ACUTE HEART FAILURE: FAILURE TO LAUNCH

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Critical Care

https://en.wikipedia.org/wiki/Failure_to_Launch
Disclosures

- I have nothing to disclose
Take Away:

- The learner should have a better understanding of the pathophysiology of heart failure
- The learner should be able to identify patients with acute heart failure
- The learner should be able to start the initial management of acute heart failure
The Heart is Dumb

■ In its simplest form the heart is merely a pump.

■ What happens when a pump breaks?

https://medium.com/indian-thoughts/the-human-heart-is-so-stupid-79bbac978b53
https://asheathersworldturns.wordpress.com/2015/01/13/mythbusters-takes-on-the-simpsons/
Back to the Basics: Delivery of Oxygen

- Why do we have a heart?
  - *To deliver oxygen to tissues.*

- DO2 = CaO2 x CO

- CaO2 = (Hgb x 1.34 x SaO2) + (PaO2 x 0.003)

https://www.wisegeek.com/what-is-oxygen.htm
Back to the Basics: Cardiac Output

Cardiac Output = Heart Rate $\times$ Stroke Volume

$CO = HR \times SV$

$5\text{L/min} = 72 \text{ beat/min} \times 70 \text{ ml/beat}$

https://slideplayer.com/slide/10693367/
Back to the Basics: Stroke Volume

Which factors determine stroke volume?

- Afterload
- Contractility
- Preload
Back to the Basics: Afterload

- Impedance during ejection
- Affected by:
  - Ventricular volume
  - Wall thickness
  - Aortic pressure
  - Aortic impedance
Back to the Basic: Contractility

- Force generated by the myocardial fibers
- "The Squeeze"

Back to the Basic: Preload

- Load – force exerted on a source or body
- Force – interaction between bodies, with direction and magnitude

- What is the type of force being exerted onto the myocardium?  
  - Pressure
- How do you affect Pressure inside the ventricle?  
  - Volume
Back to the Basics: Kinetic and Potential Energy

https://people.com/celebrity/the-lion-king-turns-20/

**Hooke’s law** is a law of physics that states that the force \( F \) needed to extend or compress a spring by some distance \( x \) scales linearly with respect to that distance.

“The Icarus of the Heart”
Back to the Basics: Pressure and Volume
Types of Dysfunction

- Systolic (HF with Reduce EF)
- Diastolic (HF with Preserved EF)

Systolic Dysfunction: The Broken Spring

https://fineartamerica.com/featured/exploding-water-balloon-3-christoffer-rathjen.html
Diastolic Dysfunction: The Rigid Container

