



ACS/Heart Failure Evaluation & Management Overview

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Goals/Objectives

- Evaluation of patient in the ED/Hospital setting
 - Presenting symptoms, diagnostic testing
 - Triaging to appropriate level of care
 - Outpatient management not covered (ie stable angina/CAD, NYHA Class 1-2 for CHF)
- Management of patient - inpatient setting
 - Treatment while awaiting specialist consultation (if applicable)



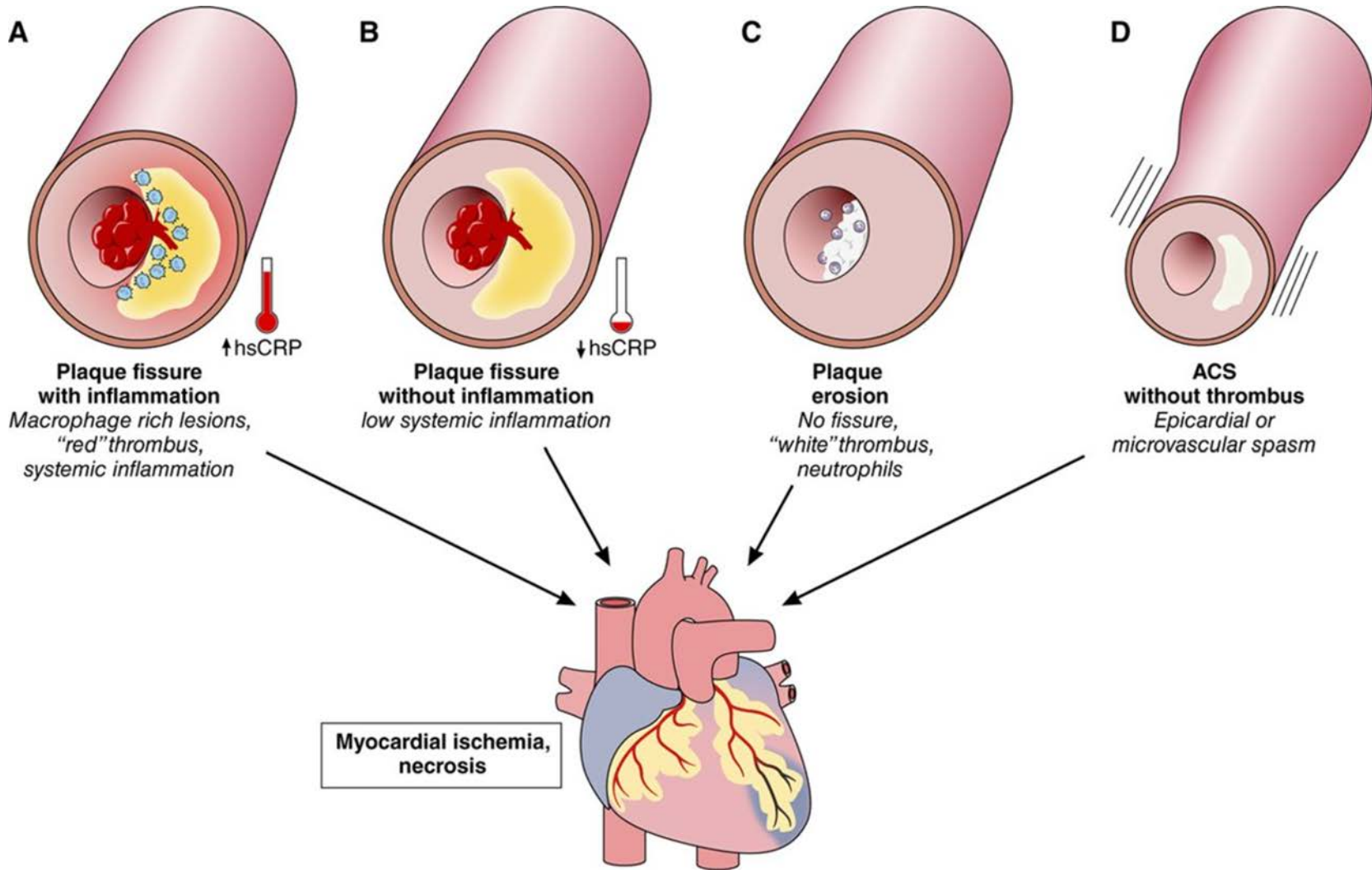
Acute Coronary Syndrome

Definitions

- Stable Angina - transient myocardial ischemia chronically
 - Stenosis and myocardial O₂ supply fixed
 - Ischemia precipitated by increase demand
- Acute Coronary Syndrome - clinical spectrum of presentations related to **acute** myocardial infarction/ischemia due to reduction of coronary blood flow
 - Unstable Angina
 - Non-ST Elevation Myocardial Infarction (NSTEMI)
 - ST Elevation Myocardial Infarction (STEMI)

Etiology/Pathophysiology

- Intracoronary Thrombus Formation
 - Plaque Rupture (most common) - usually STEMI
 - Plaque Erosion (second most common) - frequently NSTEMI
 - Calcified Nodule (rare, but sometimes in older patients)
- Nonthrombotic Etiologies (rare)
 - Vasospasm
