

Introduction

Heart failure is enormously complex. There are multiple types, manifestations, causes, and treatments. You need to consider the chronic management of a regular heart failure then decide what to do with an acute exacerbation.

Types of Failure

The first consideration to understand is **systolic** vs **diastolic**. **Systolic** failure arises when the heart can't push blood forward. It can go backwards (a **leaky** heart), be **floppy** (dilated cardiomyopathy), or be **dead** (secondary to myocardial ischemia). Plain and simple - systolic failure is a broken pump. The heart fills in diastole, hence, **diastolic** failure is when the heart **can't fill**. If something prevents the heart from relaxing and accepting blood it produces a diastolic failure. This might be from hypertrophy or infiltration.

The second consideration is left versus right failure. **Left Ventricular Failure** is a failure to pump blood into the periphery; there's a backup of blood into the lungs. **Right Ventricular Failure** causes a backup of blood into the venous circulation. Most of the time it's a combination of both.

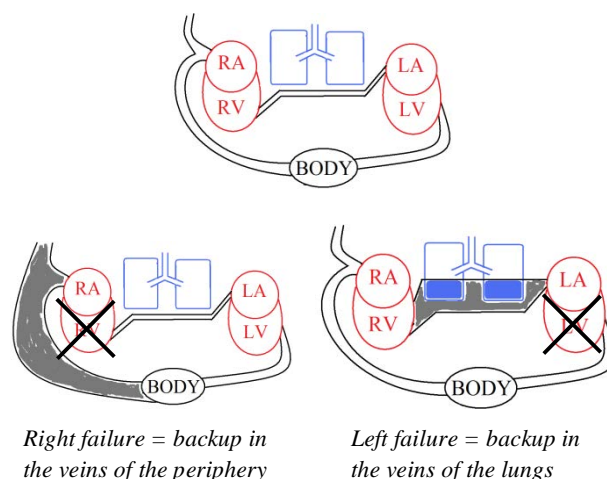
Pathogenesis and Etiology

The typical chronic failure that occurs insidiously is by far the most common. It's caused by **hypertension**. High blood pressure causes an increase in systemic vascular resistance; the heart has to pump harder and harder to push the blood. It gets bigger and beefier to compensate. But just like any muscle, it putters out and eventually fails. The heart gets bigger, rounder, and eventually goes floppy. Pathologically, constant overstimulation by **catecholamines** first helps the heart overcome the hypertension. It eventually leads to **neural hormonal remodeling**, **cardiac toxicity**, and then **fibrosis**. Other etiologies are simply a matter of memorization.

Diastolic CHF (CHF with preserved ejection fraction) is caused by the things that prevent relaxation. Generally, it's a hypertrophic or restrictive cardiomyopathy. Pericardial disease and deposition disease can do it too.

Symptomatology

Symptoms arise from where the fluid backs up. The classic patient is the triad of Exertional Dyspnea, Orthopnea, and Paroxysmal Nocturnal Dyspnea. **Exertional dyspnea** is shortness of breath limiting walking. **Orthopnea** is shortness of breath that's worse when lying flat. **Paroxysmal Nocturnal Dyspnea** is when the patient wakes up in the middle of the night gasping for breath. Because most patients have left and right failure together, **rales** (fluid on the lungs) may get mixed with **peripheral edema** and **hepatomegaly**. Symptoms like an **S3 heart sound** and **Jugular Venous Distension** are signs of acute exacerbation. In the chronic setting, it's critical to determine what **class** they are. Here, we use NYHA, as it directs treatment.



Failure	Path	Etiology	EF
Systolic Failure	Forward failure	Leaky valves = any regurgitation Dead Heart = Ischemia / infarction Floppy muscles = EtOH, HTN, Drug	↓
Diastolic Failure	Filling failure	Pericardium = Pericardial Tamponade Constrictive Pericarditis Cardiomyopathy = Restrictive Hypertrophic	↑

Symptoms

Left Ventricular Failure	Right Ventricular Failure
Orthopnea, Crackles, Rales	Hepatosplenomegaly, JVD
Dyspnea on Exertion, S3 ,	Peripheral Edema,
Paroxysmal Nocturnal Dyspnea	Dyspnea on Exertion, ↑JVP

S3 and JVD poor prognostic sign in acute exacerbation

Chronic NYHA Class

	Ø Limited	Ø Symptoms
I	Ø Limited	Comfortable at rest and walking
II	Slight Limitations	Comfortable at rest only
III	Moderate Limitations	Bed bound, sx's @ rest
IV	Totally Limited	

The ACC/AHA has a class A-D, based on the presence of structural heart disease. Don't use the A-D model, use I-IV