## Etiology of Chronic Heart Failure

<table>
<thead>
<tr>
<th>Disease of Myocardium</th>
<th>Toxic Damage</th>
<th>Immune-mediated and Inflammatory Damage</th>
<th>Infectious Disease</th>
<th>Metabolic Abnormalities</th>
<th>Genetic Abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic heart disease</td>
<td>Alcohol, cocaine, amphetamines, anabolic steroids.</td>
<td>Related to infection: Bacteria, streptococci, fungi, protozoa, parasites (Chagas disease, leishmaniasis, viruses (HIV/AIDS).</td>
<td>Lymphopenic/post myocarditis, autoimmune diseases (e.g., Graves’ disease, rheumatoid arthritis, connective tissue disorders, mainly systemic lupus erythematosus), hyperreactivity and eccentric hypertrophy (Churg-Strauss).</td>
<td>Associated to malignancy: Amyloidosis, sarcoidosis, hemochromatosis (PV), glycogen storage disease (e.g. Pompe disease), Familial hypercholesterolemia (e.g. Fabry disease).</td>
<td>Diverse forms: HCM, DCM. LV non-compaction, ARVC. Mucocutaneous cardiomyopathy. For details see respective expert documents, muscular dystrophies and laminopathies.</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>Heart metals: Copper, iron, lead, cobalt.</td>
<td>Not related to infection: Lymphopenic/post myocarditis, autoimmune diseases (e.g., Graves’ disease, rheumatoid arthritis, connective tissue disorders, mainly systemic lupus erythematosus), hyperreactivity and eccentric hypertrophy (Churg-Strauss).</td>
<td>Not related to malignancy: Amyloidosis, sarcoidosis, hemochromatosis (PV), glycogen storage disease (e.g. Pompe disease), Familial hypercholesterolemia (e.g. Fabry disease).</td>
<td>Nutritional: Deficiencies in thiamine, L-carnitine, vitamin A, iron, phosphorus, calcium, copper malabsorption (e.g. malnutrition, AIDS, anemia normocytic).</td>
<td>Diverse forms: HCM, DCM. LV non-compaction, ARVC. Mucocutaneous cardiomyopathy. For details see respective expert documents, muscular dystrophies and laminopathies.</td>
</tr>
<tr>
<td>Primary valvular heart disease</td>
<td>Medications: Cyclosporin and immunosuppressants, non-steroidal anti-inflammatory drugs, anesthetics.</td>
<td>Related to infection: Lymphopenic/post myocarditis, autoimmune diseases (e.g., Graves’ disease, rheumatoid arthritis, connective tissue disorders, mainly systemic lupus erythematosus), hyperreactivity and eccentric hypertrophy (Churg-Strauss).</td>
<td>Not related to malignancy: Amyloidosis, sarcoidosis, hemochromatosis (PV), glycogen storage disease (e.g. Pompe disease), Familial hypercholesterolemia (e.g. Fabry disease).</td>
<td>Genetic: Hypereosinophilic syndromes, Down syndrome, Marfan syndrome, Turner syndrome, Noonan syndrome.</td>
<td>Diverse forms: HCM, DCM. LV non-compaction, ARVC. Mucocutaneous cardiomyopathy. For details see respective expert documents, muscular dystrophies and laminopathies.</td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td>Endothelial dysfunction.</td>
<td>Related to infection: Lymphopenic/post myocarditis, autoimmune diseases (e.g., Graves’ disease, rheumatoid arthritis, connective tissue disorders, mainly systemic lupus erythematosus), hyperreactivity and eccentric hypertrophy (Churg-Strauss).</td>
<td>Not related to malignancy: Amyloidosis, sarcoidosis, hemochromatosis (PV), glycogen storage disease (e.g. Pompe disease), Familial hypercholesterolemia (e.g. Fabry disease).</td>
<td>Genetic: Hypereosinophilic syndromes, Down syndrome, Marfan syndrome, Turner syndrome, Noonan syndrome.</td>
<td>Diverse forms: HCM, DCM. LV non-compaction, ARVC. Mucocutaneous cardiomyopathy. For details see respective expert documents, muscular dystrophies and laminopathies.</td>
</tr>
<tr>
<td>Other (5%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Diverse forms: HCM, DCM. LV non-compaction, ARVC. Mucocutaneous cardiomyopathy. For details see respective expert documents, muscular dystrophies and laminopathies.</td>
</tr>
</tbody>
</table>

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## Classification of Heart Failure: ACC/AHA Stage vs NYHA Class

<table>
<thead>
<tr>
<th>ACC/AHA Heart Failure Stage</th>
<th>NYHA Functional Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. At risk for heart failure but without structural heart disease or symptoms</td>
<td>None</td>
</tr>
<tr>
<td>B. Structural heart disease but without heart failure</td>
<td>I. Asymptomatic</td>
</tr>
</tbody>
</table>
| C. Structural heart disease with prior or current heart failure symptoms | II. Symptomatic with moderate exertion  
III. Symptomatic with minimal exertion |
| D. Refractory heart failure requiring specialized interventions | IV. Symptomatic at rest |

