

The Four Pillars to Successfully Treat Heart Failure

Emily Benton, PhD, NP-C
University of Colorado
Division of Cardiology

- I have no disclosures

Objectives

1. Utilize evidence-based practice guidelines to appropriately evaluate and diagnose heart failure
2. Identify the four pillars of heart failure GDMT
3. Implement GDMT in the heart failure population

How frequently will you come in contact with a heart failure patient?

A: Never

B: Occasionally (<1 time per year)

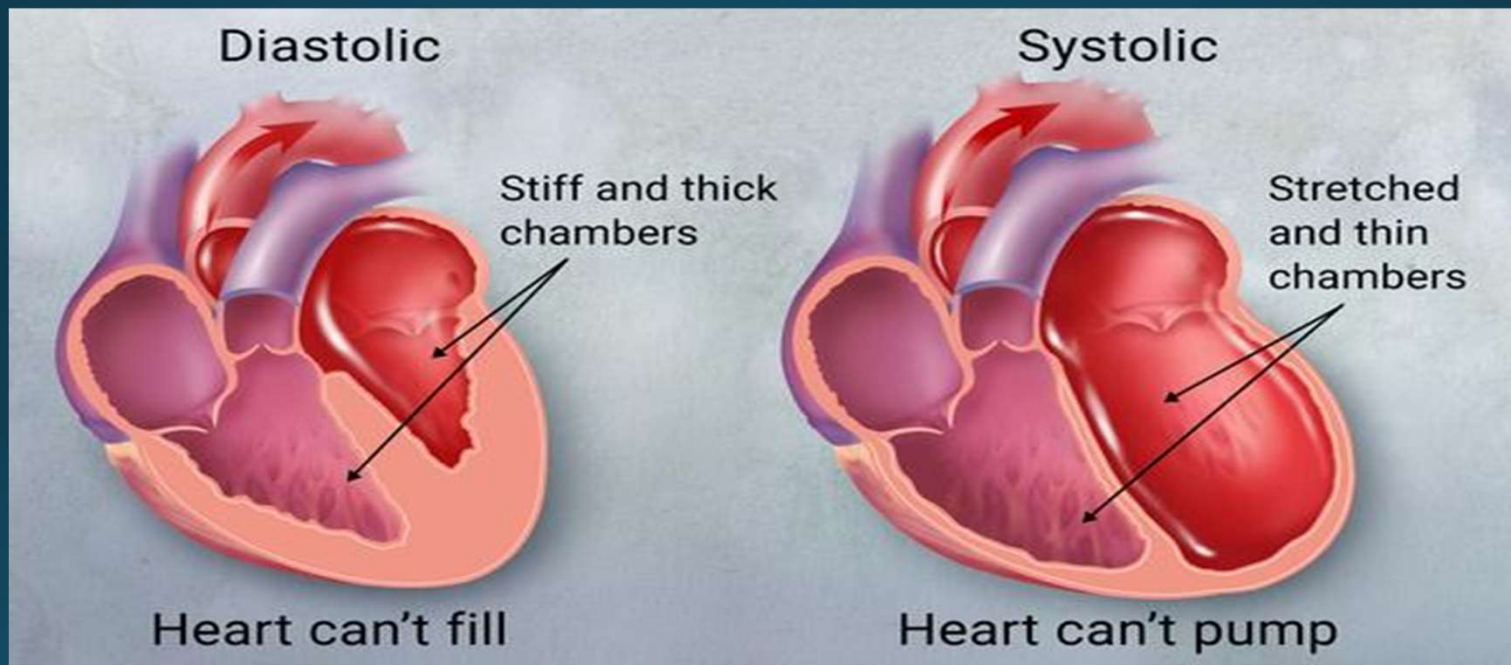
C: Frequently (>5 times per year)

D: Continuously

Understanding Heart Failure (HF)

- Over 6 million people currently living with HF and over 500,000 people are diagnosed with HF each year (CDC, 2021)
- One of the most expensive and deadly diseases you will see and treat
- In order to properly treat HF, you need to understand the source of failure and how to properly treat it
- Normal heart ejection fraction (EF) is $>55\%$

Heart Failure



HFpEF

HFrEF

Heart Failure with REDUCED Ejection Fraction (HFrEF)

- HFrEF is most commonly identified with a left ventricular EF <40%
- Two causes:
 - Ischemic
 - Non-ischemic
- It is critical to identify the cause of the heart failure, tailor treatment, and initiate GDMT in order to improve outcomes

WHY are we
looking for it?

