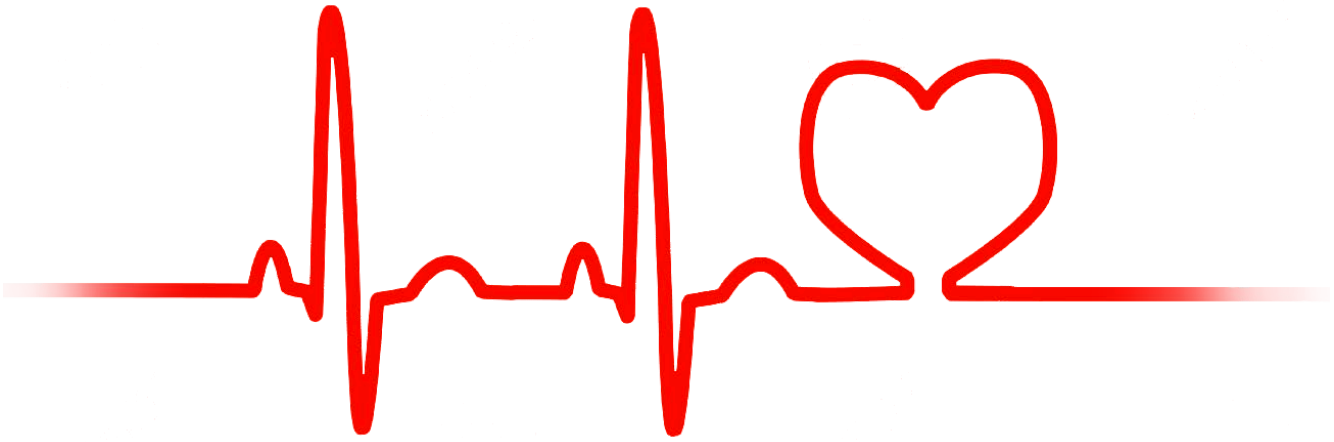
CARDIAC CARE UNIT SURVIVAL GUIDE

SUNY Upstate Medical University
2022



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Introduction to the CCU

The CCU day team consists of an attending cardiologist, a cardiology fellow**,** one senior medicine resident and one or two medicine interns from 7 am to 7 pm. The night team consists of a senior resident and cardiology fellow who covers from 7 pm to 7 am. Responsibilities include admitting new patients and serving as primary team until transfer to medicine or discharge.

Admission Note Tips

- Provide cardiac history in chronological order
- Echo: TTE or TEE, EF, Valves, wall motion abnormalities, diastolic parameters
- Catheterizations: Right heart cath values (pressures in RA, RV, PA or PCWP/PCOP), coronary angiography (left main, LAD, Lcx, RCA), intervention and complications
- CABG: Anatomy is critical; if OSH surgery, obtain copy of operative report or CABG angiogram
- Document peripheral vascular exam (femoral, popliteal, DPs, PTs)- these are important references if there is a procedure complication or access on the other side is needed
- Document your own EKG interpretations

Admissions from Cath or EP lab

- Always ask for the results of procedures and any complications
- Heparin restarted?
- What was the access? Where and what size? Does the arterial sheath need to be pulled? When? Duration of PTT
- Post Cath Check: Always check site of access (groin/radial or brachial) for distal pulses, hematoma, bruits. Follow CBC for decrease in hematocrit. If drops significantly post cath, consider retroperitoneal hematoma
- For access sheath removal, complications or questions contact fellow on call

Delivering CCU presentations

Presentations in the CCU can vary depending on your preference, the attending, and the reasons for CCU admission.

Remember it is not expected that you know all the laboratory values of your patient, we have computers to pull them out on! It is more important that you evaluate the patient and come up with a plan for the day.

Following is an example of a patient admission presentation:

1. Identifying statement for each patient
2. Does the patient follow with a cardiologist?
3. Pertinent PMH with special emphasis in cardiac history

Mr. Jones is a 60 y.o male admitted to the CCU with a diagnosis of NSTEMI . His primary cardiologist is Dr. Heart at UHCC. His past medical history includes HTN, HLD, 50 packs per year smoking h/o and obesity.

4. Timeline of events

Mr. Jones woke up at 3am with severe, 10/10, pressure-like sensation on the left side of the chest, radiated to his back and left arm, worsened with exercise, associated with shortness of breath, and orthopnea. Which prompted him to call EMS. Upon arrival to the hospital, he was hemodynamically stable, saturating 95% on RA. In the ED he received sublingual nitroglycerin which decreased his pain significantly. His troponins were elevated at 30 and 300. The EKG showed T wave inversions on V3, V4, and V5. A CTA chest ruled out aortic dissection. NSTEMI was diagnosed, he was loaded with ASA and Clopidogrel and heparin drip was started. He was kept NPO overnight for possible Cath today.

5. Subjective and objective overnight events

There were no events overnight. This morning the chest pain has resolved. He is feeling much better than before. On physical exam he is hemodynamically stable and appears euvolemic. His troponins were elevated up to 3,000. His electrolytes are WNL. An EKG from this morning does not show any new changes.

6. Assessment and Plan
- One liner summary followed by a list of problems in order of importance and explain the rationale for proposed differential diagnoses.
- List the actions that need to be taken

List of problems:
NSTEMI: The patient had anginal symptoms with significant troponin elevation and EKG changes. Our plan for today is : Continue heparin drip and aspirin, cardiac catheterization today, order an echocardiogram, and continue to trend the troponins, will order HBA1c, TSH, lipid panel to look for modifiable risk factors.

HTN: his home medications are carvedilol 6.5mg and lisinopril 20mg . Will continue.

HLD: Will increase the Rosuvastatin from his home dose of 10mg to 40mg.

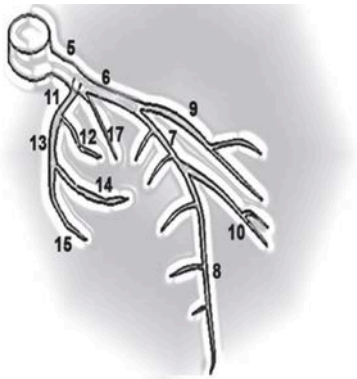
Basics of Coronary Anatomy

The gold standard for evaluation of coronary arteries is invasive coronary angiography of the left heart. The goal of coronary angiography is to identify coronary anatomy and atherosclerotic burden.

Left Coronary Artery: Left main coronary artery (5) bifurcates early into left anterior descending artery (LAD-6) and left circumflex artery (LCX-11). LAD runs on anterior part of the interventricular septum. It has two sets of branches – diagonal branches and septal perforators. The left circumflex runs in the AV grooves toward the posterior aspect of the heart. Branches off the LCx are called obtuse marginal branches (OM -12, 13, 14) and supply the lateral wall. In left dominant systems, the LCx also gives rise to the PDA (in right dominant systems, the PDA branches off the RCA). When the left main trifurcates into 3 branches, the middle branch is a ramus. It supplies the area typically supplied by the obtuse marginal of the Lcx.

Left Coronary Artery Branches

5- Left main coronary artery
6- Proximal LAD
7- Mid LAD (after take off of D1)
8- Distal LAD
9- First diagonal branch (D1)
10- Second diagonal branch (D2)
11- Proximal Left Circumflex
12- First Obtuse Marginal (OM1)
13- Mid left circumflex (after OM1)
14- Second Obtuse Marginal (OM2)
15- Distal Left Circumflex
17- Ramus intermedius

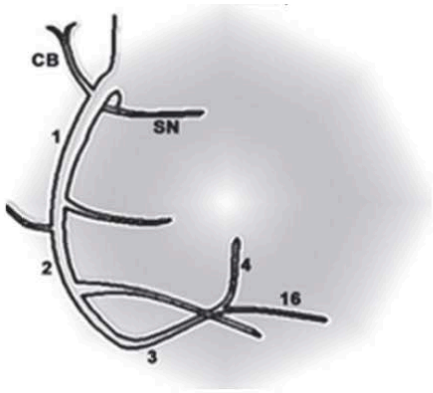


Right coronary artery (RCA): Gives rise to the PDA which determines dominance. RCA supplies the sinus node and AV node and thereby patients with RCA occlusion can suffer from conduction complications

Dominance: Most individuals (85% of population) are right dominant. Dominance is determined by which artery gives off the PDA (RCA vs LCx).

Right Coronary Artery Branches

1- Proximal RCA
2- Mid RCA
3- Distal RCA
4- Posterior descending artery (PDA)
16- Posterior left ventricular branch (PLV)
CB- Conal branch (first branch off the RCA)
SN- Sinonodal branch (feeds SA node)



Chest Pain

Chest pain is one of the most common causes of emergency department visits. The differential diagnosis is broad and includes cardiac, gastrointestinal, pulmonary, musculoskeletal and psychiatric etiologies. Below are tools to help you triage acute chest pain.

