Cardiology

- Rule out MI
- CHF
- Endocarditis
- EKG reading made easy
- Syncope

Rule out MI

Admit to Telemetry

- Tele-nurse may reach out to you for > 6 PVCs/min, atrial fibrillation, ventricular fibrillation, > 3 beats of VT.
- Admit as “observation status” if the patient will likely be discharged within 24 hours.

Activity

- Bed rest until ruled out (bedside commode okay for low-risk patient).
- Remember to promote ambulation once myocardial ischemia resolves.

Diet

- NPO except meds if possible cardiac catheterization or functional study in the AM. Applies to most patients, especially as ECGs and biomarkers are being trended.
- Hold beta-blockers before exercise or dobutamine stress testing.
- No nitrates or caffeine before vasodilatory (dipyridamole/ adenosine/regadenason) pharmacologic stress testing. Beta blockers are ok (but hold if unsure what type of test until am).
- Metformin should be held in all patients in case they need to undergo coronary angiography, place on SSI; theoretical risk for lactic acidosis

Nursing

- O2 via nasal cannula, starting at 2 L/min. Goal O2 > 95%.
- ECG on admission, trend as frequently as indicated (with symptoms, to capture evolving infarct, biomarker check, etc)
- CXR on admission.
- Chest pain protocol: vital signs, ECG, NTG 0.4 mg SL q5 minutes x 3, notify MD. ECG during and upon resolution of symptoms.

Labs

- Trend troponin ~q6-8hrs until peak, chem-panel, PT/PTT, lipids, TSH, and HgA1C

- CK-MB and myoglobin are not useful in diagnosis of ACS with contemporary troponin assays.

Medications

- Aspirin 325 mg PO (chewed), 81mg thereafter
- Statin: Atorvastatin 80 mg PO
- B-blockers: Metoprolol tartrate 25-100 mg PO q12hr, consider IV if hypertensive. Target HR < 70. Avoid in bradycardia, severe bronchospasm or hypotension/concern for shock
- See Acute Coronary Syndrome: ST Segment Elevation or New LBBB for further details and contraindications.
- Pain Control (pain means ongoing ischemia/infarct):
- Reduce demand if indicated (blood pressure and heart rate control)
- Ensure proper anticoagulation (i.e. heparin is therapeutic); Plavix loading
- Nitrates (titrate up as needed): nitro SL (NTG 0.4 mg q5 minutes x 3)-> nitro paste -> nitro gtt (CCU level of care). Avoid in suspected RV infarct
- Morphine PRN (watchout for hemodynamic effects)

If in doubt, discuss with seniors!

Consult with cardiologist (i.e. catheterization, GP IIb/IIIa gtt, CCU care), especially for persistent chest pain in the concerning patient.

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CHF

Definition

- A condition in which the heart is not able to pump enough blood to meet the body's metabolic needs or does so in the face of elevated filling pressure due to abnormalities of ejection or abnormalities of filling.

Classification

- In patients presenting with new (or possibly unexplained worsening symptoms of heart failure), document systolic function with echocardiography (HFpEF vs HFrEF), and then exclude ischemic underlying etiology (MI or CAD) with angiography (Ischemic Dilated Cardiomyopathy vs Non-ischemic Dilated Cardiomyopathy).
- Classification of heart failure is important not only for more effective communication but for optimization of medical management

Overview of Heart Failure with Reduced Systolic Function

- Ischemic Etiologies: Coronary Artery Disease (major) and congenital coronary anomalies; myocardial bridging
- Non-Ischemic Etiologies: HTN, valvular heart disease, idiopathic, tachycardia mediated, toxins (EtOH, cocaine, meth, anthracyclines, etc), end-stage infiltrative disease (amyloid, hemochromatosis, sarcoid), endocrine (hypo/hyperthyroidism), myocarditis (viral, Chagas, HIV), radiation, peripartum, vitamin deficiencies (thiamine), genetic/familial,
- Etiologies of Exacerbations: poor dietary and/or medication adherence, myocardial ischemia, inadequate drug therapy, HTN, arrhythmia (e.g., atrial fibrillation), valvular heart disease, fluid overload, PE, infection, thyrotoxicosis, physical or emotional stress.

Evaluation of Acute Heart Failure

- Retrieve old ECHO, stress tests, ECGs, and cath reports.
- Take a thorough history (dyspnea, orthopnea, PND, edema, weight gain, abdominal discomfort, nausea, anorexia, baseline weight).
- Physical examination and clinical assessment:
- Is the patient warm or cold (reduced perfusion)? Look for fatigue, altered mental status, Cheyne-Stokes respiration, pallor, cool extremities, narrow pulse pressure, lactic acidosis, hyponatremia, renal failure.

