Management of Acute Mitral Regurgitation (MR) and Systolic Heart Failure

OACNS 2026 pharmacology conference

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Objectives

- The learner will identify management recommendations for functional mitral regurgitation and systolic heart failure.
- The learner will discuss the medications recommended in guideline directed medical therapy.
- The learner will be able to discuss the recommended timing for initiation and ramping up of GDMT

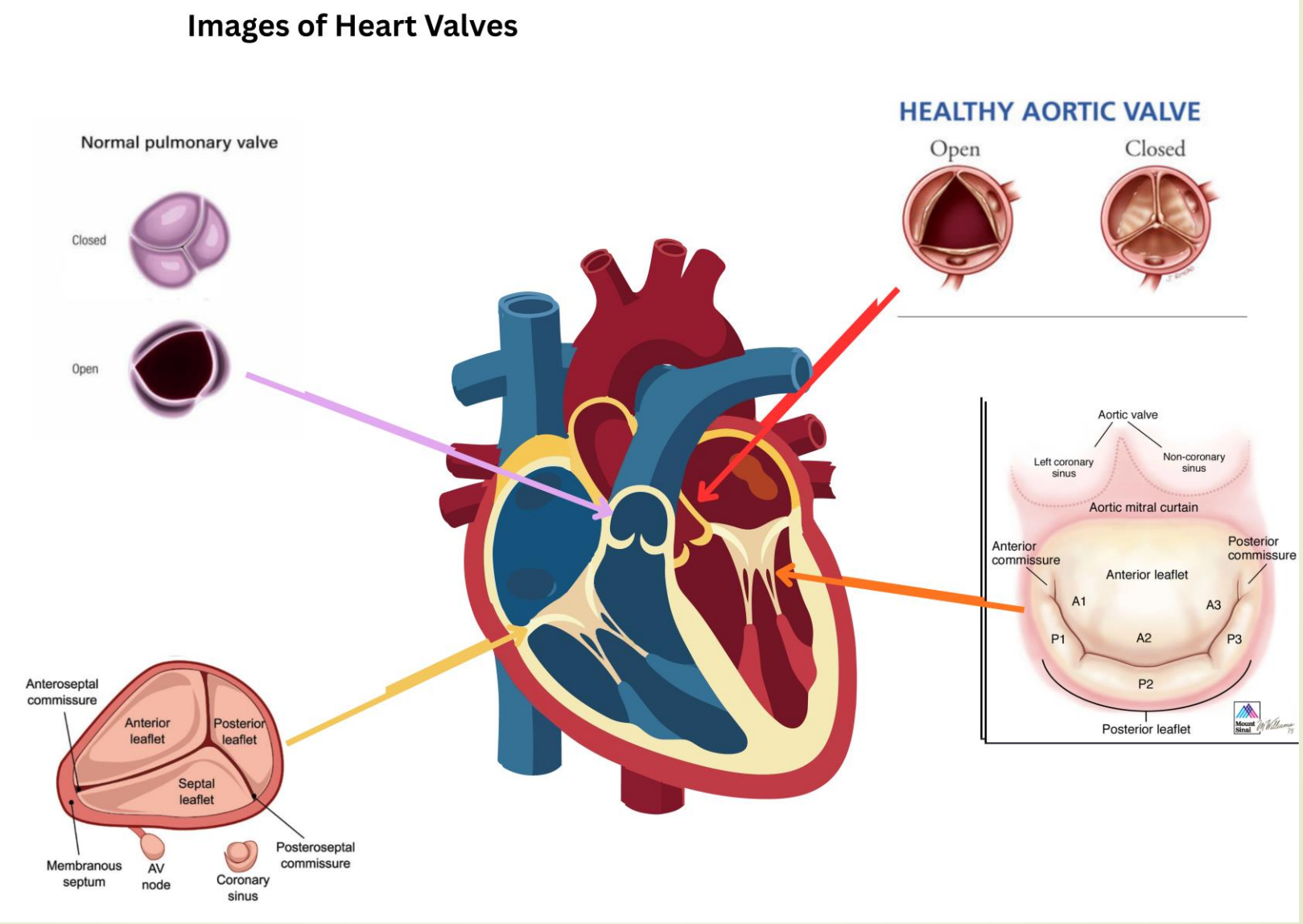
Case Study 1

- 40 y/o male patient
- Presents with recent history of SHOB, fatigue, swelling
- New onset Afib RVR (HR 140)
- Assessment findings
  - New holosystolic murmur at the apex, 3/6
  - Pitting edema 2+ bilat
  - Crackles in the bases bilat
  - SHOB at rest with talking
- CXR
  - Shows congestion bilat
- NTproBNP 23,000
- All other lab normal
- Echo
  - Severe MR, LVIDd 5.6
  - EF 15%
- Home meds: none
- Exercises, uses pre-workout daily
- Drinks energy drinks

Case study 2

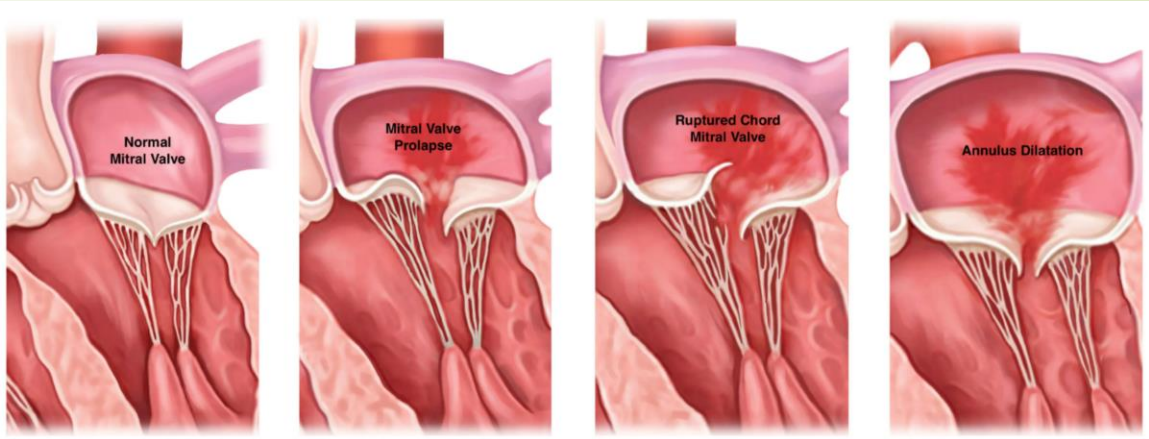
- 45 y/o male patient
- Presents to clinic for consult for his new MR
- Assessment findings:
  - Holosystolic murmur at apex/axilla 3/6
  - Bilat LE edema 1+
- c/o SHOB over the last month with minimal exertion, fatigue, swelling
- Works construction
- Echo shows severe MR, EF 10%, LVIDd 6.2
- NTproBNP 13,000, liver enzymes slightly elevated
- CXR clear
- Meds: metoprolol tartrate 25mg BID, lisinopril 5mg daily, Lasix 20mg daily.
- History:
  - Drinks most days 2-3 beers
  - On the weekends drinks ~12 pack each day

Heart Valves



Mitral Regurgitation

- Degenerative MR
  - Structural dysfunction (primary)
    - Prolapse
    - Flail
    - cleft
- Functional MR (secondary)
  - Annular dilation
  - LV/LA dilation



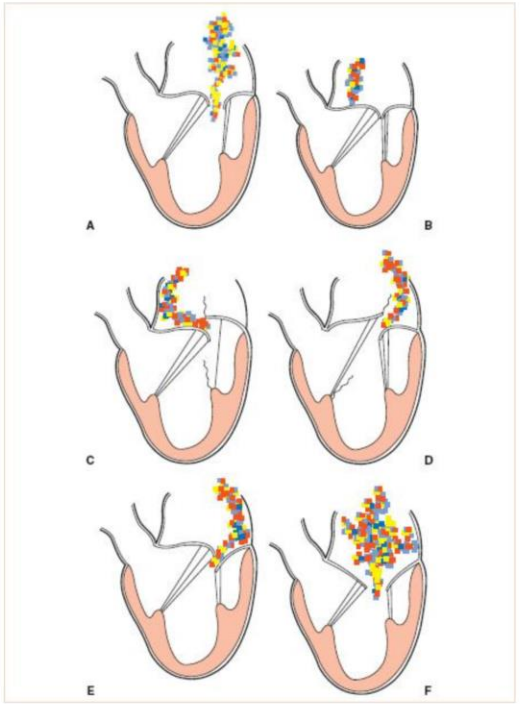
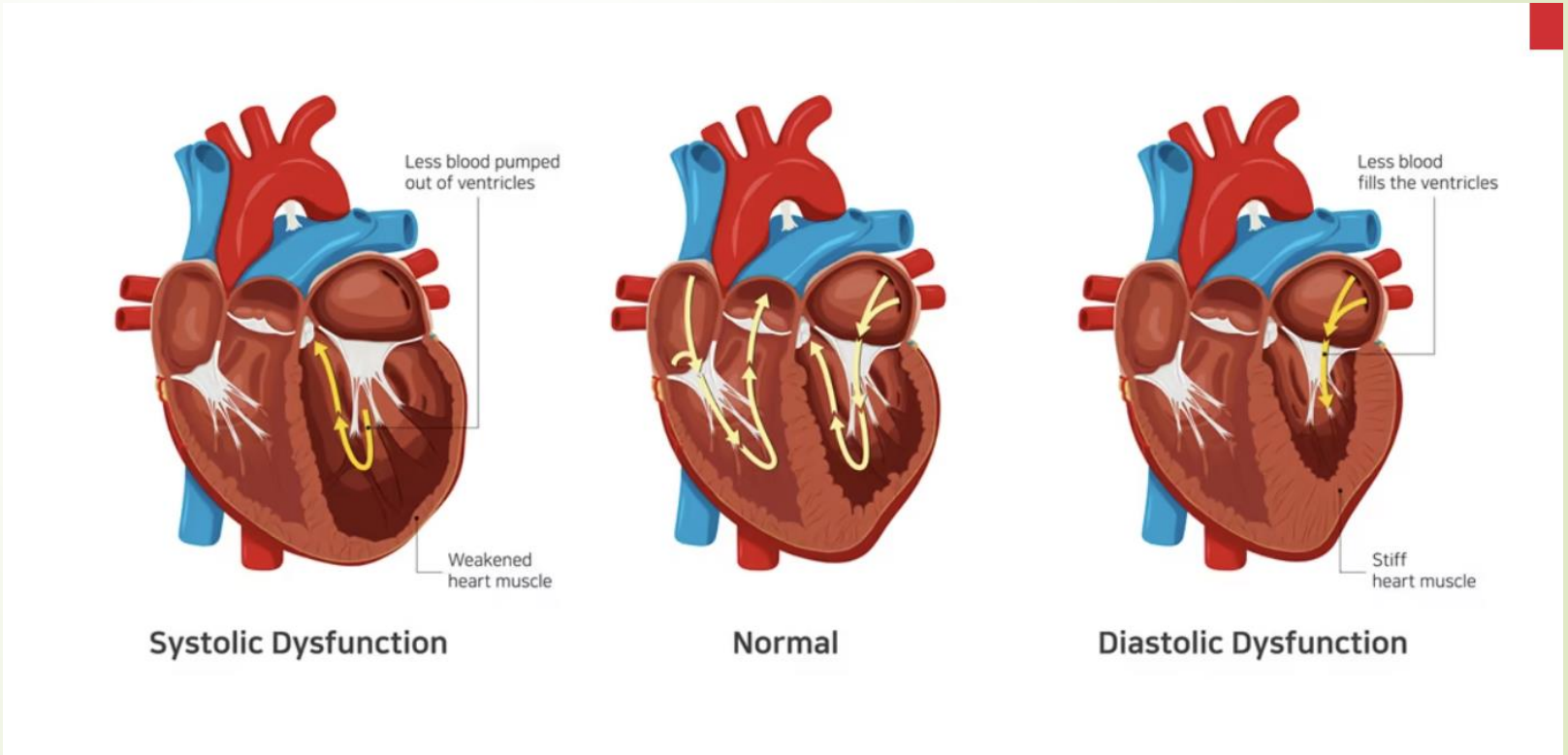