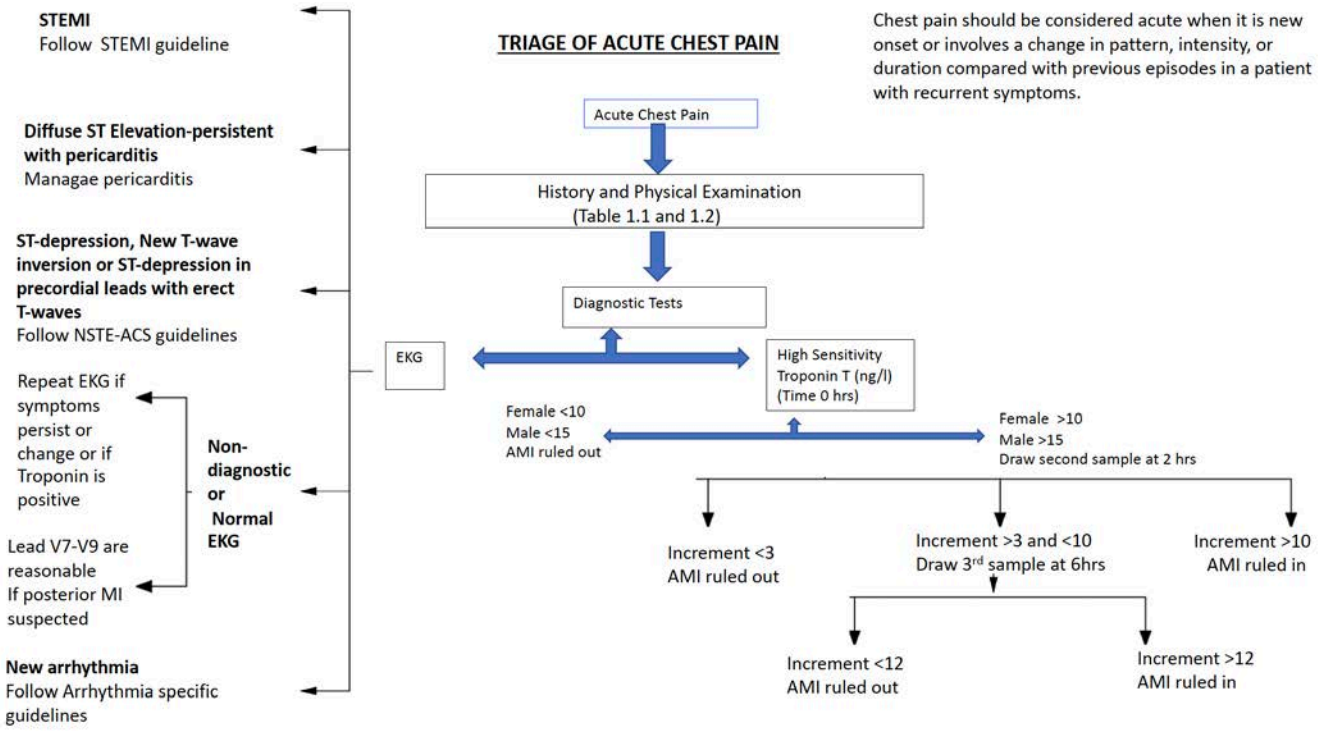


Table 1.1 History (SOCRATES)	Table 1.2 Physical examination
Site Retrosternal	ACS Diaphoresis, tachypnea, tachycardia, hypotension, crackles, S3,
Onset: ACS-Over few minutes; Aortic Dissection (AD)-Sudden; Pericardial-gradual; Gastrointestinal (GI)- sudden/gradual	Aortic Dissection Extremity pulse differential, Xray-widened mediastinum
Characteristics ACS-Constricting, heavy, tightness, pressure, squeezing; AD-Ripping/tearing; Pericardial- sharp/stabbing; GI-gripping/burning	Pulmonary embolism Tachycardia, dyspnea, chest pain with inspiration
Radiation ACS-Arm, neck, jaw, epigastrium; AD-back, between shoulders; Pericardial-back, left shoulder; GI-often back	Pericarditis Fever, friction rub
Associated symptoms ACS/AD-sweating, breathlessness, nausea, vomiting, feeling of impending death; Pericardial- flu like prodrome; GI-upper abdominal pain, heartburn	Esophageal rupture Subcutaneous emphysema, pneumothorax, unilateral absent or decreased breathe sounds
Timing ACS/AD-prolonged; Pericardial-variable, GI-night time common	Peptic ulcer disease/Gall bladder disease Epigastric tenderness, right upper quadrant tenderness
Exaggerating and relieving factors ACS-usually spontaneous, may occur with stress and exercise. Relief with nitroglycerine is not necessarily diagnostic of ACS; AD-spontaneous, no relieving factors; Pericardial-sitting up and lying down affect intensity of pain, NSAIDS relieve the pain; GI- lying flat may trigger pain, not relieved by rest	Pneumonia Fever, tachypnea, dullness to percussion, bronchial breathe sounds
Severity ACS-usually severe. AD-very severe, pericardial-moderate to severe, GI-mild	Pneumothorax Dyspnea, unilateral absence of breathe sound
	Costochondritis Tenderness of costo-chondral joint
	Herpes-zoster Pain in dermatomal distribution and characteristic rash

The HEART Score (History, EKG, Age, Risk Factors, Troponin) score is a well validated ED clinical decision tool that helps prognosticate and triage patients presenting with chest pain suspicious for cardiac features. It identifies patients who are either already sick or at risk of becoming sick by estimating risk of MACE (major adverse cardiac events) within 6 weeks of presentation to the ED. Refer to MDCalc to calculate the score.

Typical chest pain	Atypical/Non-cardiac chest pain
Characterized by chest tightness, squeezing, heaviness, pressure, weight	Characterized by sharp, pricking, knife-like, pulsating, choking
Radiating to shoulder, neck, inner arm	Positional, chest wall may be tender to palpation, radiation pattern may be highly variable
Lasts for 10-20 minutes	Lasts for variable duration
Provoked by exertion or emotional stress Relieved by nitroglycerine or rest	Can be provoked or unprovoked Does not relieve with nitroglycerine or rest

Acute Coronary Syndrome

Definition: clinical or pathological event caused by myocardial ischemia where there is evidence of myocardial injury or necrosis.

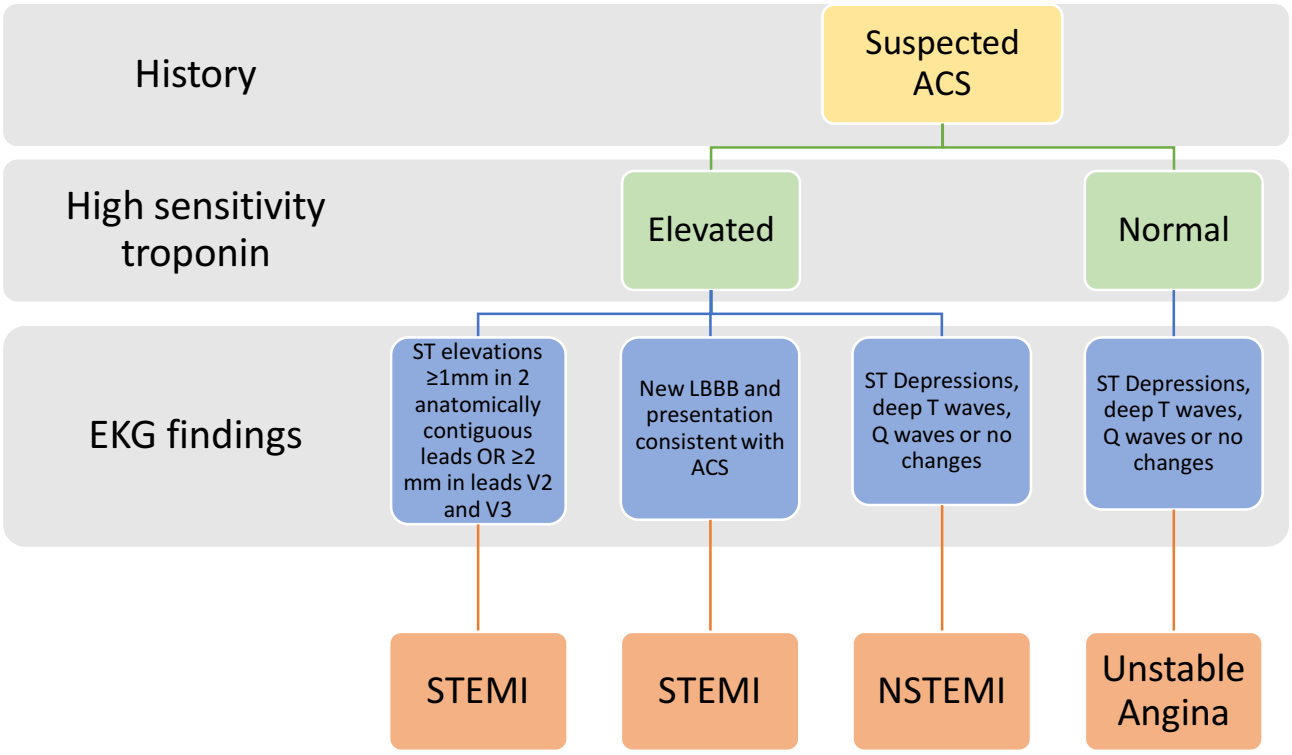
History:

1. Rest angina which is more than 20 minutes in duration
2. New onset angina limiting physical activity
3. Increasing angina that is more frequent, longer in duration or occurs with less exertion than previous angina
4. Atypical presentation seen in female, older adults, and diabetics

Diagnostic work-up:

1. Obtain a 12-lead EKG
    1. Can be repeated q10-15mins if high suspicion of ACS with non-diagnostic initial EKG
2. High sensitivity troponin

Diagnosis: 2 out of 3 criteria (history, elevation in cardiac biomarkers, EKG findings)



Initial interventions are usually done in the field/ ED and include

- Assess and stabilize **A**irway, **B**reathing, **C**irculation. Establish IV access
- **O2** as needed for sats>90%. Telemetry
- Rapid ACLS for sustained ventricular arrhythmia
- **ASA 325** chew and swallow (unless Aortic dissection is being considered). Rectal, if PO is not feasible.
- S/L **Nitro** 0.4mg q5mins for 3 doses (if no signs of hemodynamic compromise like RV infarct AND no use of PDE inhibitors). Can start IV Nitro if persistent pain/discomfort.
- Obtain **troponins,** electrolytes, crt, H/H, coag (for pts with coagulopathies or on anticoagulation)
- If LV failure is suspected, reduce afterload with nitro S/L or IV gtt @ 40 mcg/min, loops (Lasix), or BiPAP
- **Beta-Blockers** (Metoprolol tartrate 25 mg PO) if no signs of HF, not at high risk of HF, no hemodynamic compromise or bradycardia or severe airway reactive disease. If hypertensive: Can use IV Lopressor 5 mg q5mins for 3 doses as tolerated.
- **Morphine** (2-4 mg slow IV q5-15mins) for persistent discomfort/ anxiety
- **Atorvastatin 80 mg** as early as possible (before PCI if not already on statins. If pt is on moderate intensity statin, switch to high intensity.

STEMI is a MEDICAL EMERGENCY! Select reperfusion therapy →

PCI (strongly preferred esp. cardiogenic shock, HF, late presentation or C/I to fibrinolysis

Fibrinolysis (if PCI unavailable within 120 mins of first medical contact AND symptoms <12 hours AND no C/I

NSTEMI or Unstable Angina
Management depends on mortality risk →

PCI very high to intermediate risk patients within 24-48 hours or within 2 hours if persistent chest pain, hemodynamic instability or ventricular tachycardia

Stress testing stable low risk patients to evaluate need for coronary angiography

Prognosis for ACS patients can be found using the **GRACE** or **TIMI** risk score (MDCalc). All patients should receive an antiplatelet and anticoagulation in addition to aspirin. Choice of drug, dose, timing and duration is individualized and depends on the reperfusion strategy chose. Please ask your CCU fellow before starting these!

Antiplatelets used in ACS:

- **Plavix (Clopidogrel)**
  - 300 mg load if age ≤ 75 years
  - 75 mg load for age > 75 years if f received fibrinolysis.
  - Plavix load of 600 mg if going for PCI and high risk of bleeding so that Brilinta and Effient can't be used.
- **Brilinta (Ticagrelor**) 180 mg load is preferred if no fibrinolysis
- **Effient (Prasugrel**) 60 mg load if primary PCI is planned, and NO prior stroke or TIA or age ≤ 75 years or wt <60 kg)
- Sometimes patients will be started on a Gp IIb/IIIa inhibitor Integrilin (Eptifibatide) gtt during and few hours post-PCI w/o any prior antiplatelets.

You will hear the names of PLATO, TREAT, TRITON-TIMI trials commonly referred when choosing an antiplatelet drug prior to PCI.

Anti-coagulation used in ACS:

1. For patient undergoing primary PCI:

- Assuming patient will receive Brilinta/Effient: **UFH i**s preferred 50-70 U/kg bolus (max 5000 U). Additional heparin maybe given in cath lab. (Usually, it's ordered as heparin gtt low dose with bolus. However, may not need the bolus if received Integrilin intra-cath)
- If patient receives Plavix: Either heparin or **Bivalrudin** is reasonable.

- If treated with fibrinolysis:

- **Lovenox** preferred for patients not at high bleeding risk. Patients in whom PCI is possible after fibrinolytics, **UFH** is reasonable.
- High risk of bleeding and who are not likely to require PCI: **fondaparinux** or UFH.

- Not receiving reperfusion therapy:

- Lovenox or UFH (same bolus as above followed by IV infusion 12 U/kg/hr to achieve goal aPTT 50-70 sec).

