UCLA CCU HOUSESTAFF HANDBOOK

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General CCU Information

Intern Responsibilities

- Primary goal for the UCLA CCU intern is to know or have readily available all possible vitals, swan numbers, labs, imaging studies, consult recommendations, etc on your patients before you round. A good intern will be able to run through all pertinent information for each patient without having to turn to the resident for answers. The less your resident has to speak on behalf your patients, the better the intern.
- Remember 80 hr work-week; maximum of 30 consecutive hrs
- Morning pre-rounds to be completed prior to 7:45 am on those days morning didactic sessions are scheduled (see posted schedule, no lecture on Wed.)
- Obtain sign-out from post-call intern about any events on your patients overnight
- Assess all your patients, including vital signs (BP, HR, Temp, I/O, weight). Obtain all labs, overnight imaging, relevant physical examination (JVP, rales, etc.). Know all information about your patient.
- Check with the night nurse about events that occurred overnight and share your plan for the patient, even if it is not complete.
- Check with CCU nurse regarding overnight arrhythmias picked up on monitor for all your patients; go to tele nurse and print pertinent strips to bring to rounds
- Review the chart, including the recommendations of all consults. Discuss these on rounds with the rest of the team.
- Develop a treatment plan for all patients to discuss on rounds.

Admissions

- The admission note should consist of the H/P as well as your impression and treatment plan. Review this with your resident. You have the choice to either dictate your H/P or type it online (https://cdsprod.mednet.ucla.edu:447/eSig/logon.htm)
- For the history: include details of the symptoms leading to the admission, whether the patient has had angina or CHF, the patient's exercise tolerance, and whether the patient has a history of palpitations or syncope. Review the patient's cardiac procedures (CABG, PTCA, stents), most recent coronary angiogram, stress test, and Holter results.

Discharges

- Housestaff should plan to complete all the discharge paperwork 1 day prior to patient discharge
- Make sure all home health issues dealt with (i.e home oxygen, home Lovenox, PT/OT, etc) and post discharge appointments made
- Go to http://medres.med.ucla.edu/ and click on Discharge tab
- Please make sure if your patient was admitted with CHF or ACS, fill out the appropriate checkboxes in the discharge paperwork.

Sign-outs

- Go to http://medres.med.ucla.edu/ for signouts
- You can use Dr. Peter Yang's medication an lab clean up tool: http://www.dryang.org/clean.php
- Include attending of record, code status, goals (esp. I/O) for the night. Every day, you should update medications, pressors, etc. Also, make sure pending studies, labs, consult, etc are listed for your resident.
- If you are post-call (i.e. leaving at 1:00 pm) or if you are off the next day, please write in the orders section the name of the intern who will be covering for you and sign out your pager appropriately.
- Please make sure before you leave that the Pharmacy has the discharge prescriptions for those patients expected to leave the next day.
- When patients are signed-out to you, they become your patients. You are not covering. The patient is now yours.

Reasons to call resident or fellow and/or attending

- Unstable patient that needs to be intubated or needs an urgent procedure
- Patient's condition is not improving:
    - New or worsening oliguria
    - Recurrent angina or angina with EKG changes

- New or increasing troponins
- New or increasing inotrope requirements
- New or worsening CHF
- New or worsening arrhythmia or firings of the AICD
- Pulmonary pressures approaching systemic blood pressures

- Patient needs to be transferred to a higher level of care

You will <u>never</u> be wrong by calling the resident, fellow or attending if you have a question.

Signing Out Your Pager

In house, dial 231 - Off-campus, dial 1-800-233-7231 (1-800-BEEP-231)
Enter * followed by your pager number
Enter #
Enter 1
Enter 1
Enter the pager number that will be covering you
ICU Intern 90040
CCU Intern 90089
You can also sign out to a referral number, such as your cell phone (more useful for residents)
After Enter #
Enter 2
Enter 1
Enter the referral number Note: if you don't want people to see the number, you can refer your calls to the page operator (310) 825-6301 (make sure to call page operator to tell them which number to direct your calls to)

Signing Back on to Your Pager

In house, dial 231 - Off-campus, dial 1-800-233-7231 (1-800-BEEP-231) Enter * followed by your pager number
Enter #
Enter 1
Enter 2

Data Collection and Documentaion

- Go to http://mednet.ucla.edu/
- There are three main systems to view patient information, cView, Essenstris, and PCIMIS. PCIMIS is the old medical record system, though still is very useful.
- For Documentation (H/Ps, Progress Notes, etc) use Clinical Document System (CDS).

Things found in Essentris

- Vitals, Basic labs in treatable form (CBC, Chem 10, LFT's, blood gases, coags)
- For unit patients, Swan numbers, vent settings, drip rates
- Ins and Outs
- Notes for PT, OT, Speech/swallow, Nutrition, Social Work, DC planning), Invasive devices (has lines, Foleys, etc. and # of days in place)

Things found in cView

- Labs, Microbiology
- Use 72H and 24H summary for prerounding
- Results summary to select particular labs to display, useful for trending or to locate obscure labs
- Documents
- All medicine H&Ps, daily progress notes, discharge summaries, most medicine consult notes and initial consultations from other services, some consult follow up notes, Radiology reports, Final echocardiogram and ECG reports
- For Pathology reports, it best to call the path resident for prelim read.
- Medication Profile (current meds for inpatients only, to get details of prn dosing you need to go to actual MAR)
- PACS (to see actual radiology images) Tip: also a good way to see if a study has been done or not. Tip: sometime the last image of an ultrasound has a tech wet read like "No DVT." EKG reports (to print prior ECG's) Visit history (mainly useful on admission to identify PMD and which consultants follow)
- Things found in Paper Chart- Prelim echo reads (in front of chart), Consult notes for some services (especially non-medicine services and follow up notes), Attending notes for certain attendants (Froch, Saleh, Kelley, Heart Transplant/CM attendings Anesthesia records from OR cases, Prelim Endoscopy/Bronchoscopy notes(sometimes with pretty pictures), ECG'S (under Cardiodiagnostics tab)

Resident Responsibilities

- Your interns are a reflection of you. If they look uninformed or unprepared during presentations, you're who the attending will look to for explanations. The more you teach and prepare them, the more your silence during morning rounds will be golden.

- Your job on call (especially at night) will be triaging new admits. First and foremost, if the patient looks sick, call the fellow. Secondly, if in doubt, call the fellow. You will never be faulted for being overly cautious, but you might for being overly confident. Carry a copy of the chest pain flow chart (will be in the CCU nuts and bolts). This is a great resource that will reduce the number of inappropriate CCU admits.

- Practice and know your ACLS/BCLS There is a high likelihood that you will witness multiple codes during your CCU rotation, and an almost equal chance that you will run one or more.

- Most useful topics to beef up on prior to starting the rotation: CHF, chest pain/NSTEMI/STEMI, pressors and hemodynamics, ECGs

- Be able to interpret the following cardiology diagnostic testing procedures: cardiac catheterization data, nuclear/stress test imaging data, and echocardiogram

- Demonstrate understanding of risk factor modification for CAD (GRACE & TIMI), management of patients with NSTEMI and STEMI, management of the critically ill cardiac patient with decompensated heart failure, management of cardiac arrhythmias, and interpretation of telemetry.

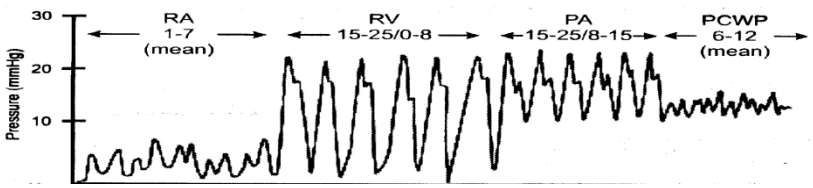
- Ensure that UCLA's CHAMP protocol for heart failure and Acute MI protocol are followed and properly documented. Otherwise, you will get a not so nice email from Myrtle Yamamoto.

- Teach. You learn the most from your peers. The best time to teach is while you are seeing a patient. Review ECGs and other radiographic images with your interns during your assessment of patients.

