

Translating Global Guidelines

BP targets, drug selection & GDMT Optimisation in Heart Failure

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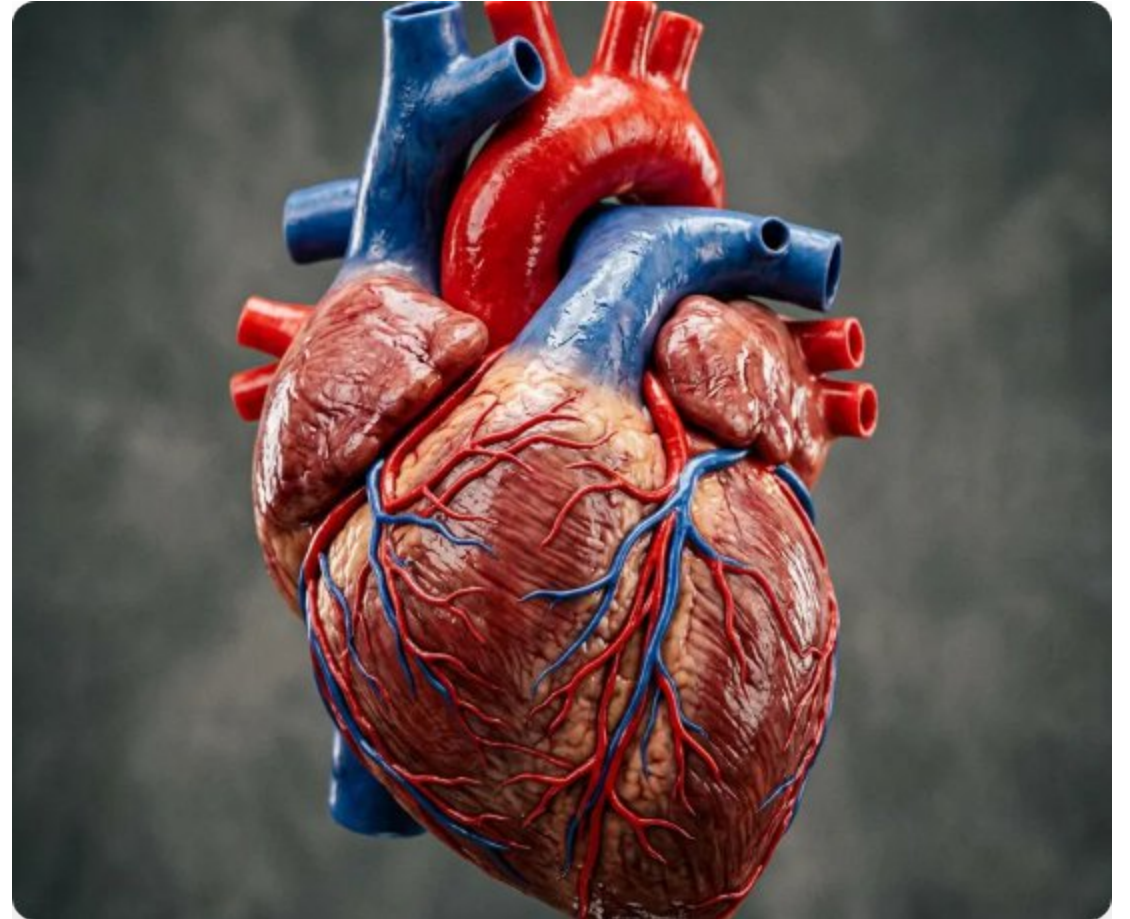
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The Clinical Paradox: Competing Priorities

In patients with Heart Failure with reduced Ejection Fraction (HFrEF), clinicians must constantly reconcile two opposing physiological forces:

- **The Antecedent:** Hypertension is a primary driver of incident heart failure (particularly HFpEF).
- **The Consequence:** Once HFrEF develops, systemic blood pressure naturally falls secondary to left ventricular pump failure.
- **The Paradox:** The most effective disease-modifying therapies (GDMT)—which profoundly reduce mortality—inherently lower systemic blood pressure further.

Our clinical objective shifts from "treating hypertension" to "managing hypotension" to permit neurohormonal blockade.