- Is the patient dry or wet (congested)? (See chart below.)
- Left Sided Congestion (crackles, pulmonary edema, left ventricular S3, elevated JVP) +/- Right-sided congestion: elevated JVP, lower extremity edema, hepato-jugular reflex, pulsatile liver, right ventricular S3
- Labs: CBC, chem-10, coagulation studies, troponin, TSH, ECG (e/o ischemia, hypertrophy, pericarditis, new bundle branch block), CXR (vascular prominence, effusion, cardiomegaly).
- Diagnostic utility of B-type natriuretic peptide (BNP):

1. *BNP is released from the heart (mainly the LV) in response to increased wall tension.*
2. *BNP measurement has limited utility after a good clinical assessment. If the diagnosis of HF is clinically apparent, a BNP test is not indicated. Measurement of BNP levels should not be used as a screening test. If the clinical picture is consistent with HF, do not let a low or normal BNP level necessarily deter appropriate diagnosis and therapy.*
3. *In one study, BNP < 100 pg/mL was helpful in ruling out cardiac causes of dyspnea (<50 pg/mL had 96% negative predictive value for CHF) while BNP > 400 pg/mL is helpful for ruling in cardiac causes of dyspnea. For values <100 pg/mL and >400 pg/mL, consider other causes of dyspnea as well as CHF.*
4. *The level of BNP correlates with the severity of HF. However, patients who are stable and well-compensated may run high BNP levels chronically.*
5. *Obese patients have decreased BNP levels due to adipose clearance.*
6. *Higher baseline levels of BNP are seen in older patients and women. BNP is falsely low in obesity, diuretics, ACEi, beta-blockers, and aldosterone antagonist use. BNP is low in constrictive pericarditis*

> Acute Management of Heart Failure Exacerbation.

Therapy should be tailored to the hemodynamic profile (Volume status vs Perfusion) as described below:

1. *Volume Overload (Wet) with Good Perfusion (Warm)* -> Most common hospital presentation of CHF. IV diuretics ± nitrates, afterload reduction (ACEI/ARB, hydralazine, nitrates), ± aldosterone antagonists and beta-blockers when optimized.
2. *Volume Overload (Wet) with Poor Perfusion (Cold*) -> Likely requires ICU care. IV diuretics, ± afterload reduction, ± inotropes/inodilators. In severe or refractory cases, sometimes PA catheter-guided therapy* is needed, PUF/CVVH, consideration of advanced therapies (LVAD, transplant evaluation).
3. Non-volume Overload (Dry) with Poor Perfusion (Cold) -> Represents 10% of cases, challenging to treat. Often associated with cardio-renal syndrome. Inotropes/inodilators, afterload reduction, and advanced therapies can be considered.
4. *Non-volume Overload (Dry) with Good Perfusion (Warm*) -> Compensated HF. Usually can be treated as an outpatient. Maintain volume status and prevent disease progression with rx. Chronic management with beta-blockers, ACEI/ARB, aldosterone antagonists, loop diuretics.

In general patients should be closely monitored:

- Telemetry monitoring (high risk for ventricular arrhythmia)
- Oxygen via NC or face-mask, goal >92% saturation. If needed, CPAP or BiPAP confers mortality benefit and reduces the need for mechanical ventilation.
- Monitor K, Mg, Cr (remember Cr may be elevated on admission secondary to venous congestion at the glomerulus; if HF-related, Cr should fall with diuresis). Do not hold diuretics for BP.
- Restrict sodium (<2g) and also fluids (<1500cc) if hyponatremic; daily weights, strict I&Os.

> Overview and Specifics of Treatment

1. Immediate Considerations: Diuresis, Inotropes, Afterload reduction
2. Guideline Directed Therapy: ACE-i, Beta blockers, Aldosterone antagonists, Hydralazine/Nitrates
3. Devices: ICD, CRT, CRT-D
4. Advanced Therapies: Mechanical circulatory support, Transplant

Diuresis:

Loop diuretics:

