Heart Failure

CARDIAC OUTPUT

- Cardiac output = stroke volume x heart rate
- Depends on:
  - **Preload** (volume)
  - **Afterload** (resistance)
  - **Myocardia contractility** (UMPH)
    - → **Digoxin** can increase contractility
  - **Heart rate** (speed)
    - → **Beta blockers** can help slow down heart rate
- Drug target the above 4 factors

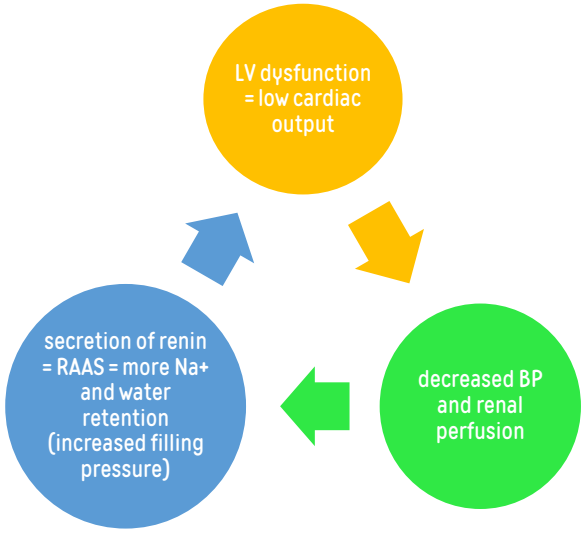
RISK FACTORS

- HTN
- Heart problems
  - valve disorders
  - CAD
  - **MI**; ischemic heart disease in the number one cause
    - → Chronically clogged coronary arteries lead to heart ischemia → heart has to work harder → HF
  - Structural disorders
- Lifestyle –alcohol, smoking, diet
  - Alcohol can poison muscle fibers
- **Pulmonary disorders**
- Other co-morbidities

POPULATION AT RISK

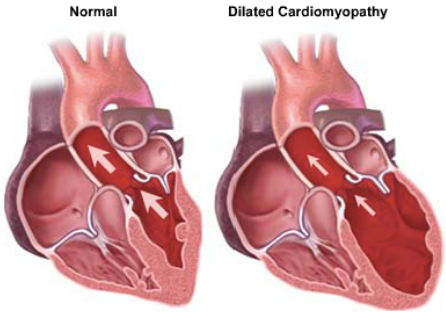
- Pts with low **socioeconomic status**
- Geriatric populations
  - s/s recognition is low due to pts thinking it's part of the aging process
  - Elders pts have harder time detecting SOB
  - Common s/s is **mental state alteration** and **confusion**

VICIOUS CYCLE OF CONGESTIVE HEART FAILURE

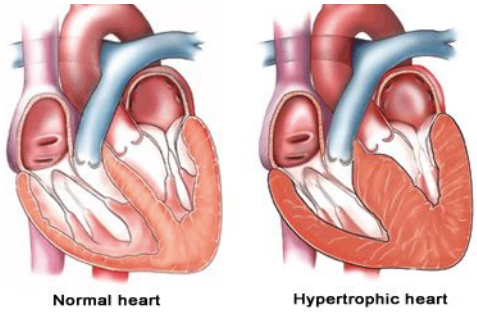


CARDIOMYOPATHY

- Dilated: inflammation and degeneration of **myocardial fibers**
  - Left ventricular dilation and atrial enlargement → contractile dysfunction
    - → **Stasis of blood** in left ventricle
    - → Impaired **systolic function**
  - Risk of **sudden cardiac death** (SCD) from lethal **dysrhythmias**
    - → **Dysrhythmias** develop due to impaired conduction

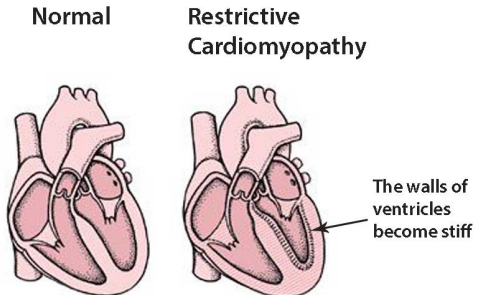


- Hypertrophic: asymmetric ventricular hypertrophy **without ventricular dilation**