 - Coronary arteritis
 - Spontaneous Coronary Artery Dissection
 - Acute intraplaque hemorrhage without thrombosis)



Symptoms

- New onset pressure-like chest pain that occurs at rest or with minimal activity
 - Retrosternal and can radiate to arm , neck or jaw(Likelihood Ratio 2.7)
 - Associated with diaphoresis (LR 2.0)
 - Associated with dyspnea or nausea (LR 1.9)
- ⅓ of ACS patients may not present with chest pain
 - Atypical Symptoms - dyspnea, nausea, weakness, epigastric discomfort/pain
 - Caution in older patients, female, patients with Diabetes to consider ACS
- Less likely Myocardial Ischemia
 - Pleuritic pain (LR 0.2)
 - Sharp, stabbing pain localized to a single location (LR 0.3)
 - Pain reproduced by palpation (LR 0.2-0.4)

Differential Diagnosis

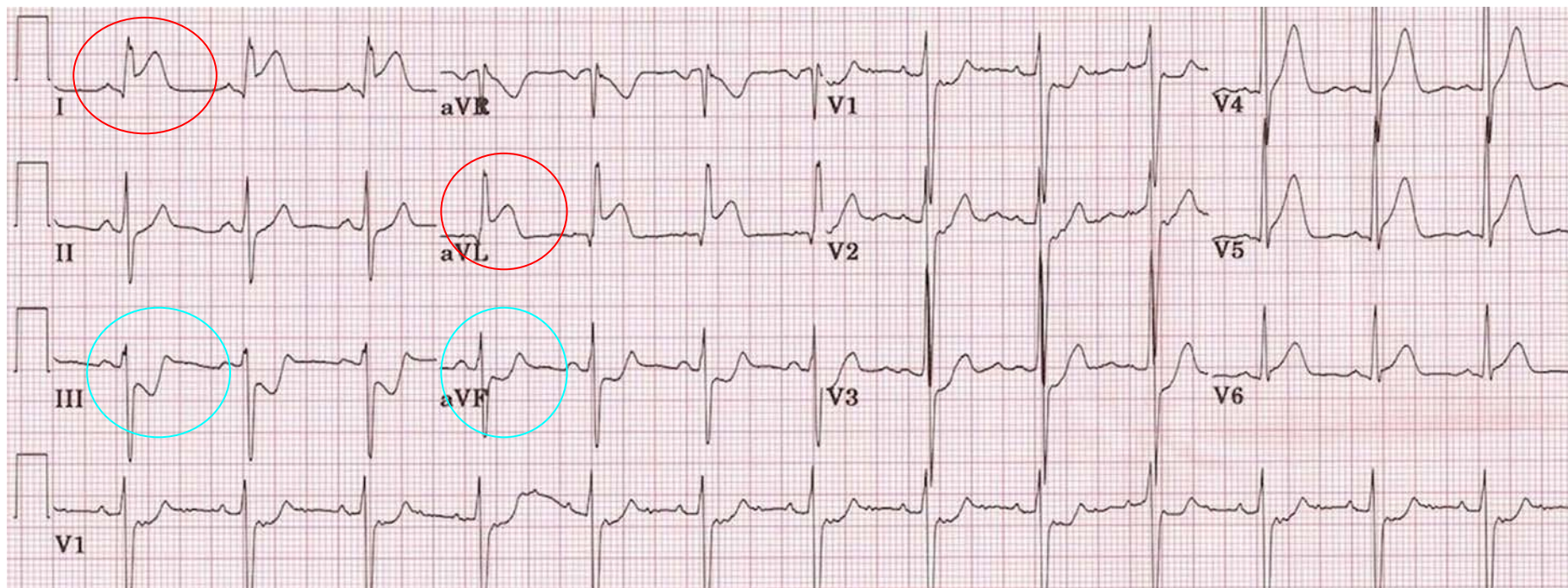
- Cardiac
 - **Aortic Dissection, Expanding Aneurysm**, Myocarditis, Pericarditis
- Pulmonary
 - COPD, **Pneumothorax, Pulmonary Embolism**, Pleuritis
- GI
 - GERD, Esophageal Spasm, **Esophageal Rupture**, Peptic Ulcer, Pancreatitis
- Musculoskeletal
 - Costochondritis, Trauma, Cervical Disc Radiculopathy
- Miscellaneous
 - Herpes Zoster, Sickle Cell Crisis, Depression/Anxiety, Drug Intoxication, Pheochromocytoma