Triggers of Acute Decompensated Heart Failure

- Acute coronary syndrome
- Tachyarrhythmia (e.g., atrial fibrillation, ventricular tachycardia)
- Excessive rise in blood pressure
- Infection (e.g., pneumonia, infective endocarditis, sepsis)
- Non-adherence with salt/fluid intake or medications
- Bradyarrhythmia
- Toxic substances (alcohol, recreational drugs)
- Drugs (e.g., NSAIDs, corticosteroids, negative inotropic substances, cardiotoxic chemotherapeutics)
- Exacerbation of chronic obstructive pulmonary disease
- Pulmonary embolism
- Surgery and periooperative complications
- Increased sympathetic drive, stress-related cardiomyopathy
- Metabolic/hormonal derangements (e.g., thyroid dysfunction, diabetic ketosis, adrenal dysfunction, pregnancy and peripartum related abnormalities)
- Cerebrovascular insult
- Acute mechanical cause: myocardial rupture complicating ACS (free wall rupture, ventricular septal defect, acute mitral regurgitation), chest trauma or cardiac intervention, acute native or prosthetic valve incompetence secondary to endocarditis, aortic dissection or thrombosis.

ACS = acute coronary syndromes; NSAIDs = non-steroidal anti-inflammatory drugs.


Profiles of Acute Decompensated Heart Failure

- 6 profiles (2005 European Society of Cardiology Guidelines for the Diagnosis and Treatment of Acute Decompensated Heart Failure
- Mild to Moderate
- Hypertensive ADHF
- ADHF with severe pulmonary edema
- Cardiogenic shock
- High-output heart failure
- Right sided heart failure

Mild to Moderate

- 70% of all HF admissions
- Mild to mod pulmonary edema
- JVD, hepatojugular reflex, R sided pleural effusion more frequent than L
- BLE edema


Hypertensive

■ 10% of acute decompensated heart failure
■ Rapid onset of heart failure with systolic BP of > 140 mmHg
■ Usually older women
■ Usually a preserved EF


ADHF with Severe Pulmonary Edema

- 15% of admissions for ADHF have pulmonary edema
- 3% have severe pulmonary edema
- “Flash pulmonary edema”

https://www.med-ed.virginia.edu/courses/rad/cxr/pathology2chest.html
Cardiogenic Shock

- 8% of patients with ADHF
- Systolic BP < 90
- Hypoperfusion

High-Output

- Uncommon
- No overt signs of hypoperfusion (warm extremities)
- Anemia, thyrotoxicosis, advance liver failure, Paget disease

https://www.health.harvard.edu/heart-health/how-thyroid-hormone-affects-the-heart
Right Sided

- Occurs in severe TR
- RV dysfunction
- Chronic lung disease (Cor pulmonale)
- Pulmonary HTN

https://www.researchgate.net/figure/Transthoracic-echocardiography-from-apical-four-chamber-view-shows-enlarged-right_fig1_45187288
ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012

The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association (HFA) of the ESC.

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ESC: explicitly participating in the development of the document

AANCA: American Academy of Cardiovascular Assistant (AANCA): European Association for Cardiovascular Prevention and Rehabilitation (EACPR); European Association of Percutaneous Cardiovascular Interventions (EAPCI); Working Groups: Acute Cardiac Care; Cardiovascular Pharmacology and Drug Therapy; Cardiovascular Surgery; Congenital Heart Disease; Hypertension; the Heart and Microcirculation; Preventive Cardiology; Phlebology and Venous Thrombosis; Valvular Heart Disease; Cardiovascular Imaging; Cardiovascular Nursing and Allied Professions; Cardiology Practice; Cardiovascular Primary Care.

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Diagnostics

- History and Physical Exam
- Vitals
- BNP, N-terminal proBNP
- Echocardiogram
- ECG
- Troponin
- CXR
- Chems, CBC, Lactate

https://www.featurepics.com/online/Medical-Chart-1960573.aspx
Figure 1. Biomarkers Indications for Use

Colors correspond to COR in Table 1.
*Other biomarkers of injury or fibrosis include soluble ST2 receptor, galectin-3, and high-sensitivity troponin. ACC indicates American College of Cardiology; AHA, American Heart Association; ADHF, acute decompensated heart failure; BNP, B-type natriuretic peptide; COR, Class of Recommendation; ED, emergency department; HF, heart failure; NT-proBNP, N-terminal pro-B-type natriuretic peptide; NYHA, New York Heart Association; and pts, patients.
Which Patients Do I Admit?
Inpatient Monitoring
Figure 12.2: Initial management of a patient with acute heart failure. *Acute mechanical cause: myocardial rupture complicating acute coronary syndrome (true wall rupture, ventricular septal defect, acute mitral regurgitation), chest trauma or cardiac intervention, acute native or prosthetic valve incompetence secondary to endocarditis, aortic dissection or thrombosis, see above.*
Heart Failure Society of America
Goals of Treatment