FIGURE 8.3 Carpentier's classification of mitral regurgitation (MR) based on leaflet motion. In type 1, the leaflet motion is *normal* and the MR jet tends to be central **(A,B)**. In type 2, there is *excessive* leaflet motion and the MR jet is typically directed away from the diseased leaflet **(C,D)**. In type 3 lesions, the leaflet motion is *restricted* and is further subdivided into type 3a (structural) **(E)** and type 3b (functional) **(F)**. In type 3 lesions, the regurgitant jet may be directed towards the diseased leaflet if only one leaflet is affected, or it may be central if both mitral leaflets are equally affected. (*Courtesy Dr. Gregory M. Hirsch.*)

Congestive heart failure

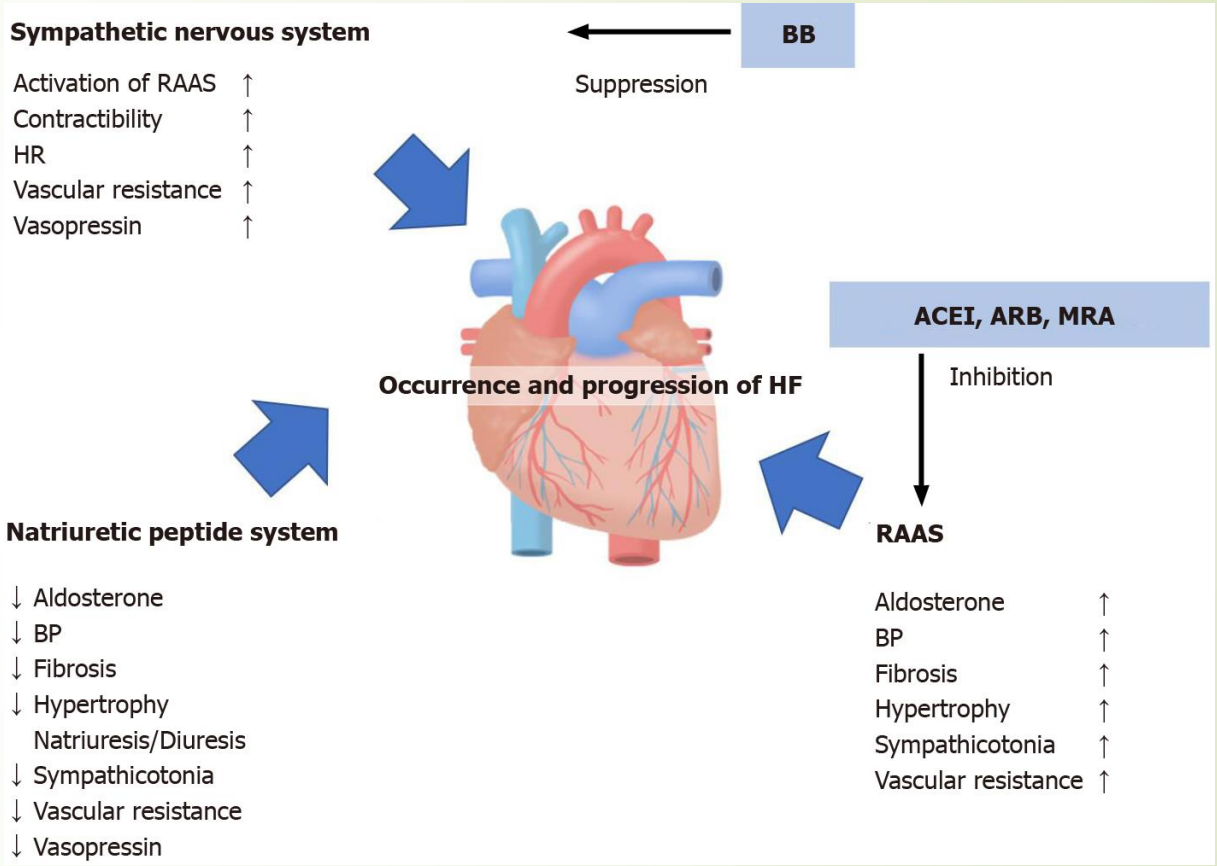
- Systolic vs diastolic
- HFrEF (</= 40%) vs HFmrEF (41-49%) vs HFpEF (>/= 50%)
- HFimpEF (previous HFrEF but now EF >40%)



https://drraghu.com/resources/heart-failure/what-are-the-symptoms-of-diastolic-dysfunction/

Heart Failure

- Mechanism of CHF:
  - Vasoconstriction/fluid retention caused by:
    - RAAS system
    - SNS system
  - Myocardium is both volume/pressure overloaded
  - Natriuretic peptides (NPs) are secreted by the myocardium
    - Promote vasodilation and diuresis



Usuda D, Higashikawa T, Hotchi Y, Usami K, Shimozawa S, Tokunaga S, Osugi I, Katou R, Ito S, Yoshizawa T, Asako S, Mishima K, Kondo A, Mizuno K, Takami H, Komatsu T, Oba J, Nomura T, Sugita M. Angiotensin receptor blocker neprilysin inhibitors. *World J Cardiol* 2021; 13(8): 325-339 [PMID: 34589168 DOI: 10.4330/wjc.v13.i8.325]

Functional MR: Mechanisms

- Left ventricular forces
  - Mitral annular dilation
  - Tethering
  - Low EF
- Cardiac Electromechanical dysynchrony
  - Tethering
- Left atrium forces
  - LA dilation (posterior > Anterior)
  - Mitral annular dilation
  - Increased LA pressure cause LA stretch and fibrosis w/altered atrial/annular dynamics
  - Posterior leaflet bending and anterior leaflet flattening

Circ Heart Fail. 2022;15:e009689. DOI: 10.1161/CIRCHEARTFAILURE.122.009689

Implications of Functional MR

- Most common form of MR
  - 12% patients post MI
  - 24% of patients with chronic systolic congestive heart failure
- Hemodynamically significant in 30% of patients
- Independent driver of prognosis
  - Even with mild functional MR– a/w twofold increase in
    - All-cause/cardiac mortality and hospitalization for heart failure
- LV dilation
  - Critical determinant for secondary MR

European Journal of Heart Failure (2021) 23, 1325–1333 doi:10.1002/ejhf.2097

4+ Pillars of CHF

Pillars

- SGLT2-I
- Beta-blocker
- ARNI/ACE-i/ARB
- MRA
- + Loop Diuretic

Additional considerations

- Hydralazine/Isosorbide dinitrate
- Guanylate cyclase inhibitor (vericiguat)
- Ivabradine
- Digoxin
- PUFA– polyunsaturated fatty acid
- Potassium binders
- CRT and CRT-D

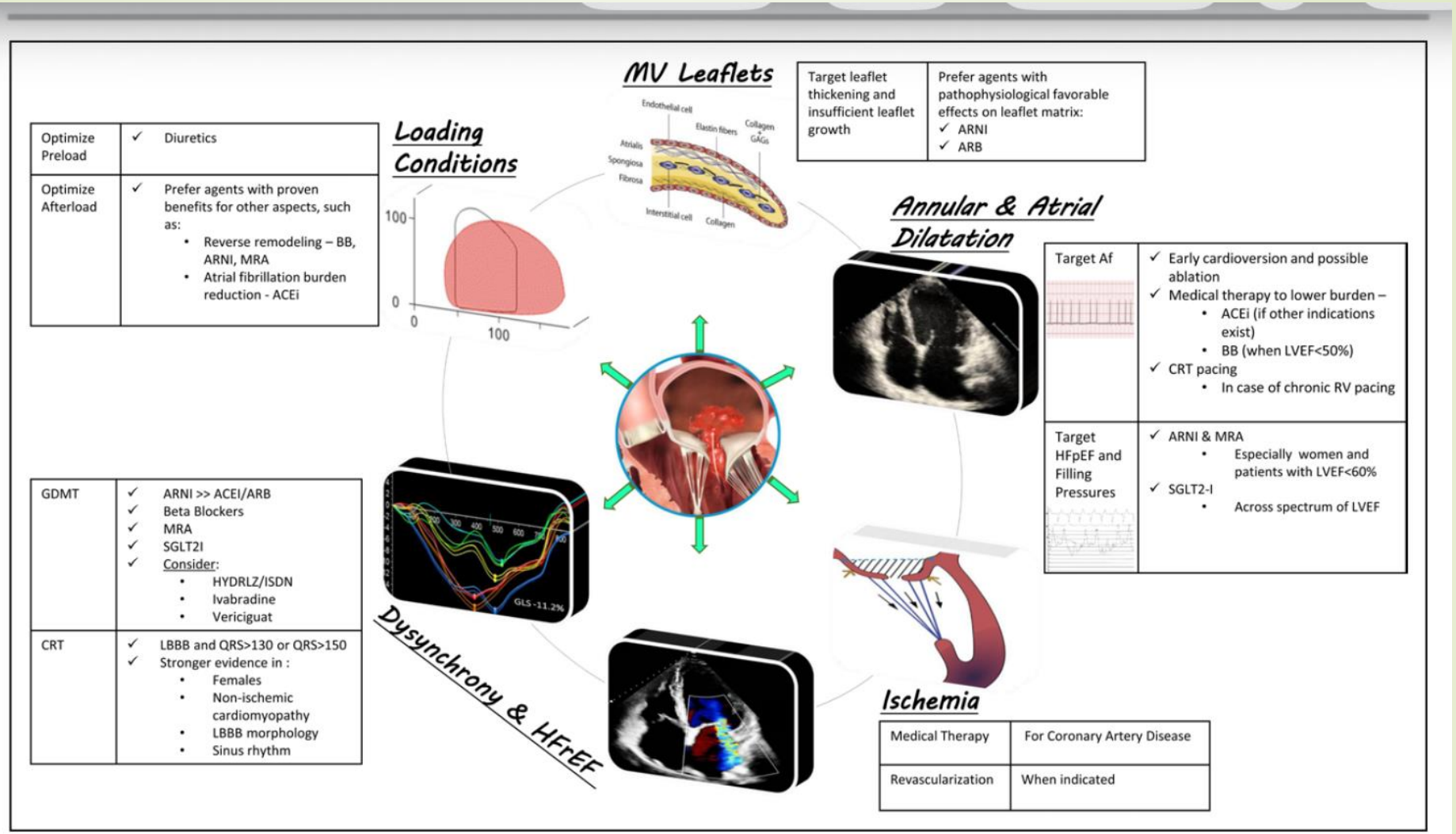
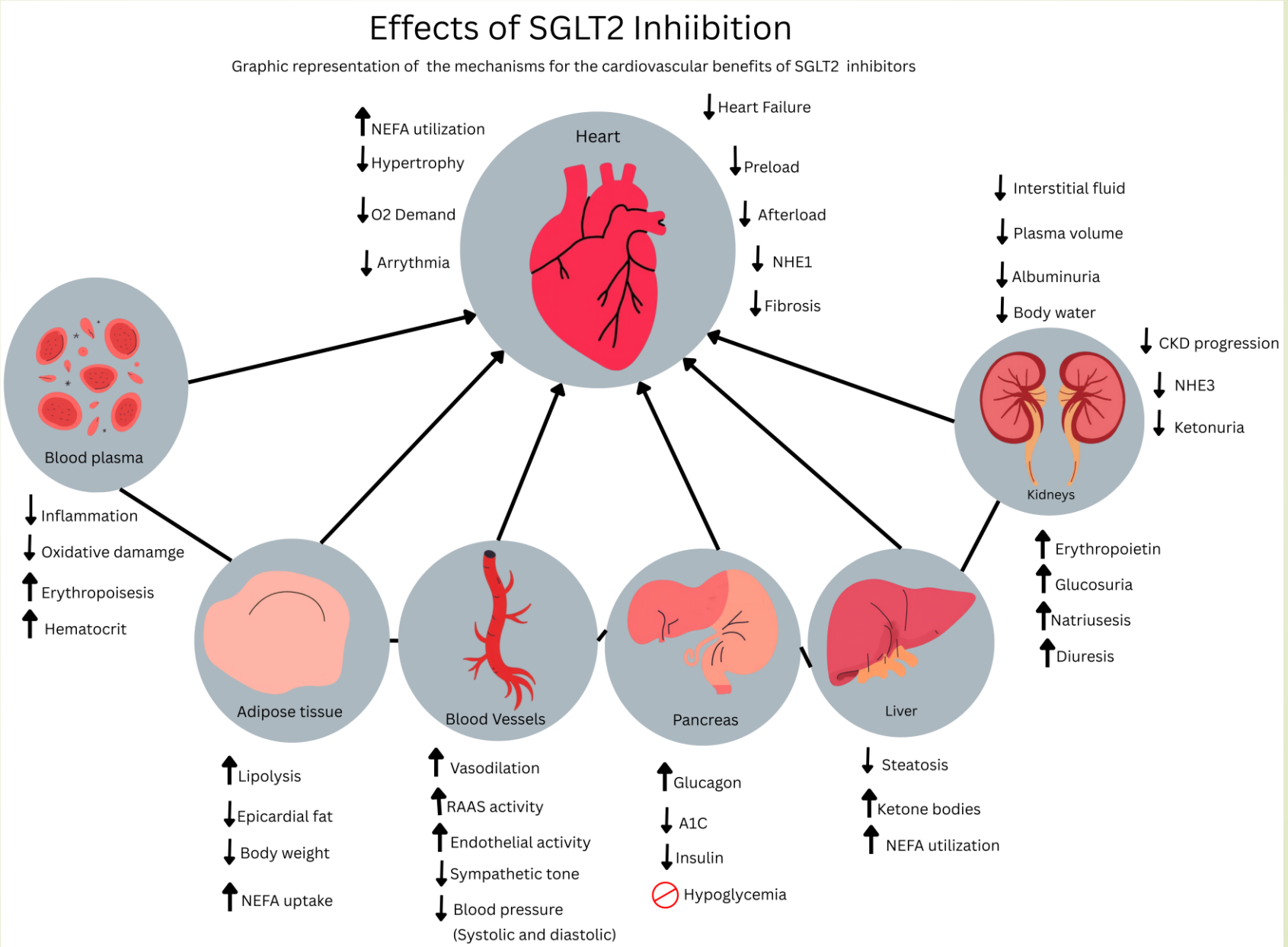