- Impaired **diastolic function** → impaired ventricular **filling**
- Risk of **SCD** (esp. in young adults that are healthy)
  - → They **septum** hypertrophies and bulges out to the left ventricle → ventricles have to work harder to push the septum out of the way to eject blood to aorta → **heart needs more blood to generate the pressure and force**
    - ✓ When pt engages in exercise, heart beats faster → not enough filling time (diastole) → **heart doesn't have enough blood to generate pressure → muscle blocks the passage to aorta → HF**
- Because heart is bigger, it needs more O2 supply → worsening of HF

- Restrictive: systolic function is unchanged



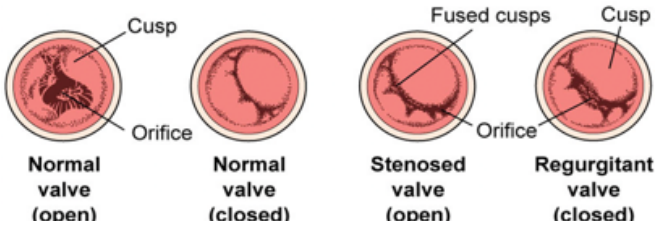
- Least common
- Heart has connective tissue growing continuously in the ventricles → heart can't relax enough to receive blood
- Impaired **diastolic filling and stretch**

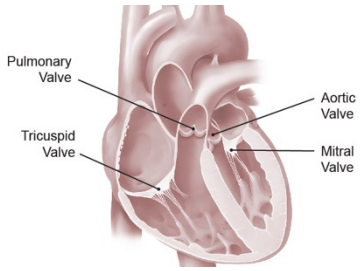
TYPES OF HF

Left	Right
- Most common - LV failure → **blood backs up into left atrium and into pulmonary veins** **LUNG ISSUE** - Pulmonary edema - Pulmonary crackles - SOB	- Primary cause is **left-sided failure** - RV fails → blood backs up into the right atrium and venous circulation **PERIPHERY ISSUE** - JVD - Blood backs up in systemic areas and organs (**hepatomegaly → RUQ tenderness)** - Ascites (if liver damage is severe)
Can also cause **dizziness** (low brain perfusion) and **low urine output** (low kidney perfusion)	

Systolic	Diastolic
- **Def:** impaired left ventricular **contractility** - Decreased **ejection fraction;** normal range is 55-75%; anything below **40% = HF** - **Decreased CO**	- Ventricle is normal size but hypertrophy → loss of left ventricular **diastolic relaxation (filling)** → filling of the ventricles is impaired - Ejection fraction is **normal**

VALVULAR DISORDERS





- You can have both stenosis and regurgitant valves (common)
- Body does a good job in adapting to faulty valves by contracting faster and stronger to maintain normal CO → **hypertrophy**
- **Stenosis** is when valve has a too narrow opening and **regurgitation** is when valve doesn't close all the way

Mitral Valve Disorders

- Left-sided valve, so s/s related to **decreased CO**
  - Fatigue, dyspnea with exertion, weakness, syncope
  - S/s will progress to **right sided HF as well** since blood will eventually back up to the right side (after lungs)

- More common that right valvular diseases due to more pressure in left heart
- 3 types:
    - **Mitral valve prolapse**: valve leaflets buckle back into left atrium
        - → Most common is US
        - → Usually asymptomatic and is **genetic**
        - → **S/s**: pain, palpitation, panic disorders, etc.
        - → You may be able to hear a **clicking nose** when the valve balloons up into the left atrium
    - **Mitral valve stenosis**: adhesion of valve cause thickening of valve fissure
        - → Commonly caused by **rheumatic heart disease**
    - **Mitral valve regurgitation**
        - → Common causes: MI, rheumatic heart disease, mitral valve prolapse, infective endocarditis
            - ✓ MI: if the papillary muscles are killed during episode
        - → **Left chambers must work harder to pump blood out → hypertrophy of heart**

Aortic Valve Disorders

- Left sided valvular disorder, so **decreased CO**
- Stenosis vs regurgitation
    - Stenosis: fusion of commissures and **calcification** of valves → leaflets stiffen
        - → Usually due to **aging**
        - → Obstructs BF **out from left ventricle to aorta**
        - → Causes ventricular **hypertrophy → pulmonary HTN *(blood backs up into lungs)* → HF**
    - Regurgitation: incomplete closure of valve
        - → Causes backflow of blood from aorta to left ventricles → **LV is overloaded → hypertrophy → HF**
- Both stenosis and regurgitation will eventually lead to **LSHF**