- For Veno-dilation and natriuresis. With advanced right heart failure and diuretic resistance, use IV as oral absorption is diminished by bowel edema. Decongestion of hepatic and renal vascular beds will improve organ function. Reduction in LV wall stress and mitral regurgitation will improve cardiac output and BP.
- Initial dosing: 2.5 times normal oral diuretic dose (DOSE trial). If inadequate response (< 100 – 200mL UOP in first 30 mins), double dose and closely monitor UOP
- Consider furosemide/bumex gtt in patients with severe volume overload and/or sub-optimal response to bolus dosing of diuretics.
- Furosemide gtt: Give initial 20-80 mg IV bolus (higher doses for patients with worse renal function; lower doses for diuretic naïve patients). Then start gtt at 5 mg/hr (range 5-20 mg/hr). FENa can be monitored to determine effectiveness of natriuresis (maximum FENa = 20%).
- Bumex gtt: Give initial 1-2 mg bolus. Then start bumex gtt at 0.5 mg/hr (range 0.2-2 mg/hr).
- If inadequate diuresis with loop diuretic alone, try adding a thiazide diuretic such as chlorothiazide IV/PO 125-500 mg or metolazone 2.5-10 mg PO, diuril IV dosed 30 min before the loop diuretic. Metolazone PO should precede IV loop diuretic by at least 2-4 hours. Monitor serum electrolytes (Na, K+, and Mg2+) closely, replete as necessary.
- Markers of adequate diuresis include resolution of dyspnea, decreased JVP, Cr normalization, return to baseline weight. JVP should be closely monitored to avoid ‘bumping patient’s creatinine.’
- Transition to PO diuretics and monitor response to PO prior to discharge

Ultrafiltration:

Fluid removal with no effect on serum electrolytes, consideration reserved for in cases of acute decompensated heart failure inadequately responsive to aggressive diuretic regimen. Increased risk of serious adverse advents and no difference in weight loss at 96 hours when compared to pharmacologic therapy (CARRESS-HF).

Optimizing Hemodynamics/Inotropes:

Dobutamine:

- Beta-adrenergic agonist (predominantly beta-1), inotrope, mild arterial vasodilator. Generally the inotrope of choice in cardiogenic shock.
- Minimal effects on left sided filling pressures. At higher doses (>10-15 mcg/kg/min) can exert significant alpha-adrenergic activity increasing SVR and worsening forward flow. Effectiveness curtailed with concomitant beta blockers.
- Adverse effects: increased myocardial oxygen demand, hypotension, ventricular arrhythmias
- Tolerance can develop with beta receptor down-regulation.
- Normal dose range: 2.0-5.0 mcg/kg/min, can be run peripherally
- Titrate to UOP, CO, SvO2 if a right heart catheter is in place.

Dopamine:

- Low dose acts through dopamine receptors increasing renal blood flow and natriuresis (1-3 µg/kg/min). Intermediate doses act through beta-adrenergic receptors to increase HR and contractility (2-10 µg/kg/min). High doses stimulate alpha-adrenergic receptors increasing afterload, this may be detrimental in HF (>10 µg/kg/min).
- Use only to stabilize hypotensive patients.
- Can run peripherally for <24 hours as long as a viable PIV has been established

Milrinone:

- Phosphodiesterase-3 inhibitor, inodilator
- Hemodynamic profile: Reduces right- and left-sided filling pressures, reduces pulmonary and systemic vascular resistance, direct positive inotropic effect. Longer half-life than dobutamine, more effective in beta-blocked patients and suspected dobutamine unresponsiveness due to beta receptor down-regulation.
- Adverse effects: Toxicity profile similar to dobutamine, although slightly less tachycardia and myocardial oxygen demand.
- Normal dose range: 0.375-0.75 mcg/kg/min; starting dose in renal dysfunction is 0.25 mcg/kg/min.
- Do not give a bolus prior to starting infusion even though doing so is suggested in the package insert. Bolus dosing is associated with more hypotension and is rarely indicated.

Digoxin:

- Na-K-ATPase inhibition improves contractility, anti-andrenergic effect also beneficial. The only safe PO inotrope. Symptomatic improvement and decrease in hospitalizations, but no mortality benefit in HF (DIG trial).
- Normal dose range: 0.125-0.25 mg daily, loading dose not indicated in HF.
- Optimal serum concentrations: 0.5-0.8 ng/mL in men, 0.5-0.9 ng/mL in women
- Adverse effects: confusion, visual disturbances, arrhythmias (conduction disease, pAT)

Nitrates:

- Preferential venodilator useful in acute setting to reduce pulmonary edema and other congestive symptoms. Reduces LV filling pressure and SVR.
- Sublingual, nitropaste, or IV nitroglycerin (start at 10-20 mcg/min and titrate as permitted by BP).
- When stable, convert to PO nitrate (e.g. start with isosorbide dinitrate 10 mg PO TID).
- Avoid with concurrent PDE-5 inhibitor, NO reductase inhibitor, severe aortic stenosis, cyanide toxicity.
- Adverse effects: headache (common), hypotension, tachyphylaxis

Guideline-Directed Therapy

ACE inhibitors:

- For afterload reduction and neuro-hormonal effects. Have a proven mortality benefit (CONSENSUS trial), prevent re-hospitalization, stop adverse LV remodeling in chronic HF (SOLVD trial). This effect is independent of the anti-hypertensive effect.
- If ACE inhibitor causes cough, try ARB (angiotensin II receptor blocker). Always attempt re-challenge with ACE inhibitor in future as ACE confers improved mortality benefit over ARB.
- Dosing: Captopril 3.125 – 6.25 mg PO q8hr and increase dose by dose as BP permits.
- Monitor K+, and Cr (up to 30% increase in Cr is acceptable).
- Once stable, switch to equivalent dose of once-daily ACE inhibitor.
- Target dosing: Lisinopril 40mg PO Qday.

Beta-blockers:

- For hemodynamic benefits, beneficial LV remodeling, decreasing frequency of PVCs. Multiple trials showing mortality benefit, reduced hospitalizations, symptom mitigation (MERIT-HF, PRECISE, CIBIS-I & II, etc.).
- Metoprolol ER, carvedilol, and bisoprolol have symptom and mortality benefit and slow disease progression for NHYA class II-IV chronic HF.
- Do not initiate in acute setting (OPTIMIZE-HF trial). Once patient is adequately diuresed, initiate beta blocker with appropriate up-titration on outpatient basis.
- Do not withdraw in exacerbation unless patient is hypotensive or in cardiogenic shock.
- Initial dosing: Start with metoprolol ER 12.5-25 mg PO daily or carvedilol 3.125 mg PO BID and titrate up as permitted by HR and BP.

- Target dosing: Metoprolol ER 200 mg daily or carvedilol 25 mg BID (50 mg BID if > 85 kg). COMET trial showed greater benefit with carvedilol vs. short-acting metoprolol. Also recent work showed that HR may be more important than final dose.

Aldosterone Antagonists:

- Survival benefit derived from potassium sparing action lowering risk of hypokalemia-associated arrhythmia as well as blocking of mineralocorticoid activity.
- Indicated for the following populations:
- NYHA class II HF + LVEF ≤ 30% (EMPHASIS-HF trial)
- NYHA class III-IV HF + LVEF < 35% (RALES trial)
- recent MI + LVEF ≤ 40% + symptomatic HF or DM (EPHESUS trial)
- Contraindications: Cr > 2.5 mg/dL in men, Cr > 2.0 mg/dL in women, K+ > 5 meq/L.
- Initial dosing: Spironolactone 25 mg daily (every other day dosing for eGFR < 50 mL/min), titrate up to 50 mg daily if K+ < 5 meq L at 4 weeks.
- Target dosing: Spironolactone 25-50 mg daily
- Can consider changing to eplerenone (much more expensive) if undesired endocrine side effects (e.g. gynecomastia) with spironolactone.
- Monitor K+.

Hydralazine + Nitrates:

- Hydralazine is a preferential arterial vasodilator.
- Indicated for black patients with NYHA class III-IV HF and LVEF < 40% on maximal medical therapy.
- Initial dosing: Hydralazine 25 mg TID + isosorbide dinitrate 20 mg TID or combination isosorbide dinitrate-hydralazine 37.5 mg-20 mg TID. Titrate up every 2-4 weeks as permitted by BP.
- Target dosing: Hydralazine 75 mg TID + isosorbide dinitrate 40 mg TID.
- Can consider isosorbide mononitrate (30-120 mg daily) as alternative to TID-dosed isosorbide dinitrate.

ICD and CRT

Indicated only after medical optimization and removal of applicable vices (no active drug use) – please see applicable section.

Implantable cardiac defibrillator (ICD): Primary prevention of sudden cardiac death in populations at increased risk for life-threatening ventricular arrhythmias. Mortality benefit for NYHA class II-III HF + LVEF ≤ 35% despite optimal medical management for at least 3 months (SCD-HeFT trial).

Cardiac resynchronization therapy (CRT): HF frequently leads to intraventricular conduction delay and ventricular dyssynchrony. Resynchronization (biventricular pacing) improves pump function with mortality benefit, symptom improvement, and decreased hospitalizations for NYHA class II-III HF + LVEF ≤ 35% + LBBB + QRS ≥ 150 ms despite optimal medical management (MIRACLE, CARE-HF, COMPANION, MADIT-CRT trials).

Most patients who meet criteria for ICD also meet criteria for CRT and vice versa. Combination devices (CRT-D) confer superior mortality benefit when compared to ICD or CRT alone (REVERSE, MADIT-CRT, COMPANION trials).