Post-PCI and stenting care:

- Ensure patients are on **DAPT** (ASA 81mg daily + Plavix 75 mg daily/ Brilinta 90 mg BID/ Effient 10 mg daily). Patients also receiving anticoagulation like Eliquis/ Xarelto for A. Fib/DVT/PE may receive only Plavix+Eliquis/Xarelto as no benefit has been noticed from triple therapy and increases the risks of bleeding. However, some attendings will prefer a short course of triple therapy too.
- Ensure high intensity **statins.** Consider starting **Beta blockers** if not already.
- Consider **ACEI** for LVEF<40%, HF, HTN, DM2 or wall motion abnormalities
- Common workup ordered: **Lipid panel, A1c, TSH FT4, TTE.**
- If the coronary lesions are not amenable to PCI, cardiac surgery evaluation for CABG may be needed

Common complications of Acute Myocardial Infarction (AMI)

Persistent chest pain

- Consider re-infarction. Check CKMB if troponin has not peaked/repeat EKG. It is important to look at the post PCI EKG and check troponins until they peak to assess for re-infarction if they have recurrent chest pain

Post-infarction pericarditis

- Avoid NSAIDS in post MI patients as it may impair LV healing
- EKG findings should show diffuse ST elevations and PR depressions

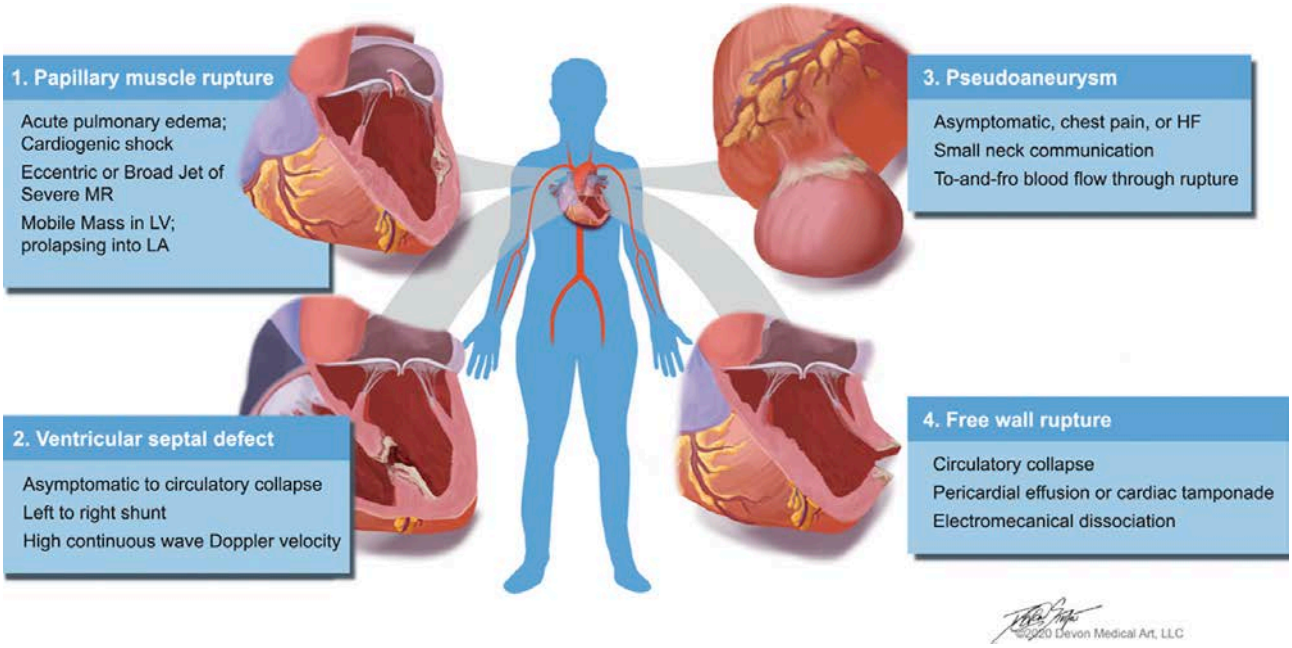
Ventricular Arrhythmias

- Ventricular tachycardia and Ventricular Fibrillation are common in post MI patients
- Accelerated idioventricular rhythm (AIVR) is a ventricular rhythm consisting of 3 or more consecutive monomorphic beats with gradual onset and gradual termination which is a common benign post revascularization arrhythmia within 24 hours

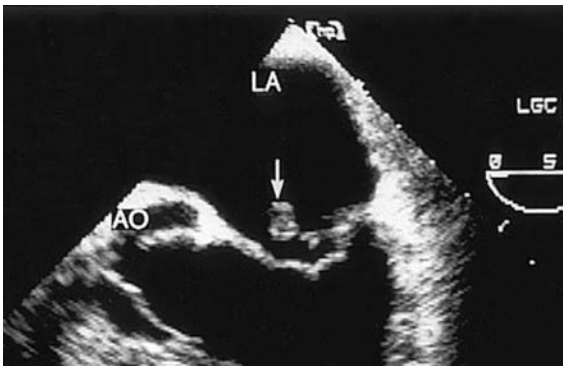
Mechanical Complications

- Advances in therapy have improved outcomes for patients with acute myocardial infarction. However, patients with large infarcts or those who do not receive timely revascularization remain at risk for mechanical complications of acute MI.
- The most common mechanical complications are listed on the following page.

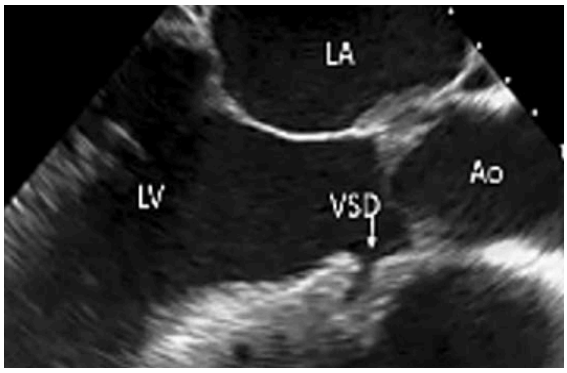
Complications of AMI (contd)



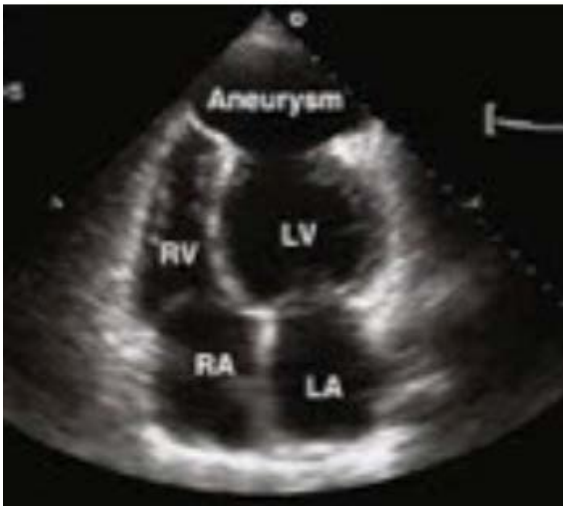
Mechanical Complications of AMI	Management
Papillary muscle rupture and acute mitral regurgitation (Fig 1)	-Afterload reduction with diuretics, nitrates and nitroprusside -Intra-aortic balloon pump (IABP) to increase forward flow and reduce regurgitation fraction -Emergent surgical mitral valve replacement (preferred) or repair within 24 hours
Left Ventricular free wall rupture	-Inotropic support, echo guided pericardiocentesis, IABP -Emergent surgical evaluation and repair
Ventricular septal defect (Fig 2)	-Immediate reduction of afterload with IABP or LVAD followed by urgent surgical or percutaneous repair. Urgency dependent on cardiogenic shock and presence/severity of end organ dysfunction
True left ventricular aneurysm (Fig 3)	-Reduce afterload with ACEI/ARB, beta blockers, diuresis -Reduce thrombosis risk with anticoagulation -Surgical intervention via aneurysmectomy if patient has CHF, Vtach or, refractory angina
LV Pseudoaneurysm	-Urgent surgical or percutaneous repair, depending on symptoms
Left ventricular thrombus (Fig 4)	Anticoagulation initiated immediately -Surgery depending on size of clot and associated symptoms



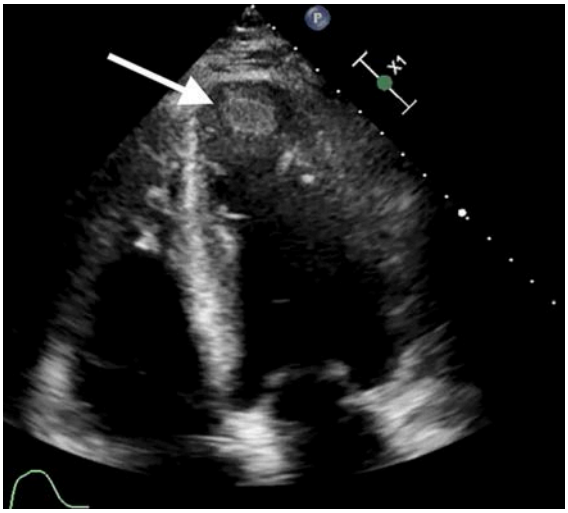
TEE examination in left ventricular PM rupture of the posterior muscle. AO indicates aorta; LV, left ventricle; RV, right ventricle; and RVO, right ventricular outflow tract. (Fig 1)



TEE imaging showing the small ventricular septal defect (VSD) in the upper part of the septum with a left to right shunt and severe aortic insufficiency (Fig 2)



TTE imaging showing very large LV Aneurysm
(Fig 3)



TEE imaging showed left ventricular apical thrombus (Fig 4)

Post Cardiac Cath Care

When accepting a patient to the CCU from the cath lab, it is important to ask the following questions:

1. Type of access used for the procedure (arterial vs venous, femoral vs radial vs jugular)
2. Any sheaths in place
3. Medications that patients received in the cath lab and planned course for antiplatelet agents
4. Drips currently running(i.e. heparin) and when they should be stopped
5. Stents placed and type, size and location

Monitor daily for the following complications

1. Hematoma or bleeding (especially retroperitoneal)
2. Access site pseudoaneurysm
3. AV fistula
4. Limb ischemia
5. Infection
6. Neuropathy (lateral femoral cutaneous nerve)
7. Contrast Induced Nephropathy
8. Chest Pain (to monitor for in-stent thrombosis however occurs in less than 1% of all patients

Congestive Heart Failure (CHF)

Definition: a complex of signs and symptoms caused by structural or functional impairment of ventricular filling and/or ejection of blood
Heart Failure with Reduced Ejection Fraction: Heart failure with reduced stroke volume and reduced ejection fraction (EF <40%)
Heart Failure with Preserved Ejection Fraction: heart failure with reduced stroke volume, normal/reduced EDV and preserved EF (LVEF >40-50%)
Heart Failure with mid-range Ejection Fraction: EF of 41-49%. ICD codes do not include HfmREF and thus for documentation we may include acute/acute on chronic systolic heart failure as the diagnosis
Right heart failure: CHF due to right ventricular dysfunction resulting in congestion of blood in the systemic venous system
Left heart failure: CHF due to left ventricular dysfunction resulting in tissue hypoperfusion and increased pulmonary capillary pressure

Clinical Features of left sided Heart Failure	Clinical Features of right sided heart failure
Symptoms: Dyspnea, orthopnea, paroxysmal nocturnal dyspnea, cardiac wheezing: **Physical Exam:** Bilateral basilar rales, laterally displaced heart beat, coolness and pallor of lower extremities	**Symptoms:** peripheral pitting edema, abdominal pain/jaundice as signs of congestive gastropathy **Physical Exam:** JVD, Kussmaul sign, hepatosplenomegaly and heptojugular reflux