Hemodynamics and Pharmacotherapy

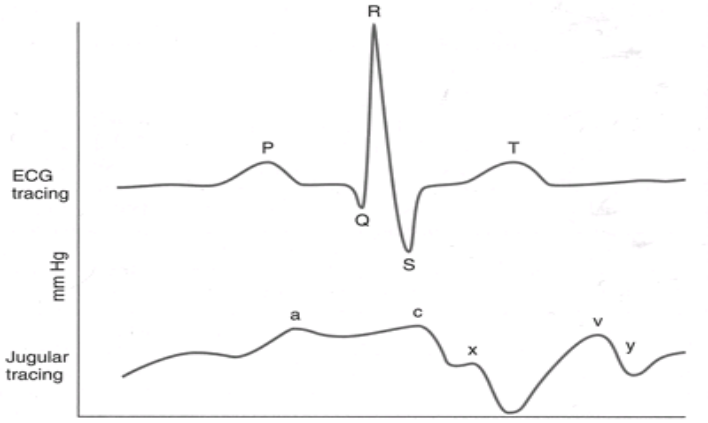
Basic Definitions

Measured Values	**Derived Values**
CVP: Central Venous Pressure 4-7 mm Hg → Reflects Right Atrial (**RA)** Pressure **RV systolic** 15-25mmHg **RV Diastolic** 0-8mmHg **PA Systolic Pressure** 15-25mm Hg **PA Diastolic Pressure** 8-15mm Hg **PCWP:** Pulmonary Capillary Wedge Pressure 6-12mm Hg PCWP =Estimates left atrial heart pressure and left ventricular end diastolic pressure **CO:** Cardiac Output 3.5 - 5.5 L/min **MV02:** Mixed venous partial pressure of oxygen 70-75% Drawn from end of pulmonary artery catheter. Used to calculate how well oxygen is extracted by the tissue. This mixed venous value holds true as long as there is no shunt present.	**CI:** Cardiac Index 2.4 - 4.0 L/min **SVI:** Stroke Volume Index 40 - 70 ml/beat/m2 **SVR:** Systemic Vascular Resistance 900-1200 dynes.sec.m2/cm5 Wood Units= (dynes.sec.m2/cm5)/80 **PVR:** Pulmonary Vascular Resistance 100 - 200 dynes.sec.m2/cm5. Ideally PVR should be <150 dynes.sec.m2/cm5. CO=HR x SV CI= CO/BSA(body surface area) Mean Arterial Pressure (MAP)= (SBP+DBP x2)/3 SVR=(MAP-CVP)/(CO x 80) Trans-pulmonary gradient= Mean pulmonary artery pressure (MPAP) - PCWP. **If transpulmonary gradient >15 (intrinsic pulmonary vascular disease, patient needs lung/heart transplant)**



Central Venous Pressure (CVP) Waveform

- The normal CVP waveform contains three peaks ('A', 'C', 'V') and two descents ('X', 'Y'). The **'A'** wave occurs at the end of ventricular diastole and is the result of atrial contraction. The etiology of the **'C'** wave is debatable. It was originally attributed to transmission of the systolic pressure wave from the adjacent carotid artery, although more recent opinion suggests that it is due to tricuspid valve closure. The **'X'** descent wave is caused by the downward movement of the ventricle during systolic contraction. It occurs before the T wave on an EKG. The **'V'** wave occurs as a result of venous filling of the atrium during late ventricular systole. It occurs as the T wave is ending on an EKG. The **'Y'**descent is produced by the tricuspid valve opening in diastole with blood flowing into the right ventricle. It occurs before the P wave on an EKG.



- In **atrial fibrillation**, a waves will be absent, and in atrioventricular disassociation, a waves increased ("cannon awaves") as the atrium contracts against a closed tricuspid valve.
- In **tricuspid regurgitation**, the c wave and x descent will be replaced by a large positive wave of regurgitation as the blood flows back into the right atrium during ventricular contraction. This can elevate the mean central venous pressure, but it is not an accurate measurement. A better way of estimating CVP in this case would be to look at the pressures between the regurg waves for a more accurate mean.
- In **cardiac tamponade**, all pressure will be elevated, and the y descent will be nearly absent

FOR HEMODYNAMIC PHARMACOLOGY please refer to UCLA yellow book pages 45 and 46.

Types of Shock

Adapted from Dr. Goldhaber lecture series. Aug 2008

Definition: Failure of adequate tissue perfusion leading to cell injury and death.

PE: will show inadequate perfusion incude cool extremities, abnormal mental status, oliguria, hypotension tachycardia.

- Hypovolemic: Post cath groin bleed and/or retroperitoneal hematoma, GI bleed from ICU stress, aortic dissection and rupture, LV free wall rupture post MI
- Cardiogenic: Acute MI, CHF, valvular dysfunction, myocarditis, bradycardia, tachycardia, cardiac contusion
- Obstructive: PE (saddle embolus), Tamponade, Constrictive pericarditis, pneumothorax
- Distributive Shock: systemic vasodilation from sepsis, anaphylaxsis, spinal injury (neurogenic), adrenal insufficiency
- Cytotoxic: Carbon Monoxide, CN

Treatment of Cardiogenic Shock during Acute MI (infarcts >40% LV, mortality >70%):

1. Exclude other causes of shock (swan)
2. Steep pressure/volume relationship and elevated PCWP to start makes fluids and ineffective therapy.
3. Inotropes with nitroglycerine for unloading
4. IABP
5. Revascularization by PTCA reduces mortality by 20-50%. Thrombolysis less effective.

RV infarct:

1. Associated with inferior MI (proximal RCA), with high mortality
2. Usually clear lungs and normal PCWP, elevated JVP
3. Responds to inotropes and chronotropes. Classic therapy of fluid resuscitation usually in profound RV failure and pulmonary hypertension. Fluid only to keep PCW at 15
4. Atrial Pacing.

The table below goes over the different states of shock and the different hemodynamic states:

TYPES	CVP or RA (preload)	PA	PCWP	CO (pump function)	SVR (Afterload)	MAP	Other Pertinent Findings
Hypovolemic	↓	↓	↓	↓	↑	↓	-decreased MVO2 (tissue pefusion) -cold extremities
Cardiogenic	↑ ↓	↑	↑	↓	↑	↓	-decreased MVO2 (tissue pefusion) -cold extremities
Tamponade	↑	↑	↑	↓	↑	↓	-Diastolic pressure equalization
Constrictive Pericarditis	↑	↑	↑	N or ↓	N or ↑	N or ↓	-Diastolic pressure equalization
RV Infarction	↑	N or ↓	N or ↑	N or ↓	↑	N or ↓	PA_d>PCW
Acute MR	N or ↑	↑	↑	↓	N or ↑	N or ↓	Large V wave
Massive PE	↑	↑	N or ↓	↓	↑	↓	-JVD maybe elevated
Acute LV Failure	Normal	↑	↑	↓	N or ↑	N or ↓	
Sepsis (Early)	↓	↓	↓	↑	↓	N or ↓	-Warm extremities -Wide Pulse pressure

ACUTE MI Pathway

See yellow book 47-48 for chest pain differential and AMI 50-53

Suspected myocardial infarction on electrocardiogram:
- ST segment elevation ≥ 1 mm in ≥ 2 leads not known to be old.
- Pathologic Q waves in ≥ 2 leads not known to be old.
- Left bundle branch block not known to be old.

OR
- First troponin positive.Note: If none of the ECG criteria (above) are met, a troponin result is required for team

No

Yes

Suspected Ischemia on electrocardiogram:
- ST segment depression ≥ 1 mm in ≥ 2 leads not known to be old.
- T wave inversion in ≥ 2 leads not known to be old.

Yes

No

POINTS:
- Systolic blood pressure < 110 mmHg.
- Bilateral rales above the bases.
- Worsening of previously stable angina.
- New or recurrent angina following a revascularization

0 Points | 1 Point | ≥ 2 Points | 0-1 Point(s) | ≥ 2

Very low risk (<1%) | Low risk (~4%) | Intermediate risk (~8%) | High risk (>16%)

If no concurrent diagnosis (i.e. altered mental status, pneumonia, new arrhythmia, severe hypertension) or alternative diagnosis for chest pain, then consider triage to **chest pain team.** Otherwise, triage to CCU Team for primary cardiac diagnosis (i.e. arrhythmia) or to general medicine.