Advanced Therapies

Mechanical circulatory support

1. Intra-aortic balloon pump (IABP): Temporary treatment for HF refractory to medical management, systolic unloading and improved coronary perfusion, particularly useful in patients with ischemia or mitral regurgitation, placed in cath lab (see corresponding section).
2. Left ventricular assist device (LVAD): Can be used as a bridge to recovery, a bridge to transplant, or “destination” therapy for outpatient use. Placed in OR.

Cardiac transplantation: Consider early involvement of transplant/advanced HF team in patients with new, rapidly progressive, severe HF, or advanced HF refractory to treatment.

> Overview and Management of Heart Failure with Preserved Systolic Function

Etiologies

- Etiologies include: ischemic heart disease (most common), hypertensive heart disease, diabetic cardiomyopathy, high output cardiac failure (e.g., severe anemia, AV fistulae, thyrotoxicosis), restrictive cardiomyopathy (radiation injury, infiltrative disease [e.g., amyloidosis], metabolic storage disease [e.g., Fabry’s disease], endocardial fibrosis), and hypertrophic cardiomyopathy, valvular heart disease (e.g., AS, MR). Exclude HF due to constrictive pericarditis or due to isolated right heart failure (pulmonary HTN, cor pulmonale)
- Etiologies of exacerbation: Anything that increases LV wall stiffness will predispose to exacerbation of diastolic HF. Common precipitants include ischemia, tachyarrythmia, HTN.

Evaluations

- CBC (anemia), chem-10 (CKD), LFTs (cirrhosis), TFTs (thyrotoxicosis), lipid panel, A1c, urine microalbumin, SPEP/UPEP as indicated
- Diastolic dysfunction can be assessed non-invasively by echo (LVH, impaired filling, valve disease, atrial enlargement, rule out constrictive pericarditis) or invasively by cardiac catheterization.
- If echo is unrevealing, consider cardiac catheterization to evaluate for occult arterial disease and, occasionally, endomyocardial biopsy.
- On exam, look for HTN, S4, signs of LVH.

Treatment

- Similar to treatment for systolic HF as outlined above. Address underlying etiology (ischemia, HTN, infiltrative process, etc)
- Evidence is weak for guiding treatment with commonly used drugs for systolic failure; many studies show marginal or no benefit, nevertheless optimization is important.
- Treat congestion (diuretics, ultrafiltration/dialysis, salt restriction)

Discharge Planning

- All HF patients should receive the pneumococcal and influenza vaccines.
- Advance directive education, palliative care involvement (depending on severity and co-morbities), goals of care discussions for patients with recurrent hospitalizations.
- Early outpatient follow-up and/or telephone nurse follow-up after discharge help reduce readmissions.
- Heart failure education, including dietary modification (fluid and salt restriction), scale and instruction regarding daily weights, diuretic self-titration teaching, avoidance of cardio-toxic recreational drugs, explain rational of all medications.

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Endocarditis

Overview

Diagnosis requires 2 major, 1 major with 3 minor, or 5 minor criteria from the modified Duke Criteria listed below:

MAJOR CLINICAL CRITERIA:

1. Persistently positive blood cultures with typical IE organisms. Bacteremia is continuous and high grade. Two blood cultures are positive > 90% of the time. Repeat the blood cultures every 48 hours until sterile. Prevalence of endocarditis among patients with S. aureus bacteremia is approximately (13-25%); TTE is now recommended in all patients with S. aureus bacteremia.
2. Evidence of valvular vegetation, or abscess, or dehiscence on TEE/TTE.
3. New regurgitant murmur.
4. Serologic dx (Coxiella IgG titer >1:800 or positive Bartonella or C.psittaci titers) or single positive culture of Coxiella burnetii (Q fever).

MINOR CLINICAL CRITERIA:

1. Predisposing condition (see below).
2. Fever (temperature >38.0 C).
3. Vascular events (septic emboli, pulmonary emboli, mycotic aneurysm, CNS or conjunctival, and Janeway lesions).
4. Immunologic events (Osler’s nodes, glomerulonephritis, Roth spots, + Rheum Factor).
5. Microbiologic data not meeting major criteria.

Etiology / Risk Factors

Predisposing conditions include prosthetic valves, previous IE, IDU, structural heart disease (e.g., valvular abnormalities including MV prolapse), hemodialysis and indwelling catheters.

Evaluation

- **Clinical manifestations** are inconsistent, so suspect the condition in those with risk factors. Fever may be absent in up to 10% of patients. 70% have heart murmurs (50% new murmur, 20% worsening of old murmur). Only ~10% of patients have classic

peripheral stigmata of endocarditis (Janeway lesions, Osler nodes, splinter hemorrhages).