- Symptom control and management
- Normal oxygenation
- Optimization of volume status
- Identify and treat underlying trigger
- Optimize chronic therapy
- Minimize side effects of management
- Identify patients that can benefit from revascularization, device therapy, anticoagulation therapy
- Education of patients
- Disease management programs


Colucci WS et al. Treatment of acute decompensated heart failure: General Considerations. UpToDate. 11.2016-2.2019
Initial Management: Systolic and Diastolic Dysfunction

- Diuresis
- Oxygen
- Hemodynamic stability
  - Vasodilation
  - Vasopressors
  - Arrhythmia management

Colucci WS et al. Treatment of acute decompensated heart failure: General Considerations. UpToDate. 11.2016-2.2019
Why Loop Diuresis

- Early diuresis with IV furosemide (< 60 mins) can lower inpatient mortality compared to later treatment (2.3 v 6%, p = 0.002)
- Reduces intravascular volume. Lowers CVP and PCWP.
- +/- venodilation effect secondary to prostaglandin release

Loop Diuresis

- Careful in hypotension/shock
- Dose should be individualized, loop naïve consider starting IV doses of:
  - *Furosemide* 20-40 mg
  - *Bumetanide* 1 mg
  - *Torenemide* 10-20 mg

Loop Diuresis

- Chronic loop diuretic dosing may require > 2.5 x maintenance oral dosing
- Bolus dosing vs continuous infusions.
- Follow Kidney function and electrolytes (replete as needed)

Vasodilation

- Used to decrease elevated filling pressures
- Decrease LV afterload pressure
  - Severe HTN
  - Acute AR
  - Acute mR

https://psychonautwiki.org/wiki/Vasodilation
Vasodilation

- Nitroglycerin
  - venous > arterial dilation
  - High doses decrease SVR, LV afterload = possible increased SV and CO
  - IV dosing 5-10 mcg/min increase by 5 mcg/min q 5 mins as tolerated to symptom improvement up to 200 mcg/min
  - Tachyphylaxis can occur within hours
  - Hypotension
  - HA
  - Do not give with recent PDE-5 inhibitors

Colucci WS. Treatment of acute decompensated heart failure: components of therapy. 
UpToDate 12.2018-2.2019
Vasodilation

- **Nitroprusside**
  - *Venous = arterial dilation*
  - *Decrease LV filling pressures and SVR*
  - *Decrease afterload*
  - *Initial dose 5-10 mcg/min titrate as tolerated up to 400 mcg/minutes*
  - *Metabolized to cyanide, thiocyanate*
Vasodilation

- **Nesiritide**
  - *Increased hypotension without improvement in outcomes, no longer recommended for use*
  - (ASCEND-HF, NEJM 2011)
Water Metabolism

- Sodium restriction (unknown specific level goals), usually < 2-3 g depending on which guideline you follow (2013 ACC/AHA, 2012 ESC)
- Limiting fluids 1.5-2 L/day (2013 ACC/AHA guidelines) in general. If hyponatremia present may need to alter dosing of water

https://newsnetwork.mayoclinic.org/discussion/helpful-ways-you-can-reduce-your-sodium-intake/
Morphine

- Decreases anxiety, decreases WOB
- Very little quality of evidence of benefit with some evidence of harm
- Increased risk of MV, ICU admit and in hospital mortality


Hypotension with HFrEF

- Consider Inotrope when hypoperfusion present
- Vasopressors may be required to increase SVR to perfuse organs
  - Increased in LV afterload
- LVEF < 25%, consider mechanical support (IABP, ECMO, VAD) to bridge to decision or bridge to recovery

2013 ACCF/AHA guidelines and 2012 ESC guidelines
Hypotension with HFpEF

- No inotropes
- Vasopressors if hypoperfusion present
- Diuresis if volume overloaded
- Dynamic LVOT need BB, vasopressors and gentle hydration (preload dependent)
  - No inotropes

