) despite optimal therapy
- LVEF <35% despite 3 months of GDMT (may need ICD)
- Diagnostic uncertainty

2022 AHA/ACC/HFSA guideline for the management of heart failure. *Journal of the American College of Cardiology* 79.17 (2022)

Remember acronym to assist in decision making for referral to advanced heart failure specialist:

I-NEED-HELP (also see *Table 6*)

I: Intravenous inotropes

N: NYHA IIIB/IV or persistently elevated natriuretic peptides

E: End-organ dysfunction

E: Ejection fraction ≤35%

D: Defibrillator shocks

H: Hospitalizations >1

E: Edema despite escalating diuretic agents

L: Low blood pressure, high heart rate

P: Prognostic medication – progressive intolerance or down-titration of GDMT

2024 ACC expert consensus decision pathway for treatment of heart failure with reduced ejection fraction. *Journal of the American College of Cardiology* 83.15 (2024)

Agenda

Hemodynamics first

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Transition planning

Did you know that you can hear the blood in your veins?

Did you know that you can hear the blood in your veins?

You just need to listen varicosely.

Transition Planning

- Resolution of congestion and optimized perfusion
- GDMT started / titrated
- Prior Authorizations completed for medicines
- Home diuretic regimen determined
- Cardiology consulted / recommendations
- Close follow up ideally in 1-2 weeks

Figure 2: Pre-discharge Checklist

Medications	Follow-up	Patient Education
☐ GDMT initiation: ACEi/ARB/ARNI, β-blocker, MRA, SGLT2i	☐ Telehealth/in-person visit within 1 week	☐ Medication education
☐ Assessment of oral diuretic efficacy	☐ Heart failure clinic referral	☐ Nutrition counseling
☐ Iron deficiency repletion	☐ Labs: creatinine, electrolyte panel, glucose, BNP	☐ Physical exercise education
☐ Assess for potential drug–drug interactions	☐ Cardiac rehabilitation referral	☐ Daily weight and blood pressure monitoring
		☐ Substance use/tobacco cessation counseling

Optimizing Guideline-directed Medical Therapies for Heart Failure with Reduced Ejection Fraction During Hospitalization. US Cardiol. 2021 Apr 23;15:

FIGURE 10 Education for Patients, Families, and Caregivers

EDUCATION FOR PATIENTS, FAMILIES, AND CAREGIVERS

- ☐ Current medications
  - Indication
  - Dose/frequency
  - Potential side effects
  - Potential adherence barriers
- ☐ Activity level
- ☐ Dietary sodium restriction _____mg/day
- ☐ Fluid restriction ☐ Yes _____L/day or ☐ No
- ☐ Daily weight monitoring
  - Has scale ☐ Yes ☐ No
  - Records daily weights ☐ Yes ☐ No
- ☐ Assessment for peripheral edema
- ☐ Smoking cessation counseling for current or recent smokers
- ☐ Substance use counseling, if applicable
- ☐ List of warning signs of decompensation
- ☐ What to bring to each outpatient appointment
  - List of meds
  - Recordings of daily weights
- ☐ Who to call for increased weight / worsening symptoms / ICD discharge

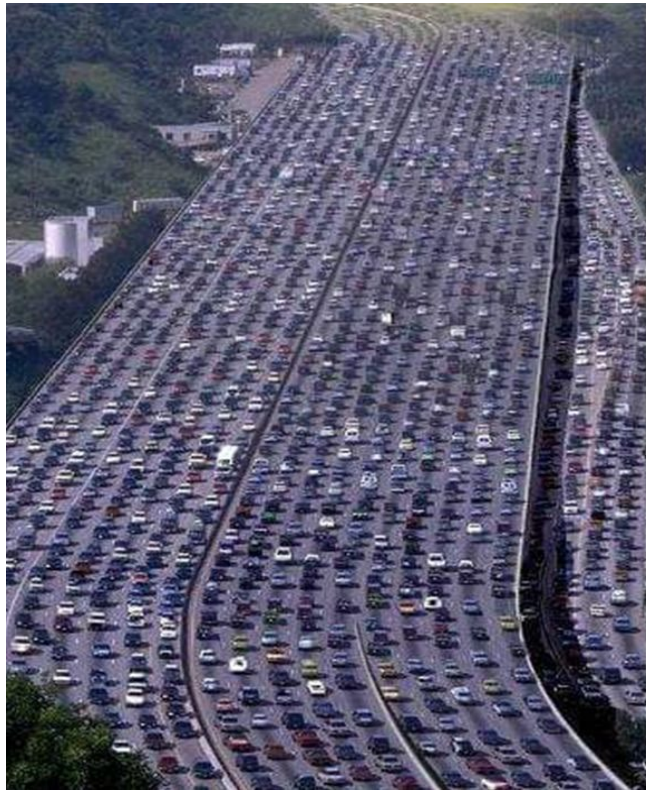
  _____
- ☐ Diuretic management plan
- ☐ Plans for continuation of care
  - Cardiology specialty clinic follow-up appointment _____/_____/_____