- **Physical Exam**
- Cardiac: exam can be dynamic so reassess for new or changing regurgitant murmurs.
- Skin: the most common rash is petechiae; can be on the conjunctiva, palate, or buccal mucosal surfaces. Splinter hemorrhages are non-blanching, often at distal nail bed. Osler nodes are small tender nodules on fingers and/or toes. Janeway lesions are small, macular, painless hemorrhages on palms or soles.
- Eyes: Roth spots are pale, oval retinal lesions surrounded by hemorrhage.
- Full neurologic exam to document baseline and to evaluate for focal findings.
- Other: evaluate for arthritis, vertebral tenderness, and tender splenomegaly.
- **Microbiology** for native versus prosthetic valves differs slightly, although Staph aureus is the most common organism in both.
- **Early vs Late prosthetic valve infections**: early infections are typically acquired from surgical-related bacteremia within days to weeks. Because the valve and sutures are not yet endothelialized, bacteria easily adhere to these surfaces. They are more likely to cause valve dehiscence or abscess.
- Early organisms (<2 mo): Staph > CoNS (Staph epidermidis).
- Late organisms: CoNS and Staph aureus are roughly equivalent.
- Infections in valves > 12 mo after surgery are similar to those of native valves, with the exception of bioprosthetic valves, which may breakdown over time and become more susceptible to infection.
- **Echo**: in native valve endocarditis, the sensitivity of TTE is ~60% and TEE is ~90%. Specificity is ~99% for both. Guidelines recommend TEE first but practically, we usually begin with TTE and consider TEE if negative if there remains a high pre-test probability. Consider TEE in patients with:
- High concern for native-valve endocarditis and a negative TTE.
- Prosthetic valves (TTE is 15-30% sensitive), cardiac device (e.g. pacemaker, AICD) or prior IE.
- Limited thoracic windows (obesity, COPD, mechanical ventilation).
- Concern for perivalvular complications (e.g. myocardial abscess).
- **Culture negative endocarditis**: defined as negative cultures after 7 days. One of the most common causes is antibiotic administration before cultures. The most common infectious causes in America are Bartonella spp and Coxiella burnetii (Q fever). Other pathogens: Chlamydia, Legionella, Brucella. Note, the HACEK organisms are no longer a common cause of culture negative endocarditis as they can be cultured by most microbiology labs.

Management

- All patients need prolonged courses of antibiotics and may need surgery.
- ID should be consulted for all cases of documented endocarditis.
- Empiric therapy for native valve IE in most patients is vancomycin (targeted to a goal trough of 15-20) plus ceftriaxone 2gm IV q24h, started after cultures have been drawn. Subsequently tailor antibiotics to culture results.

- Oral antibiotic step-down therapy: Not yet standard of care but POET (https://www.nejm.org/doi/full/10.1056/NEJMoa1808312) study found that oral step down therapy was noninferior in their population to full-course IV therapy. Caveats included only Streptococcus, MSSA, CoNS, or Enterococcus faecalis organisms and few PWID.
- **Surgical indications** in native valves include:
- Valve dysfunction (i.e. aortic or mitral) causing heart failure.
- Perivalvular extension with development of abscess, fistula, and/or heart block.
- Fungi or orther highly resistant organisms that are difficult to treat with antibiotics alone.
- Persistent bacteremia despite maximal treatment, indicating a lack of source control.
- Recurrent embolization with persistent vegetations despite appropriate treatment.
- Large vegetations (>1cm) with severe valvular regurgitation (Kang et al, NEJM 2012).
- **Complications**: suspect with persistent fever for > 48 hours despite treatment or persistent bacteremia despite appropriate treatment.
- Cardiac: remember to obtain baseline ECG to assess for heart block or conduction delay (e.g. PR prolongation) due to perivalvular abscess (more common in aortic valve disease). Other complications include cardiac ischemia (embolism to coronary ostia). Heart failure is now the most common cause of death in endocarditis.
- Embolic: systemic embolization is usually due to left-sided endocarditis or via PFO. Can occur in up to 30% of patients with Staph aureus endocarditis. Risk factors include S. aureus, S. bovis, mitral valve disease, veg > 10mm and increased veg mobility on echo. While a history of aspirin use may be protective, starting anti-platelet treatment after diagnosis may cause increased bleeding (e.g. hemorrhagic conversion of septic emboli) and is not recommended.
- Persistent fever: while on antibiotics for endocarditis: consider metastatic infection including abscess (splenic, perivalvular, renal, and psoas), septic pulmonary emboli, pleural effusion, CNS infection, vertebral osteomyelitis, septic arthritis (esp. sacroiliac, pubic, manubriosternal joints), drug fever, and catheter-associated phlebitis.