Inotropes

- No evidence for clinical effect or safety
- Routine use can be harmful (increased hypotension, atrial arrhythmia increased in hospital mortality 3.8 v 2.3% and 60 day (10.3 vs 8.9%)

Cuff MS et al. Short Term intravenous milirone for acute exacerbation of chronic heart failure:a randomized controlled trial. JAMA, 2002;287(12):1541


Inotropes

- **Dobutamine**
  - *Beta 1 agonist, minimal effects on B2 or Alpha 1*
  - *Increase SV, CO*
  - *Decrease SVR and PCWP*
  - *Started 2.5 mcg/kg/min, titrated up to 20 mcg/kg/min*

https://rk.md/2018/dobutamine/
Inotropes

- Milrinone
  - *PDE inhibitor*
  - *Decrease SVR and PVR (afterload)*
  - *Increase CO*
  - *50 mcg/kg loading over 10 mins, followed by 0.375 mcg/kg/min-0.750 mcg/kg/min*

Inotropes

- **Dopamine**
  - *Dopamine receptors*
  - *Beta 1*
  - *Alpha1*
  - *1 micrograms/kg/min up to 10 micrograms/kg/min*
Vasopressors

- May need to be used despite the increase in afterload and decrease CO
  - Perfusion is important!
- Although no evidence of clinical efficacy and safety
# Vasopressors

<table>
<thead>
<tr>
<th>Vasopressor / Inotrope</th>
<th>alpha-1</th>
<th>beta-1</th>
<th>beta-2</th>
<th>Dopamine Receptor</th>
<th>Physiologic Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Norepinephrine</td>
<td>+++</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>↑↑ SVR +/- CO</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>+/-</td>
<td>++++</td>
<td>++</td>
<td>0</td>
<td>↑ CO ↓ SVR</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>+++</td>
<td>++++</td>
<td>++</td>
<td>0</td>
<td>↑↑ CO ↓ SVR (low dose) ↑ SVR (high dose)</td>
</tr>
<tr>
<td>Dopamine (mcg/kg/min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 to 3</td>
<td>0</td>
<td>+</td>
<td>0</td>
<td>++</td>
<td>CO ↑ CO ↑ SVR</td>
</tr>
<tr>
<td>5 to 10</td>
<td>+</td>
<td>++</td>
<td>0</td>
<td>++</td>
<td>↑ CO ↑ SVR</td>
</tr>
<tr>
<td>&gt; 10</td>
<td>++</td>
<td>++</td>
<td>0</td>
<td>++</td>
<td>↑↑ SVR</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>+++</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>↑↑ SVR +/- CO</td>
</tr>
</tbody>
</table>

**CO**: Cardiac Output  
**SVR**: Systemic Vascular Resistance  
+++ Strong effect  
++ Moderate Effect  
+ Weak Effect  
0 No effect

Mechanical Support and Heart Transplantation

- Automatic implantable cardioverter defibrillator (AICD)
- Intra-aortic Balloon Pump (IABP)
- Extracorporeal membrane oxygenation (ECMO)
- Ventricular Assist Device (VAD)
- Heart Transplantation

Table 13.1 Terms describing various indications for mechanical circulatory support

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bridge to decision (BTD)</td>
<td>Use of short-term MCS (e.g., ECLS or ECMO) in patients with cardiogenic shock until haemodynamics and end-organ perfusion are stabilized, contra-indications for long-term MCS are excluded (brain damage after resuscitation) and additional therapeutic options including long-term VAD therapy or heart transplant can be evaluated.</td>
</tr>
<tr>
<td>Bridge to bridge (BTB)</td>
<td>Use of MCS (usually LVAD) to improve end-organ function in order to make an ineligible patient eligible for heart transplantation.</td>
</tr>
<tr>
<td>Bridge to candidacy (BTC)</td>
<td>Use of MCS (LVAD or BiVAD) to keep patient alive who is otherwise at high risk of death before transplantation until a donor organ becomes available.</td>
</tr>
<tr>
<td>Bridge to transplantation (BTT)</td>
<td>Use of MCS (typically LVAD) to keep patient alive until cardiac function recovers sufficiently to remove MCS.</td>
</tr>
<tr>
<td>Bridge to recovery (BTR)</td>
<td>Use of MCS (LVAD) as an alternative to transplantation in patients with end-stage HF ineligible for transplantation or long term waiting for heart transplantation.</td>
</tr>
<tr>
<td>Destination therapy (DT)</td>
<td>Long-term use of MCS (LVAD) as an alternative to transplantation in patients with end-stage HF ineligible for transplantation or long term waiting for heart transplantation.</td>
</tr>
</tbody>
</table>