CCU Team

Treatment Algorithm for Acute Myocardial Infarction with or without Left Ventricular Dysfunction

Acute Myocardial Infarction

NSTEMI

Aspirin and clopidogrel
IV heparin or LMWH
IV/PO beta-blockers
Early invasive management (intermediate or high risk)
GP IIb/IIIa inhibitor*
ACE inhibitor (12 to 24 hours)
Statin (first 24 hours)
*check interventional cardiologist prior to starting especially if PCI will be done

STEMI

Aspirin (and clopidogrel if PCI)
IV heparin or LMWH
IV/PO beta-blocker
Primary PCI or thrombolytics
ACE inhibitor (12 to 24 hours)
Statin (first 24 hours)

Measure LVEF

EF ≤40%

Left Ventricular Dysfunction with or without Heart Failure

Aspirin, clopidogrel, or both
Anticoagulation (selected indications)
Carvedilol
ACE inhibitor
Statin (irrespective of LDL)
Spironolactone or eplerenone (if HF symptoms or diabetes)
Diet (omega-3 fatty acids)
Exercise/cardiac rehabilitation
Smoking cessation
Rigorous control of BP and diabetes
Sudden death risk stratification

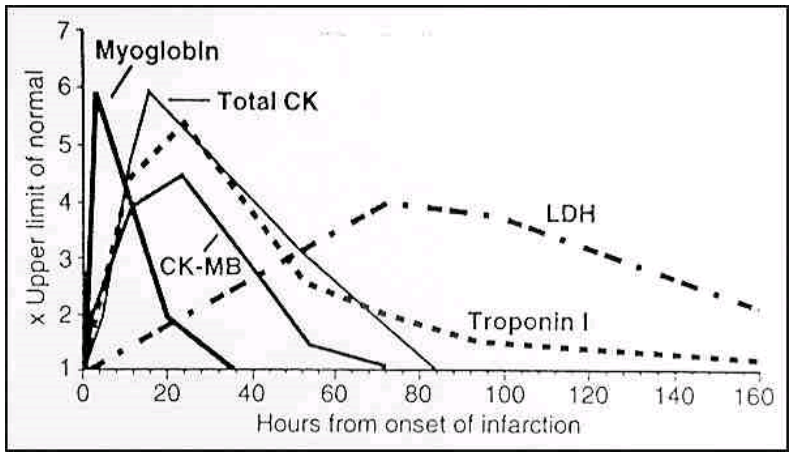
EF >40%

No Left Ventricular Dysfunction

Aspirin, clopidogrel, or both
Anticoagulation (selected indications)
Beta blocker
ACE inhibitor
Statin (irrespective of LDL)
Diet (omega-3 fatty acids)
Exercise/cardiac rehabilitation
Smoking cessation
Rigorous control of BP and diabetes

Adapted from UCLA Clinical Practice Guideline-2005

Cardiac Markers



Troponin Levels

- Troponin levels are now considered the criterion standard in defining and diagnosing MI, according ACC/AHA. Trop T and I have a greater sensitivity and specificity than CK-MB levels in detecting AMI..
- Serum levels increase within 3-12 hours from the onset of chest pain, peak at 24-48 hours, and return to baseline over 5-14 days.
- Serum troponin maybe elevated long term in a patient with chronic renal failure (CRF). If suspect AMI in a patient with CRF, consider CK-MB

Creatine Kinase (CK) level

- 3 isoenzymes of CK, including creatine kinase with muscle subunits (CK-MM), which is found mainly in skeletal muscle; creatine kinase with brain subunits (CK-BB), predominantly found in the brain; and myocardial muscle creatine kinase (CK-MB), which is found mainly in the heart.
- Serial measurements of CK-MB was previously the standard criterion for diagnosis of AMI.
- CK-MB levels increase within 3-12 hours of onset of chest pain, reach peak values within 24 hours, and return to baseline after 48-72 hours. Levels peak earlier if reperfusion occurs.
- Sensitivity is approximately 95%, with high specificity. However, sensitivity and specificity are not as high as for troponin levels, and the trend has favored using troponins for the diagnosis of MI.

Myoglobin Levels

- Urine myoglobin levels rise within 1-4 hours from the onset of chest pain.

- Myoglobin levels having high sensitivity but not specific. They can be useful within the context of other studies and in early detection of AMI in emergency setting

LDH

- LDH has been succeeded by other tests. It begins to rise in 12 to 24 hours following AMI, and peaks in 2 to 3 days, gradually dissipating in 5 to 14 days. Measurement of LDH isoenzymes is necessary for greater specificity for cardiac injury. There are 5 isoenzymes (1 through 5). Ordinarily, isoenzyme 2 is greater than 1, but with myocardial injury, this pattern is "flipped" and 1 is higher than 2.

Fundamentals of Stress Testing

(Adapted from Dr. Michelle Kittleson's Stress Test Lecture October 2008)

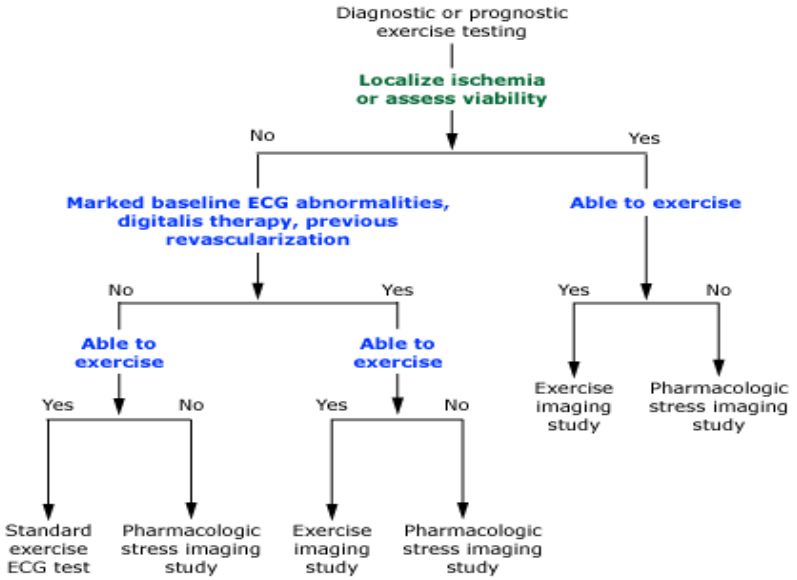
1. **INDICATIONS:** *Why Am I ordering a stress test?*
    a. Diagnosis CAD
        i. Ischemia
        ii. Viability
    b. Assessing Functional Capacity
        i. Valvular Heart Disease (e.g. AS,AR)
        ii. Hypertrophic Cardiomyopathy
        iii. Cardio vs. Pulmonary Disease
    c. Risk Stratification Post MI
        i. Submaximal Stress 3 to 7 days post MI to assess need for Cath
        ii. Symptom-Limited Stress 2 to 6 weeks post MI to assess activity level
    d. Assess efficacy of medical therapy
        i. Afib (HR control)
        ii. Post MI (BP,HR control)
2. **CONTRAINDICATIONS:** *Why shouldn't I order a stress test?*
    a. Complications
        i. Death: 1 in 10,000 patients
        ii. Cardiac Arrest: 2 in 10,000 patients
    b. Absolute Contraindications
        i. Severe/Symptomatic AS
        ii. Acute MI (within 2 days)
        iii. Unstable Angina
        iv. Decompensated HF
        v. Unstable Arrhythmias
        vi. Acute PE
        vii. Suspected Aortic Dissection
    c. Relative Contraindications
        i. Left Main CAD
        ii. Moderate AS
        iii. HOCM
        iv. Electrolyte Abnormalities
        v. Afib with RVR
        vi. 3rd degree AV block
        vii. Resting BP > 200/110
3. **TYPES OF STRESS TESTS:** *Which test should I order?*
    a. Exercise
        i. EKG (ECHO or Nuclear)
            1. Advantages
                a. stress of choice
                b. cheap
                c. no needed IV's
                d. added information on functional capacity
            2. Disadvantages
                a. Lower sensitivities/specificities
                b. Can't use with abnormal baseline EKGs
                    i. LBBB
                    ii. Vpaced
                    iii. LVH with strain pattern
                    iv. Digoxin associated ST depressions
    b. Pharmacological
        i. Dobutamine (ECHO or Nuclear)
            1. Mechanism: β1 agonist (increase HR and contractility)
            2. Advantages:
                a. Assess Wall Motion and Valvular function
                b. Can do rapidly (30 –45 minutes)
                c. More Specific
                d. Cheaper, NO IV's, NO radiotracers
            3. Disadvantage:
                a. Possible side effects: Palpitations, Tachyarrythmias
                b. Pt can not take beta blockers night prior to test
            4. Contraindications (False Positive 2/2 to abn septal wall motion)
                a. LBBB
                b. Vpacing
                c. Symptomatic Aortic aneurysms
        ii. Adenosine (Nuclear only)
            1. Mechanism: Vasodilator