Figure 2. Targeted medical therapy for functional mitral regurgitation (FMR).
Medical therapy including cardiac resynchronization therapy (CRT) targeting different aspects in treatment of FMR is portrayed. ACEI indicates angiotensin-converting enzyme inhibitor; Af, atrial fibrillation/flutter; ARB, angiotensin receptor blocker; ARNI, angiotensin neprilysin inhibitor; BB, β-blocker; GAG, glycosaminoglycan; GDMT, guideline-directed medical therapy; GLS, global longitudinal strain; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; HYDRLZ/ISDN, hydralazine/isosorbide dinitrate; LBBB, left bundle branch block; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; MV, mitral valve; and SGLT2i, sodium glucose transport 2 inhibitor.

Circ Heart Fail. 2022;15:e009689. DOI: 10.1161/CIRCHEARTFAILURE.122.009689

SGLT2i


Effects of SGLT2 Inhiibition
Graphic representation of the mechanisms for the cardiovascular benefits of SGLT2 inhibitors
Heart
NEFA utilization
Hypertrophy
O2 Demand
Arrythmia
Heart Failure
Preload
Afterload
NHE1
Fibrosis
Blood plasma
Inflammation
Oxidative damamge
Erythropoisesis
Hematocrit
Kidneys
Interstitial fluid
Plasma volume
Albuminuria
Body water
CKD progression
NHE3
Ketonuria
Erythropoietin
Glucosuria
Natriusesis
Diuresis
Adipose tissue
Lipolysis
Epicardial fat
Body weight
NEFA uptake
Blood Vessels
Vasodilation
RAAS activity
Endothelial activity
Sympathetic tone
Blood pressure
(Systolic and diastolic)
Pancreas
Glucagon
A1C
Insulin
Hypoglycemia
Liver
Steatosis
Ketone bodies
NEFA utilization

SGLT2i

- Farxiga (dapagliflozin)
- Jardiance (empagliflozin)
- Inpefa (sotagliflozin)
  - Approved in 5/23- not in the guidelines

- Considerations
  - Don't affect the blood pressure much
  - Start regardless of EF
  - Inpatient:
    - SBP >100
    - No inotropic support x24hrs
    - No increase in IV diuretic therapy
    - No IV vasodilator
    - DO NOT start with evidence of hypovolemia
  - Kidney disease
    - eGFR >20
      - empagliflozin/sotagliflozin eGFR>25
    - OK to continue on dialysis
  - Type 1 diabetes---NOT Indicated

Adverse Events

- MOST common AE
  - Genital mycotic infections
    - Manage with good hygiene
    - Rinse after voiding

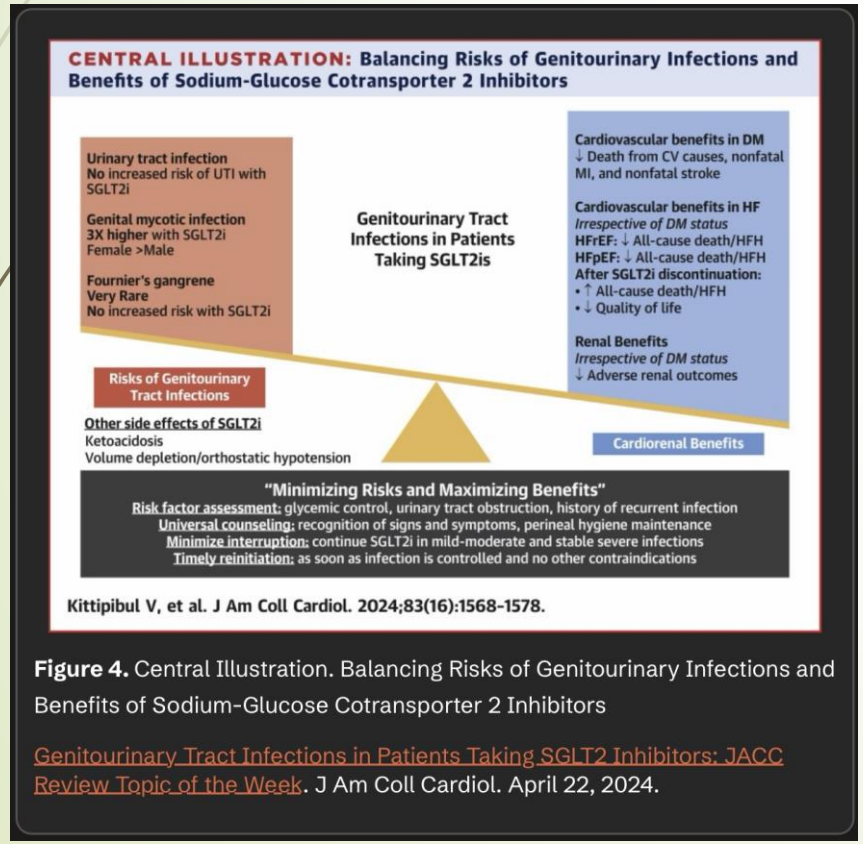
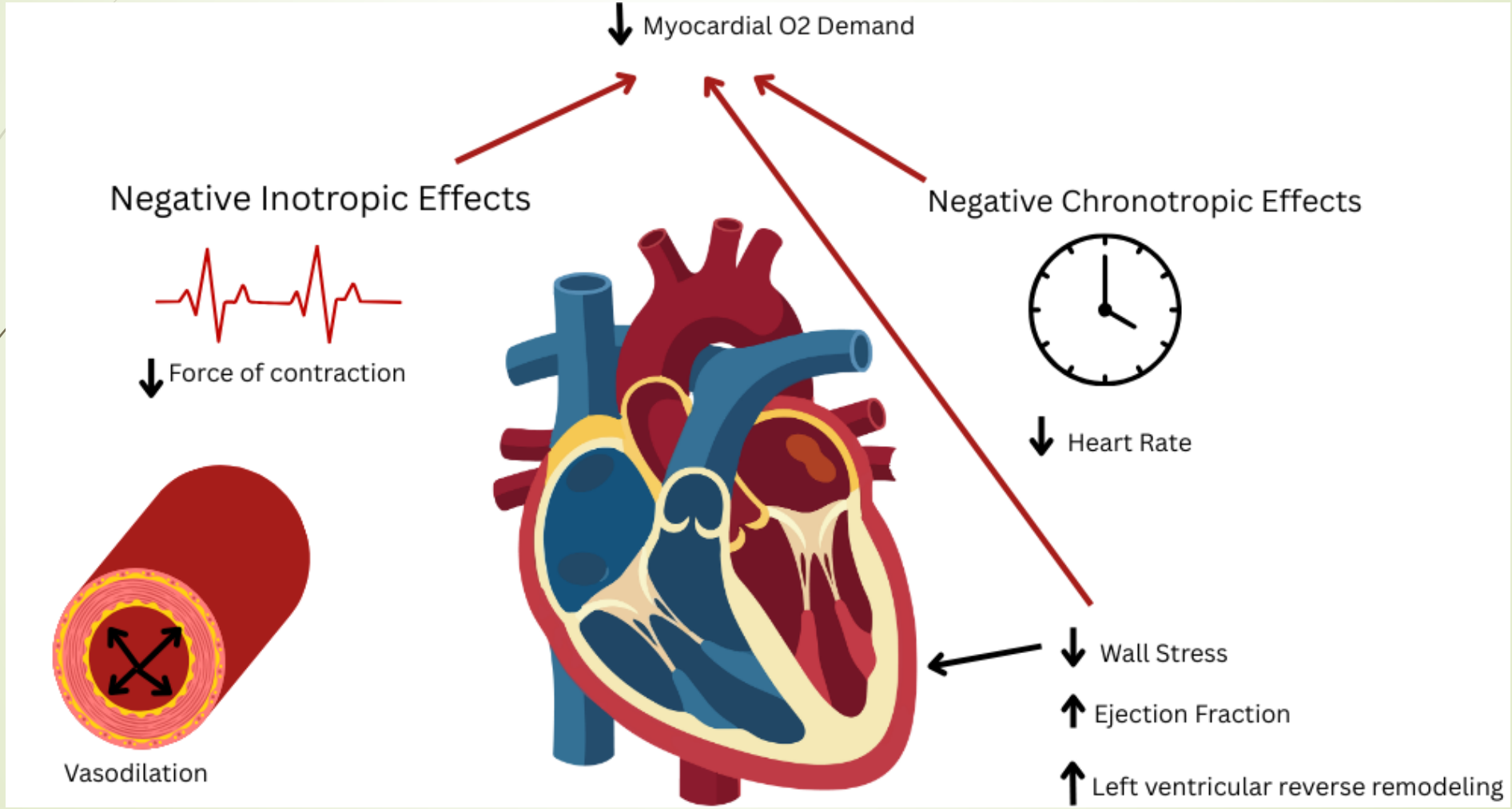


Figure 4. Central Illustration. Balancing Risks of Genitourinary Infections and Benefits of Sodium-Glucose Cotransporter 2 Inhibitors

Genitourinary Tract Infections in Patients Taking SGLT2 Inhibitors: JACC Review Topic of the Week. J Am Coll Cardiol. April 22, 2024.

- Other AE
  - Diabetic ketoacidosis
    - Euglycemic diabetic ketoacidosis
  - Urinary tract infection
  - Volume depletion
    - SGLT2i augment urine output and diuretic response
    - Diuretic requirements should be evaluated.