Diagnosis

- EKG
 - Usually obtained with onset of suspicious symptoms (remember atypical symptoms as well in specific populations)
 - Serial EKGs q15-30 min if patient persistently symptomatic due to evolving EKG changes secondary to ischemia
 - ST Elevation - new ST elevation in at least 2 anatomically contiguous leads with ≥ 1 mm in all leads other than V2 to V3. For V2 to V3, elevation must be ≥ 2 mm in men ≥ 40 , ≥ 2.5 mm in men < 40 , ≥ 1.5 mm in women
 - NSTEMI - typically ST segment depression but only occur in approximately $\frac{1}{3}$ of patients. T wave changes more common but less specific unless they are new a deep T wave inversions (≥ 3 mm)

Diagnosis

- EKG (cont'd)
 - Left Bundle Branch Block/Paced rhythms particularly difficult to interpret due to alteration of ventricular depolarization producing secondary ST-T wave changes (QRS Prolongation, T Wave inversion, ST Depression)
 - Sgarbossa criteria is a scoring system used in these cases with score ≥ 3 specific but not sensitive
 - ST Elevation ≥ 1 mm concordant with QRS complex (5)
 - ST Depression ≥ 1 mm from V1-V3 (3)
 - ST Elevation ≥ 5 mm that is discordant with QRS (2)



Diagnosis (Cont'd)

- Cardiac Enzymes measure myocardial injury/necrosis in plasma
 - If enzymes are present then NSTEMI, if not unstable angina
- Older biomarkers such as CK-MB aren't used anymore - not very specific
- Troponin I and T much more sensitive than CK-MB
- Troponin above 99th percentile defines acute myocardial injury
- Troponin concentrations usually rise 2-3 hrs after acute MI
- Usually measure troponins to observe rise and then fall in NSTEMI
- Currently High sensitivity troponins are usually in place these days which reduces frequency of checks to 3 hrs instead 5-6
- High sensitivity troponins also making Unstable Angina less apparent

Management - STEMI with PCI on site

- Usually Cath Lab Activated, cardiology stat consult
- Critical for Revascularization within 90 minutes of first medical contact if PCI is available
- ASA 325 mg
- Loading dose of oral P2Y₁₂ antagonist - Prasugrel 60 mg, Ticagrelor 180 mg preferred over Clopidogrel 600 mg undergoing emergent PCI - PLATO trial
 - In the rare case patient needs a CABG, would delay surgery depending on surgical team comfort with antiplatelet loading dose
- Heparin anticoagulation to prevent extension of thrombus (50-70 units/kg max 5000 bolus), then continue heparin gtt until PCI
- LMWH also possible but less preferable due to longer acting time and renal limitations