- Chief complaint?
- Asthma?
- Recurring Bronchitis?
- Family History?
- Recent heart attack?

Review of Symptoms

- Fatigue
 - Shortness of Breath
 - Orthopnea
 - Does your patient sleep with an adjustable bed?
 - Cough (non-productive)
 - Edema
 - Bloating
 - Leg swelling
 - Depression
 - Decreased activity tolerance
- ▶ PND
 - Waking up gasping for air; usually afraid to lie flat due to breathing difficulty
 - ▶ Unexplained rapid weight fluctuations
 - Weight gain with swelling
 - Inability to lose weight despite not eating
 - ▶ Palpitations
 - ▶ Brain fog
 - ▶ Loss of appetite

Physical Exam (Objective)

Early in HF

- Tachypnea
- Abdominal distention
- Edema in lower extremities

Advanced or Decompensated HF

- Tachycardia
- Can not talk in full sentences
- Cool extremities
- Clammy on exam
- Hypotensive
- JVD
- Abdominal distention
- Edema
- Murmur (S_3 / S_4) *if advanced HFrEF*

Your exam is concerning for HF, how do we confirm and diagnosis HF?

- ECHO
- Labs:
 - BNP
 - CBC, CMP (end organ dysfunction)
 - coags (pt/INR)
- CXR

Identifying the cause of HF

WHY

- Most of the time....this does not occur over night!
 - Be cautious in your younger patients!!!!
- Incidental finding

Reversible

- The earlier you identify the cause, the more successful your treatment will be
- Sometimes, the cause of HF is not the heart....
 - Chemo/radiation
 - Trauma
 - Thyroid
 - Supplements
 - Substance Use (Etoh, meth, etc.)

Etiology of Heart Failure

Ischemic (ICM) Etiology

- Acute event: MI, embolic (post-COVID, etc.)

Non-Ischemic (NICM) Etiology

- Viral
- Endocrine
- Substance abuse (ETOH, meth)
- Rhythm (afib, persistent tachycardias)
- Infiltrative: Amyloid, non-compaction, HOCM, sarcoid
- Structural (valvular disease)
- Genetic
- Unknown.....