Diagnosis

When first attempting to diagnose CHF there are two tests that should be used. The **BNP** is useful to say, “volume overload or not.” It’s a blood test and requires no advanced training to interpret. The standard test is the **2D echocardiogram**, which can distinguish between systolic failure (ejection fraction <55%) and diastolic failure (preserved ejection fraction). There are more definitive tests available. A **nuclear study** calculates the exact ejection fraction and identifies areas of ischemia (it’s a stress test). **Left Heart Catheterization** (even more definitive of EF and coronary artery disease) can be performed with a right heart cath to demonstrate elevated pulmonary artery pressures. ECG (demonstrates old ischemia / arrhythmia), CXR (demonstrates cardiomegaly or pulmonary edema), and troponins (acute ischemia) aren’t inappropriate, but they’re also not necessary.

Treatment with Reasoning

There are two goals: reduce fluid (preload) and reduce afterload. To reduce fluid, it’s important to restrict salt intake (< **2g/day** of NaCl) and reduce fluid intake (< **2L H2O/day**). Everybody gets this. Once the patient reaches class II, keep the fluid off by using diuretics like **furosemide**. At class III, **Isosorbide Dinitrate** is added.

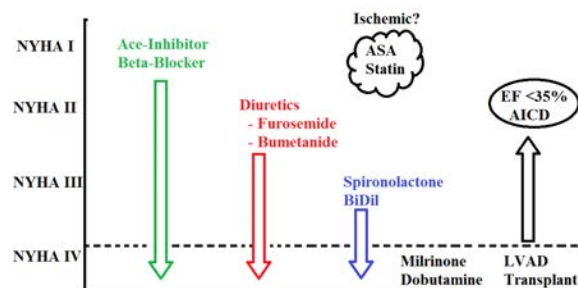
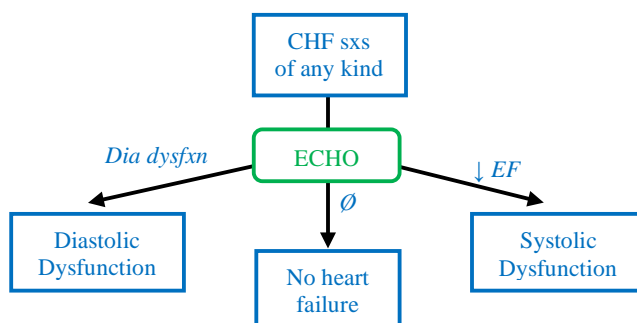
Afterload reduction is achieved with **ACE-inhibitors** (also Angiotensin Receptor Blockers). When CHF gets really bad (Class III and greater), add **Spironolactone** or **Hydralazine**. Isosorbide Dinitrate (preload) and Hydralazine (afterload) are given as a combination medication BiDil®.

When the situation is dire (class IV) it’s time to add **inotropes** like **Dobutamine** (which is a continuous infusion) while preparing for a **transplant** or **ventricular assist device** bridging them to transplant. Ambulatory infusion devices are available.

To reduce the risk of sudden cardiac death, **Beta-blockers** are used to reduce arrhythmia and neuro-hormonal remodeling. Other considerations are the placement of an **AICD** if the **EF < 35%** and they’re NOT class IV. **Digoxin** can be used if there’s need of symptom relief (knowing it won’t change mortality).

Acute Exacerbation

The precipitant of a CHF exacerbation (which usually means **volume overload**) can be a product of **dietary noncompliance**, **medication noncompliance**, blood pressure control, **ischemia** or **arrhythmia**. The goal is the same as for chronic management: afterload reduction (aka blood pressure control) and preload reduction (diuresis and nitrates). Ruling out acute ischemia (which should be treated as an MI) and other causes of dyspnea is important. But the person who is overtly overloaded (**JVD**, **crackles**, **peripheral edema**) with an **elevated BNP** needs aggressive diuresis with **IV Furosemide** and blood pressure control. Never start or increase a Beta-Blocker during an exacerbation.



Patient	Treatment
Everybody	Salt <2g per day H ₂ O < 2L per day ACE-i or ARB (best mortality benefit) Beta-Blocker
Preload Reduction	Diuretics such as Furosemide Nitrates such as Isosorbide Dinitrate Dietary Modifications (NaCl, H ₂ O)
Afterload Reduction	ACE-i or ARB Hydralazine Spironolactone

Special	Treatment
EF < 35%	AICD (must be Class I-III)
Ischemic	ASA and Statin
Class IV	Inotropes like Dobutamine (ICU) VAD bridge to transplant Transplant