            2. Advantages:

a. Assess Radiotracer Uptake and Blood Flow
b. More Sensitive

3. Disadvantage
   a. Possible side effects: hypotension, chest pain, flushing, wheezing
   b. Test takes 2-4 hours, tracer last 2 days
   c. Radioactive tracers, need for IV
   d. Caffeine, Theophylline decrease sensitivity
4. Contraindications:
   a. Bronchospasm (pts with severe COPD/Asthma)
   b. Heart Block
   c. Severe AS

c. Summary

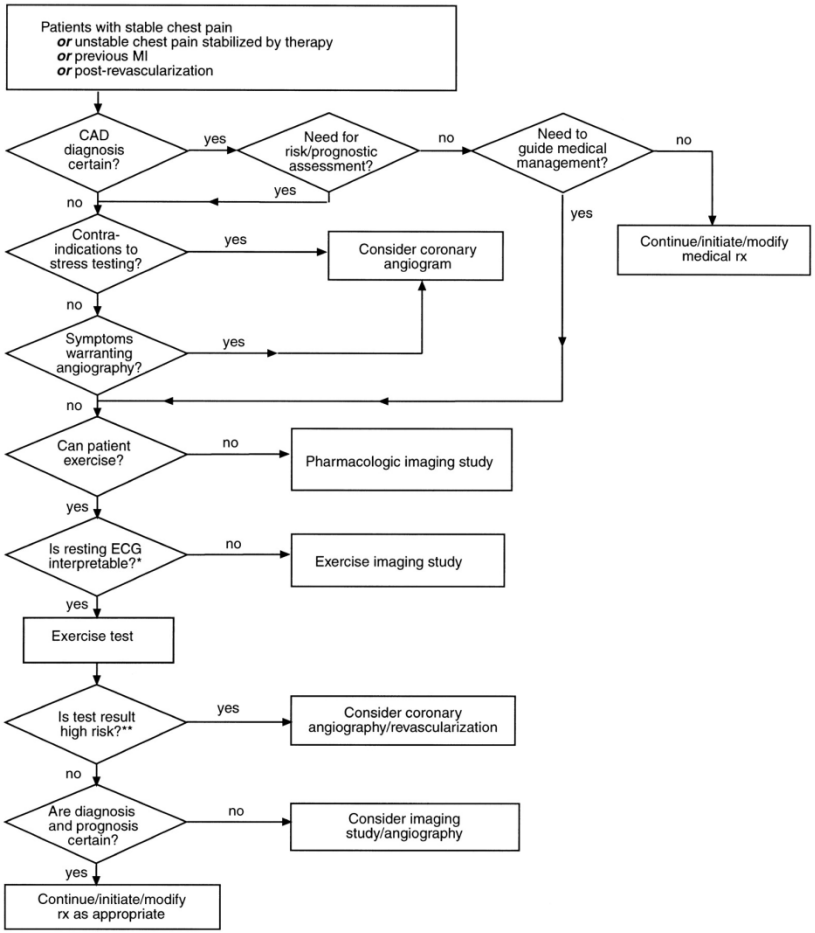


4. **INTERPRETATION:** *What does this mean for my patient?*

a. Treadmill
   i. Stress Test is only negative if patient reaches target heart rate
   ii. Target Hear Rate = 0.85 * (220 – age)
   iii. Hold Beta Blocker for assessing for ischemia
   iv. Exercise time is prognostic
   v. DUKE Treadmill Score:
      1. Duration of Exercise – 5 X max ST deviation – 4 X treadmill angina index (0, none till 2, limiting angina)
      2. Scores
         a. Low-risk — score ≥+5 (99% 4 yr survival)
         b. Moderate-risk — score from -10 to +4 (95% 4 yr survival)
         c. High-risk — score ≤-11 (79% 4 yr survival)

Angiogram Basics

- For full information on the ACC/AHA Guidelines go to http://circ.ahajournals.org/cgi/content/full/99/17/2345
- Image below adapted from ACC/AHA guidelines for angiogram



Post Catheterization Groin Checks

- If nursing calls you with concern, check the patient's skin, color, temperature and peripheral pulses below the puncture site
- If bleeding occurs at the site, apply direct manual pressure to the site and notify resident and/or cardiology fellow.

<u>When to contact your resident or attending:</u>

- Pt has a large hematoma unchanged or increasing in size even with manual pressure or pressure via femstop or sandbags.
- Uncontrolled oozing from the site even with pressure application, unpalpable or undopplerable pulses on either foot (unless this is pts. baseline on unaffected leg), or loss of CMS for extended period of time even with no pressure application.
- Signs of retroperitoneal bleed, pseudoaneurisms, or vasovagal response unrelieved by fluids and atropine.

Sheath pulls need to be monitored closely as fatalities can occur from complications!

FEMORAL SHEATH PULLS

***Never pull a sheath unless it has been discussed with the cardiology fellow and the interventional attending.**

Before the sheath is pulled monitor the groin site for hematomas, excessive oozing from the groin site, as well as pulses and CMS of both the affected leg as well as the opposite leg. Pulses should always be palpable or dopplerable. Pull the sheath as soon as possible. The longer it stays in, the more one's risk of complications increases. Make sure 1mg atropine and 1L NS is in the room before pulling the sheath incase the patient vasovagals. Pre-medicating the patient before the procedure (if VS are appropriate) can decrease one's risk of vasovagaling.

During the sheath pull, both manual pressure or pressure via femstop may be used to achieve hemostasis. Femstops are best to use for someone with a high blood pressure. Pressure must be held for a minimum of 5 minutes for each venous sheaths and 20 minutes for each arterial sheaths. Hemostasis must be achieved before removing pressure or before pulling out the next sheath if multiple sheaths exist (a venous and arterial sheath may be pulled at the same time). Continue to monitor VS, pulses, CMS, and the groin site for excessive oozing,hematomas and pseudoaneurisms. Monitor the patient for back pain, hypotension, and tachycardia in case of retroperitoneal bleed. **Continue monitoring for these complications after the sheath has been pulled.**

Cardiac Hospitalization Atherosclerosis Management Program CHAMP Protocol

Adapted from UCLA Comprehensive Atherosclerosis Treatment Program, UCLA Division of Cardiology, 2005 Gregg C. Fonarow, MD

- Patients with established coronary artery, cerebral vascular, and peripheral atherosclerosis are at high risk for vascular events and cardiac death regardless of identifiable risk factors and regardless of whether they have undergone revascularization. Combination therapy targeting the underlying atherosclerotic disease process can markedly improve clinical .outcome in patients with atherosclerosis, whereas failure to employ these therapies increases patient mortality. Compliance and treatment utilization can be enhanced by employing secondary prevention measures prior to hospital discharge. Patients should not be discharged from the hospital (including chest pain, unstable angina, acute myocardial infarction, cardiac catheterization, angioplasty, coronary bypass, and ischemic heart failure hospitalizations) without initiation of definitive atherosclerosis treatment.

Goals with CHAMP

- LDL < 70 mg/dL Once achieved, document with biannual or annual lipid panel(Secondary goal HDL > 40 mg/dL, Triglycerides < 150 mg/dL)
- BP < 140/90 mmHg Document on each follow-up visit, with additional monitoring as indicated
- BP < 130/80 mmHg If diabetes or renal failure (if diabetes and renal insufficiency BP < 125/75)
- No smoking: Current status with regards to smoking should be documented in all current/formers mokers. Recommendation for smoking cessation and nicotine replacement/Zyban®/behavior modification attempts should be documented
- HbA1C <7.0% Diabetes management, tight control in diabetics
- 30-60 min, daily Physical activity
- BMI 21-25 kg/m2 Achieve weight goal via Mediterranean or therapeutic lifestyle change (TLC) diet.

Treatment Regimen in patients with established Atherosclerosis

Aspirin and/or Clopidogrel: Antiplatelet therapy reduces the risk of vascular events in patients with atherosclerosis. Patients should continue on ASA, 81 mg to 162 mg per day indefinitely after discharge.
Contraindications include true aspirin allergy with nasal polyposis and active bleeding. Patients with acute coronary syndromes should be treated with combined aspirin and clopidogrel (75 mg daily) for 12 months or indefinitely. Patients that have contraindications or intolerance to ASA should be treated with **clopidogrel** 75
mg daily. Patients with a recurrent event despite ASA should be considered for aspirin plus clopidogrel treatment.
In patients with coronary artery disease, ASA lowers the risk of myocardial infarction, unstable angina, need for revascularization, and death. Pooling data from the four largest trials suggests a 48% reduction in the risk of myocardial infarction and a 51% reduction in the risk of death. This benefit continues beyond ten years. CURE demonstrated the additional benefit of 3 to 12 months of clopidogrel in combination with aspirin in acute coronary syndrome patients.