Key Points

- Predisposing factors include prosthetic valves, previous IE, IDU and catheters.
- Monitor for EKG changes, evidence of heart failure and systemic emboli.
- The microbiology for native versus prosthetic valves differs slightly, although Staph aureus is the most common organism in both.
- Consider surgery in cases of heart failure, severe AI/MR, fungi or highly resistant organisms, perivalvular disease, >10mm mobile vegetation, or failed medical therapy.

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EKG reading made easy

Rates

- Normal: 60-100 bpm
- Tachycardia: > 100 bpm
- Bradycardia: < 60 bpm

Readings

- Count the number of large squares present within one R-R interval.
- Divide 300 by this number to calculate heart rate.

OR

- Count the number of complexes on the rhythm strip (each rhythm strip is typically 10 seconds long).
- Multiply the number of complexes by 6 (giving you the average number of complexes in 1 minute).

Note:

- A patient’s heart rhythm can be regular or irregular.
- Irregular rhythms can be either:
- Regularly irregular (i.e. a recurrent pattern of irregularity)
- Irregularly irregular (i.e. completely disorganized)

The cardiac axis

The cardiac axis describes the overall direction of electrical spread within the heart.

In a healthy individual, the axis should spread from 11 o’clock to 5 o’clock.

To determine the cardiac axis you need to look at leads I, II, and III.

Right Axis deviation

- Right axis deviation (RAD) involves the direction of depolarisation distorted to the right (between +90º and +180º).
- The most common cause of RAD is right ventricular hypertrophy. Extra right ventricular tissue results in a more robust electrical signal being generated by the right side of the heart. This causes the deflection in lead I to become negative and the deflection in lead aVF/III to be more positive.
- RAD is commonly associated with conditions such as pulmonary hypertension, as they cause right ventricular hypertrophy. RAD can, however, be a normal finding in very tall individuals.

Left Axis deviation

- Left axis deviation (LAD) involves the direction of depolarisation distorted to the left (between -30° and -90°).
- This results in the deflection of lead III becoming negative (this is only considered significant if the deflection of lead II also becomes negative).
- Conduction abnormalities usually cause LAD.

P Wave

The next step is to **look at the P waves**

- Sawtooth baseline → flutter waves
- Chaotic baseline → fibrillation waves
- Flatline → no atrial activity at all
- The **PR interval should** be between **120-200 ms** (3-5 small squares).

Note: A prolonged PR interval suggests the presence of atrioventricular delay (AV block).

If the **PR interval** is **shortened**, this can mean one of two things:

- the SA node is not in a fixed place and some people's atria are smaller than others.
- an **accessory pathway** and can be associated with a **delta wave** (see below which demonstrates an ECG of a patient with Wolff Parkinson White syndrome).

QRS Complex

When assessing a QRS complex, you need to pay attention to the following **characteristics**:

- Width
- Height
- Morphology

Width can be described as **NARROW** (< 0.12 seconds) or **BROAD** (> 0.12 seconds)

Height can be described as either **SMALL** or **TALL**:

- **Smallcomplexes**are is defined as < 5mm in the limb leads or < 10 mm in the chest leads.
- **Tallcomplexes**imply ventricular hypertrophy (although can be due to body habitus e.g. tall slim people).

To assess **morphology**, you need to assess the individual waves of the QRS complex.

Note – the presence of a delta wave does NOT diagnose Wolff-Parkinson-White syndrome. This requires evidence of tachyarrhythmias AND a delta wave.

Q Wave

Isolated Q waves can be **normal**.

A **pathological Q wave** is **> 25% the size of the R wave that follows it** or **> 2mm in height and > 40ms in width**.

R Wave

Assess the R wave progression across the chest leads (from small in V1 to large in V6).

The transition from the S > R wave to the R > S wave should occur in V3 or V4.

Poor progression (i.e. S > R through to leads V5 and V6) can be a sign of previous MI but can also occur in very large people due to poor lead position.

ST-Segment

The **ST segment** is the part of the ECG **between the end of the S wave and the start of the T wave**.

In a healthy individual, it should be an isoelectric line (neither elevated nor depressed).

ST-elevation is significant when it is **greater than 1 mm** (1 small square) **in 2 or more contiguous limb leads** or **>2mm in 2 or more chest leads**. It is most commonly caused by **acute full-thickness myocardial infarction**.

ST depression ≥ 0.5 mm in **≥ 2 contiguous leads** indicates **myocardial ischemia**.