Beta Blocker Effects in Heart Failure



Beta Blocker

Cheat sheet

	B1	B2	A1	Na+	K+	NO ↑
Cardio selective						
Metoprolol	X					
Atenolol	X					
Bisoprolol	X					
Non-selective						
Propranolol	X	X		X		
Nadolol	X	X				
Sotalol	X	X			X	
Non-selective + alpha blocker						
Carvedilol	X	X	X	mild		
Labetalol	X	X	X	mild		
Special/3rd Gen						
Nebivolol	X					X ↑

Blocked	Mechanism when blocked
B1	• ↓ Chronotropy (slower HR) • ↓ Inotropy (weaker squeeze) • ↓ Dromotropy (Slower AV node conduction)
B2	• Bronchoconstriction (loss of airway smooth – muscle relaxation) • Peripheral vasoconstriction in skeletal muscle beds • Blunted glycogenolysis/gluconeogenesis--- Masks hypoglycemia signs
A1	• Arterial & Venous vasodilation--- afterload/BP • Reflex tachycardia possible if B1 blocked incompletely • Orthostatic hypotension
NA+	Slows phase-0 depolarization in fast fibers--- wider QRS at high doses
K+	• Prolongs phase 3 repolarization–longer action potential & QT interval • Prevents re-entry, increased risk of Torsades if QT is too long
NO ↑ (Not Blocked)	• Endothelial NO release--- vasodilation via cGMP • ↓ Peripheral vascular resistance w/o big HR drop • Better endothelial function & exercise tolerance

Beta-Blocker in Heart Failure/Functional Mitral Regurgitation

- Coreg
  - Selective antagonize alpha-1 adrenergic receptors
  - Antagonizes beta-1 and beta-2 adrenergic receptors
  - Selective alpha and non-selective betablocker
- Coreg CR

- Metoprolol succinate
  - Selectively antagonizes beta-1 adrenergic receptors
- Bisoprolol
  - Selectively antagonize beta-1 adrenergic receptor

- Selectively inhibit B1 receptors in the heart: lower affinity to the B1 receptors in lungs/blood vessels. Fewer side effects r/t bronchoconstriction as compared to non-selective

? Choices

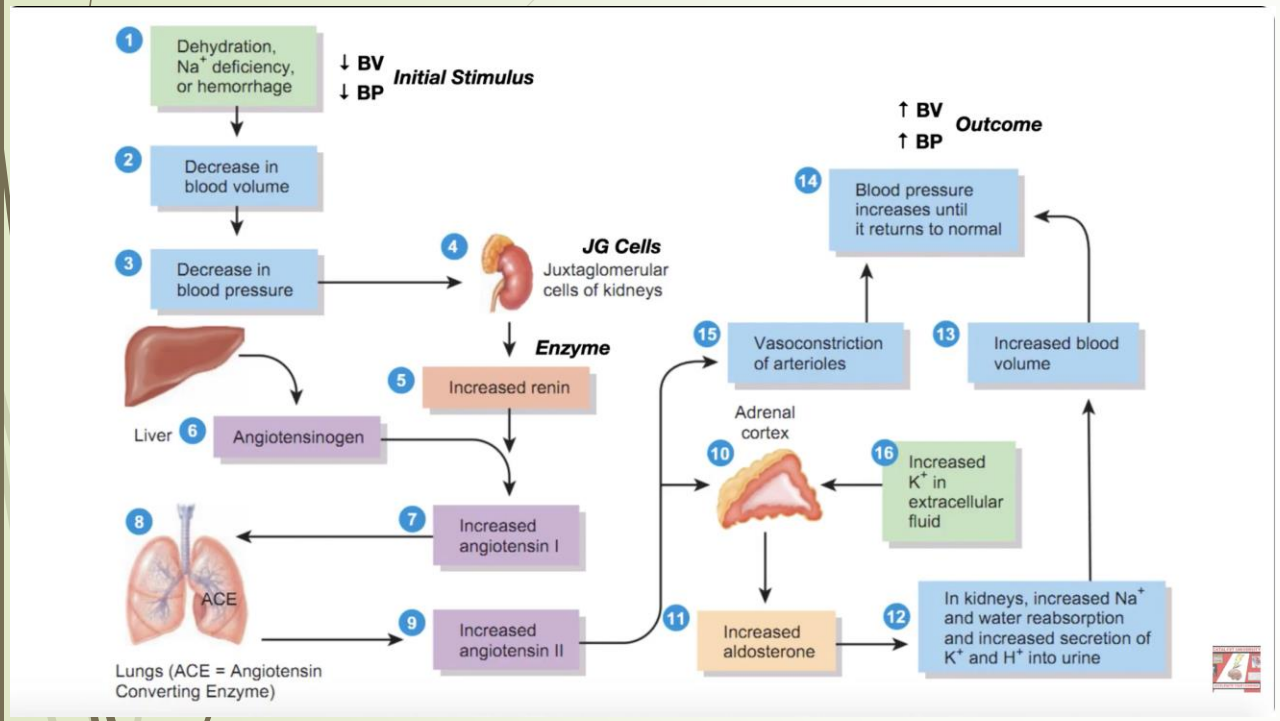
Which to choose:

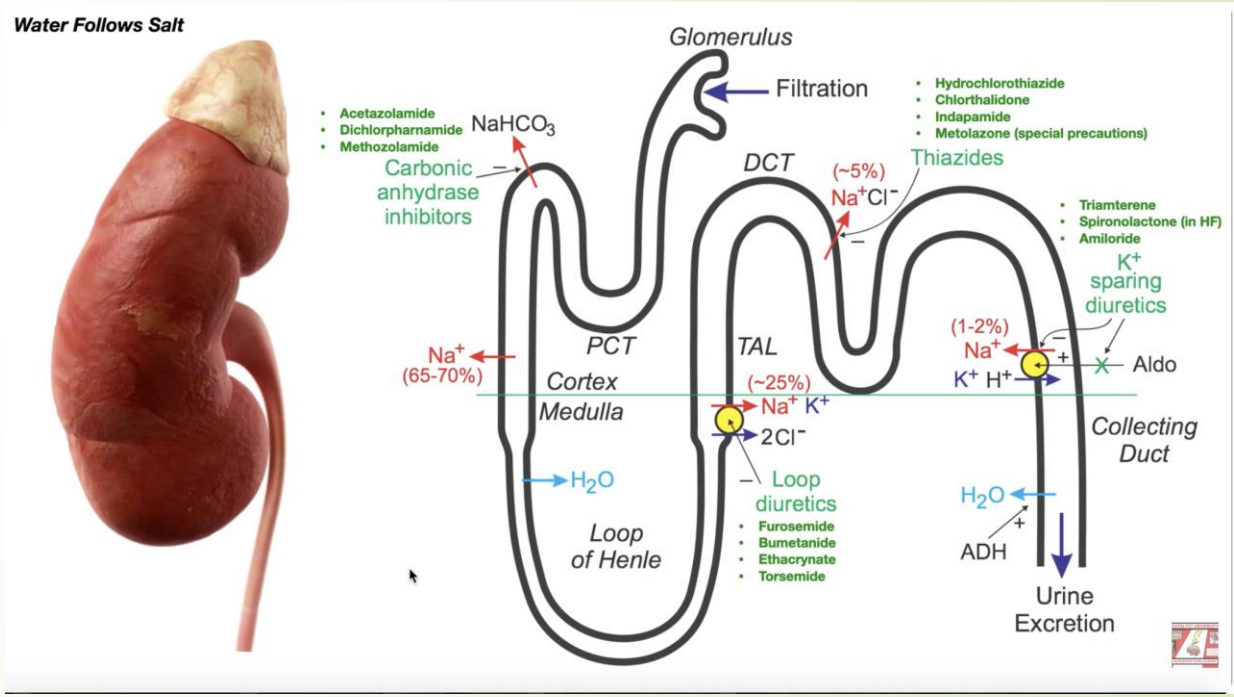
- Reactive airway:
  - metoprolol/bisoprolol
- Diabetes: carvedilol
  - d/t alpha blocking effects
  - Decrease insulin levels, decrease HbA1c, reduce incidence of new onset diabetes (Circ)
- Afib—any
- Can be easier to reach target dose with carvedilol vs Metoprolol/Bisoprolol

- Side effects:
  - Dizziness
  - Fatigue
  - Bradycardia
  - Hypotension

- Bisoprolol 10mg ~ Carvedilol 50mg ~ Metoprolol succ 200mg
- 1mg Coreg ~ 4mg meto succ
- No equivalency for bisoprolol

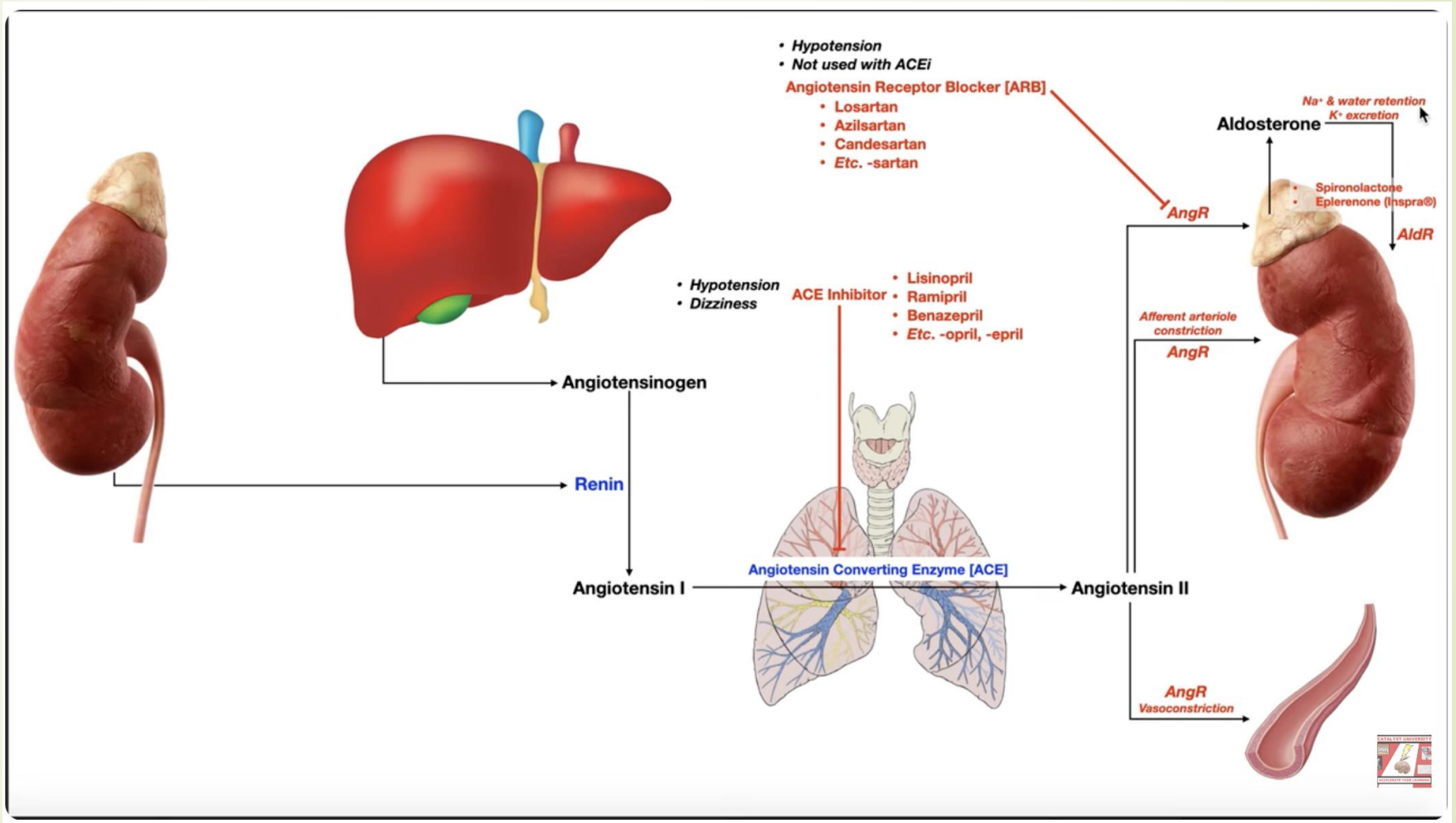
RAAS System





The Renin-Angiotensin-Aldosterone [RAAS] Pathway EXPLAINED, Catalyst university (YouTube). https://www.youtube.com/watch?v=_S59qSuosgE