Diagnostics

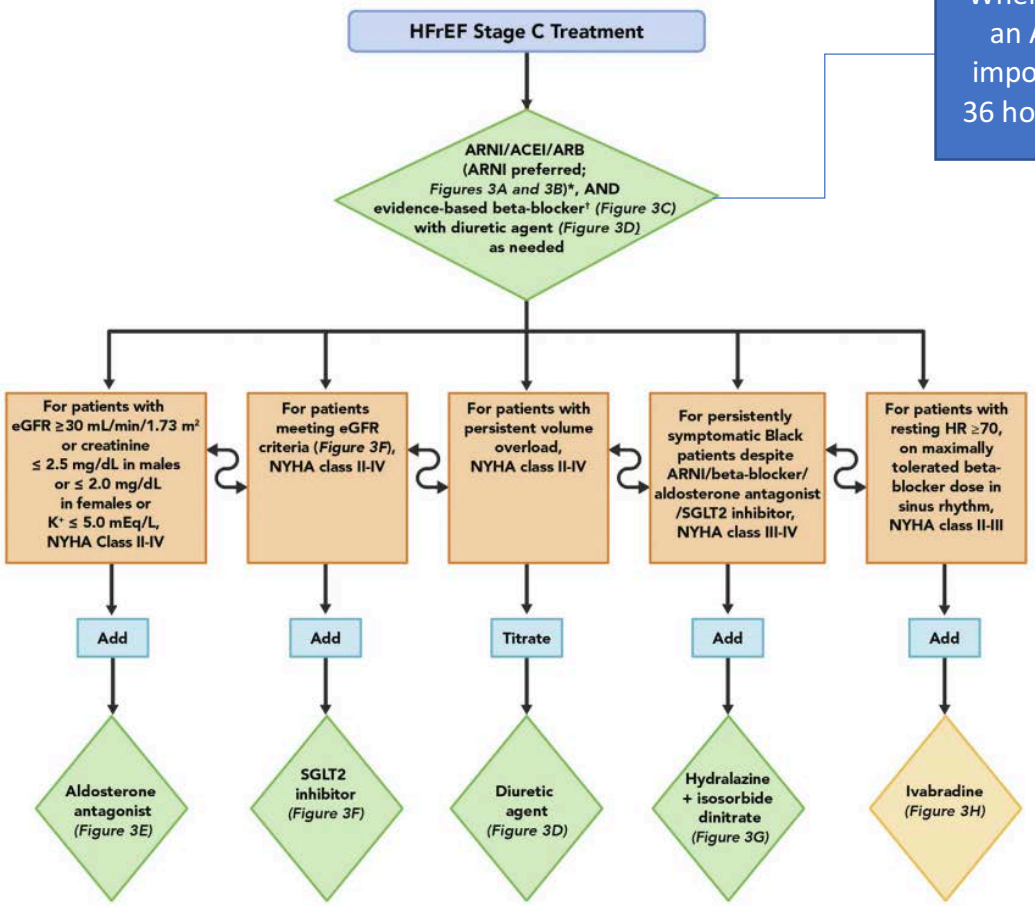
1. Based on clinical signs and symptoms. Support diagnosis with cardiac biomarkers (proBNP), EKG, Chest Xray, Echocardiogram
2. If CHF is confirmed, investigate for underlying causes (consider coronary angiogram, chest imaging and advanced cardiac imaging)
3. Modifiable risk factors (hypertension, coronary artery disease)

Treatment

- Refer to diagram on the following page for treatment of HFREF. This review article from JACC is also a great comprehensive review: https://www.jacc.org/doi/pdf/10.1016/j.jacc.2020.11.022**.**
- Note that there is a paucity of evidence supporting treatment regimens for those with HFPEF.

When transitioning from an ACE-I to ARNI, it is important to allow for a 36 hour wash out period!

FIGURE 2 Treatment Algorithm for Guideline-Directed Medical Therapy Including Novel Therapies



*ACEI/ARB should only be considered in patients with contraindications, intolerance or inaccessibility to ARNI. In those instances, please consult Figure 3 and text for guidance on initiation.

†Carvedilol, metoprolol succinate, or bisoprolol.

ACEI = angiotensin-converting enzyme inhibitors; ARNI = angiotensin receptor-neprilysin inhibitors; ARB = angiotensin receptor blocker; eGFR = estimated glomerular filtration rate; HFrEF = heart failure with reduced ejection fraction; HR = heart rate; K^+ = potassium; NYHA = New York Heart Association; SGLT2 = sodium-glucose cotransporter-2.

Green color identifies a Class I therapy from clinical practice guidelines, whereas yellow color indicates a Class II therapy.

Invasive Interventions: ICD, CRT, LVAD

Patients with CHF are at risk of sudden cardiac death from arrhythmias such as ventricular tachycardia (VT) or ventricular fibrillation (VF) and heart failure may be worsened by cardiac dyssynchrony. At this point, we may consider referral to a heart failure specialist for ICD placement, cardiac resynchronization therapy or mechanical circulatory support such as the left ventricular assist device (LVAD).

Acutely Decompensated Heart Failure

Definition: Clinical syndrome of new or worsening signs and symptoms of heart failure. Clinical features are commonly classified according to perfusion and presence of congestion at rest.

	No evidence of congestion	Evidence of congestion
Adequate perfusion	WARM AND DRY	WARM AND WET
Hypo-perfusion	COLD AND DRY	COLD AND WET

Assess for clinical features suggestive of hypoperfusion (narrow pulse pressure, cool extremities, peripheral cyanosis, altered mental status, below baseline blood pressure) to identify patients with or at risk of cardiogenic shock.

	Heart Failure Unlikely	Heart Failure likely
BNP	<100	>500
Nt-proBNP	<300	>1000

Initiation of Treatment:

- Diuretic naïve patients: IV furosemide or bumetanide.
  Those taking diuretics at home: Administer 1-2.5 times the patient's usual oral dose IV as a bolus or continuous infusion
- Assess effect of diuretics every 6 hours
- If urinary output is <100 ml/hour → consider doubling the diuretic dose
- If urinary output is > 100- 150 ml/hour →continue current diuretic dose
- Goal output: 2L/day

Refractory AHF

- Consider combination therapy with thiazide diuretic (metolazone, HCTZ, chlorthiazide) /add vasodilatory such as IV nitroglycerin/sodium nitroprusside. Consider inotropes if low EF and poor response to IV diuresis such as milrinone, dobutamine, dopamine.
- As congestion is resolving, clinical symptoms improving, or renal function worsening, consider less frequent dosing or transition to oral diuretic
- Ultrafiltration as last resort

Ongoing Management

- Identifying any underlying triggers
- Fluid restriction 1.5-2L/day, sodium restriction <3 g/day
- Monitor and replete serum electrolytes (K, Mg, Na) every 12-24 hours
- Monitor renal function daily: elevated creatinine not a contraindication to diuretic therapy in patients with AHF as renal function typically improves with effective diuresis in cardiorenal syndrome
- Consider consult to heart failure nurse and schedule follow up with cardiology

Cardiogenic Shock

Definition: Clinical condition of inadequate tissue (end-organ) perfusion due to the inability of the heart to pump an adequate amount of blood. The reduction in tissue perfusion results in decreased oxygen and nutrient delivery to the tissues and if prolonged, potentially end organ damage and multi system failure.

Manifestation:

- Systolic blood pressure <90 (or vasopressor requirement
- Cardiac Index <2.2 L/min
- Cardiac power output <0.6W
- Lactic acidosis

Etiology:

- Impaired myocardial contractility: Myocardial ischemia or myocardial infarction, myocarditis, drugs
- Abnormalities of cardiac rhythm: Tachycardia, bradycardia
- Cardiac structural disorder: Acute mitral or aortic regurgitation, ruptured interventricular septum, prosthetic valve malfunction

Management:

1. Consider **pulmonary artery catheter** for more accurate measurements to distinguish between cardiogenic, hypovolemic, distributive or obstructive shock

2. **Intravenous inotropes and vasopressors**
   - Adrenergic agents – norepinephrine and epinephrine
   - Inotropic agents – dobutamine and dopamine (given propensity to increase myocardial oxygen demand, ischemic burden, malignant arrhythmias these agents should be used in lowest possible doses for shortest duration)
   - Phosphodiesterase inhibitors – milrinone and levosimendan (which modulates positive inotropic effects through a combination of calcium sensitization and selective PDE-3 inhibition

3. **Mechanical Circulatory Support**
   - Examples include IABP, Impella (used commonly in our CCU), VA-ECMO, Tandem Heart LA-FA. Only device with biventricular support is VA-ECMO
   - To be used in acute severe or refractory cardiogenic shock
   - Benefits include reduction of LV stroke work and intracardiac filling pressures
   - Lactate levels, cardiac power output and pulmonary arterial pulsatility index help facilitate both selection and weaning strategies

4. **LVAD and Heart Transplantation**
   - Requires early and ongoing assessment of need for cardiac replacement therapy, which is necessary in those with refractory CS

Tachyarrhythmia and Resuscitations

Definition: Ventricular rate of 100 or more beats per minute
Symptoms: dyspnea, angina, lightheadedness or syncope and decreased level of consciousness
Diagnostic Tests: Electrolytes (including magnesium), thyroid function tests, serum concentration of digoxin (if applicable) urine toxicology screen, EKG, CXR and Transthoracic Echocardiogram

Step 1: Is the patient hemodynamically stable or unstable?

Stable
Non emergent approach to diagnose patient's rhythm, identify reversible causes

Hemodynamically Unstable
1. Sinus Rhythm → non-emergent approach to diagnose patient's rhythm, identify reversible causes
2. Not sinus rhythm →urgent cardioversion

Step 2: Is the QRS complex narrow or wide? (<120 ms or > 120 ms)
Step 3: Is the QRS complex regular or irregular?
Step 4: ALWAYS COMPARE with prior EKG to check baseline morphology

Narrow QRS complex (<120 ms)
arrhythmia originates within the atria and rapidly activates ventricles via His Purkinje

Regular
- Sinus vs inappropriate sinus tachycardia
- Supraventricular tachycardia (AVNRT, AVRT, Atrial Tachycardia)
- Atrial Flutter

Irregular
- Atrial Fibrillation
- Atrial Flutter with variable conduction
- Multifocal atrial tachycardia

Wide QRS complex (>120 ms)
arrhythmia originates within ventricles; or originates in atria and travels to ventricles either via abnormal his-purkinje or through accessory pathway

Regular (typically have RBBB or LBBB morphology)
- Monomorphic Vtach (VT)
- SVT with aberrant conduction

Irregular
- Polymorphic Vtach (VT)
- Ventricular Fibrillation /V Flutter
- Afib with aberrant conduction/bundle block block

Points to Consider:

1. Give sedation before cardioversion if patient is awake. It is painful! Consider versed.
2. Hypotension can sometimes be from tachycardia. If otherwise asymptomatic, try fluids and beta block/CCB to improve HR. Often BP improves with better control of HR.
3. Post cardioversion, patient will need to be on anticoagulation for 4 weeks.

Narrow Complex Regular Tachycardia	**Narrow Complex Irregular Tachycardia**
<u>Sinus vs Inappropriate sinus tachycardia</u> • Appropriate → treat underlying etiology (coronary ischemia, pulmonary embolism, respiratory or cardiac failure, hypovolemia, anemia, hyperthyroidism, fever, pain or anxiety) • Inappropriate → treat with IV beta blocker. <u>Supraventricular Tachycardia (AVNRT, AVRT, Atrial Tachycardia)</u> • Hemodynamically unstable → immediate synchronized cardioversion • Consider vagal maneuvers (Valsalva or carotid sinus massage) • Hemodynamically stable → therapy with AV nodal blocking agent (1. adenosine 2. verapamil 3. beta blockers). Run EKG/rhythm strip to assess underlying rhythm! <u>Atrial Flutter with 2:1, 3:1 or 4:1 block</u> • Consider if ventricular response of approx. 150 beats per minute indicating 2:1 block • New onset → assess need for cardioversion (if hemodynamically unstable), rate controlling therapy (beta blocker or non-dihydropyridine calcium channel blocker) and antithrombotic therapy. Rhythm control can also be used to prevent recurrence.	<u>Atrial Fibrillation</u> • Please see management in the Atrial Fibrillation section <u>Atrial Flutter with variable conduction</u> • Similar to atrial fibrillation, see previous section on atrial flutter <u>Multifocal atrial tachycardia (MAT)</u> • Definition: P waves with three or more different morphologies • Associated with significant pulmonary or cardiac illness. Most episodes do not precipitate hemodynamic compromise or limiting symptoms, so therapy aimed at underlying disease • Patients with MAT associated hypokalemia or hypomagnesemia should undergo electrolyte repletion • If symptomatic, requires ventricular rate control including non-dihydropyridine calcium channel blocker and beta blocker. However, rate control therapy is typically unsuccessful without treating underlying disorder.