Statins and other lipid lowering agents:

- Statins have potent vascular and cardiac protective effects and are indicated in all patients with atherosclerosis or diabetes. Statins reduce vascular inflammation and stabilize the vulnerable atherosclerotic plaque, thereby markedly reducing the risk of vascular events.
- Clinical trials have shown mortality reduction in patients with baseline LDL levels of 70 mg/dL and above. Initiation of statin therapy in patients with documented atherosclerosis results in a reduction in myocardial infarction, unstable angina, stroke, need for revascularization, hospitalization, and all cause mortality compared to patients treated with diet alone.
- Early benefits (within 8 - 16 weeks) can be seen in patients presenting with ACS when started on immediate, high dose potent statin treatment (e.g. atorvastatin 80 mg/d) as shown in MIRACL and PROVE-IT.
- In ACS patients, high dose potent statin treatment, regardless of baseline LDL is recommended. In non-ACS patients where the baseline LDL is pending or not known, empiric doses may be used. Patients who fail to achieve target lipid levels (**LDL < 70 mg/dL**) at 6 weeks after initiation of therapy should have their dose increased or an additional agent (ezetimibe, niacin, or cholesterol binding resin) added.
- The combination of a statin and ezetimibe may also be used as first line therapy to achieve LDL goal, with the exception of patients with ACS in whom high dose, potent statin therapy is preferred. The target lipid levels in patients with AVD or diabetes are LDL cholesterol < 70 mg/dL HDL cholesterol >40 mg/dL, and triglycerides (TG) < 150 mg/dL. The ideal LDL in all patients is likely LDL < 70 mg/dL (ongoing trials are evaluating this further). The benefits of statins are seen in men and women, older and younger patients, diabetics and nondiabetics.
- ***Patients with atherosclerosis and/or diabetes will live longer when treated with statins. In the 4S trial there was a 34% risk reduction in major cardiac events, a 42% risk reduction in cardiovascular mortality and a 30% reduction in all cause mortality associated with statin treatment. The LIPID trial demonstrated that even patients with "low or normal" levels of total cholesterol and LDLcholesterol (LDL 70-170 mg/dl) have mortality reduction with statin treatment. The HPS trial demonstrated that patients with LDL < 100 at baseline, derive similar risk reduction to those with higher LDLs.***

ACE Inhibitors:

- ACEI have potent vascular and cardiac protective effects, are indicated in all patients with atherosclerosis. Patients with coronary, peripheral, cerebral vascular disease, and diabetes have reduced risk of MI, stroke, heart failure, and death when treated with an ACEI
- All post CABG, post PTCA, post unstable angina, post MI, stable CAD, PVD, CVD, and diabetic patients should receive an ACEI unless a specific contraindication is documented. Patients with acute myocardial infarction have improved early survival and less heart failure when treated with ACEI. All MI patients without contraindications should be started on ACEI within 12-24 hours and treated long term. Patients with left ventricular dysfunction should be started and maintained on an ACE inhibitor indefinitely.
- Contraindications include history of angioedema, cardiogenic shock, hyperkalemia, and pregnancy. Angiotensin receptor antagonists should be used in ACEI intolerant patients.
- ***The HOPE and EUROPA trials demonstrated that in patients with CAD, CVD, PVD or diabetes the use of an ACE inhibitor was associated with a reduction in cardiovascular events, cardiovascular mortality, and all cause mortality. The PEACE trial was underpowered. This benefit was seen in patients without hypertension and with normal left ventricular ejection fractions. Long term treatment with ACEI is thus indicated in any patient with atherosclerosis.***

Beta Blockers:

- Beta blockers should be considered in all patients with atherosclerosis, since they reduce the risk of myocardial infarction and make it more likely that a patient will survive an infarction.
- These agents should be considered first line agents for the symptomatic control of angina. In addition these agents prolong survival in patients with previous myocardial infarction as well as reduce the risk of unstable angina in patients with coronary artery disease. These agents also attenuate the remodeling process post myocardial infarction and reduce the risk of developing heart failure.
- Use target doses as clinically tolerated. In patients with LVEF < 0.40 with or without heart failure symptoms, carvedilol is preferred.

- Contraindications include symptomatic bradycardia, 2nd/3rd degree AV block without pacemaker, cardiogenic shock, acutely decompensated heart failure, severe asthma or COPD, diabetic with recurrent life threatening hypoglycemic episodes. Please note that diabetes, peripheral vascular disease, mild/moderate asthma or COPD, asymptomatic bradycardia, and heart failure are not contraindications and should not preclude the use of beta blockers.

Fish Oil (Omega-3 Fatty Acids):

- Omega-3 fatty acids have been demonstrated to have a variety of cardiovascular protective effects. Fish oil supplementation has been demonstrated in clinical trial to reduce the risk of cardiovascular events by 10 to 20%. This benefit was additive to cardiovascular protective medications. It is recommended that all patients with atherosclerosis or diabetes be treated with omega 3 fatty acid supplementation, with therapy beginning in the hospital. Patients may be treated with fish oil capsules containing 800 to 1000 mg of omega-3 fatty acids (eicosapentaenoic acid, [EPA] and docosahexaenoic acid, [DHA]) PO daily. Alternative supplements include flax seed oil or canola oil.

Aldosterone Antagonists:

- indicated in patients with AMI and left ventricular ejection fraction < 0.40 and who have signs or symptoms of heart failure or diabetes.
- attenuate remodeling and have been demonstrated to benefit patients with acute myocardial infarction with left ventricular dysfunction with heart failure symptoms. Patients should be clinically stabilized prior to initiation of the aldosterone antagonist.
- This therapy in only indicated in patients with systolic dysfunction (LVEF < 0.40), not all ACS patients. Start low dose and need to closely monitor potassium levels and renal function. Hyperkalemia is an absolute contraindication. Use extreme caution if Cr > 2.5 mg/dL in men or >2.0 mg/dL in women.
- Starting either spironolactone at 6.25 mg PO daily with target dose of no more than 25 mg daily or Eplerenone 25 mg daily starting dose with target dose of 50 mg daily.
- ***The EPHESUS trial demonstrated a 15% reduction in mortality with the selective aldosterone antagonist eplerenone in AMI patients with LVEF < 40% with heart failure signs or symptoms.***

EKG Guide

This is the criteria you should use to read all EKGs

- **<u>Determine HR</u>**- 1 small box=.04ms – 1 large box=.20 ms Sinus rhythm- 60-100 BPM

  For regular HR 50-300: using R-R interval, count down in this interval for every large box: 300, 150, 100, 75, 60, 50

  For regular HR <50: measure the number of large boxes between the R-R ratio and divide by 300. Ex: for an R-R interval with 8 boxes between them the HR would be 300/8=38 BPM

  For irregular HR find average HR using 6 second method: count the number of QRS complexes in a 6 second time frame (30 large boxes) then x10 to find the average # of BPM

- **<u>Determine Rhythm</u>:** Regular rhythm vs. irregular rhythm. If irregular check for regularly irregular rate vs. irregularly irregular rate. Look for premature atrial and ventricular contractions. Check for P waves, should be 1:1 ratio with QRS complexes. >1:1 ratio 2° AVB type 1 & 2, 3° AVB

  No P wave = Atrial fibrillation vs atrial flutter

- **<u>Determine QRS Axis:</u>** Axis can be determined using any of the limb leads

  1) Normal axis -30° to 100°(+) Lead I & II

2) Left axis deviation -30° to -90° (+) Lead I (-) Lead II

3) Right axis deviation+90° to +180° (-) Lead I (+) II

4) indeterminate axis deviation -90° to -180° (-) Lead I, II

Causes for Axis Deviations:

Left axis deviation: Normal Variation (often with age), Mechanical shifts (expiration, high diaphragm in pregnancy, ascites, and abdominal tumor), left anterior fascicular block, LBBB, left ventricular hypertrophy, PVCs, congenital heart disease (atrialseptal defect, endocardial cushion defect), Hyperkalemia, Preexcitation syndromes, Inferior wall MI

Right axis deviation: Normal variation (vertical hearth with an axis of 90),Mechanical shifts (inspiration and emphysema), left posterior fascicular block, right ventricular hypertrophy, RBBB, Dextrocardia, Preexcitation syndrome, later wall MI, PVCs (in extreme right shifts)

Determine Intervals

PR interval. Normal is 0.12sec - 0.20 sec.