Evaluation of Heart Failure

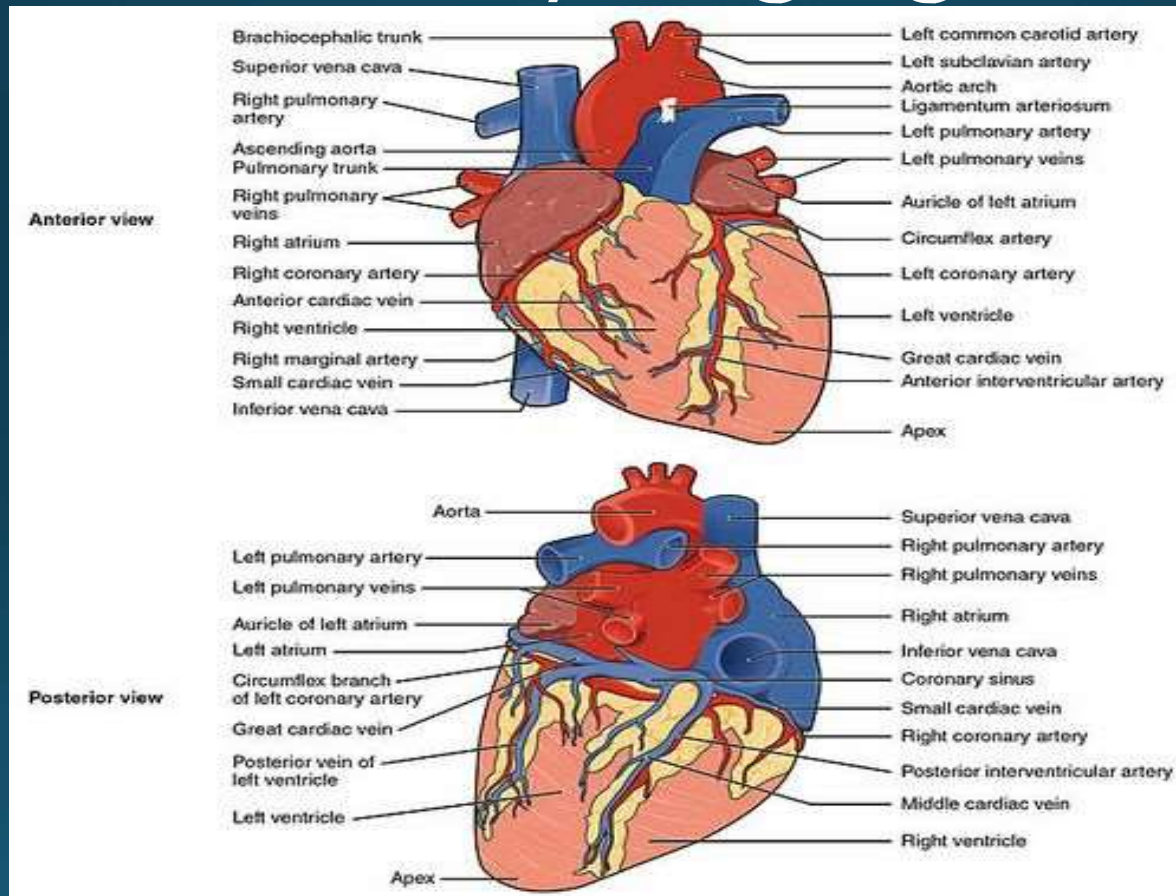
Heart Failure Evaluation

- CXR (\$)
 - Size of heart, evidence of pulmonary edema
- TTE (\$\$\$)
 - Evaluate overall heart function
- ECG (\$\$)
- CBC, BMP, INR, BNP, TSH (\$\$\$)
- Tox screens (\$\$)
- Troponins (\$)

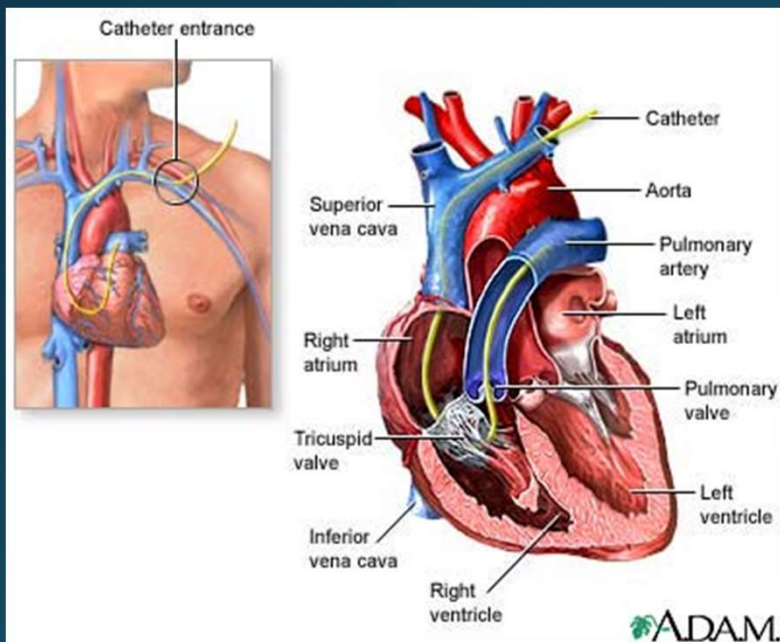
Invasive Evaluation

- Coronary Angiogram (\$\$\$)
- Right heart catheterization (\$\$\$)
- Cardiac MRI (\$\$\$)
- CT coronary (\$\$\$)
- Genetic testing (\$\$\$)

Coronary Angiogram



Right Heart Catheterization



- RA ~ 2-6mmHg
- PA ~ 20-30s/8-15mmHg
- PCWP ~ 6-12mmHg
- PA sat% ~ 60-70%
- Thermal/Fick CO/CI

B-type Natriuretic Peptides (BNP)

BNP

- Released with increase stretch of heart (volume overload)
 - Normal < 100pg/mL
- More commonly used
- Shorter 1/2 life (20 minutes)
- Altered with obesity (falsely low)
- Less costly

NT-ProBNP

- Released with increase stretch of heart (volume overload)
 - Normal <125pg/mL
- Center specific
- 90 minute 1/2 life

Trends are key! A persistently elevated BNP is a poor prognostic indicator

Initiating Heart Failure Specific Guideline Directed Medical Therapy (GDMT)