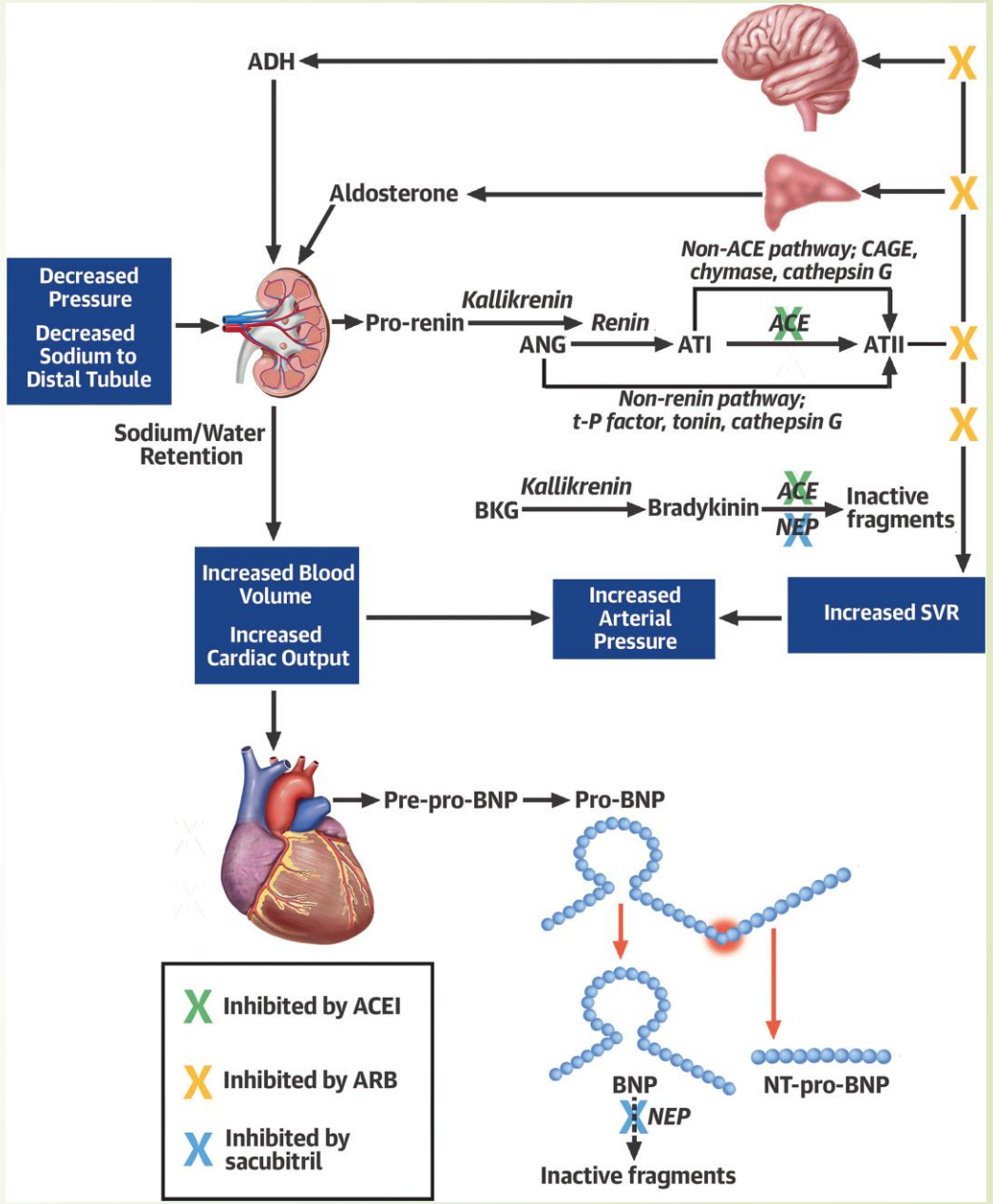
ACE/ARB/Aldosterone inhibitors (RAAS meds)



Pharmacology of the Renin-Angiotensin-Aldosterone [RAAS] Pathway, Catalyst University; YouTube https://www.youtube.com/watch?v=aKPd2MAaGfM

ACE/ARB/ARNI

- Activation of RAAS (renin-angiotensin-aldosterone system)
  - Angiotensin II (ATII) converted from angiotensin I (ATI) by angiotensin-converting enzyme (ACE)
  - Binds to angiotensin II type-1 receptor in vasculature, brain, heart, kidney, adrenal gland, and nervous system
  - ACE
    - Breakdown of bradykinin to inactive fragments
  - Neprilysin (NEP)—neutral endopeptidase
    - Catalyzes activated peptide degradation
      - A-type natriuretic peptide (ANP), B-type natriuretic peptide (BNP), and bradykinin
      - Contributes to the breakdown of angiotensin II
  - ACE-I: increase bradykinin/decrease angiotensin II
  - ARB: block angiotensin II at the level of tissue receptors
  - Sacubitril: inhibits neprilysin, which increases BNP and bradykinin



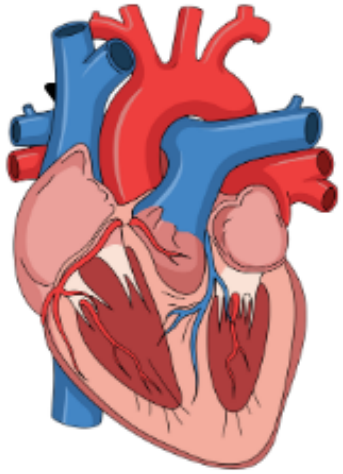
Page, R. The Real-World Price of Switching to an ARNI: A Case for De Novo Sacubitril/Valsartan Initiation∗ . JACC. 2023 Jan, 81 (4) 332–335. https://doi.org/10.1016/j.jacc.2022.11.024

ARNI

Entresto

↓ Functional MR

LV reverse remodeling
improved leaflet coaptation
reduce effective orifice area (EROA)



↓ All cause mortality

↓ Preload

↓ Afterload

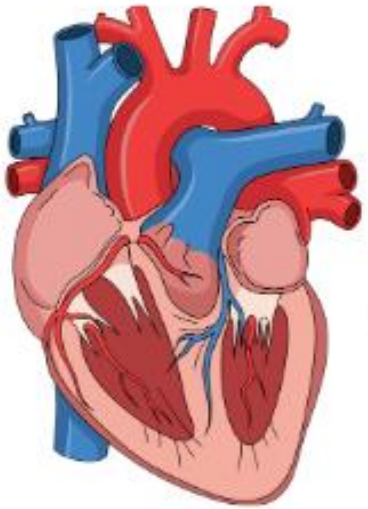
> through natriuretic effects

↓

LV reverse remodeling
improved leaflet coaptation
inhibit tissue growth factor beta to counteract leaflet thickening

ACE-I/ARB

ACE-I/ARB



↓ Preload

< ↓ Regurgitant volume

↑ Effective stroke volume

Lower BP

Protect kidney function (blocking production of Angiotensin II)

Decrease aldosterone secretion

Reduce fluid volume

Ease workload of the heart

↓ Peripheral vascular resistance

↑ Bradykinin. (ACE-I)

Adverse Effects

- Dry cough (ACE-I)
- Hyperkalemia
- Hypotension
- Angioedema

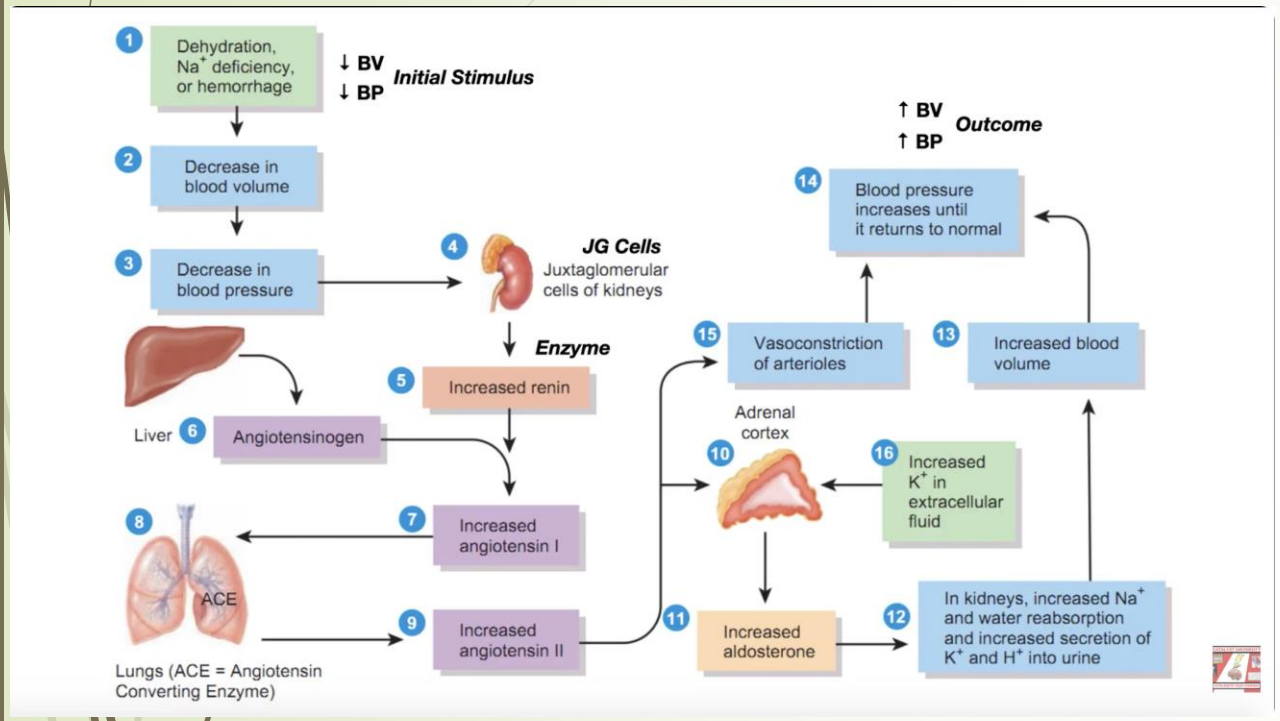
- Contraindication
  - pregnancy

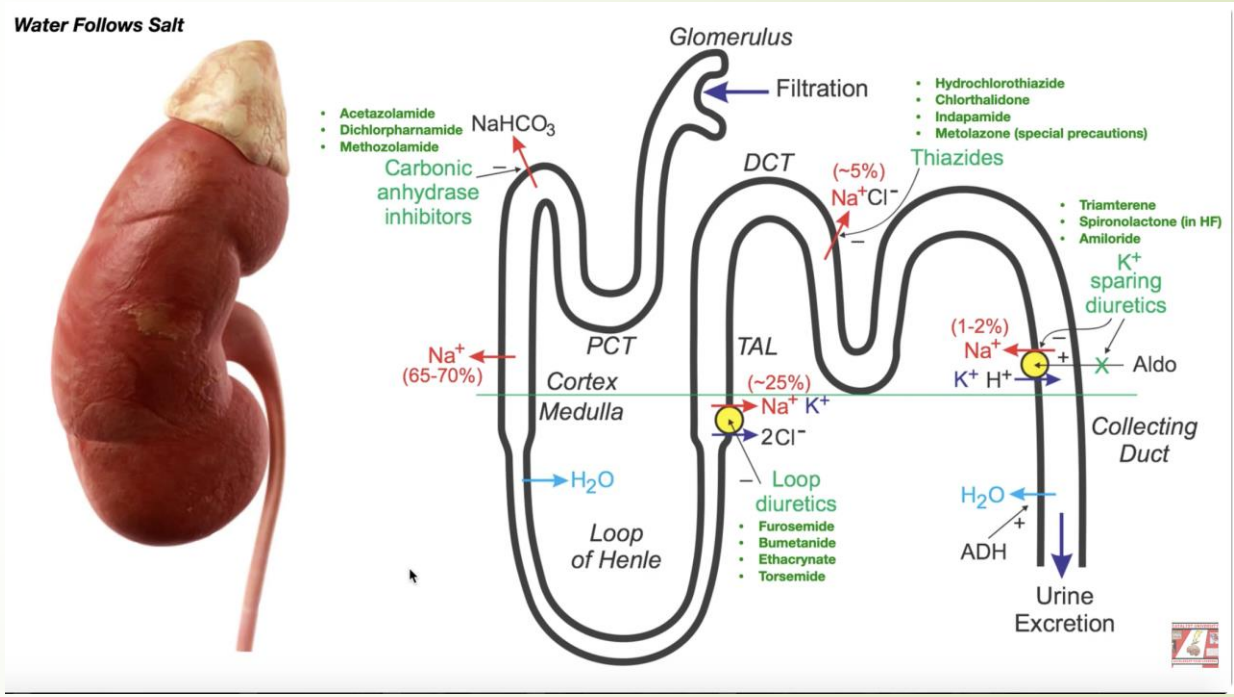
Which to use ?