Translating ESC 2024 & ACC/AHA Targets

HF Phenotype	Target SBP	Clinical Rationale (2024/2025 Guidelines)
HFpEF	120–129 mmHg	Hypertension is the primary driver of diastolic dysfunction. Intensive lowering improves cardiovascular outcomes (SPRINT criteria).
HFmrEF	< 130 mmHg	A transitional phenotype; aggressively treat elevated blood pressure to prevent structural progression to HFrEF.
HFrEF	Permissive	No rigid numerical target. Treat to maximum tolerated GDMT doses. Accept asymptomatic hypotension to achieve mortality benefits.

HFrEF: Navigating the "Fantastic Four"



ARNI / ACEi / ARB

Profound BP Effect

Critical for reverse LV remodeling. ARNIs provide superior mortality benefit but induce more clinically significant hypotension than ACEi/ARBs. Requires careful dose titration.



Beta-Blockers & MRAs

Modest BP Effect

Beta-blockers profoundly affect HR but have a lesser effect on BP. Do not withhold for low BP if HR permits. MRAs have minimal hypotensive effect at standard HF doses.



SGLT2 Inhibitors

Mild BP Effect

Induces mild osmotic diuresis resulting in a ~3-4 mmHg SBP drop. Exceptionally well-tolerated even in patients with borderline hypotension upon initiation.

Sequencing & Up-Titration Strategy

2. Initiate

Start multiple pillars simultaneously at low doses rather than sequentially maximizing single agents. Increases early tolerability.

4. Maintenance

Accept SBP > 85-90 mmHg if end-organ perfusion is maintained (warm extremities, normal mentation, stable creatinine).

1. De-Prescribe

Immediately stop CCBs and alpha-blockers. Reduce loop diuretics if the patient is clinically euvolemic to create "BP room".

3. Rapid Titration

Reassess clinical status and labs every 1-2 weeks. Aim to reach target GDMT doses within 4 weeks if tolerated.

HFpEF: The BP-Centric Phenotype

Unlike HFrEF where low BP is expected and tolerated, **hypertension is the cardinal hemodynamic driver of HFpEF.**

Strict blood pressure control (targeting an SBP of 120-129 mmHg) is not just preventative; it is actively therapeutic in reducing left atrial stretch and pulmonary congestion.

Drug Selection Pivot:

Do not rely on generic antihypertensives. Selection must pivot toward therapies with proven outcome benefits in HFpEF (SGLT2i, MRAs, and ARNIs) to achieve BP targets while providing disease modification.



"True" vs. "Permissive" Hypotension

True Hypotension

SBP < 90 mmHg *accompanied by symptoms of hypoperfusion* (dizziness, syncope, acute kidney injury, cold/clammy extremities). This state mandates immediate GDMT down-titration or temporary cessation.

Permissive Hypotension

SBP 85-95 mmHg where the patient remains *completely asymptomatic* with preserved renal function.

Clinical Imperative:

Do not stop GDMT for isolated, asymptomatic low numerical blood pressure readings. The mortality benefit outweighs the number on the cuff.



Overcoming the Potassium Barrier



Maintaining the Blockade

RAAS inhibitors and MRAs frequently cause hyperkalemia, which remains a primary reason life-saving GDMT is prematurely discontinued or under-dosed in clinical practice.

Current global guidelines strongly advocate against reflexively stopping MRAs for mild hyperkalemia (K^+ 5.1-5.5 mmol/L).

The Solution: Implement dietary restriction and utilize novel potassium binders (e.g., Patiromer, Sodium Zirconium Cyclosilicate) to medically manage the electrolyte shift, thereby enabling the continuation of critical neurohormonal blockade.

The GDMT Optimisation Algorithm

1. Hemodynamic Assessment

Is the patient congested?

Increase loop diuretics to relieve symptoms.

Is the patient hypovolemic?

Decrease or stop the loop diuretic. Resolving over-diuresis frequently rescues blood pressure, creating the hemodynamic space necessary for GDMT up-titration.

2. Pharmacological Adjustments

Stagger Dosing:

Separate administration times (e.g., morning vs. evening) to prevent peak hypotensive overlap.

Class Switching:

Transitioning from ACEi/ARB to ARNI yields superior outcomes, but requires heightened vigilance for increased hypotensive episodes during the transition phase.

Key Takeaways for Clinical Practice

1. In HFrEF, push GDMT to target doses; tolerate asymptomatic hypotension.
2. In HFpEF, strict BP control (120-129 mmHg) is mandatory for disease modification.
3. De-prescribe non-GDMT drugs and adjust diuretics to enable neurohormonal blockade.
4. Treat hyperkalemia medically to avoid stopping life-saving MRA/RAASi therapy.

Thank You

Questions & Discussion

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