Tricuspid and Pulmonic Valve Disorders

- Not very common; usually stenosis (regurgitation is very rare)
- Right sided valve damage causes **right sided heart failure s/s**
- Tricuspid vs pulmonic stenosis
    - **Tricuspid stenosis** –almost always caused by **rheumatic fever** or **IVDA**
        - → Growing instances due to IVDA
        - → Can also lead to **regurgitation**
    - **Pulmonic stenosis** –almost always a **congenital defect**
- Murmurs
    - Pulmonic valve regurgitation
        - → You'll hear a murmur during diastole since valve will not **close** properly
    - Mitral stenosis
        - → Murmur during diastole since the valve won't **open** properly

CHRONIC VS ACUTE HEART FAILURE

Chronic	Acute
- Progressive worsening of ventricular function - Chronic **neurohormonal activity causing remodeling of myocardium** - S/s depends on the degree of damage, age, etc. - Irreversible unless transplant is done - Goal is to **slow down or halt** the process	- Severe **LV impairment** - Can be either a **rapid onset** or **progression from chronic HF** - Most serious complication is **pulmonary edema**

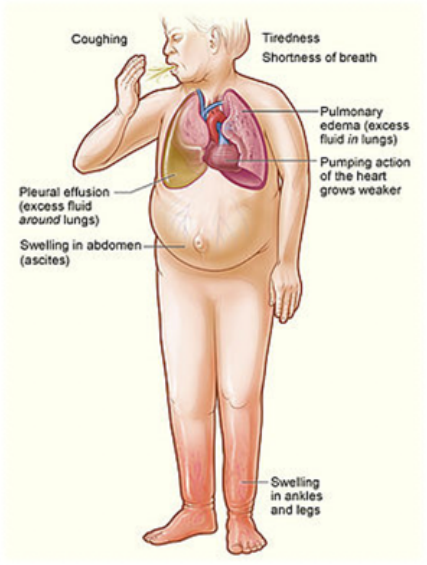
- Decompensation happens when pt with chronic HF can no longer regulate HF due to stressors (ex: high Na+ intake during holidays)

Chronic HF

- Manifestations
    - **Early signs**: increased HR and RR (slightly)
    - Fatigue, dyspnea, orthopnea (very non-specific)

→ Paroxysmal nocturnal dyspnea

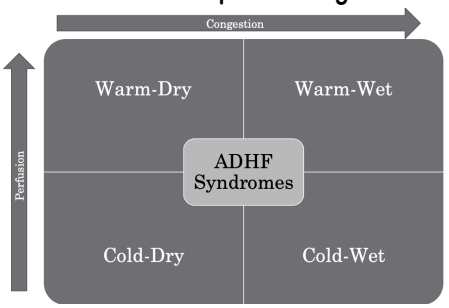
- **Persistent coughing** (dry, unrelieved by position changing or over the counter meds)
- Dependent edema
- **Nocturia** (due to blood being pooled during day but when pt lies flat, fluid goes back to heart more easily → more blood can perfuse kidneys)
- **Late signs:**
  - → Dusky, cool, and damp skin
  - → Confusion and decreased memory
  - → Weight changes (fluid retention but can have decreased appetite)
    - ✓ May suggest **smaller meals or give shakes** – pay attention to fluid overload with shakes
- **S4 and S4 sounds**



- Classification/grades of severity of HF
  1) Class 1: mild; no limitation of physical activity
  2) Class 2: mild; slight limitation of physical activity (fatigue, palpitation, or dyspnea)
  3) Class 3: moderate; **marked limitation** of physical activity (ex: pt gets SOB with just walking to bathroom)
  4) Class 4: severe; unable to do any physical activity without discomfort; **symptoms of cardiac insufficiency at rest**