Wide Complex Regular Tachycardia

- Most concerning is VTach (VT), arrhythmia assumed VT until proven otherwise

Monomorphic Vtach (concerning only if sustained → three or more consecutive beats that last longer than 30 seconds)

- Pulseless → ACLS algorithm, defibrillate
- Unstable but with pulse →synchronized external cardioversion
- Refractory or recurrent wide complex tachycardia → IV class Ib or III antiarrhythmic drugs, such as amiodarone, lidocaine/mexilitine or procainamide
- Nonsustained Vtach → rule out MI, replete electrolytes, ensure fluid balance

SVT with aberrant conduction

- Narrow complex SVT rhythms present with wide complex in setting of aberrant conduction
- Perform vagal maneuver or administer rate controlling agent and repeat EKG to assess sinus node conduction
- Initial management similar to that of SVT with narrow QRS complex

Wide complex irregular tachycardia

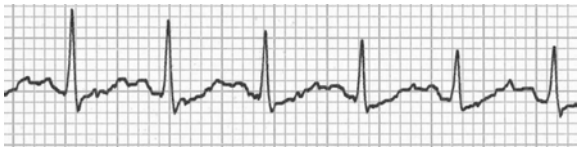
Polymorphic Ventricular Tachycardia/Torsades des Pointes

- Hemodynamically unstable → Urgent defibrillation
- Hemodynamically stable ? CHECK QTC. If QTC >500, avoid amiodarone without running it by the fellow! In a prolonged QTC do the following:
  - IV mag sulfate (initial dose of 1-2 grams IV over 15 minutes, followed by infusion)
  - If not responsive to IV magnesium then use isoproterenol (initial dose 0.05 to 0.1 per minute) used as temporizing measure to achieve heart rate of 100 beats per minute.
  - Refractory to isoproterenol→ attempt temporary transvenous pacing at 100 bpm
  - Ensure to remove QTC prolonging agents.
- Hemodynamically stable and normal QTC
  - Beta blockers if blood pressure tolerates
  - IV amiodarone to prevent recurrence
  - Mag less likely to be effective for polymorphic VT if baseline QT interval normal
  - Most likely cause is myocardial ischemia, consider urgent coronary angiography

Ventricular Fibrillation

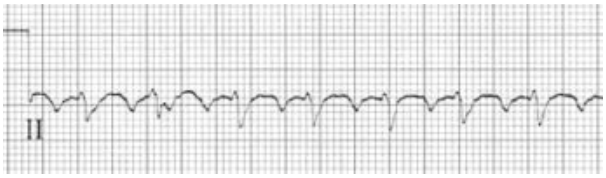
- Defibrillation → follow ACLS algorithm

Sinus Tachycardia



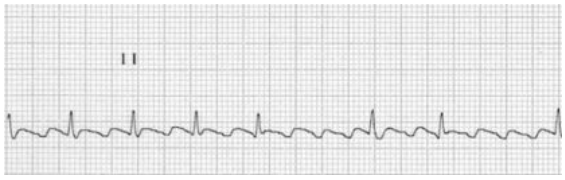
With very fast heart rates the P waves may be hidden in the preceding T wave, producing a 'camel hump' appearance.

Atrial Flutter with 2:1 Block



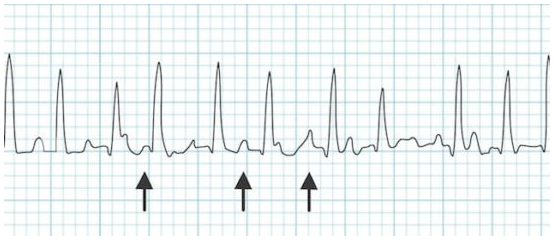
Inverted flutter waves in lead II at a rate of 300 bpm (one per big square)
2:1 AV block resulting in a ventricular rate of 150 bpm

Atrial Flutter with variable block



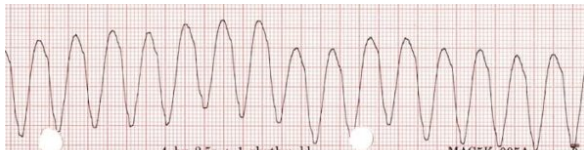
The degree of AV block ranges from 2:1 to 4:1

Multifocal Atrial Tachycardia



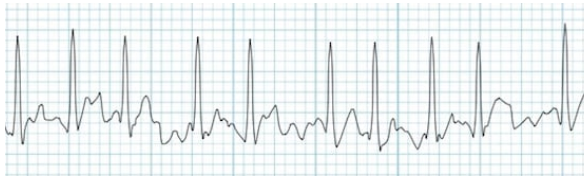
Rapid Irregular rhythm > 100 bpm
Atleast 3 distinct P wave morphologies

Monomorphic Vtach



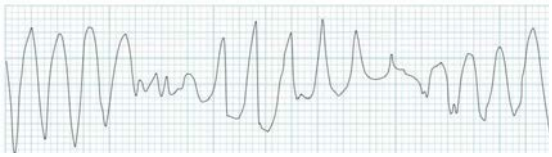
Uniform QRS complexes, Broad >200 ms

Atrial Fibrillation



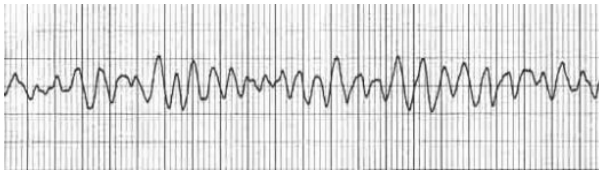
Irregularly Irregular Ventricular Rate without visible P waves

Torsades des Pointes



For TDP to be diagnosed, patient must have evidence of both polymorphic Vtach and prolonged QTC

Ventricular Fibrillation



Chaotic irregular deflections without identifiable P-QRS-T waves

Atrial Fibrillation

Definition: Supraventricular tachyarrhythmia characterized by uncoordinated atrial activation that results in an irregular ventricular response
Presentation: Most are asymptomatic, less commonly individuals develop palpitations, dizziness, syncope, fatigue or dyspnea
Diagnostics: Obtain EKG (irregularly irregular, absent p-waves), monitor on tele. Investigate the underlying cause

RATE CONTROL	RHYTHM CONTROL
Goal: HR <110/minute **Pharmacological options:** 1st line: beta blockers, nondihydropyridine calcium channel blockers (avoid use in patients with decompensated heart failure or low ejection fraction) 2nd line: digoxin (preferred as first line therapy in patients with decompensated HF, avoid in AKI, dose adjusted for renal dysfunction) 3rd line: amiodarone (reserved for patients in whom all other options have failed) **Surgical options:** AV nodal ablation and implantation of a permanent ventricular pacemaker. Indications include recurrent Afib, refractory to medical rate control and patients who do not tolerate the pharmacological options for Afib management	**Goal:** termination of atrial fibrillation, restoration of sinus rhythm to prevent atrial remodeling **Indications:** 1) failure of rate control 2) new onset atrial fibrillation **Contraindications:** 1) Long standing persistent Afib 2) reversible cause such as digoxin toxicity or electrolyte imbalances 3) high risk of thromboembolic events **Electrical options:** Synchronized cardioversion – can be performed in emergency in unstable patients or electively in stable patients **Pharmacological options:** Inpatient regimens using intravenous or oral antiarrhythmics → dofetilide, ibulitide, flecainide, propafenone, amiodarone and sotalol (requires inpatient monitoring after initiation) **Interventional options:** Pulmonary vein isolation **Other things to consider:** initiate anticoagulation before cardioversion to reduce stroke risk. Consider TEE if anticoagulation duration suboptimal. Following cardioversion, consider daily antiarrhythmic drugs to maintain SR.

Anticoagulation

During cardioversion

- Hemodynamically unstable → Anticoagulate ASAP, should not delay electrical cardioversion
- Valvular Afib → anticoagulated before procedure, continue long term
- Non valvular Afib → risk stratify using CHADSVASC to determine need for long term

Indications for TEE use

- Used prior to cardioversion to evaluate for thrombus and reduce risk of thromboembolic events, TEE visualizes atria and left atrial appendage
- Indications prior to cardioversion
    - New onset atrial fibrillation or atrial flutter for >48 hours or unknown duration
    - No previous anticoagulant use or subtherapeutic anticoagulation
    - CHF exacerbation or hemodynamic instability
    - Symptomatic Afib
    - High stroke risk (history of stroke, left atrial thrombus, HOCM or rheumatic fever)
- Interpretation
    - Thrombus identified: Patients should ideally receive >3 weeks of anticoagulation and a repeat TEE prior to any procedure
    - No thrombus identified: 3 weeks of preceding anticoagulation is not required
- Interventional alternatives to anticoagulation
    - Description → occlusion of the left atrial appendage (most common location for the formation of thrombus)
    - Options include
        - Percutaneous left atrial appendage occlusion
        - Surgical occlusion of left atrial appendage
    - Consider in patients who have contraindications to long term anticoagulation and have increased risk of stroke.

Bradyarrhythmia and Resuscitations

Definition: Ventricular rate of 60 or less beats per minute

Step 1: Determine if pulse is present

NO PULSE
START CPR

PULSE PRESENT
1. Identify rhythm
2. Treat hypoxemia
3. Obtain 12 lead EKG
4. Determine if patient is stable or unstable

Step 2: Determine if patient is unstable and act accordingly (Skip to Step 3 if stable)

1. Signs of unstable bradycardia → Administer atropine 0.5 -1 mg for a total of 3 mg
   - Signs of hypo-perfusion
   - Respiratory distress
   - Pulmonary edema
   - Shock
   - Altered mental status

2. If Atropine is ineffective, prepare for emergency transvenous pacing while considering the following temporizing measures
   - Epinephrine
   - Dopamine
   - Isoproterenol
   - Transcutaneous pacing (requires sedation as it is very painful!)
   - If on Beta blockers and CCB consider glucagon for reversal

3. Correct Underlying Etiology, see below

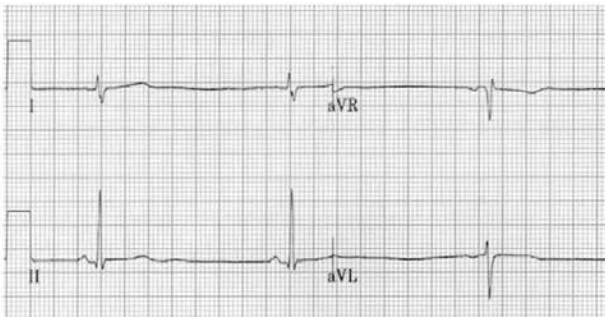
Step 3: If patient is stable, determine if symptomatic

1. Symptomatic, stable bradycardia: most patients can be observed and will not require intervention
   - Patients with severe symptoms: administer atropine
   - If second degree AV block, MOBITZ II or third degree AV block is present and the patient is symptomatic: start transcutaneous pacing or transvenous pacing
2. Asymptomatic bradycardia
   - Usually no treatment is required. If second degree AV block, MOBITZ II or third degree AV block is present: consider transcutaneous pacing or transvenous pacing.