Shortened PR interval is <0.12sec. Causes include AV nodal rhythm, low atrial nodal rhythm, WPW Syndrome, Lown-Ganong-Levine (LGL), HTN, Normal variant

Prolonged PR interval. >0.20 AV Block, hyperthyroidism

QRS Interval

QRS interval- 0.06-0.10 sec = normal interval
QRS Prolongation 0.10- 0.12sec
>0.12sec=bundle branch block or Ventricular tachycardia

RBBB- slow RV activation: Secondary R wave (R') in V1& V2. Broad Lateral S waves (I,V5, V6) Normal QRS axis

LBBB- slow LV activation: QS wave in V1 & V2 Large, positive, widened R waves in I, aVL & V6. ST & T waves opposite QRS. QRS axis is normal or slightly to left

Left Anterior Hemiblock Shift in axis to left (usually greater than -45°) Small R waves in inferior leads II, III, aVF. Small Q waves may be notched in leads I,aVL, V5, V6 QRS may or may not be > 0.12 seconds.

Left Posterior Hemiblock Shift in axis to the right approx 110° early activation by normally conducting anterior and septal fascicles= initial small R waves in I, V5, V6 Mid-temporal and terminal activation= rightward axis +90° to +180°, qR morphology in leads II, III, aVF, rS morphology in leads I and aVL QRS may or may not be >0.12 sec

QT interval/QTc: QT > ½ R-R interval = abnormal QTc – QT interval corrected for the HR. QTc= QT/√R-R (Men < 0.44 sec & Women < 0.46)

Check for P wave abnormalities:

Lead II- should be up (+) &≤ 2.5 small boxes tall &≤0.12 sec long, P wave in aVR should be negative. V1 P wave often biphasic with neither P wave components>1.5 mm tall

LAE P wave> 0.12sec in lead II or >1.5mm tall in second P wave component of notched P wave in V1

RAE P wave >2.5mm tall in lead II or first P wave component in notched P wave in V1 >1.5mm tall

Do the p waves all look the same? Consider WAP if at least 3 different looking P waves exist with a rate <100 and MAT with HR >100

Check for Q wave formation = cardiac death - primary indication pt. has had MI-norm seen 24 hrs after MI Q wave = neg deflection infront of R wave (significant if >0.04 sec wide or >1/3 height of R wave)

Check for Abnormalities in QRS complex: QRS in V1-V3 should be≥ 1 small box wide or >25% amplitude of corresponding R wave except in lead III
Check for R wave progression: if Q = R later than V4 than poor R wave progression

Check for ST segment abnormalities: > 1 mm elevation = infarction
> 1 mm depression = ischemia

Determining area of infarct

Anterior/septal MI: Leads V1-V4

Lateral MI: I, aVL, V5, V6

Inferior MI: II, III, aVF Posterior MI: V1, V2

Right Ventricular MI: V3R, V4R, V5R, V6R

Check for T wave abnormalities: often the first to change even before ST segment changes T wave inversion indicated cardiac ischemia, should be positive in lead I, II, V3-V6 (V2 is vent. Septal)

LVH

There are several sets of criteria used to diagnose LVH via electrocardiography

The Sokolow-Lyon index
- S in V_1 + R in V_5 or V_6 (whichever is larger) ≥ 35 mm
- R in aVL ≥ 11 mm

The Cornell voltage criteria for the ECG diagnosis of LVH involves measurement of the sum of the R wave in lead aVL and the S wave in lead V_3. The Cornell criteria for LVH are:

- S in V_3 + R in aVL > 28 mm (men)
- S in V_3 + R in aVL > 20 mm (women)

Romhilt-Estes point score system

- Probable left ventricular hypertrophy is diagnosed if 4 points are present and definite left ventricular hypertrophy is diagnosed if 5 or more points are present.

Criterion	Points
Any limb R wave or S wave >/=2.0 mV (20 mm) OR S in V1 or S in V2 >/= 3.0 mV (30 mm) OR R in V5 or R in V6 >/= 3.0 mV (30 mm)	3
ST-T wave changes typical of LVH Taking digitalis Not taking digitalis	 1 3
Left atrial abnormality P terminal force in V1 is 1 mm or more in depth with a duration >/= 40 ms (0.04 sec)	3
Left axis deviation >/= -30[a]	2
QRS duration >/= 90 ms	1
Intrinsicoid deflection in V5 or V6 >/= 50 ms (0.05 sec) *	1

Sensitivity and specificity for selected ECG criteria of LVH

Criterion	Sensitivity (%)	Specificity (%)
Sokolow Lyon Voltage	22	100
Cornell Voltage Criteria	42	96
Cornell Voltage Duration Criteria	51	95
RaVL > 11 mm	11	100
Romhilt-Estes > 4 points	54	85
Romhilt-Estes > 5 points	33	94

RVH
- Large R wave in V1 and V2 (R>S) and/or smaller R waves in V5,V6 (R<S)
- Deep S wave in V5 and V6
- V3R and V1 and V2, R > S V4R
- R:S ratio > 1:1
- ST segment depression and T wave inversion in right precordial leads is usually seen in severe RVH such as in pulmonary stenosis and pulmonary hypertension.

Cardiac Arrhythmias

***add to existing section of Bradycardia and Tachycardia in UCLA yellow book**

Atrioventricular Heart Blocks (AVB)

1st degree AVB

EKG changes
- Prolonged PR interval >0.20 seconds

Causes
- CAD, MI, AV node ischemia as well as digitalis or other drugs affecting the AV node.

Treatment
- Treatment is generally only required if bradycardia develops or if the PR >.25seconds and continues to increase.
- Monitor for worsening blocks such as second and third degree AVB

2nd degree AVB type 1 (Wenckebach/Mobitz I)

EKG changes
- Increasing PR interval with each QRS with an eventual P wave with no QRS following.

Causes
- Injury/ischemia to the AV node (AV node slows progression/stops progression of the SA impulse to the ventricules.
- Commonly seen temporarily after an MI

Treatment
- Treatment is not normally required. Continue close monitoring for worsening blocks

If Bradycardia consider:
- Atropine to increase the AV conduction rate
- Epinephrine to increase SA node and overall HR
- Pacing- consider transcutaneous or temporary transvenous pacemakers

2nd degree AVB type 2/ Mobitz II

EKG changes
- Multiple p waves for each QRS complex
- P waves "march out" meaning they occur at set intervals
- PR interval before the QRS complex remains constant/fixed

Causes
- Blocked conduction from AV node to the bundle branches r/t disease of AV node, AV junctional tissue, or His-Perkinje system

Treatment to consider
- Atropine- 0.5 mg q3-5 minutes for a total of 3mg
- Epinephrine- 1mg q5 minutes
- Temporary pacemaker
- Permanent pacemaker is often placed as well since conduction to the ventricles is unpredictable and can quickly lead to 3rd degree AVB and sudden cardiac arrest

3rd degree AVB/Complete Heart Block

EKG changes
- HR 30-40 BPM unless junctional escape mechanism is present
- PR interval unconstant
- P waves march out but QRS complexes do not correlate with them since they are depolarized independently
- Not all P waves are followed by QRS complex
- Regular atrial and ventricular rates

Causes
- Ischemia or injury to the AV node, junctional tissues, or His-Purkinje tissue
- Ischemia r/t CAD, acute MI, drug toxicity (digitalis), systemic disease, electrolyte imbalances, renal insufficiency/ESRD/ARF etc.

Treatment
- Atropine and Epinephrine (see doses above) for bradycardia
- Fluid replacement for hypotension

- Temporary pacemaker if r/t acute cause
- Cardiology consult for evaluation-if heart block is r/t chronic cause, a permanent pacemaker is warranted.

Ventricular Arrhythmias

Ventricular Tachycardia

Brugada Criteria
***** Brugada criteria consists of four criteria

- Lack of an RS complex in the precordial leads
- Whether the longest interval in any precordial lead from the beginning of the R wave to the deepest part of the S wave when an RS complex is present is greater than 100 ms
- Whether atrioventricular dissociation is present
- Whether both leads V1 and V6 fulfilled classic criteria for ventricular tachycardia.
- sensitivity of the four consecutive steps was 0.987, and the specificity was 0.965 for diagnosis of VT
- If none of these criteria are present, then the likely rhythm is SVT with aberrant conduction.