- Guidelines emphasize no differences between ACE-I vs ARB
  - On effects on symptoms or survival
  - Some trials show better BP control with ARB
  - ARBs have better tolerability
  - No need for washout between ACE-I and ARB

- ARNI: superior for reducing CV death and heart failure hospitalization in HFrEF
  - 36hr washout when switching from ACE-I, not needed with ARB
  - Don't give with history of angioedema
- If on HIGH dose ACE-I/ARB (enalapril >10mg daily, Valsartan >160mg daily) start at 49/51mg BID
- If on low to medium dose: start 24/26mg BID
- If de novo: start low 24/26mg BID

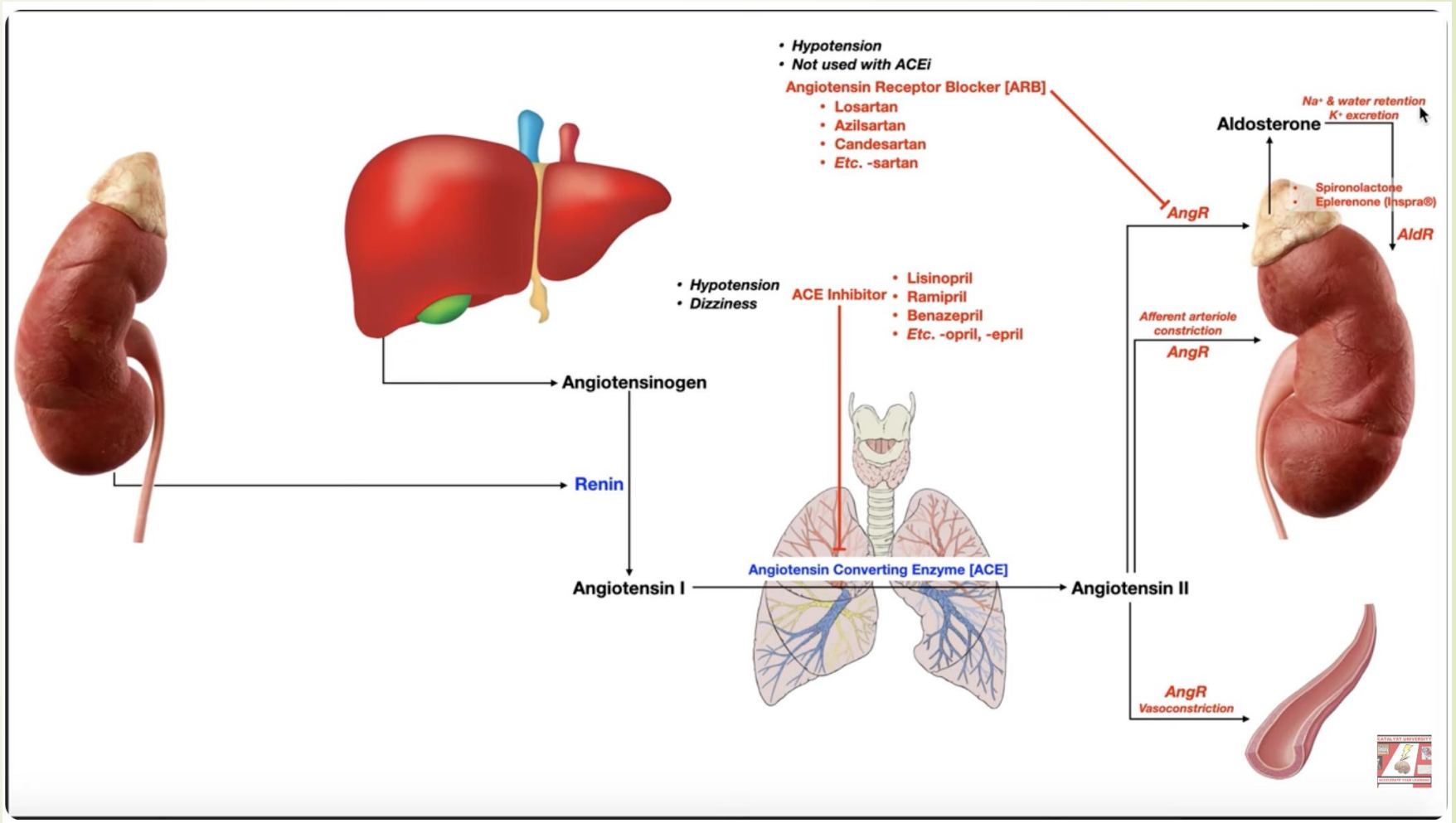
RAAS System (MRA)





The Renin-Angiotensin-Aldosterone [RAAS] Pathway EXPLAINED, Catalyst university (YouTube). https://www.youtube.com/watch?v=_S59qSuosgE

ACE/ARB/**Aldosterone inhibitors** (RAAS meds)



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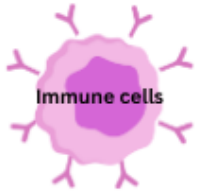
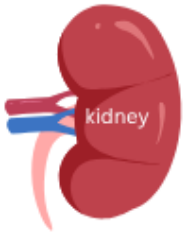
MRA



↓ Inflammation

Macrophage polarization
Promote shift from M1 phenotype (proinflammatory) to M2 macrophage (reparative)



Cardiorenal protection
In CKD/T2DM

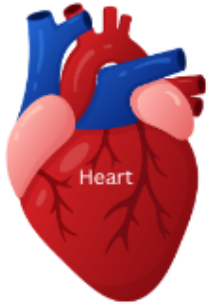
↓ Cardiorenal Fibrosis

↓ proteinuria (albuminuria)

↓ Inflammation

MRA

Work synergystically with SGLT2i for better cardiorenal protection



↓ Cardiac remodeling

↓ Cardiovascular death

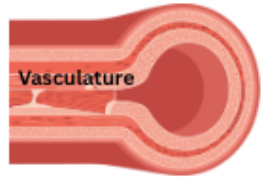
↓ Cardiac hospitalizations

Endothelial function

↑ BP control

↑ blood circulation

↓ strain on vasculature



↓ Fibroblast Activation

↓ Cardiac fibrosis

Inhibition of myofibroblast activation
Myfibroblast---excessive scar tissue

↓ Collagen Deposition

↓ Profibrotic markers

Improved myocardial markers d/t fibrosis markers

↓ LVH

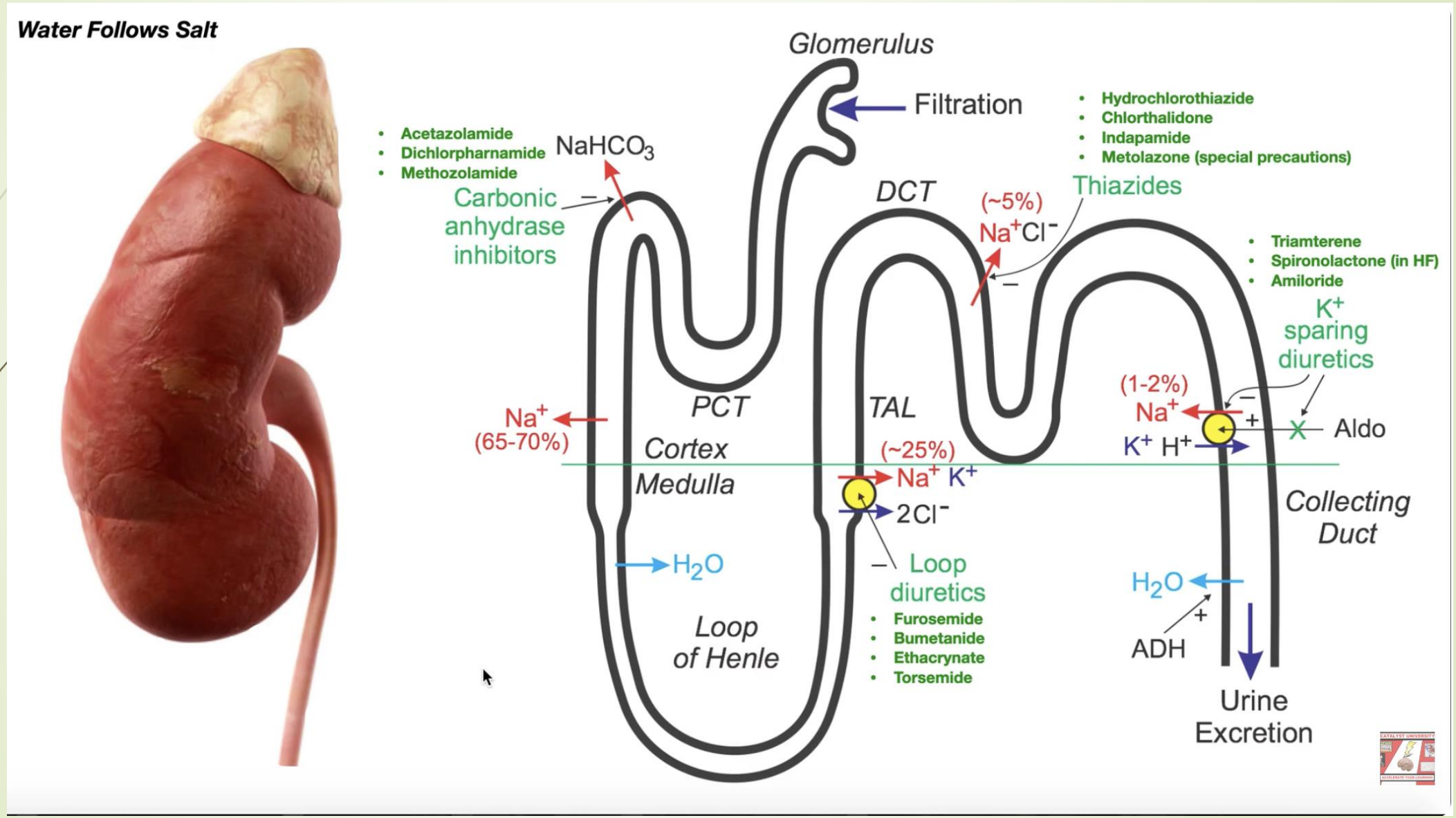
slow CKD

MRA (Mineralocorticoid Receptor Antagonist)

- Spironolactone
  - Dose: 25-50mg/day
    - Renal dosing cut by half
  - Adverse effects
    - Hyperkalemia
    - Gynecomastia
- Eplerenone
  - Dose 25-50mg/day
    - Renal dosing cut by half
  - Adverse effects
    - Hyperkalemia
    - Decreased risk for vaginal bleeding and/or gynecomastia
- Block stress hormones
- Contraindicated
  - eGFR <30
  - K >5.0

- Finerenone (not in guidelines- yet)
  - Selective, non-steroidal MRA
    - Unique mode of interaction w/mineralocorticoid receptor
    - High affinity and selectivity for mineralocorticoid receptor
    - Minimal activity w/other hormone receptors
      - Androgen, progesterone, glucocorticoid, estrogenic receptors
    - Short half life, doesn't generate active metabolites
  - More balanced distribution between cardiac/renal tissues
  - Tissue distribution profile/pharmacodynamics
    - ? More favorable risk: benefit ratio
    - low incidence of hyperkalemia
- Dosing
  - 20-40mg daily
  - Renal dosing- cut in half