Acute Decompensated HF

- 3 phases:
  1) Early: increased **pulmonary venous pressure**
     - → Causes increased RR
     - → Decreased O2 sat (but PCO2 level may be fairly normal since CO2 diffuses more easily than O2)
  2) Later: interstitial **edema**
     - → Tachypnea
  3) Further progression: **alveolar edema**
     - → Respiratory **acidemia** –at this point pt is no longer able to blow off CO2
- Pulmonary Edema S/s
  - Anxiety
  - Pale, cyanotic
  - Cool and clammy skin
  - Dyspnea, orthopnea, tachypnea
  - Use of **accessory muscles**
  - Coughing with **frothy, blood-tinged sputum** –remember that this is **different from dry cough from chronic HF**; indication that HF is **progressing**
  - Crackles and wheezes
  - Tachycardia (SNS s/s)
  - Hypotension or hypertension
  - **Abnormal S3 and S4**
- ADHF syndromes
  - We don't want **cold or wet**
  - Warm= good perfusion
  - Dry = no congestion



Pretty rare but means that lungs are congested AND low perfusion (severe)

DIAGNOSING HEART FAILURE

Because s/s are not specific for different valves, may need different screening

- **BNP** –most important biomarker that helps distinguish between resp. vs cardiac issue

    - **Mechanism**: pressure of the heart → stretching of the heart wall → release of BNP → body try to get rid of fluid
- CBC, BMP, cardiac markers, liver function test (LFT), RFP, thyroid, lipid check
    - Cardiac markers can detect signs of myocardial ischemia; **troponin** can usually increase a little due to **stressors**
    - LFT can check for **hepatomegaly**
- Exercise stress test
- **Echocardiogram**
- Cardiac catherization
- CXR –can visualize **hypertrophy/cardiomegaly**
- 12 lead ECG –can detect **ventricular hypertrophy** and **dysrhythmia** since HF are higher risks
    - **Atrial fib**: atrium is not contracting → can't squeeze the remaining blood into ventricles → decrease of EF; usually asymptomatic for normal ppl, **but ppt with HF can worsen condition due to more EF decrease**
- Electrolyte monitoring (esp. **K+)**
    - Esp. if pt takes other meds like **ACE-I**
    - **Digoxin** can exacerbate **hypokalemia**

COLLABORATIVE CARE FOR CHRONIC HF

- Treat underlying cause
- Education
    - Fluid restriction teaching can be hard since **RAAS will trigger thirst**
- Nutrition: **Na+<2g/day**
- **Weight daily and record** –important
    - Make sure pt has same cloth, measure at same time (morning is best)
- O2 supplementation (some pts may need **home O2**; make sure to educate fire safety)
- Rest to decrease O2 need by heart
- ICD or pacemaker
- **Ultrafiltration** to remove fluid
- Transplant

DRUGS FOR HEART FAILURE: "ABCDs"

- Ace-inhibitors/ARBs
    - Blocks RAAS → decrease **ventricular remodeling**
    - Watch out for **angioedema** and **hypotension**
- Beta-blockers
    - Stops SNS effects on heart –slows heart
    - **Contraindicated for acute HF** since it can actually worsen it
    - Monitor for bradycardia
- Cholesterol related to **CAD**
    - **Statins** are most commonly used
- Diuretics
    - Monitor for **K+ levels**
- Digitalis **(digoxin –very high risk)**
    - **Positive inotropic effect** –increase contraction
    - **Negative chronotropic effects** –slows down heart
- Vasodilators
    - **Hydralazine** and **Isosorbide Dinitrate**
    - Used when pts **cannot have ACE-I**

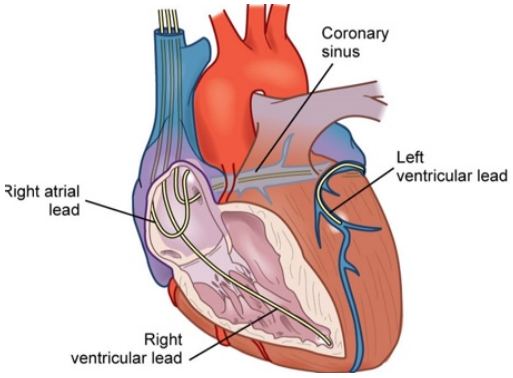
Newer Drugs

- Angiotensin receptor /Neprilisin inhibitors (an enzyme that breaks down **ANP** and **BNP**)
    - Sacubitril (ARB)/Valsartan
    - Trials demonstrated that it's better than ACE-I
- **Ivabradine**
    - Slows down **SA nodes** to slow down heart rate
    - Used for pts with **resting HF>70bpm** and are on **max amount of BB**