New York Heart Association Classification (I-IV) ~ FUNCTIONAL

- Class I
 - No HF symptoms
 - Class II
 - HF symptoms with moderate exertion
 - Class III
 - HF symptoms with minimal exertion
 - Class IV
 - HF symptoms at rest, any physical activity produce discomfort
- Trick is to figure out severity of symptoms....ie your patient says they get short of breath hiking vs moving from couch to chair 10 feet away

Heart Failure Stages ~ STRUCTURAL

- Stage A
 - High risk, no symptoms
- Stage B
 - Structural disease, no symptoms
- Stage C
 - Structural disease, previous or no symptoms
- Stage D
 - Refractory HF symptoms requiring intervention

Heart Failure Specific Guideline- Directed Medical Management (GDMM)

- 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure (Heidenreich et al., 2022)
- Improved survival and long-term benefits just getting on small doses ~ *some is better than none*
- Significant reduction in ALL CAUSE mortality and hospitalizations with GDMM
- GDMM will NOT necessarily improve the EF, HOWEVER; it does work with the rest of the body and prevents / protects end organ damage (ie worsening kidney function)

Where to start?



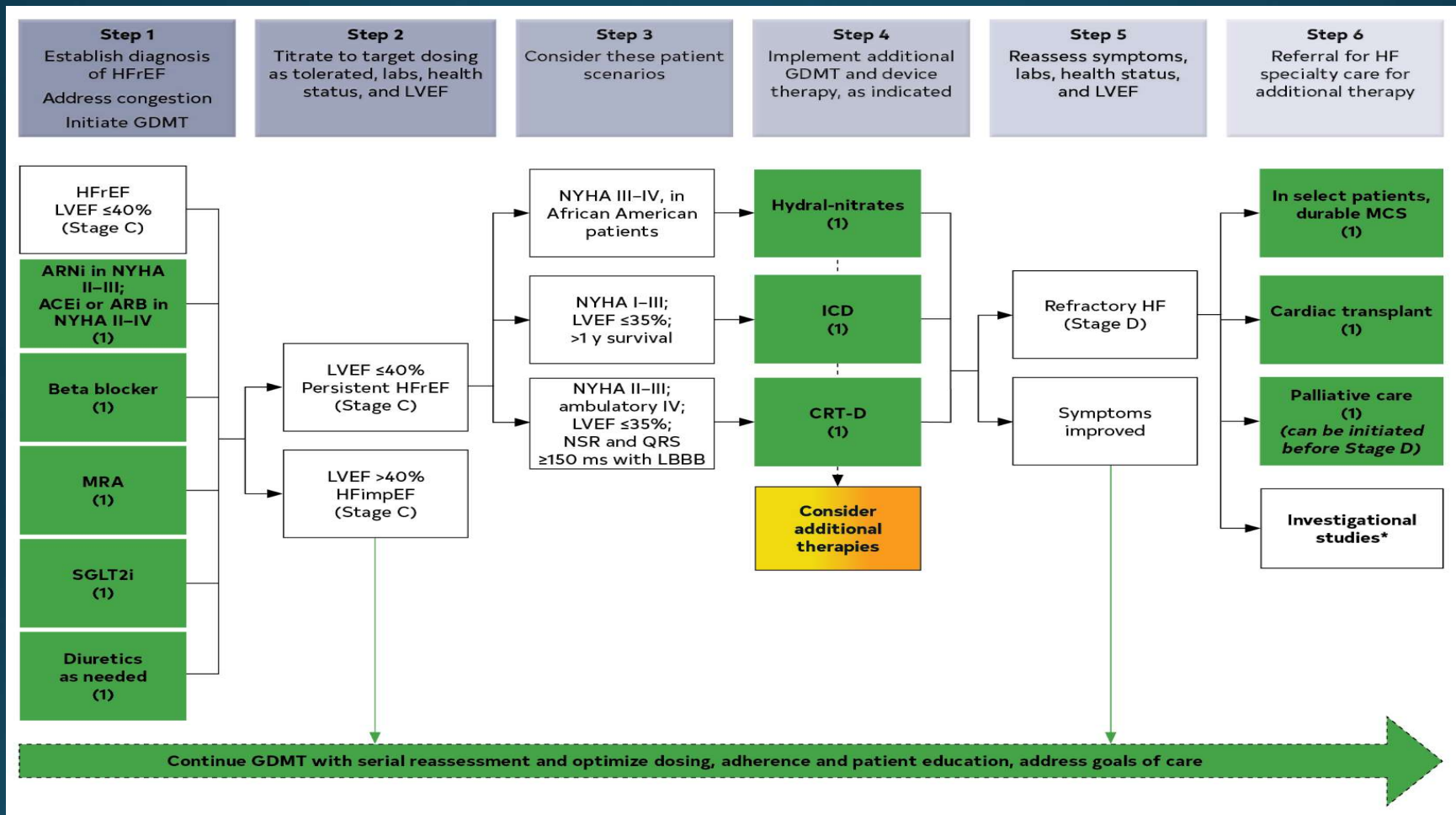
What GDMT would you start first?