**Biventricular assist device**; **BTB** = bridge to bridge; **BTC** = bridge to candidacy; **BTD** = bridge to decision; **BTR** = bridge to recovery; **BTT** = bridge to transplantation; **DT** = destination therapy; **ECLS** = extracorporeal life support; **ECMO** = extracorporeal membrane oxygenation; **HF** = heart failure; **LVAD** = left ventricular assist device; **MCS** = mechanical circulatory support; **VAD** = ventricular assist device.
Chronic Therapy?— HFpEF

- If Hemodynamically stable continue antihypertensives
- If hypotensive- pressors
- If severely hypertensive – vasodilators


Chronic Therapy? - HFrEF

- Pt must be hemodynamically stable and without any other contraindications (relative or absolute) can be resumed or titrated
  - ACE, ARB, BB, Mineralocorticoid receptor antagonist (MRA)
- Should not be started during hemodynamic instability
- ACE (or ARB) and BB should be started once HD stable and prior to discharge
- MRA can be added before discharge or soon after
AKI in ADHF

- Look for underlying cause
- Medication adjustment
  - Decrease diuretic, ARB/ACE
- Consider renal consult
- Consider dialysis

Discharge Criteria

- High risk of readmission.

Discharge Instructions:
- Sodium restrictive diet
- Discharge Medication Education
- Acuity level
- Close follow up
- Daily Weights
- Plan if things worsen

Colucci WS et al. Treatment of acute decompensated heart failure: General Considerations.
UpToDate. 11.2016-2.2019
Outpatient Therapy for HFrEF:

- ACE or ARB, angiotensin receptor-neprilysin inhibitor (ARNI)
- Beta Blockers
- Mineralocorticoid receptor antagonist
- NO CCB

Colored boxes correspond to COR: red = moderate; green = low.

See text for important note: Hydralazine; green box should be carefully monitored.

Table 5: Drugs Commonly Used for HFrEF (Stage C HF)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Initial Daily Dose(s)</th>
<th>Maximum Dose(s)</th>
<th>Mean Dose Achieved in Clinical Trials</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE inhibitors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5 mg TID</td>
<td>10–20 mg TID</td>
<td>16.6 mg QD</td>
<td>(129)</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>5–10 mg QD</td>
<td>40 mg QD</td>
<td>N/A</td>
<td>---</td>
</tr>
<tr>
<td>Perindopril</td>
<td>2.5–5 mg BID</td>
<td>20–80 mg QD</td>
<td>32.5–35.0 mg QD</td>
<td>(129)</td>
</tr>
<tr>
<td>Quinapril</td>
<td>5 mg BID</td>
<td>20 mg BID</td>
<td>N/A</td>
<td>---</td>
</tr>
<tr>
<td>Ramipril</td>
<td>1.25–2.5 mg QD</td>
<td>10 mg QD</td>
<td>N/A</td>
<td>---</td>
</tr>
</tbody>
</table>

| ARB                 |                       |                 |                                       |            |
| Candesartan        | 4–8 mg QD             | 32 mg QD        | 24 mg QD                              | (137)      |
| Losartan           | 25–50 mg QD           | 100 mg QD       | 120 mg QD                             | (138)      |
| Valsoiran          | 20–40 mg BID          | 160 mg BID      | 234 mg QD                             | (134)      |