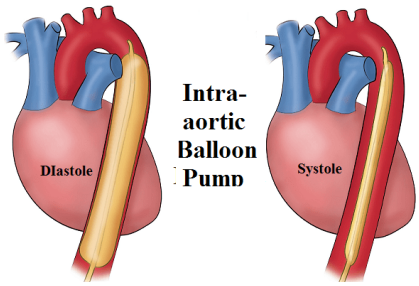
INTERPROFESSIONAL CARE: CARDIOSUPPORTIVE SYSTEMS/DEVICES

- **CardioMems system** –continuously monitor pulmonary artery pressure and the HR to catch increase of **pressure so that we can prevent PE**
- Implantable cardioverter defibrillator (ICD)
- Biventricular pacing/cardiac resynchronization therapy (CRT)

- Regulate irregulate bad synchronization between left and right ventricles to maximize CO



- **Ventricular assist device (VAD)**: device that helps the heart to pump blood throughout body
  - Used as a bridge to transplant or as destination therapy
- Impella pump: like a mini version of VAD; inserted into **femoral artery** into the **LV; from there it pulls fluid and puts it to aorta**
- Balloon pump
  - During **diastole,** balloon inflates to push back blood to coronary arteries and body
  - During **systole**, balloon deflates to allow blood to go from ventricle to aorta



TREATMENT OF ACUTE DECOMPENSATED HF

- **Increase oxygenation**
  - Give O2, raise HOB, manage anxiety
- **Reduce volume overload**
  - Diuretics (**furosemide**) –main goal is to pull fluid out of lungs
    - → You may have to give **nitroglycerin** (vasodilator) in order for Lasix to work since the pt's SNS system will prevent urination
- **Improve ventricular function**
  - Vasodilators (IV nitroprusside, NTG)
    - → High BP contribute to increased **afterload**
  - Monitor continuously due risk of dysrhythmia with meds
- **Increase force of contraction**
  - IV inotropes (**Milrinone** and **Dobutamine**)
    - → Not first line of therapy due to **risk of tachycardia**

MANAGEMENT FOR ACUTE HF PTS

- Watch hemodynamic parameters
  - **Heart rate**
  - **Arterial BP**
  - **Pulmonary pressures**
  - **Central venous or right arterial pressure**
  - **CO**
- Invasive hemodynamic monitoring system is implemented in **critical care settings**

Collaborative Care for Valvular Heart Disease

- Conservative therapy
  - Preventing recurrent **rheumatic fever** and **infective endocarditis**
  - Care depends on severity and the valve damaged
  - Goal is to prevent:
    - → **HF exacerbation**
    - → **Pulmonary edema**
    - → **Thromboembolism**
- Medical/surgical interventions (done for **severe** situations)
  - Percutaneous transluminal valvuloplasty
  - Valve repair or replacement

Valve Replacement

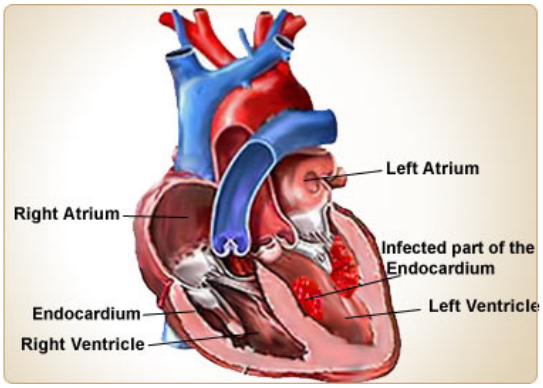
- **Def:** replacing damaged valve with prosthetic
- Classified as **mechanic** or **biologic**

Mechanical	Biological
- **Last longer** - **Higher risk for** thromboembolism, thus **needs long-term** anticoagulation therapy **(INR values should be between 2.5-3.5)**	- Reduced risk of **thromboembolism** - Shorter life - Pts with atrial fibrillation needs long-term **anticoagulation therapy**

- Nursing interventions for post-op
  - Infection check
  - Monitor heart and put pt on telemetry due to high risk of **dysrhythmia**
  - Check for **kidney perfusion** (UOP) just to make sure that CO is now adequate