A: MRA (spironolactone or eplerenone)

B: SGL2i (dapagliflozin, empagliflozin)

C: ARNI (sacubitril-valsartan) or ACEi/ARB

D: Beta-Blocker (carvedilol, metoprolol succinate, bisoprolol)



Treatment recommendations for patients with HFrEF are displayed. Step 1 medications may be started simultaneously at initial (low) doses recommended for HFrEF. Alternatively, these medications may be started sequentially, with sequence guided by clinical or other factors, **without need to achieve target dosing before initiating next medication**. Medication doses should be increased to target as tolerated.

Heidenreich et al. (2022)

GDMT:

Aldosterone Antagonist (Mineralocorticoid Receptor Antagonist)

MRA: potassium retention, mild diuretic aid

Spironolactone (Aldactone)	Eplerenone (Inspra)
25mg – 100mg PO daily	25mg – 100mg PO daily
↑ diuresis w/ higher dose	↑ diuresis w/ higher dose
↑ potassium	↑ potassium

Prior to initiation: Creatinine <2, K 3.5-4.5, no gynecomastia

GDMT: sodium-glucose cotransporter-2 (SGL2i)

SGL2i: weight loss, aid in diuresis, unclear cardiac benefits, renal protective

Dapagliflozin (Farxiga)	Empagliflozin (Jardiance)
10mg PO daily	10mg or 25mg PO daily
↑diuresis	↑ diuresis
↔ A1C	↔ A1C

Prior to initiation: GFR >20, no type-1 DM, no h/o *frequent* UTIs

GDMT: ACEi/ARB/ARNI

ACEi/ARB/ARNI: Renin-angiotensin-aldosterone system (RAAS) inhibition, after load reduction, renal protective (esp. with DM), cardiac protective with remodeling

ACEi (lisinopril, captopril)	ARB (losartan, valsartan)	ARNI Sacubitril-valsartan
↓BP	↓ BP	↓ BP effect

Prior to initiation: BMP (creatinine <2, normal potassium); blood pressure >90mmHg

ARNI: sacubitril-valsartan (ENTRESTO)

- One of the most important GDMT available!!!
 - Insurance coverage \$\$\$
 - Prior to initiation:
 - Creatinine (ok if GFR >30); Potassium WNL; BP >90mmHg
 - BNP – need baseline to trend with treatment
 - MUST BE OFF ACEi >36 hours prior to starting (angioedema risk)
 - 24/26mg PO BID, 49/51mg PO BID, 97/103mg PO BID (max)
 - Works as a neprilysin inhibitor which activates the natriuretic peptide system = ↑ vasodilatation, ↑ diuresis, ↑ afterload reduction = less stress on heart

Nicolas, D., Kerndt, C., & Reed, M (2021). Sacubitril-valsartan. Retrieved on <https://www.ncbi.nlm.nih.gov/books/NBK507904/>

GDMT: Evidence Based Beta Blockers

EVBB: beta1-adrenoreceptor overstimulation, anti-arrhythmic effects, reduction in heart rate, cardiac remodeling benefits

Carvedilol (Coreg)	Metoprolol Succinate (Toprol XL)	Bisoprolol
3.125mg – 50mg PO BID	12.5mg -100mg PO BID	2.5mg – 20mg PO daily
↓BP	↔ BP effect	↔ BP effect

Prior to initiation: normal volume status, what is baseline heart rate & rhythm, blood pressure >90mmHg