Treatment of VT with a pulse/stable

- Synchronized Cardioversion
- Amiodarone 150mg IV over 10 min, then 1mg/min x6hrs, then 0.5mg/min x 18 hrs.
- Check electrolytes
- 12 lead EKG if possible

Treatment of PULSELESS VT

- IMMEDIATE Defibrillation
- CPR between shocks, IV placement, intubation
- Medication treatment =E.V.A.
- **E**pinephrine- 1mg q5min
- **V**asopressin 40 units x1- can be used to replace 1st or 2nd dose of epinephrine
- **A**miodarone 300mg IV, then 150 mg IV, can repeat 150mg IV x1

Torsades de pointe
EKG changes

- Paroxysms of 5-20 beats, with a HR faster than 200 beats per minute. Sustained episodes are occasionally seen.
- Progressive change in polarity of QRS about the isoelectric line occurs with complete 180 degree twist of QRS complexes in 10-12 beats.
- Usually, a prolonged QT interval and pathological U waves are present, reflecting abnormal ventricular repolarisation. The most consistent indicator of QT prolongation is a QT of 0.60 s or longer or a QTc (corrected for heart rate) of 0.45 s or longer.
- A short-long-short sequence between the R-R interval occurs before the trigger response.
  - QRS complexes look different-rotate on the axis
  - QT interval >0.40/QTc interval >0.44 seconds in males and >0.46 seconds in females prior to the start of VT

Causes

- Hypomagnesemia, hypokalemia
- Long QTc/QT intervals r/t congenital long QT syndrome
- medications that increase the QT interval (see www.torsades.org for a complete list of drugs and their risk for causing torsades)

Treatment

- Magnesium can be given at 1-2 g IV initially in 30-60 seconds, which then can be repeated in 5-15 minutes. Alternatively, a continuous infusion can be started at a rate of 3-10 mg/min.
- Acceleration of the heart rate can be achieved by using B1-adrenergic agonists such as isoproterenol or overdrive electrical pacing with temporary transvenous pacing

Ventricular fibrillation

EKG changes

- No P waves, QRS complexes, or T waves
- Asymmetrical fibrillatory waves of different sizes depending on fine/coarse v-fib

Causes

- Ischemic heart disease, cardiomyopathies (hypertrophic and dilated) , CHF, valvular disease, digitalis toxicity, electrolyte imbalance, Lown-Ganong-Levine syndrome, WPW, RVOT syndrome, Long QT syndrome, and Brugada Syndrome.

Treatment

- event of cardiac arrest, the immediate implementation of ACLS guidelines
- Defibrillate the patient (adult, 200-300 J)
- Administer amiodarone (300 mg IV). If does not work, then use lidocaine (1-1.5 mg/kg bolus).

Heart Failure in CCU

In addition to yellow book heart failure add this updated ACE protocol

Hemodynamic Management of Cardiomyopathy Patients (ACE Protocol)

1. Hemodynamics are examined after RHC placed: if PCW>16, and/or cardiac index<2.2 L min/m² on two sets of hemodynamic measurements obtained in the CCU, the swan ganz catheter should remain in place.
2. lf SVR<1300, PCW>20, RA>10, and CI>2.2 then begin diuretics alone and restart previous ACE inhibitors.
If the hemodynamics subsequently deteriorate (SVR>1500), discontinue oral vasodilators (except nitrates) and begin Nipride gtt.
3. lf the SVR>13OO and PCW>20 or Cl<2.2, begin Nipride with the following hemodynamic goals: **SVR<1200, PCW<15, RA<7, while maintaining SBP>80**
4. If patient has very low cardiac index (<1.4 or CO <4) in the setting of low SVR or if patient is in shock, this meets criteria for initiation of inotropic agents. (Patient with low CI or pulmonary HTN, PA>70, require transfer to the CCU).
5. **Nipride** starting dose is **0.3 mcg/kg/min**, titrating upwards by 0.3mcg/kg/min q 5-10 mins to a **maximum dose of 4mcg/kg/min**, or until optimum aerodynamics are achieved. Diuresis with IV Lasix should also be initiated as well.
6. One should obtain hemodynamic goals (seen by RHC measurements)on Nipride and IV Lasix and these optimal hemodynamics should be maintained for a minimum of 2-4 hrs prior to starting oral vasodilators.
7. The initial PO vasodilator of choose is Captopril. Captopril titration as follows: **Captopril 6.25 mg. Increase to 12.5 mg after 2 hours**, if tolerates meaning SBP >80 and asympyomatic, **after additional 2 hours give 25 mg, then increase by 25 mg every 6 hours** (e.g. 50mg, 75mg, then 100mg) only as necessary to taper off Nipride and match the hemodynamic goals achieved on Nipride, up to a maximum of 100mg PO Q6 hours. Do not continue to titrate Captopril dose once off Nipride unless specific indication (i.e. SVR>1500 of SBP>100). Avoid hypotension or advancing ACEI dose despite low SVR due to risk of renal insufficiency.
8. **Isordil** to be started with initial unloading therapy in patients with CAD (10mg or their admit dose of Nitrated). For patients without coronary artery disease, once Captopril dose his reached 25 mg and still on Nipride or for elevated filling pressures despite continued diuresis, lsordil to be started at **10 mg tid.** lf patient tolerates, this maybe titrated, but only if indicated by high filling pressures or high SVR. Do not increase nitrates beyond 20mg tid, unless indicated. If indicated isordil may increase by 10-20mg q8hr, to a maximum of dose of 80 tid.
9. **If optimum hemodynamics are achieved and sustained for 6-8 hours** (absolute minimum 4 hours) on Captopril or Captopril/lsordil regimen, **Swan can be discontinued.**
10. lf optimum hemodynamics are not achieved on Captopril/lsordil regimen or if captoril is not tolerated due to serious side effects (i.e. severe symptomatic hypotension, renal insufficiency, allergy), the decision regarding the addition of **Hydralazine** should be made by the CCU attending in injunction with the Cardiomyopathy attending. Continue Captopril at the greatest tolerated dose (usually 100mg q6h) or switch to longer acting ACE inhibitor. Hydralazine is started at 25mg, after 2 hours 50mg, then 50mg q6h, increase by 25mg every 6 hours (e.g. 75mg, l00mg, then 150mg) as needed to maximum 150mg PO q6hr. Isordil is to be continued or started. Hydralazine may also be given tid.
11. **Aldosterone antagonist** is indicated unless hyperkalemia or significant renal insufficiency (Cr > 2.5 in men and > 2.0 mg/dL in women. Begin at low dose (e.g. spironolactone 6.25 mg/d or eplerenone 12.5 mg/d) and very

closely monitor serum K+ and renal function.
12. **Diuresis is of utmost importance in these patients', Lasix should be given 2 to 4 times a day with supplemental potassium**. Monitor I/Os closely multiple times per day.
13. Potassium and magnesium to be closely followed, K+ should be kept 4-5.5 meg/dL Mg++ should be kept >2.0 meg/dL.
14. Patients should receive a 2 gram sodium diet and 1500-2000 cc fluid restriction.

Pacemaker basics

Indications for Pacemaker

Guidelines for implantation of cardiac pacemakers have been established by a task force formed jointly by the American College of Cardiology, the American Heart Association, and the Heart Rhythm Society (ACC/AHA/HRS) but is still a datable subject. Generally, it is helpful to divide the indications for pacemaker implantation into three specific classes, as defined by the ACC/AHA/HRS guidelines

- Class I — Conditions in which permanent pacing is definitely beneficial, useful, and effective. In such conditions, implantation of a cardiac pacemaker is considered acceptable and necessary, provided that the condition is not due to a transient cause.
- Class II — Conditions in which permanent pacing may be indicated but there is conflicting evidence and/or divergence of opinion; class IIA refers to conditions in which the weight of evidence/opinion is in favor of usefulness/efficacy, while class IIB refers to conditions in which the usefulness/efficacy is less well established by evidence/opinion.
- Class III — Conditions in which permanent pacing is not useful/effective and in some cases may be harmful.

The most common indications for pacemaker placement are sinus node dysfunction followed by AV block. Other less common indications include carotid sinus syncope and post-ablation

Sinus Node Dysfunction Indications

The indication for permanent pacing in patients with sinus node dysfunction is usually due to the bradycardia with the following symptoms: syncope, seizures, heart failure, dizziness, and confusion.

Class I — The following conditions are considered class I indications for pacemaker placement:

- Sinus bradycardia in which symptoms are clearly related to the bradycardia (usually in patients with a heart rate below 40 beats/min or frequent sinus pauses).
- Symptomatic chronotropic incompetence.