Step 4: Subsequent Management

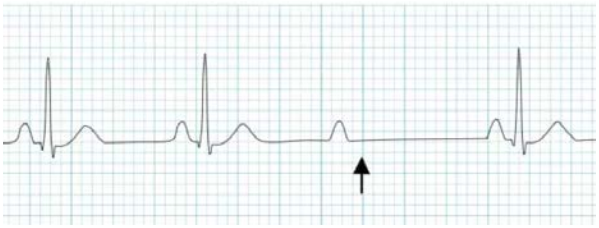
- Perform a focused history and examination
- Identify and treat underlying etiology:
- Hypoxemia
- Sinoatrial dysfunction
- Drugs: Parasympathetic Agents (acetylcholine, carbachol, acetylcholinesterase inhibitors) , Sympatholytic drugs (beta blockers, methyldopa, clonidine, opioids and sedatives, cimetidine, digitalis, calcium channel blockers, amiodarone
- Myocardial infarction (especially right coronary artery)
- Obstructive Sleep Apnea
- Exaggerated vagal activity
- Increased intracranial pressure, strokes, cervical, thoracic spinal trauma
- Infections – Lyme, Chagas disease, legionella, psittacosis, Qfever, typhoid, typhus, babesiosi, malaria, leptospirosis, dengue
- Hypothyroidism
- Genetic: long QT syndrome, catecholaminergic polymorphic ventricular tachycardia syndrome
- Consult cardiology and consider indications for permanent pacemaker (e.g third degree AV block)

Sinus Bradycardia



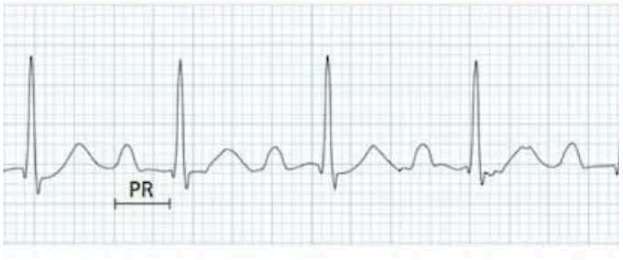
Regular sinus rhythm, rate of 35

Second Degree Heart Block Mobitz



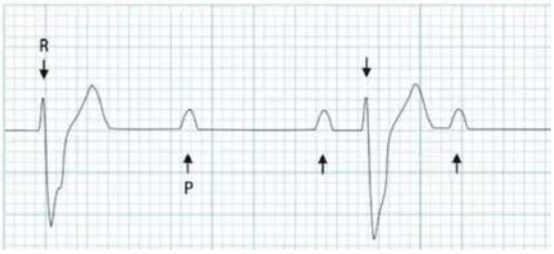
Intermittent non-conducted P waves without progressive prolongation of the PR interval

First Degree Heart Block



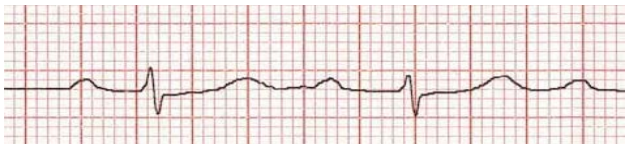
PR interval > 200 ms
There is delay, without interruption, in conduction from atrial to ventricles

Complete Heart Block



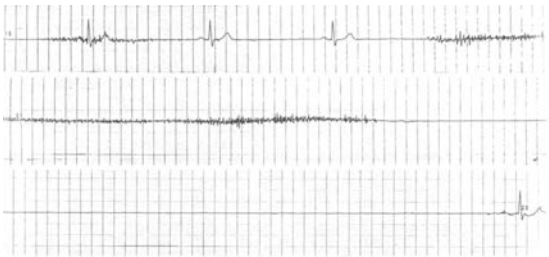
AV dissociation, with the atrial rate (~100 bpm) independent of the ventricular rate (~40 bpm)

Second Degree Heart Block Mobitz Type I



Progressive prolongation of the PR interval culminating in a non-conducted P wave. Note that PR interval is longest immediately before the dropped beat and PR interval is shortest immediately after the dropped beat

Sinus Pause



Prolonged absence of sinus node activity (absent P waves) > 3 seconds.

Temporary Cardiac Pacing

Temporary cardiac pacing involves electrical cardiac stimulation to re-establish circulatory integrity and normal hemodynamics that are acutely compromised by bradyarrhythmia or tachyarrhythmia until it resolves or until long-term therapy can be initiated. Any symptomatic indication for permanent cardiac pacing is potentially an indication for temporary cardiac pacing including:

1. Acute myocardial infarction with bradycardia
2. Electrolyte disturbances, toxicities, and drug-induced causes for bradycardia
3. Injury to the sinus or AV node or His-Purkinje system after heart surgery.
4. Lyme disease and Chagas disease with AV block
5. Heart transplantation
6. Cardiac trauma, Subacute bacterial endocarditis with an aortic valve abscess damaging the His-Purkinje system and causing AV block
7. Acute heart block may occur during transcatheter aortic valve replacement
8. Repetitive monomorphic ventricular tachycardia requiring overdrive pacing

Valvular Disease: Aortic Stenosis

Definition: Reduced systolic opening of the aortic valve

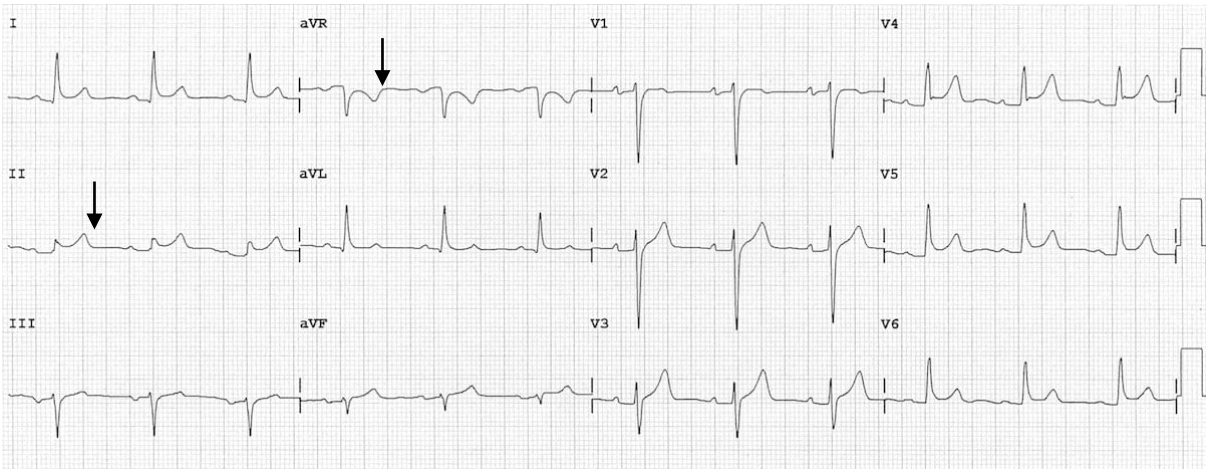
Severe aortic stenosis is defined as aortic valve area ≤1 cm^2, transvalvular jet velocity ≥4 m/sec and mean aortic pressure gradient ≥40 mmHg **(Remember 1, 4, 40!)**

Common etiologies include congenital aortic valve disease, calcific degeneration and rheumatic heart disease. Suspect aortic stenosis in a patient with exertion syncope, angina and dyspnea! Pulsus parvus et tardus along with systolic ejection murmur in the second right intercostal space with radiation to the carotids can be found on physical exam.

Below are the most common indications and contraindications for aortic valve replacement.

Indications for Aortic Valve Replacement	**Contraindications for Aortic valve replacement**
1.Class I recommendation, level B evidence	**1. Cardiac Contraindications**
a. Severe high gradient AS with symptoms b. Asymptomatic patients with severe AS and LVEF < 50% c. Severe AS when undergoing other cardiac surgery	• **Valvular:** **a.** Congenital unicuspid, bicuspid or non-calcified aortic valve **b.** Severe mitral regurgitation **c.** Mixed aortic valve disease (concomitant aortic regurgitation), or significant aortic disease • **Non valvular:** **1.** Left ventricular ejection fraction < 20%, **2.** Severe pulmonary hypertension with right ventricular dysfunction **3.** Echocardiographic evidence of intra-cardiac mass, thrombus or vegetation
2.Class IIa recommendation, level B evidence	**2. Non cardiac contraindications**
a. Asymptomatic severe AS and low surgical risk b. Symptomatic with low-flow/low-gradient severe AS	**i.** Life expectancy less than 12 months owing to a non-cardiac cause **ii.** MRI confirmed CVA or TIA within last six months **iii.** End-stage renal disease **iv.** Need for emergency surgery

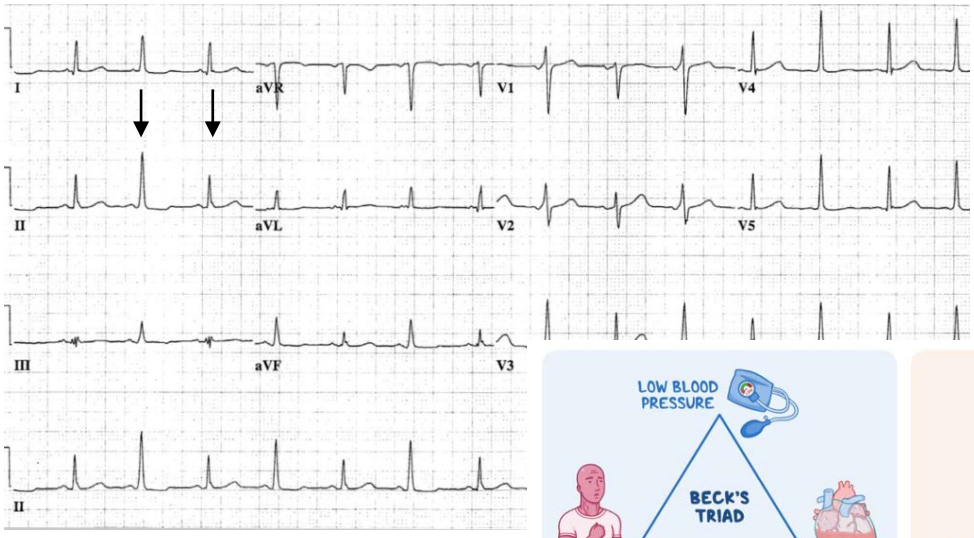
Acute Pericarditis



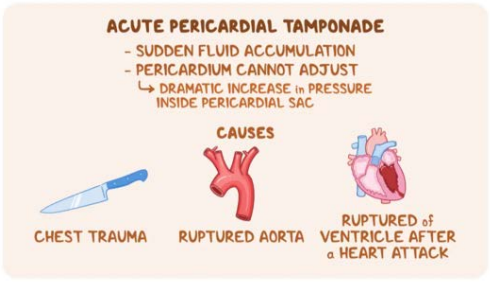
Diffuse concave-upward ST-segment elevation, ST-segment depression in aVR, and PR-segment depression is best demonstrated in leads II and V3. Note lack of reciprocal ST-segment changes, an important feature differentiating acute pericarditis from acute myocardial infarction

Presentation: • Pleuritic chest pain relieved when leaning forward • Persistent fever • Recent viral illness or COVID vaccine • Hemodynamic compromise suggesting cardiac tamponade • Immunocompromised or immunosuppressed • Acute trauma	**Etiologies:** • Idiopathic most likely viral, neoplastic, tubercular, autoimmune or bacterial causes
Diagnosis: • EKG as above • CXR • ESR/CRP • TTE (absence of effusion does not rule out diagnosis)	**Management:** • NSAIDs • Colchicine • Treat underlying cause and if necessary drain associate effusion • Consider prednisone in severe cases or in pericarditis caused by uremia, connective tissue disease or autoreactivity • Consider gastroprotective therapy in patients at risk for GI bleeding

Cardiac Tamponade



Alternating QRS complex height best seen in lateral precordial leads; rare to see EKG changes



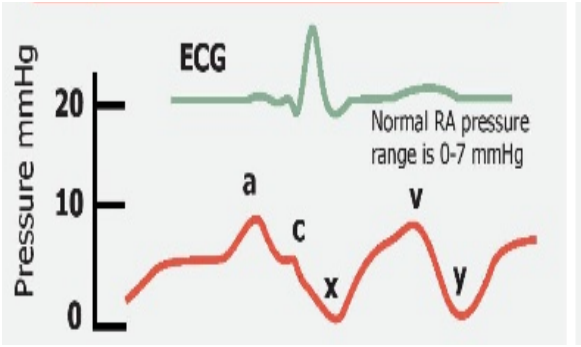
Definition: elevated intra-pericardial pressure from a pericardial effusion compressing the heart (especially the right ventricle)