GDMT: Diuretics

Diuretics: weight loss, volume removal via diuresis

Furosemide (Lasix)	Bumetanide (Bumex)	Torsemide (Demadex)
10mg – 100mg PO BID	1-6mg PO BID	10- 100 mg PO BID
↑ dose = ↑ diuresis	↑ dose = ↑ diuresis	↑ dose = ↑ diuresis

Prior to initiation: creatinine (if ↑ will likely need higher dosing), current weight, diuretic response goal (ie 3L negative or 5# weight loss)

GDMT in different settings

Inpatient

- MRA
 - Potassium retention with diuresis
- SGL2i
 - Hold if multiple procedures planned
2/2 euglycemic DKA
 - Augments diuresis
- ARNI/ACEi/ARB
 - Off inotropes, stable BP
- EVBB
 - Last but not least.....maybe only in clinic

Outpatient

- ARNI
 - Usually a better BP in outpatient setting
- MRA
- SGL2i
- EVBB
 - Usually tolerated more in outpatient setting
 - Euvolemic

On Good GDMT, now what??

- Frequent follow up with medication titration
 - Up to twice weekly appointments are sometimes necessary, especially new diagnosis, recent hospital discharge, up-titrating meds
- Repeat an ECHO in 6 months (be prepared that the EF has not changed)
- Repeat BMP with each titration (ACEi/ARB/ARNI, MRA, diuretics)
- Trend BNP

Optimization of HF after GDMT

- Very high risk of sudden death
- Arrhythmias are a sign of the heart asking for help
 - Natural progression of the disease
- Atrial vs ventricular
 - Symptomatic?
 - Affect quality of life

Cardiac Resynchronization Therapy Defibrillator (CRT-D)

- Optimization of ventricular function through electrical optimization
- Helps the heart to beat in synchrony
 - Must be on GDMT for 3 months
 - Reasonable survival over 1 year
 - QRS <150ms
 - Persistently low EF (<35%) despite optimization
 - Persistent and symptomatic VT

Implantable Cardioverter Defibrillator (ICD)

- ICDs can be safe and benefit the patient:
 - Must be on stable GDMT for 3 months
 - Reasonable survival over 1 year
 - Persistently low EF (<35%) despite optimization
 - Persistent and symptomatic VT

Progression of Heart Failure

Evidence of Worsening Heart Failure

- Patients endorse: ↑ SOB, ↑ fatigue, ↓ appetite, ↑ weight (fluid), ↓ activity tolerance
- Hypotension:
 - BP < 85mmHg and symptomatic
 - As HF worsens, target BP 80-90mmHg WITHOUT symptoms
 - Decreased doses of GDMT = poor prognostic sign
- Activity Tolerance:
 - Unable to climb a flight of stairs or ambulate without frequent stops; more sedentary but symptomatic
- Loss of appetite
 - Despite not eating, weight is going up (fluid accumulation and muscle loss)
- Diuretic Resistance
 - Despite increasing doses, they are not making urine and gaining fluid

Treatment of Worsening HF

- Volume assessment
 - Increase diuretics or change agent if not absorbing/responding well
 - Consider RHC
- Decrease beta-blocker
 - If you are in shock or decompensated HF – continuing the beta-blockade is like driving with your foot on the gas pedal and brake at same time
 - First to be removed, last to be added before discharge!
- Decrease Afterload Reduction to help with hypotension
- Does your patient have evidence of end-organ dysfunction (creatinine above baseline or liver dysfunction)??

HF Hospitalization(s)

- Any admission for HF is concerning for overall prognosis
 - 3 heart failure specific admissions in 6 months
 - ~consider referral for advanced therapies: cardiac transplant and/or LVAD
- Volume overload is **#1** reason for admission
- Hospitalizations are very costly with high expectations and close monitoring by government
 - Failure to discharge patients on GDMT = \$\$\$ penalty for institution
 - Failure to optimize HF patients = recurrent admissions = increased financial burden
 - HF patients **MUST** have follow up with in 7 days of discharge!

Goals of Care (GOC)

- GOC must be addressed with every HF patient
- The unpredictability and high mortality rate pose high risk for unwanted hospitalizations and treatments
- GOC conversations should occur early and often throughout diagnosis (Braun et al., 2016)
 - As APPs – this often falls on us...Taking the time to get to know your patient. We can be advocates for a procedure just as much as we are an advocate to prevent a procedure.

Questions?

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emily.benton@cuanschutz.edu