Loop Diuretics



Diuretic meds

Table 2

Diuretics Utilized in the Treatment of Heart Failure

Drug	Pharmacologic Class	Dosage	Onset of Action (Oral/IV)	Elimination Half-life in HF
Furosemide	Loop diuretic	Oral: 20 mg up to 400 mg IV: 40-mg IV load, then 10-40 mg/h infusion	1 h/5 min	2.7 h
Torsemide	Loop diuretic	Oral: 10 mg up to 200 mg IV: 20-mg IV load, then 5-20 mg/h infusion	1 h/10 min	6 h
Bumetanide	Loop diuretic	Oral: 1 mg up to 5 mg IV: 1-mg IV load, then 0.5 to 2 mg/h infusion	0.5 h/5 min	1.3 h
Ethacrynic acid	Loop diuretic	Oral: 50 mg up to 400 mg IV: 0.5-1 mg/kg/dose (max 100 mg/dose)	30 min/5 min	ND
Metolazone	Thiazide-like diuretic	Oral: 2.5 mg up to 20 mg	1 h/NA	ND
Chlorthalidone	Thiazide diuretic	Oral: 25 mg up to 200 mg	2 h/NA	ND
Hydrochlorothiazide	Thiazide diuretic	Oral: 12.5 mg up to 100 mg	2 h/NA	ND
Chlorothiazide	Thiazide diuretic	Oral and IV dosing are equivalent: 500 mg up to 1,000 mg	1 h/15-30 min	ND
Spironolactone	Aldosterone antagonist	Oral: 12.5 mg up to 50 mg[a]	ND	ND
Eplerenone	Aldosterone antagonist	Oral: 25 mg up to 100 mg	ND	ND

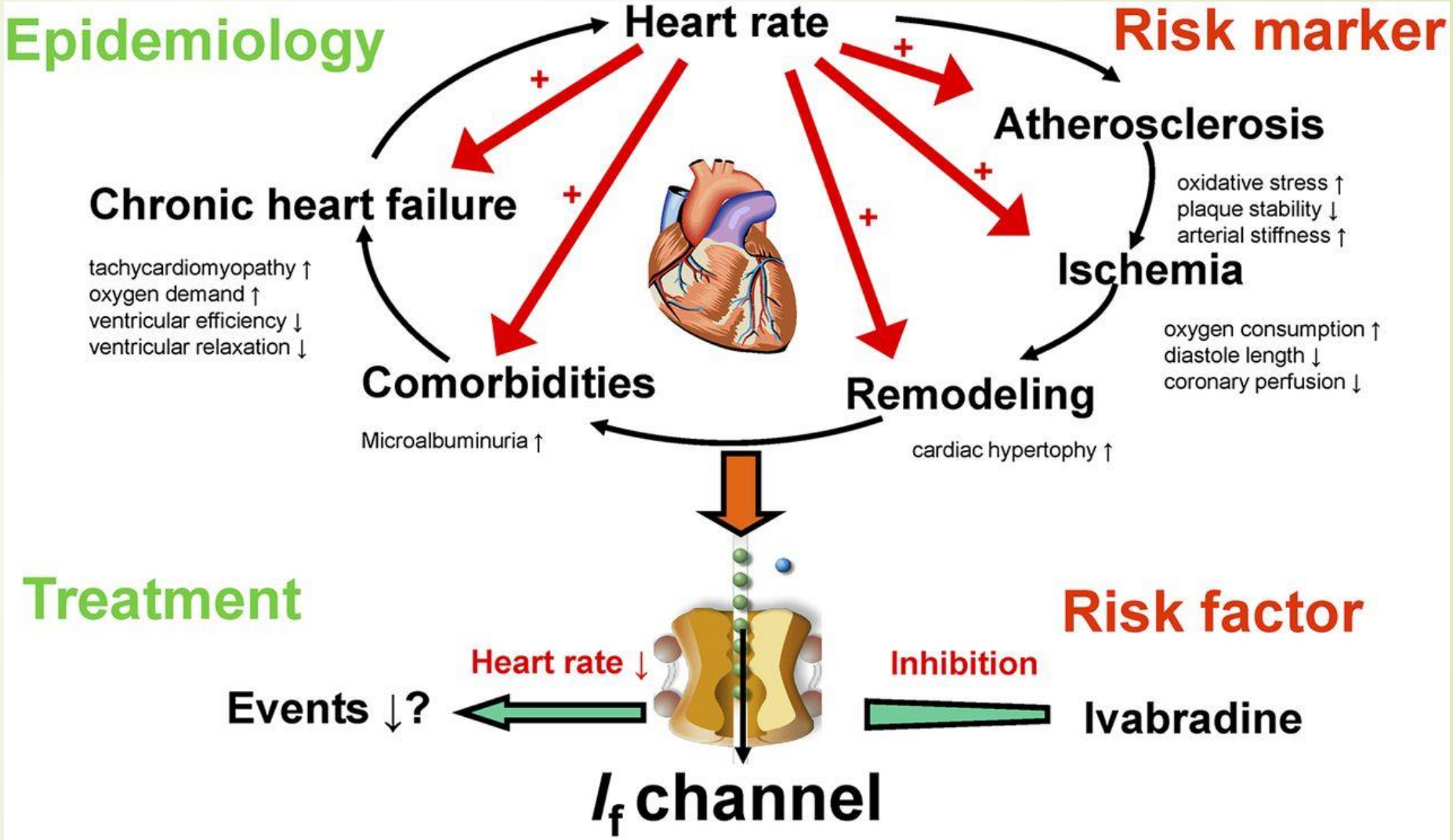
[a] Higher doses may occasionally be used with close monitoring. min: minute; max: maximum; ND: not determined; NA: not applicable.
Source: Reference 10.

Loop Diuretic	IV, equivalent dose (MG)	IV to PO conversion
Bumetanide	1	1:1
Torsemide	20	1:1
Furosemide	40	1:2

No specific evidence for long term benefit with diuretics

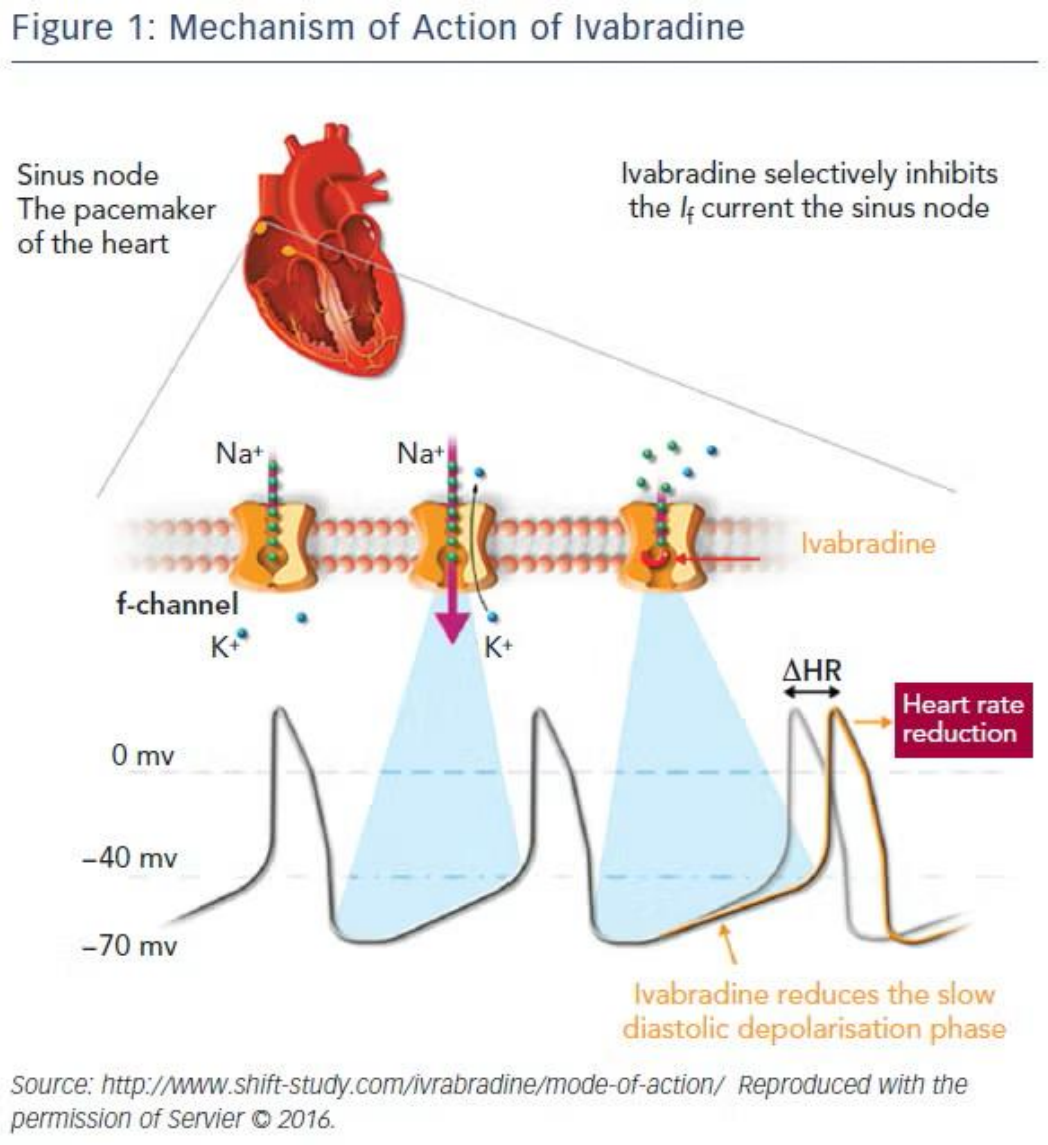
US Pharm. 2008;33(6):HS10-HS18

Heart rate effects in CHF



Böhm M, Reil J
Heart rate: surrogate or target in the management of heart failure?
Heart 2013;**99:**72-75.

Ivabradine



- Selectively and specifically inhibits the If current in SA node
- Reduce HR w/o affecting the autonomic nervous system (ANS)
  - HR reduction can increase the duration of diastole----improve myocardial perfusion.
- Slows LV remodeling, can induce reverse LV remodeling
- ? Combination of BB and Ivabradine may be important
  - BB—reduce HR and prolong diastolic duration
    - Impair isovolumetric relaxation
    - Increase alpha-adrenergic coronary vasoconstriction
  - Ivabradine: does NOT increase alpha-adrenergic coronary vasoconstriction
    - Increase Stroke volume in severe CHF
  - USE of BOTH: improves myocardial perfusion-both at rest and during exercise

Ivabradine

- Indications:
  - Symptomatic (NYHA II-III) CHF
  - EF </= 35%
  - **Sinus rhythm with HR >70**
  - On GDMT (including max tolerated BB)
- Goal: decrease heart rate
  - Reduce HF hospitalizations

- Dosage:
  - 5mg po BID
  - If Hx of conduction issues: 2.5mg po BID
  - Goal: titrate to HR 50-60bpm
    - Titrate up to 7.5mg BID if HR >70, titrate down if HR <50.
    - Stop if persistent HR <50
- Side effects
  - Bradycardia
  - Blurry vision