Management - STEMI w/o PCI onsite

- If no PCI Available, should still be transferred to PCI site if able to transport patient with first medical contact to device time under 120 minutes
- If above goal cannot be met, then will need fibrinolytic therapy within 30 minutes of patient arrival unless contraindicated
 - Absolute contraindications - Prior Intracranial Hemorrhage, malignant intracranial neoplasm, structural cerebral vascular lesions, ischemic stroke < 3 mo, suspected aortic dissection, active bleeding, closed head/facial trauma < 3 mo, Intracranial/Intraspinal surgery < 2 mo, Severe uncontrolled HTN
 - Relative contraindications - Significant HTN, Ischemic Stroke < 30 mo, Dementia, Major surgery < 3 weeks, Pregnancy, Oral Anticoagulant therapy
 - If symptoms > 4 hours, high bleeding risk, cardiogenic shock then should be transported to PCI center regardless of delays
- Alteplase, tenecteplase preferred over non-fibrin agents (streptokinase)
- Should still receive ASA 325, clopidogrel 300 mg, Heparin bolus/infusion and then still transferred to PCI facility for monitoring after fibrinolytic therapy

Management - NSTEMI

- ASA 325 mg for all comers, P2Y12 antagonist (antiplatelet) - Ticagrelor 180 mg LD followed by 90 mg BID or Clopidogrel 600 mg LD followed by 75 daily
 - Avoid Prasugrel if prior to cath due to increased bleeding (ACCOST trial)
- Anticoagulation - usual choices Unfractionated Heparin or LMWH
- Avoid Morphine if possible as it can mask symptoms, O2 only if hypoxemic
- Caution with nitroglycerin with Right ventricular infarctions or PDE5 use (Viagra)
- If patient is hemodynamically unstable from cardiogenic shock, refractory angina, electrical instability (Vfib/Vtach), then patient should undergo angiography within 2 hours
- Otherwise risk stratification will determine early invasive vs ischemia guided
- TIMI Score (or GRACE) is one scoring system used to risk stratify
 - Age > 65, >= 3 CAD risk factors, Known CAD, ASA use within past week, Severe Angina, EKG ST Changes >= 0.5 mm, Positive Cardiac enzyme

Management - NSTEMI Early Invasive

- Angiography with 72 hours of admission if intermediate or high risk
- Intermediate or High Risk ACS defined by one or more of the following
 - Recurrent angina at rest with low level of activity
 - Elevated troponin, New ST Depression, Signs/symptoms of heart failure
 - High risk findings from noninvasive testing
 - Hemodynamic instability
 - Sustained Vtach (> 30 s)
 - PCI within 6 months
 - TIMI score ≥ 3
 - Newly reduced LVEF < 40%

Management - NSTEMI/UA Ischemia Guided

- Strategy for Low Risk Patients
 - TIMI Score ≤ 2
 - Unstable Angina - no elevated troponins
- Angiography can be avoided unless patient experiences refractory angina, hemodynamic instability or objective evidence of severe ischemia
- Ischemia Guided patients should STILL undergo noninvasive testing

Late Hospital Care & Discharge

- All patients should be on ASA 81mg indefinitely
- All ACS patients should receive a P2Y12 antagonist for at least 1 year - clopidogrel, prasugrel, or ticagrelor for stented patients otherwise clopidogrel/ticagrelor
- Beta Blockers should be administered within 24 hrs and continued at discharge unless contraindicated or unable to tolerate
 - If contraindicated due to asthma exacerbation, nondihydropyridine calcium channel blockers can be substituted
- ACEi/ARB for patients with LVEF < 40% or CKD/Diabetes
- Aldosterone Antagonists for LVEF < 40% for decreased mortality
- High Intensity Statin regardless of LDL
- All patients should be referred to cardiac rehab
- Counseling regarding diet/exercise, smoking cessation