In Conclusion

Hemodynamics first
Evidence based therapies
Acute interventions
Risk stratification
Transition planning

Objective 1

Recognize key hemodynamic concepts for CHF exacerbations

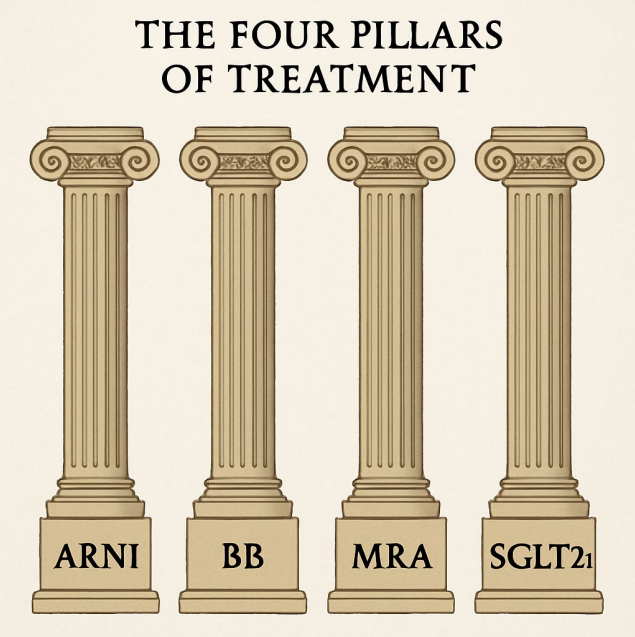


- **Perfusion** assessment is key for recognizing risk of acute decline, but shock is the minority of cases
- **Congestion** is much more common and carries significant mortality and readmission risk
- Goal is resolution of pulmonary congestion prior to discharge if possible

Hemodynamics first
Evidence based therapies
Acute interventions
Risk stratification
Transition planning

Objective 2

Distinguish classes of GDMT therapy and use evidence-based approaches for initiation and titration



- ACE / ARB / ARNI (ARNI is better)
- BB
- MRA
- SGLT2i – best evidence with HFmrEF, HFpEF
- Hydralazine and Isosorbide Dinitrate for African Americans at full GDMT who can tolerate
- Start inpatient for best outcomes!
- OK to up titrate more aggressively

Hemodynamics first
Evidence based therapies
Acute interventions
Risk stratification
Transition planning

Objective 3

Describe treatment goals for diuretics

- Start with IV loops at higher dose
  - In exacerbation takes more to achieve less
- Add Thiazides
- Goal UOP 150cc/hr
- OK to push the kidneys – some recommendations for doubling creatinine or increase by 1mg/dL

Hemodynamics first
Evidence based therapies
Acute interventions
Risk stratification
Transition planning

Objective 4

Identify cases that are appropriate for cardiology consultation

- New diagnosis
- Recurrent hospitalizations
- Arrhythmias
- Stage D symptoms (symptoms at rest) despite optimal therapy
- Intolerance of GDMT
- Poor response to diuretics
- Diagnostic uncertainty

Hemodynamics first
Evidence based therapies
Acute interventions
Risk stratification
Transition planning

Objective 5

Optimize transitions of care from the hospital

- Resolution of congestion and optimized perfusion
- GDMT started / titrated
- Prior Authorizations completed for medicines
- Home diuretic regimen determined
- Cardiology consulted / recommendations
- Close follow up ideally in 1-2 weeks

Questions?