Hydralazine/Isosorbide Dinitrate

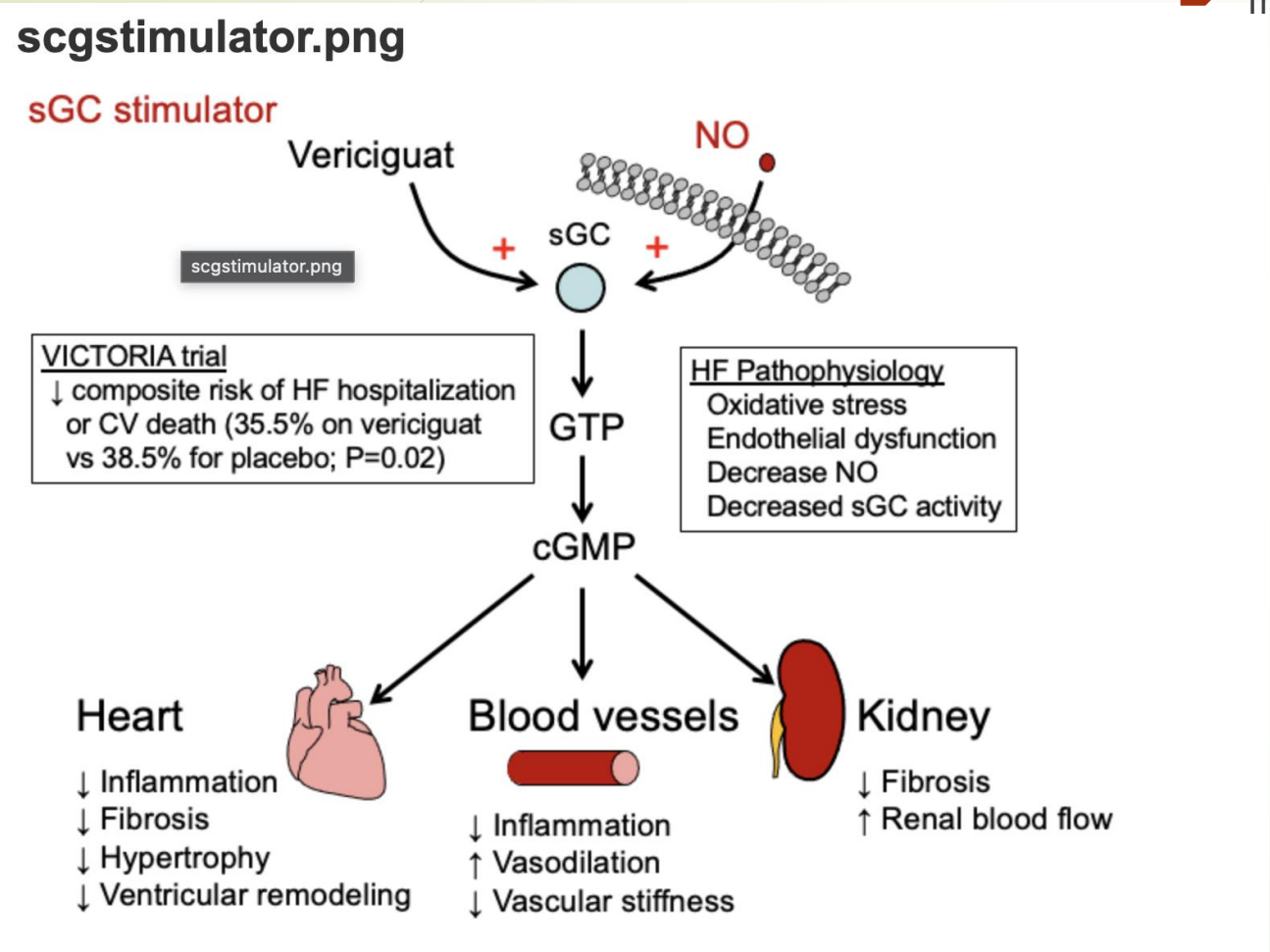
- Mechanism of Action
  - Isosorbide Dinitrate
    - Nitric oxide donor
      - Increases cyclic GMP
      - Causes primarily venous vasodilation
  - Hydralazine
    - Dilates resistance arterioles
    - May prevent development of tolerance to nitrates
  - Combination
    - Improves hemodynamics by reducing biventricular filling pressures and afterload

- In the failing heart, increased levels of reactive oxygen species can negatively impact myocardial calcium handling and contribute to the development of ventricular hypertrophy, apoptosis and necrosis, as well as negatively affect vascular endothelial and smooth muscle physiology (Munzel et al, 2017).
- **NO is known to reduce the harmful effects of reactive oxygen species** which are elevated in heart failure (Taylor et al, 2004; Cole et al, 2011; Mollace et al, 2023) Evidence indicates that elevating NO levels in the setting of HFrEF can augment cardiac contractility and reduce the progression of heart failure by exerting multiple effects including:
  - reduced tissue inflammation
  - reduced fibrosis
  - reduced hypertrophy
  - inhibition of ventricular & vascular remodeling
  - reduced vascular stiffness

Hydralazine/Isosorbide dinitrate

- Indications
  - Self Identified African American patients
    - NYHA III-IV on optimized GDMT
  - Intolerance to first line agents
- Goal
  - Improve symptoms
  - Reduce morbidity and mortality
- Efficacy attributed to lower nitric oxide bioavailability and differences in RAAS activation in African American patients
- Dosing
  - Fixed dose combination
    - Start 1 tab 37.5mg hydralazine/20mg isosorbide dinitrate TID
    - Can cut in half TID
- Side effects
  - Headache
  - Nausea
  - Dizziness
  - hypotension

Guanylate cyclase inhibitor (vericiguat)



- In Heart failure
  - Oxidative stress/endothelial dysfunction cause:
    - Decreased soluble guanylate cyclase (sGC) activity
    - Decreased Nitric oxide (NO) bioavailability
  - This contributes to myocardial and vascular dysfunction.

https://tmedweb.tulane.edu/pharmwiki/lib/exe/detail.php/scgstimulator.png?id=treatment_of_heart_failure

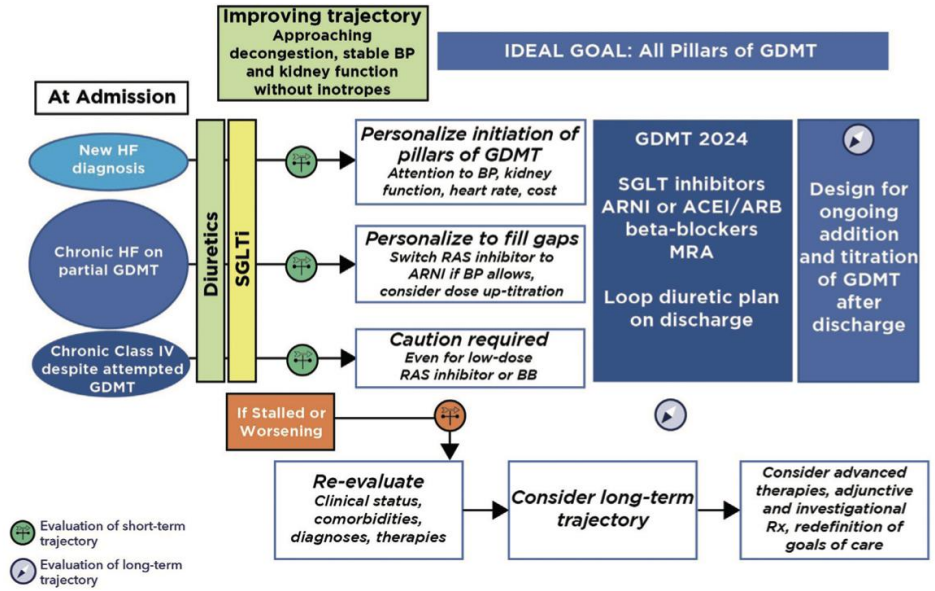
Guanylate cyclase inhibitor (Vericiguat)- WHEN?

- Indications
  - Recent CHF hospitalization (6mo) OR need for outpatient IV diuretics (3mo)
  - On GDMT and BNP >/= 300 or NTproBNP >/= 1000, in SR
  - Symptomatic CHF, EF <45%
- Goal:
  - Decrease cardiovascular death
  - HF hospitalization
- Dosage:
  - Start 5mg po daily, increase every 2 weeks to 10mg daily
  - If hypotensive: start at 2.5mg daily
- Adverse effects:
  - Anemia
  - Hypotensive
- BLACK BOX warning
  - pregnancy

Rapid initiation of meds

- Patient considerations
  - Patient phenotype
    - Wet vs Dry
- Consider starting several meds at once and/or add new each day
  - GOAL- discharge on 4 Pillars of GDMT
- Titration after discharge
  - Q1-2 weeks
  - Vitals, labs

FIGURE 7B Titration of GDMT in HFrEF



Titration of GDMT in HFrEF by initial presentation and trajectory. Patients with decompensated HFrEF should be diuresed and started on SGLT inhibitor unless contraindicated or cost prohibitive. Those with an improving trajectory (denoted by the green weathervane icon) should have optimization of GDMT. Patients may have a new HF diagnosis, in which case initiation of all 4 pillars of GDMT should be attempted. Patients with chronic HF on partial GDMT should have personalized therapy to fill in gaps, considering a switch from an ACE inhibitor/ARB to ARNI if appropriate. Caution is required for patients with chronic Class IV HF with decompensated HF; these patients may not tolerate even low doses of beta blockers and RAS, although an attempt at titration may be made. Patients whose short-term trajectory is stalled or worsening (denoted by the orange weathervane icon) should have re-evaluation of comorbidities and consideration of other diagnoses. The long-term trajectory (denoted by the compass icon) should be reevaluated, with consideration of goals of care, candidacy for advanced therapies, and experimental treatments. The ideal goal is initiation of all 4 pillars of GDMT for HFrEF in the hospital on a baseline of diuretic therapy. A plan for ongoing addition and titration of GDMT after discharge should be fashioned as well. ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor/neprilysin inhibitor; BB = beta-blocker; BP = blood pressure; d/c = discharge; GDMT = guideline-directed medical therapy; MRA = mineralocorticoid antagonist; RAS = renin-angiotensin system; SGLT = sodium-glucose cotransporter.

Case Study 1

- 40 y/o male patient
- Presents with recent history of SHOB, fatigue, swelling
- New onset Afib RVR (HR 140)
- Assessment findings
  - New holosystolic murmur at the apex, 3/6
  - Pitting edema 2+ bilat
  - Crackles in the bases bilat
  - SHOB at rest with talking
- CXR
  - Shows congestion bilat
- NTproBNP 23,000
- All other lab normal
- Echo
  - Severe MR, LVIDd 5.6
  - EF 15%
- Home meds: none
- Exercises, uses pre-workout daily
- Drinks energy drinks

Case Study 1- Treatment

- Diagnosis
  - Acute systolic heart failure
  - Acute dilated cardiomyopathy
  - Acute Afib w/RVR
  - Severe functional MR
- Treatment:
  - Manage Afib- rate, rhythm control
  - IV diuresis- aggressive
- Education
  - STOP pre-workout
  - STOP energy drinks
- Discharge
  - Lab in 1-2 weeks (BMP. NTproBNP)
  - Daily weight, BP
  - Echo in 2-3 months
  - Lifevest
  - Uptitrate meds as tolerated q2 weeks
  - Education: low salt, CHF symptoms, when to call
- Once stable: BP, heart rate
  - Start initiation of GDMT
    - Jardiance or Farxiga (1st)
    - Metoprolol Succ 12.5mg daily (1st)
      - Looking at rate control
      - If still wet, may consider ARNI
  - If tolerating---BP >100, sCr OK
    - Next day– add ARNI (24/26 mg BID or half tab BID)
  - If tolerating
    - Next day add Spironolactone 25mg ½ tab daily
  - Continue diuresis
  - Daily lab: BMP, NTproBNP every few days, CXR

Case study 2

- 45 y/o male patient
- Presents to clinic for consult for his new MR
- Assessment findings:
  - Holosystolic murmur at apex/axilla 3/6
  - Bilat LE edema 1+
- c/o SHOB over the last month with minimal exertion, fatigue, swelling
- Works construction
- Echo shows severe MR, EF 10%, LVIDd 6.2
- NTproBNP 13,000, liver enzymes slightly elevated
- CXR clear
- Meds: metoprolol tartrate 25mg BID, lisinopril 5mg daily, Lasix 20mg daily.
- History:
  - Drinks most days 2-3 beers
  - On the weekends drinks ~12 pack each day

Case Study 2- Treatment

- Diagnosis
  - Acute systolic CHF
  - Acute dilated cardiomyopathy/alcoholic cardiomyopathy
  - Severe functional MR
- Education:
  - Manage GDMT
  - STOP drinking
  - Low sodium diet
  - Daily weights
- GDMT
  - Switch Metoprolol tartrate to Metoprolol Succinate 50mg daily (1:1 daily dose equivalency)
  - Stop lisinopril. Start Entresto 24/26mg po BID in 36 hrs
    - Get lab in 2 weeks (BMP/NTproBNP)
  - Start Jardiance (or Farxiga) 10mg daily
  - In two weeks, start Spironolactone 25mg daily (or 12.5mg daily) pending BP
    - Get lab in 1-2 weeks BMP/NTproBNP
  - In two weeks, uptitrate Metoprolol OR Entresto if BP tolerates. Continue q2 weeks.
- Place Lifevest
- Repeat echo/lab in 2-3 months with clinic visit