Congestive Heart Failure

Heart Failure - Overview/Epidemiology

- Heterogeneous spectrum of pathologies
- Increasing Incidence of ~870000 new diagnosis of Heart Failure annually
- In US, responsible for ~500000 ED visits, 1 million hospital admissions
 - 25% admitted within 1 month, 50% within 6 months
- Mortality is high - ~50% 5 year survival when diagnosed

Heart Failure - Pathophysiology

- Inciting events that progressively diminishes ability of ventricles to fill or eject blood to maintain metabolic demands
- HFrEF - LVEF < 40% - Low cardiac output initiates neurohormonal sympathetic activation (Renin-Angiotensin-Aldosterone system) leading to salt and water retention.
 - Short term compensatory mechanisms but long term consequences to remodeling leading to Heart Failure
- HFpEF - LVEF $\geq$ 50% - Less understood, possibly due to HTN
- LVEF 40-50% - unclear classification

Heart Failure - Etiologies

- HFrEF
 - CAD (most common), HTN, Valvular Disease, Chronic lung disease, Nonischemic Dilated Cardiomyopathy (Familial, Genetic, Infiltrative), Toxic/Drug Induced, Viral, Disorders of Rate/Rhythm (Tachyarrhythmias, Bradyarrhythmias), Chagas Disease
- HFpEF
 - CAD, HTN, Infiltrative (Amyloidosis, sarcoidosis), Hemochromatosis, Fibrosis, Aging
- High Output States - Thyrotoxicosis, Chronic Anemia, Nutritional Disorders (Beriberi), Systemic A/V shunting
- Considerable Overlap between certain etiologies

Congestive Heart Failure - Clinical Presentation

- Now called Acute Decompensated Heart Failure
- Cardinal Symptoms are Fatigue and Dyspnea
- Orthopnea, Paroxysmal Dyspnea, Peripheral Edema, GI Symptoms of Early Satiety, Abdominal Fullness, Nausea, Ascites leading to liver congestion - RUQ Pain
- Physical Examination - JVD, Rales, Displaced PMI if cardiomegaly, S3 Heart Sound indicating volume overload,, Hepatomegaly
- DDx of possible Heart Failure mimickers - MI, pulmonary disease, Hepatic/Renal Failure, Venous Insufficiency, Pericardial disease, Obesity/Deconditioning
- Clinical Diagnosis with adjunct imaging, labs
 - Chest X-ray - cardiomegaly, pulmonary edema
 - BNP - elevated for new diagnosis, elevated over baseline for prior dx of HF
 - Other etiologies for elevated BNP, elderly patients have increased BNP
- Echocardiogram

NYHA Heart Failure Classification (Class I-IV)

J. Larry Jameson, Anthony S. Fauci, Dennis L. Kasper, Stephen L. Hauser, Dan L. Longo, Joseph Loscalzo+

TABLE 252-2 New York Heart Association Classification

Functional Capacity	Objective Assessment
Class I	Patients with cardiac disease but without resulting limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitations, dyspnea, or anginal pain.
Class II	Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.
Class III	Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary activity causes fatigue, palpitation, dyspnea, or anginal pain.
Class IV	Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of heart failure or the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.

Source: Adapted from New York Heart Association, Inc., *Diseases of the Heart and Blood Vessels: Nomenclature and Criteria for Diagnosis*, 6th ed. Boston, Little Brown, 1964, p. 114.

Acute Decompensated Heart Failure - Triage

- Determination of Volume status and cardiac output based on physical examination
 - 4 states in a 2 x 2 grid
 - Volume - Wet vs Dry
 - Cardiac Output - Warm vs Cold
- Important for determining level of care - ICU vs Floor
 - If Cold and Wet - usually needs Inotropic agents such as dobutamine, milnorone, dopamine for cardiac output support - ICU

**Evidence for congestion
(elevated filling pressure)**

Orthopnea
High jugular venous pressure
Increasing S₃
Loud P₂
Edema
Ascites
Rales (uncommon)
Abdominojugular reflux
Valsalva square wave