| ANP/ARNI           |                       |                 |                                       |            |
| Sacubitril/valsartan| 49.51 mg BID (sustained-release tablet) (therapy may be initiated at 24/26 mg BID) | 97/103 mg BID (sustained-release tablet) | 375 mg QD; target dose: 24/26 mg, 49.51 mg OR, 97/103 mg BID | (138) |

| Beta blocker       |                       |                 |                                       |            |
| Bisoprolol         | 1.25 mg QD            | 10 mg QD        | 8.6 mg QD                             | (140)      |
| Carvedilol         | 3.125 mg QD           | 50 mg QD        | 37 mg QD                              | (181)      |
| Carvedilol CR      | 10 mg QD              | 80 mg QD        | N/A                                   | ---        |
| Metoprolol         | 12.5–25 mg QD         | 200 mg QD       | 139 mg QD                             | (139)      |

Modified (Table 15) from the 2013 HF guidelines (9).
7.3.3. Pharmacological Treatment for Stage C HFrEF: Recommendations

<table>
<thead>
<tr>
<th>Class</th>
<th>LOE</th>
<th>Recommendations</th>
<th>Comment/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>B</td>
<td>Systolic and diastolic blood pressure should be controlled in patients with HFrEF in accordance with published clinical practice guidelines to prevent morbidity and (164, 165).</td>
<td>2013 recommendation remains current.</td>
</tr>
<tr>
<td>IIa</td>
<td>C</td>
<td>Diuretics should be used for relief of symptoms due to volume overload in patients with HFrEF.</td>
<td>2013 recommendation remains current.</td>
</tr>
<tr>
<td>IIa</td>
<td>C</td>
<td>Coronary revascularization is reasonable in patients with CAD in whom symptoms (angina) or demonstrable myocardial ischemia is judged to be having an adverse effect on symptomatic HFrEF despite GDMT.</td>
<td>2013 recommendation remains current.</td>
</tr>
<tr>
<td>IIa</td>
<td>C</td>
<td>Management of AF according to published clinical practice guidelines in patients with HFrEF is reasonable to improve symptomatic HF.</td>
<td>2013 recommendation remains current (Section 9.1 in the 2013 HF guideline).</td>
</tr>
<tr>
<td>IIa</td>
<td>C</td>
<td>The use of beta-blocking agents, ACE inhibitors, and ARBs in patients with hypertension is reasonable to control blood pressure in patients with HFrEF.</td>
<td>2013 recommendation remains current.</td>
</tr>
<tr>
<td>IIb</td>
<td>B-R</td>
<td>In appropriately selected patients with HFrEF (with EF &gt;45%), elevated BNP levels or HF admission within 1 year, estimated glomerular filtration rate (GFR) 30 mL/min, creatinine &gt;2.5 mg/dL, potassium &gt;5.0 mEq/L, aldosterone receptor antagonists might be considered to decrease hospitalizations (83, 166, 167).</td>
<td>NEW: Current recommendation reflects new RCT data.</td>
</tr>
</tbody>
</table>

Mechanistic studies have suggested that mineralocorticoid receptor antagonists can improve measures of diastolic function in patients with HFrEF, possibly by a similar effect on remodeling (83, 168).

The TOPCAT (Treatment of Preserved Cardiac Function Heart Failure With an Aldosterone Antagonist) trial (169) investigated the effects of spironolactone on a combined endpoint of death, aborted cardiac death, and HF hospitalization in patients with HFrEF. A small reduction (HR 0.89) in this composite endpoint did not reach statistical significance, although HF hospitalization was reduced (HR 0.83); known side effects of hyperkalemia and rising creatinine were seen more commonly in the treatment group (166). An unusual amount of regional variation was seen in this trial, prompting a post-hoc analysis (167) that showed that rates of the primary endpoint were 4-fold lower in Russia/Georgia than in North America and South America (the Americas). Rates in the Americas were comparable to those in other HFrEF trials (149, 150). The post-hoc analysis showed efficacy in the Americas (HR 0.83) but not in Russia/Georgia (HR 1.10). Moreover, a sample of the Russia/Georgia population, despite having been in the active treatment arm, had non-detectable levels of
Thank You

failure to launch

To leave the nest, some kids just need a little push.