T Waves

T waves are considered **tall** if they are:

- **> 5mm** in the **limb leads**AND
- **> 10mm**in the **chest leads** (the same criteria as ‘small’ QRS complexes)

Tall T waves can be associated with:

- **Hyperkalaemia**(“tall tented T waves”)
- **HyperacuteSTEMI**

Inverted T waves in other leads are a nonspecific sign of a wide variety of conditions:

- Ischaemia
- Bundle branch blocks (V4-6 in LBBB and V1-V3 in RBBB)
- Pulmonary embolism
- Left ventricular hypertrophy (in the lateral leads)
- Hypertrophic cardiomyopathy (widespread)
- General illness

Flattened T waves are a non-specific sign, that may represent **ischemia** or **electrolyte imbalance**.

Biphasic T waves have **two peaks** and can be indicative of **ischemia** and **hypokalaemia**.

> **U Waves**

U waves are **not** a **common** finding.

The U wave is a **> 0.5mm deflection after the T wave** best seen in **V2** or **V3**.

These become larger the slower the bradycardia – classically U waves are seen in various **electrolyte imbalances**, **hypothermia** and secondary to **antiarrhythmic therapy** (such as digoxin, procainamide, or amiodarone).

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Syncope

> **Syncope**

1. Rapid onset of transient loss of consciousness
2. Inability to maintain postural tone
3. It may be associated with a fall
4. Resolves spontaneously and quickly without intervention

> **Presyncope (Near-Syncope)**

1. Weakness, Dizziness, lightheadedness, or "graying out" of consciousness without loss of postural tone
2. Evaluate Presyncope with the same vigor as Syncope
    1. Presyncope has the same risks of adverse events as Syncope

> **Causes: Neural or Reflex Mediated Syncope (no cardiovascular risk, most common, 45% of cases)**

1. Vasovagal Syncope (Vasodepressor Syncope)
2. Situational Syncope
3. Carotid Sinus Syncope
4. Glossopharyngeal neuralgia (uncommon)
5. Trigeminal Neuralgia
6. Hypovolemia
7. Medication-related Syncope (Drug-Induced Syncope, responsible for 5-15% of Syncope causes)
8. Recreational drug use
9. Postural Tachycardia Syndrome (POTS)

    - Most common in young female women (associated with chronic Fatigue and Mitral Valve Prolapse)

10. Autonomic failure

Cardiac syncope

- Arrhythmias
- Ventricular Tachycardia

- Sick Sinus Syndrome
- Supraventricular Tachycardia
- Atrioventricular Block (second or third degree)
- Pacemaker malfunction
- Valvular disorders
- Hypertrophic Cardiomyopathy (esp. young patients)
- Aortic Stenosis
- Acute Mitral Valve Regurgitation (i.e. acute MI with papillary Muscle rupture)
- Prosthetic Heart Valve complications (e.g. Thromboembolism, valvular obstruction)

Vascular disorders

- Myocardial Infarction
- Aortic Dissection
- Abdominal Aortic Aneurysm rupture
- Pulmonary Embolism
- Pulmonary Hypertension
- Subarachnoid Hemorrhage
- Subclavian Steal Syndrome

Myocardial disorders

- Hypertrophic Cardiomyopathy
- Atrial myxoma

Examination

- Vital sign
- General
- Cardiovascular examination-murmurs, Carotid bruit, asymmetric pulses
- Abdomen and pelvis exam- pulsatile mass and decreased femoral pulses, pelvic pain, rectal exam

Labs

- Basic Chemistry Panel (Serum Electrolytes including Glucose)
- Hemoglobin or Hematocrit
- Pregnancy Test (urine HCG)
- Fecal Occult Blood Test
- Troponin I
- D Dimer if necessary.

Diagnostics

- Electrocardiogram (EKG)
- Continuous cardiac monitoring-telemetry for inpatient

Imaging

- Chest XRay
- Echocardiogram
- CT chest with contrast (if Pulmonary Embolism is suspected)
- Imaging related to injuries sustained in a Syncope- fall
- CT Head (usually low yield except indicated by history and physical examination)

Indications for head imaging include:

- Age over 65 years
- Warfarin use
- First Seizure
- Trauma above the clavicles
- Persistent neurological deficit
- Dizziness
- Sudden onset headache (Thunderclap Headaches)

Note: The San Francisco Syncope Rule (CHESS Score) or Canadian syncope risk score are used to evaluate the short-term risk of severe outcomes and may reduce the syncope hospitalization rate.

Management depends on the cause

- Fall precautions
- Telemetry if needed
- Assess ability to tolerate PO
- IV fluids if needed
- Consider intoxication

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