Evidence for low perfusion

Narrow pulse pressure
Pulses abberations
Cool forearms and legs
May be sleepy, obtunded
ACE inhibitor-related
Symptomatic hypotension
Declining serum sodium level
Worsening renal function

		Congestion at Rest?	
Low perfusion at rest?	No	No Warm and Dry A	No Warm and Wet B
	Yes	Cold and Dry L	Cold and Wet C

Diagram indicating 2 x 2 table of hemodynamic profiles for patients presenting with heart failure. Most patients can be classified in a 2-min bedside assessment according to the signs and symptoms shown although in practice some patients may be on the border between the warm-and-wet and cold-and-wet profiles. This classification helps guide initial therapy and prognosis for patients presenting with advanced heart failure. Although most patients presenting with hypoperfusion also have elevated filling pressure (cold and wet profile), many patients present with elevated filling pressures without major reduction in perfusion (warm and wet profile). Patients presenting with symptoms of heart failure at rest or minimal exertion without clinical evidence of elevated filling pressures or hypoperfusion (warm and dry profile) should be carefully evaluated to determine whether their symptoms result from heart failure.

Acute Decompensated Heart Failure - Causes

- Usually an inciting event to cause decompensation
- Important to rule out and address
 - Acute Coronary Syndrome
 - Uncontrolled HTN
 - Uncontrolled Arrhythmias
 - Valvular Heart Disease
 - Myocarditis
 - Infections
 - Medical/Dietary noncompliance
 - Drug or EtOH Abuse

Acute Decompensated Heart Failure - Treatment

- Usually Wet & Warm are admitted to Floor, Wet & Cold usually are admitted to ICU with inotropic support if HFrEF
- Volume Management
 - IV Diuretics - usually double diuretic home dose and can be augmented with thiazide diuretics
 - Studies have shown that no significant difference between IV drip but some specialists prefer drip
 - Strict Input/Output
 - Fluid Restriction
 - Low Dietary Salt
- Decrease Cardiac Demand
 - Beta Blockers as tolerated - not to be used if approaching cardiogenic shock/hypotensive
 - Shown to decrease mortality
 - Digoxin can be used but caution with elderly due to narrow therapeutic range
 - Afterload reduction with ACEi/ARB, nitrates

HFrEF - Specific Treatment

- Aldosterone receptor antagonists shown to reduce mortality and hospitalizations
 - RALES Trial (1999) - spironolactone, eplerenone also acceptable
 - HFpEF - TOPCAT (2014) no benefit in mortality but small secondary benefit for reduction in hospitalizations
- Intracardiac Defibrillator Placement
 - Primary Prevention to reduce mortality
 - Ischemic (up to 40 days post MI)
 - Non-ischemic - LVEF $\leq$ 35% with NYHA Class II/III Symptoms (9 months)
 - Secondary Prevention - History of Vfib, cardiac arrest, unstable Vtach
- Biventricular pacemaker (Cardiac Resynchronization therapy)
 - LVEF $\leq$ 35%, sinus, class II/III/IV despite medical therapy, QRS $>$ 150 ms

Acute Decompensated Heart Failure - Care Transition

- Ensuring patient is at optimal volume status, resolution of symptoms, tolerant of PO medications to ensure compliance
- Detail Discharge instructions regarding medication dosing, dietary restrictions, scales to measure daily weights, symptom recognition such as fluid overload, increased O2 usage
- Referral to cardiologist outpatient and/or Heart Failure Clinic for volume management
- Close follow up with PCP within 1 week usually with electrolyte labs
- Cardiac Rehabilitation and exercise as tolerated

References

- 1) McKean SC, Ross JJ, Dressler DD, Scheurer DB. eds. *Principles and Practice of Hospital Medicine*, 2e. McGraw Hill; 2017.
- 2) Jameson J, Fauci AS, Kasper DL, Hauser SL, Longo DL, Loscalzo J. eds. *Harrison's Principles of Internal Medicine*, 20e. McGraw Hill; 2018.
- 3) Uptodate - Initial Evaluation & Management of Suspected Acute coronary Syndrome