Presentation: • Sinus tachycardia • Hypotension • Elevated JVP • Pulsus Paradoxus • Pericardial rub • Beck's triad	**Etiologies of pericardial effusion:** • Idiopathic • Infectious: Viral (coxsackievirus, echovirus, adenovirus, EBV, CMV), Bacterial (Myocobacterium TB, Staph, Strep, Hemophilus, Neisseeria, Borrelia), Fungal or Parasitic • Autoimmune
Diagnosis: • TTE findings: chamber collapse, abnormal venous flows, exaggerated respiratory variation of cardiac and venous flows • EKG: low voltage, electrical alternans, tachycardia • CXR: enlarged cardiac silhouette with clear lung fields • Dx confirmed by clinical and hemodynamic response to pericardial fluid drainage	**Management:** **a.** Pericardial fluid drainage is definitive treatment

Pulmonary Artery Catheter

The catheter: Quadruple lumen catheter with thermodilution sensor attached to a pressure transducer with a balloon that can inflate and deflate

Pathway of insertion:
Main central vein (subclavian, IJ or femoral) →R atrium through the tricuspid valve → RV ventricle outflow tract → Pulmonary valve → Pulmonary artery

	1. a wave: contraction of atrial systole 2. c wave: closure of the tricuspid valve 3. x descent: atrial diastole (fall in RA pressure 4. v wave: ventricular systole + passive atrial filling in atrial diastole 5. y wave: RA pressure decrease due to opening of the tricuspid valve + passive filling of RV

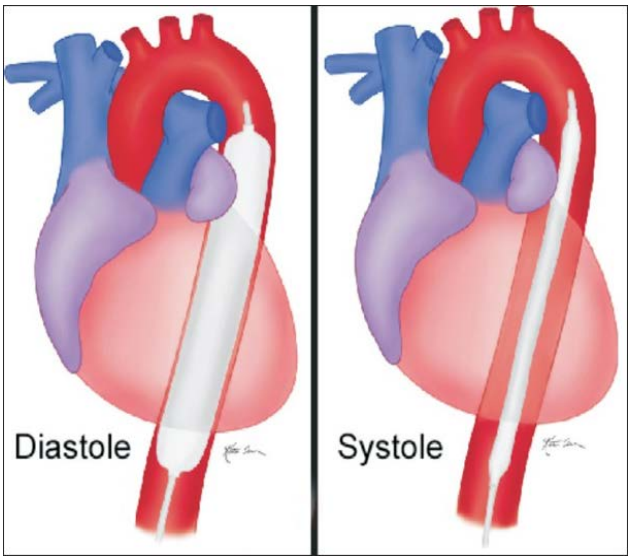
Indications for use:

Diagnosis	Management
1. Differentiation between types of shock (cardiogenic, hypovolemic, distributive, obstructive) 2. Differentiation between cardiogenic vs non-cardiogenic cause of pulmonary edema 3. Evaluation of pulmonary hypertension 4. Diagnosis of pericardial tamponade 5. Diagnosis of L-to-R intracardiac shunt	1. Titration of inotropes in cardiogenic shock or complicated MI 2. Assessment of volume status during aggressive diuresis and heart failure 3. Initiation and titration of pulmonary vasodilators in pulmonary hypertension

Contraindications:

- Right heart mass/tumours
- Tricuspid valve endocarditis
- Tricuspid stenosis or pulmonary stenosis

Intra-aortic balloon Pump



An intra-aortic balloon pump is an intravascular balloon approximately 20-50 cc in volume placed within the thoracic aorta via catheterization procedure. The balloon inflates and deflates synchronized with the cardiac cycle using EKG or aortic pressures. The balloon inflates during diastole and deflates during systole.

Inflation during Diastole -> Increase in pressure in the proximal aorta results in increased coronary flow -> increased myocardial perfusion

Deflation during systole -> Decreased pressure in proximal aorta -> decreased afterload which decreases myocardial oxygen demand

Indications	Contraindications
1. Cardiogenic shock 2. Intractable angina 3. Low CO in cardiopulmonary bypass 4. Bridge to further therapy in intractable myocardial ischemia, intractable ventricular arrythmias and heart failure	1. Significant Aortic regurgitation (AR) as balloon inflation during diastole will worsen AR 2. Aortic dissection or Aortic aneurysm 3. Uncontrolled sepsis or bleeding disorder 4. Severe PAD that cannot be treated with stenting 5. Lack of definitive therapy, IABP is only a temporary treatment

Prophylactic treatment for pts on IABP

1. Prophylactic antibiotics (cefazolin 1g IV Q8H or vancomycin 1g Q12H)
2. Therapeutic anticoagulation

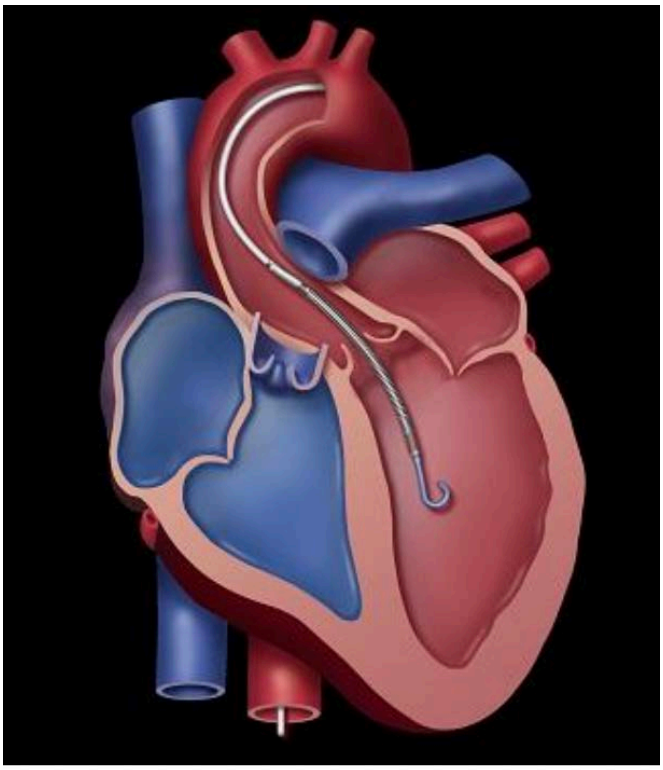
Daily assessment for pts with IABP

1. Daily chest x-ray to monitor position -> catheter tip should be at the level of bifurcation of left and right main bronchi
2. Check distal pulses TID. Check insertion site to assess bleeding
3. Assess IABP waveform daily for appropriate timing of balloon inflation
4. Monitor for hemolysis with hematocrit, platelet count and creatinine. If dropping, consider hemolytic workup
5. Anticoagulation with therapeutic heparin, Daily PT-INR

Weaning off IABP

IABP support is measured in ratios. Full IABP ratio is 1:1, i.e 1 supported beat for every 1 native beat. The ratio is decreased from 1:2 then 1:4 down to 1:8, i.e the ratio of native unassisted beats to supported beats is increased with concurrent monitoring for hemodynamic stability. If the patient is able to maintain hemodynamic stability with fewer supported beats the IABP can be removed.

Impella



The current left sided Impella device is a catheter based ventricular assist device which pumps blood from the left ventricle into the ascending aorta and helps maintain systemic circulation at 2.5 to 5L/minute. This results in immediate and sustained unloading of the left ventricle.

In comparison, the ability of the IABP to augment cardiac output is very modest; no more than 0.5L/min. By continuously drawing blood from the LV, the Impella unloads the LV thereby decreasing LV work and myocardial oxygen demand. This also results in an increase in mean arterial pressure and cardiac output, resulting in improved systemic perfusion and coronary flow. Finally, Impella leads to a decrease in pulmonary wedge pressure and a secondary reduction in right ventricular afterload,

Indications	Contraindications
1. Acute myocardial infarction complicated by cardiogenic shock 2. Facilitate high risk PCI 3. Cardiomyopathy in acute decompensation	1. Presence of thrombus in the LV 2. Mechanical heart valve 3. Severe aortic valve stenosis/moderate to severe aortic valve regurgitation 4. Severe peripheral artery disease (will preclude attempting placement via femoral artery in which case axillary artery approach can be attempted)

Prophylactic treatment for pts with Impella

1. Therapeutic anticoagulation

Daily assessment for pts with Impella

1. Peripheral pulses
2. Daily labs: Hematocrit, platelet count and creatinine
3. Anticoagulation with therapeutic heparin, Daily PT-INR

Most common complications (Assess daily)

Limb ischemia, vascular laceration necessitating surgical repair, major hemorrhage

Other mechanical circulatory support devices include TandemHeart and VAECMO which we will not include here

Procedures in Electrophysiology

Electrical cardioversion and defibrillation

Electrical cardioversion an electric shock is delivered in synchrony with patients QRS. This results in extension of refractory period of myocardial cells thereby preventing the propagation of reentrant circuits and allowing the myocardium to reset. In contrast the electric shock delivery is not synchronized to the QRS complex in defibrillation

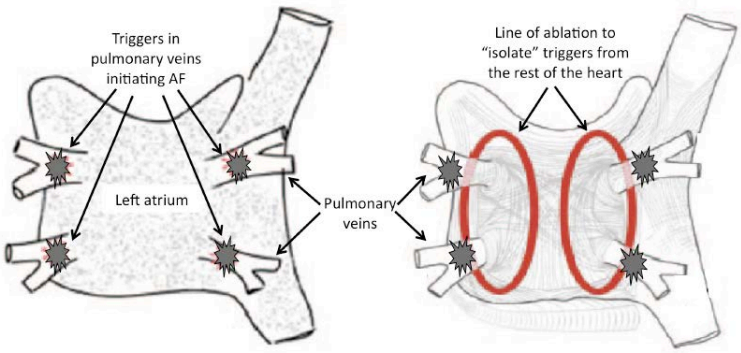
	Cardioversion	**Defibrillation**
Shock delivery	Delivery is synchronized to the QRS complex	Delivery is not synchronized and occurs randomly during the cardiac cycle
Indications	1. Atrial fibrillation 2. Atrial Flutter 3. Supraventricular tachycardia (persistent atrioventricular (AV) nodal reentry, AV reentrant tachycardia, and atrial tachycardia) 4. Ventricular tachycardia	1. Ventricular fibrillation 2. Ventricular Tachycardia with hemodynamic instability
Contraindications	1. Known Atrial thrombus 2. Digitalis toxicity (heart sensitive to electrical pulse, cardioversion can trigger more arrythmias including VFib) 3. Unknown duration of Afib/A Flutter in hemodynamically stable non-anticoagulated pt without prior TEE	

TEE	For patients who have not received adequate anticoagulation. TEE is done prior to cardioversion to assess for the presence of a left atrial thrombus. Pt is cardioverted if no thrombus is observed
Complications	1. ECG changes (ST and T wave abnormalities) 2. Arrhythmias 3. Thromboembolism 4. Myocardial necrosis 5. Myocardial dysfunction (LV dysfunction) 6. Transient Hypotension
Efficacy	-Rate of Successful conversion to sinus rhythm ~75-95%. -whether pts remain in Sinus rhythm is dependent on underlying arrhythmia, response to antiarrhythmics and underlying structural heart disease.

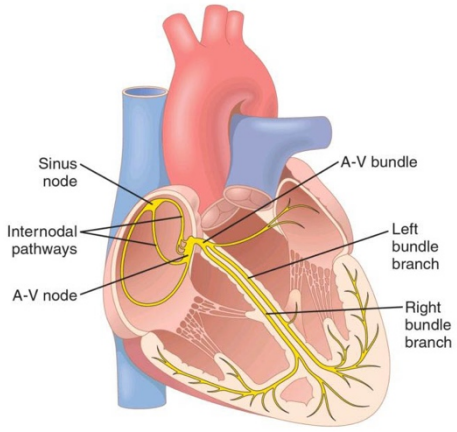
Electrophysiology study

In EP study, a catheter is inserted into the heart to record intracardiac electrical signals. These recordings can be used to identify circuits of conduction that may be causing the arrhythmia. These intracardiac signals are mapped and activation sequences are identified. During EPS procedure response to pacing can be measured, as well various cardioactive drugs (isoproterenol, procainamide, adenosine) can be used to induce arrhythmias for mapping purposes. Once mapped, ablation can be performed to stop conduction of electrical signals through the abnormal circuit.