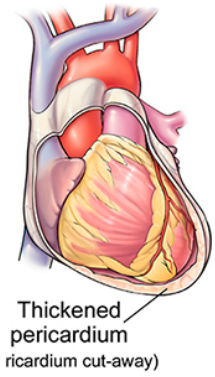
INFLAMMATORY CARDIAC DISORDERS

Infective Endocarditis



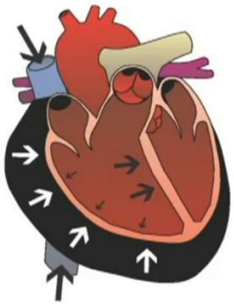
- **Def:** infection of endocardial layer of heart; since endocardial layer is continuous with heart valves → valves are infected as well (**tricuspid** is the most common one)
  - **Vegetations** form on valves or endocardial surface which may lead to **emboli** (if it dislodges)
  - Can either cause stenosis or regurgitation
- Classified as **acute** or **subacute** (may be asymptomatic)
- Can be bacterial, fungal, or viral
  - Most commonly caused by **Streptococcus viridians** and **Staph. Aureus**
- S/s:
  - Fever, chills, malaise, etc.
  - **New murmurs** since valves are not incompetent
- Risk factors
  - Age
  - IV drug use
  - Prosthetic heart valves
  - Prior endocarditis
  - Certain heart diseases
    - → Cardiomyopathies
    - → Congenital heart disease
    - → Acquired valve disease
    - → Existing cardiac lesions
  - Certain invasive procedures
    - → **PA catheters or central lines**
    - → **Dialysis**
- Clinical presentation
  - Often **non-specific**
    - → Fever, malaise, weakness, anorexia, etc. (s/s of infection)
  - **Abdominal discomfort**
  - **Arthralgia, myalgia**
  - **Cutaneous signs**
    - → Splinter bleeding
    - → Osler's nodes
    - → Roth's spots (in eye)
    - → Janeway lesions
  - **Onset or changing heart murmur** – marker!
  - Signs of **embolization** depends on the affected organ
    - → Spleen = ULQ pain
    - → Brain = s/s of stroke
    - → Limbs=pain, pallor, pulselessness
    - → Lungs=SOB
    - → Kidneys=flank pain and blood in urine
- Treatment
  - Antibiotics (make sure pt is adherent for full course)
  - Prophylactic measures for high risk pts: **pts with prosthetic HV, uncorrected congenital heart defects,** and **PA catheters**
  - Antipyretics
  - Pain relivers for arthralgia and myalgia
  - **Bed rest is usually not needed**
  - Pt education for high risk pts (infection prevention, avoiding fatigue, oral hygiene, IVDA treatment)

Pericarditis



- **Def:** inflammation of pericardial sac
- Often **idiopathic**
- Can be infectious or not
  - Non-infection: uremia, AMI, trauma, radiation
- Can be caused by hypersensitivity
  - Drug reactions
  - Rhematologic diseases (like lupus or RA)

- Clinical manifestations
    - Chest pain worse with **inspiration –hall mark** and when swallowing
        - → Usually relieved by sitting forward (unlike MI) and worse when pt
        - → Pain can radiate to trapezius
    - **Pericardial friction rub**
    - **ST segment elevation on EVERY lead** (unlike MI)
- Warning s/s that may indicate progression to **cardiac tamponade/pericardial effusion** (remember that inflamed tissue is very easy to rupture and leak)
    - **Muffled heart sounds** (due to fluid between stethoscope and heart)
    - JVD
    - **Pulsus paradoxus** –when systolic BP drops more than 10mmHg when pt inhale
    - Tachycardia, tachypnea, confusion, agitation



- Treatment
    - Antibiotics
    - NSAIDs –classic for inflammation
    - **Colchicine** (anti-inflammatory med)
    - Corticosteroids (high risk; do not use unless really needed)
    - **Pericardiocentesis**
        - → ECG lead is attached to needle

Post Procedure Care for Pericardiocentesis

- Obtain **12-lead ECG via PCXR** to get **baseline** and see if treatment was effective
- Continuous cardiac monitoring since heart is more **irritable**
- Check for resolution or recurrence of **effusion or tamponade**
    - Check v/s every 15 mins for the first hour and then every 30 mins until pt is stable
- Make sure that **pericardial fluid was sent to lab** for tests
- Proper **pericardial catheter care**
- Monitor drainage from catheter