EP study	
Indications	1. To evaluate tachyarrhythmia/bradyarrhythmia and reproduce/treat reentrant cardiac rhythms 2. Evaluate accessory pathways 3. As part of the evaluation for cardiogenic syncope
Pulmonary Vein Isolation	Electrical isolation of pulmonary veins through ablation to prevent the propagation of electrical signals that underly atrial fibrillation.
Pre-procedure	Anticoagulation for at least for 4-6 weeks prior to procedure
Indications	Symptomatic atrial fibrillation refractory to antiarrhythmics
Procedure	Goal is to create entry and exit blocks in the antrum of the pulmonary veins through ablation. Voltage is then measured and the goal is to decrease the atrial electrogram by at least 90% or less the 0.05 mV.

	
Complications	1. Cardiac tamponade 2. Pulmonary vein stenosis 3. Stroke (most often embolic) 4. Esophageal thermal injury from ablation 5. Formation of an Atrial esophageal fistula (can lead to air embolism and stroke)

Intracardiac Devices

Pacemaker	Electrical device made up of pulse generator and Leads (electrodes) that attach to heart chamber to deliver electrical signal
Indications 	**Symptomatic bradyarrhythmia** **->** dizziness, lightheadedness, syncope, fatigue, and poor exercise tolerance Establish association between bradyarrhythmia and symptoms VIA ambulatory monitoring (cardiac monitor) **Location of conduction abnormalities** • Abnormality within the AV node i.e. 1° AV block and 2° Mobitz type 1 with normal QRS may not require pacemaker. • Abnormalities below AV node i.e. 2° Mobitz type II , 3° (complete) AV block, with widened QRS complex -> likely to benefit from permanent pacemaker. **Class I** 1. Symptomatic sinus bradycardia with clear association between symptoms and bradycardia (HR<40, frequent sinus pauses) 2. Symptomatic chronotropic incompetence -> inability to achieve 85% of the age-predicted

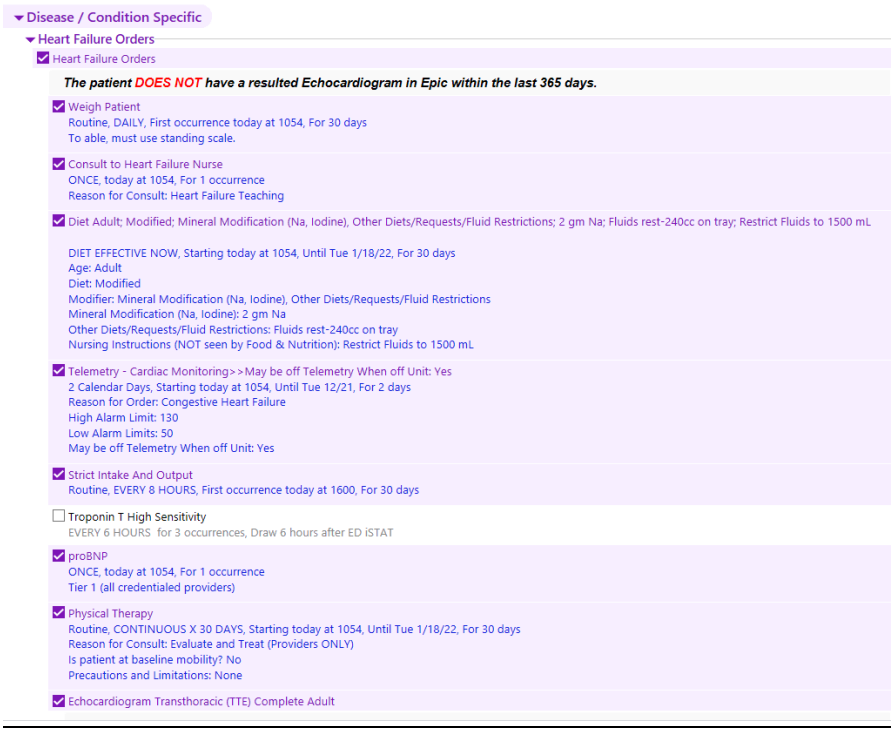
	maximum HR during a formal or informal stress test or during ADLs 3. Symptomatic Mobitz II 2° AV block with associated symptoms or a widened QRS **Class II (Sinus node dysfunction)** 1. Sinus bradycardia in a patient with symptoms but no clear association 2. Sinus node dysfunction in a patient with unexplained syncope. 3. Chronic HR <40 beats per minute while awake and minimally symptomatic patient **Class II (AV node dysfunction)** 1. Asymptomatic Mobitz II 2° AV block with narrow QRS 2. 1° AV block when there is hemodynamic compromise due to AV dissociation 2/2 to a very long PR interval. 3. Bifascicular or trifascicular block associated with syncope 2/2 transient 3° AV block (complete heart block)
AICD	Automated Implantable cardioverter-defibrillator is a device that is able to monitor the heart rhythm in real time and deliver a shock if pt enters life threatening arrhythmia.
Indications	Prevention of sudden cardiac death (SCD) in patients with VT or VF can be either **1° prevention** in pts at risk of life threatening VT/VF (already on optimal medical management) **2° prevention** in pts who have had previous episode of sustained VT/VF
Effect of Magnet on ICD	**In pacemakers** magnet application will result in asynchronous pacing with fixed AV delay. **In AICDs** magnet application results in suspension of rhythm monitoring and shock deliver. There is no effect on pacing.

Device interrogation	Used for routine evaluation of ICD. Allows you to review electrical activity recorded in device memory and ensure that device is working appropriately. Adjustments to device settings can be made.

Quality Improvement Toolkit

UPON ADMISSION

Heart Failure Admission Orders – Admission → Disease/Condition specific → Heart failure orders → Follow prompts



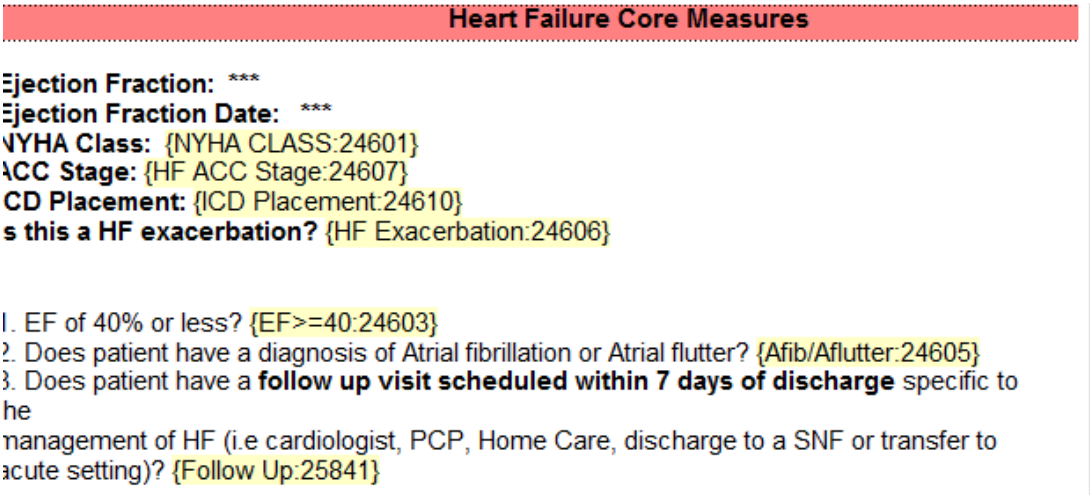
- Disease / Condition Specific
  - Heart Failure Orders
    - Heart Failure Orders

The patient DOES NOT have a resulted Echocardiogram in Epic within the last 365 days.

- Weigh Patient
  Routine, DAILY, First occurrence today at 1054, For 30 days
  To able, must use standing scale.
- Consult to Heart Failure Nurse
  ONCE, today at 1054, For 1 occurrence
  Reason for Consult: Heart Failure Teaching
- Diet Adult; Modified; Mineral Modification (Na, Iodine), Other Diets/Requests/Fluid Restrictions; 2 gm Na; Fluids rest-240cc on tray; Restrict Fluids to 1500 mL

  DIET EFFECTIVE NOW, Starting today at 1054, Until Tue 1/18/22, For 30 days
  Age: Adult
  Diet: Modified
  Modifier: Mineral Modification (Na, Iodine), Other Diets/Requests/Fluid Restrictions
  Mineral Modification (Na, Iodine): 2 gm Na
  Other Diets/Requests/Fluid Restrictions: Fluids rest-240cc on tray
  Nursing Instructions (NOT seen by Food & Nutrition): Restrict Fluids to 1500 mL
- Telemetry - Cardiac Monitoring>>May be off Telemetry When off Unit: Yes
  2 Calendar Days, Starting today at 1054, Until Tue 12/21, For 2 days
  Reason for Order: Congestive Heart Failure
  High Alarm Limit: 130
  Low Alarm Limits: 50
  May be off Telemetry When off Unit: Yes
- Strict Intake And Output
  Routine, EVERY 8 HOURS, First occurrence today at 1600, For 30 days
- ☐ Troponin T High Sensitivity
  EVERY 6 HOURS for 3 occurrences, Draw 6 hours after ED iSTAT
- proBNP
  ONCE, today at 1054, For 1 occurrence
  Tier 1 (all credentialed providers)
- Physical Therapy
  Routine, CONTINUOUS X 30 DAYS, Starting today at 1054, Until Tue 1/18/22, For 30 days
  Reason for Consult: Evaluate and Treat (Providers ONLY)
  Is patient at baseline mobility? No
  Precautions and Limitations: None
- Echocardiogram Transthoracic (TTE) Complete Adult

.**HFcoremeasures** – Utilize for note as it clearly identifies all needed aspects to follow and manage.



Heart Failure Core Measures

Ejection Fraction: ***
Ejection Fraction Date: ***
NYHA Class: {NYHA CLASS:24601}
ACC Stage: {HF ACC Stage:24607}
CD Placement: {ICD Placement:24610}
s this a HF exacerbation? {HF Exacerbation:24606}

1. EF of 40% or less? {EF>=40:24603}
2. Does patient have a diagnosis of Atrial fibrillation or Atrial flutter? {Afib/Aflutter:24605}
3. Does patient have a **follow up visit scheduled within 7 days of discharge** specific to he
management of HF (i.e cardiologist, PCP, Home Care, discharge to a SNF or transfer to acute setting)? {Follow Up:25841}

DURING HOSPITALIZATION

The Importance of Heart Failure Nurses – Once New diagnosis of Heart Failure is made, or patient has had recurrent hospitalizations for Heart Failure Exacerbations, contact Heart Failure nurses and place consult. It is Important to contact Heart Failure Nurses **EARLY** in admission to assure they are seen prior to discharge.
**Contact HF is included if Heart Failure Admission Order set is utilized.*

UPON DISCHARGE

Discharge Medications – When discharging patients from the CCU especially those who were admitted for MI and interventions were performed, order all new/old cardiovascular medication for **1 YEAR** to assure patient does not run out.

Discharge Follow Up Contact patient primary Cardiologist office to make an appointment for follow up. If patient does not have Cardiologist make sure you notify patient of new Cardiologist and make appointment in similar manner.

Cardiac Rehab – Always discuss Cardiac Rehab with patients following hospitalization, especially those with HF and MI. If not option due to transport or patient living situation offer other options possibly speak to PT/OT

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