Class II — The following are considered to be class II, or possible, indications for pacemaker placement in patients with sinus node dysfunction:

- Sinus bradycardia (heart rate <40 beats/min) in a patient with symptoms suggestive of bradycardia, but without a clearly demonstrated association between bradycardia and symptoms.
- Sinus node dysfunction in a patient with unexplained syncope.
- Chronic heart rates <40 beats/min while awake in a minimally symptomatic patient.
- A less distinct group of patients with sinus bradycardia of lesser severity (heart rate >40 beats/min) who complain of dizziness or confusion that correlates with the slower rates.

AV Node Block Indications

AV block is the second most common indication for permanent pacemaker placement. Causes include the following: fibrosis and sclerosis of the conduction system, ischemic heart disease, digitalis, calcium channel blockers, beta blockers, amiodarone, increased vagal tone, valvular disease, congenital heart disease, cardiomyopathies, myocarditis, hyperkalemia, and infiltrating malignancies .

Class I Indications — The following conditions represent severe conduction disease and are generally considered to be class I indications for pacing, regardless of associated symptoms:

- Complete (third-degree) AV block*
- Advanced second-degree AV block (block of two or more consecutive P-waves)
- Symptomatic Mobitz I or Mobitz II second-degree AV block
- Mobitz II second-degree AV block with a widened QRS or chronic bifascicular block, regardless of symptoms
- Exercise-induced second or third degree AV block (in the absence of myocardial ischemia)

* current ACC/AHA/HRS guidelines classify asymptomatic third-degree AV block with average awake ventricular rates ≥40 beats/min, in a patient with normal left ventricular size and function, as a class IIA indication for permanent pacemaker implantation. Yet many cardiologists consider this condition a definite indication for pacemaker placement. The ACC/AHA/HRS guidelines consider asymptomatic complete AV block in the setting of cardiomegaly or left ventricular dysfunction to be a class I indication

Class II Indications — Patients with less severe forms of acquired AV block may still benefit from pacemaker placement. In such patients, determinations are often based upon correlation of bradycardia with symptoms, exclusion of other causes of symptoms, and/or results of electrophysiology (EP) testing.

Other indications for possible pacemaker implantation:

- Asymptomatic Mobitz II second-degree AV block with a narrow QRS interval; patients with associated symptoms or a widened QRS interval have a class I indication for pacemaker placement.
- First-degree AV block when there is hemodynamic compromise because of effective AV dissociation secondary to a very long PR interval.

- Bifascicular or trifascicular block associated with syncope that can be attributed to transient complete heart block, based upon the exclusion of other plausible causes of syncope (specifically ventricular tachycardia)
- Patients that meet Class I indications for pacing after myocardial infarction, regardless of symptoms:
  - Third-degree AV block within or below the His-Purkinje system.
  - Persistent second-degree AV block in the His-Purkinje system, with bilateral bundle branch block.
  - Transient advanced infranodal AV block with associated bundle branch block.
- Patients that meet class I indications in patients with neurocardiogenic syncope:
  - Significant carotid sinus hypersensitivity, defined by syncope and >3 seconds of asystole following minimal carotid sinus massage

Pacemaker Modes

1st Letter	Chamber Paced 2nd Letter	Chamber Sensed 3rd Letter	Response to Sensed Beat 4th Letter	Programmability 5th Letter
Antitachycardia Function				
A	A	T	P	P (pacing)
V	V	I	M	S (shock)
D	D	D	C	D (dual: pacing + shock)
O	O	O	R	
			O	

A = Atrium V = Ventricle D = Dual (both chambers) O = None

T = Triggered I = Inhibited

D = Double (Atrial triggered and ventricular inhibited)

The first 3 letters are used most commonly. More modern pacemakers have multiple functions. A pacemaker in VVI mode denotes that it paces and senses the ventricle and is inhibited by a sensed ventricular event. Alternatively, AAT mode represents pacing and sensing in the atrium, and each sensed event triggers the generator to fire within the P wave.

The DDD mode denotes that both chambers are capable of being sensed and paced. This requires two functioning leads, one in the atrium and the other in the ventricle. In the ECG, each QRS is preceded by two spikes. One indicating the atrial depolarization and the other indicating the initiation of the QRS complex. Given that one of the leads is in the right ventricle, a left bundle-branch pattern may also be evident upon the ECG. Note that a two-wired system need not necessarily be in DDD mode, since the atrial or ventricular leads can be programmed off. Additionally, single tripolar lead systems are available that can sense atrial impulses and either sense or pace the ventricle. Thus, this system provides for atrial tracking without the capability for atrial pacing and can be used in patients with atrioventricular block and normal sinus node function.

Transplant Basics for CCU

Indications- Severe heart disease despite adequate medical therapy

- Dilated cardiomyopathy
- Ischemic cardiomyopathy
- Congenital heart disease for which no conventional therapy exists or that conventional therapy has failed
- Ejection fraction less than 25%
- Intractable angina or malignant cardiac arrhythmias for which conventional therapy has been exhausted
- Pulmonary vascular resistance of less than 2 Wood units (160 dyn·s/cm^5) or Transpulmonary gradient >15

Contraindications

- Fixed pulmonary vascular resistance of greater than 3 Wood units (240 dyn·s/cm^5)
- Active systemic infection
- Active systemic disease such as collagen vascular disease , myopathic disease, or sickle cell disease
- Active malignancy: Patients with malignancies who have demonstrated a 3- to 5-year disease-free interval may be considered, depending upon the tumor type and the evaluating program.
- An ongoing history of substance abuse (eg, alcohol, drugs, tobacco)
- Psychosocial instability
- Inability to comply with medical follow-up care

Criteria for Transplant Listing at UCLA
(Adpated from Goldhaber et al)

Patients are listed with UNOS (United Network for Organ Sharing)

Listing Options:

- Status 1A or IB-top priority for transplant
- Status lB's are allowed to be at home
- Status 2-at home waiting
- Status 7-temporarily inactive
- FYI: No status system for the lung transplants

Patient who wait for transplant dependent on:

- Height and Weight
- Blood Type
- Status
- Time spent waiting on the list
- **ABO Compatibility- most important**

UCLA's alternate list:

- Developed for patients that are not acceptable for the regular list.
- Main reason for to be on the alternate list are patients older than 70 years of age.
- Also patients who have risk factors making them unacceptable for the regular list, but without absolute contraindications.
- Patients listed as an alternate will not be eligible for status one listing if they deteriorate. Nor will they be candidates for a VAD.

Transplant Medications

Drug	Pharmacology	Side Effects
Corticosteroids (i.e Prenidsone)	Inhibition of cytokine production	Cushingoid syndrome,infections
Mycophenolate mofetil (MMF, CellCept)	Reversible inhibitor of inosine monophosphate dehydrogenase (IMPDH)	Nephrotoxic, hepatotoxic, hypertension, hyperkalemia, hyperlipidemia, hyperglycemia, hirsutism, infections
Cyclosporine (CSA, Neoral, Sandimmune)*	Calcineurin inhibitor	Hypertension, peripheral edema, GI hemorrhage, infection, malignancy, leukopenia, myelosuppression
Azathioprine (Imuran)	Antagonizes purine metabolism, which may inhibit DNA, RNA, and protein synthesis	Nephrotoxic, hepatotoxic, hypertension, hyperkalemia, hyperlipidemia, hyperglycemia, hirsutism, infections
Tacrolimus (Prograf, FK506)	Calcineurin inhibitor	Risk of malignancy, hepatotoxicity, thrombocytopenia, anemia, leukopenia
Sirolimus (Rapamycin, Rapamune)	Antimigratory/antiproliferative effects on vascular smooth muscle	Hepatotoxicity, nephrotoxicity, thrombocytopenia, hyperlipidemia, hyperkalemia
Everolimus	Analog of rapamycin with antiproliferative and immunosuppressive activity	Anemia, thrombocytopenia, hypertension, hyperlipidemia, thrombosis, hepatotoxicity
		Leukopenia, thrombocytopenia, GI upset, hyperlipidemia, infection, nephrotoxicity

*There are different brands of cyclosporine with different bioavailability, the same brand should be used as an outpatient in the inpatient setting.

Goal Immunosuppression Levels

Months after Transplant	Tacrolimus Level	Cyclosporine
0-1 months	10-15	250-350
2-3 months	8-12	200-300
>3 months	5-10	150-250
>6 months	5-10	100-200

Cylex Test

- An immune cell function test, which measure the amount of ATP a patient's WBC uses. It is used as a surrogate marker of immune function
- Very immunosuppressed: <225 (patient at risk for infection)
- Goal immunosuppression: 270-300 depending on type of organ

Lipid Lowering Drugs

DRUGS	LDL	TG	HDL	Comments
Statins	**30-60%**	10-30%	10%	First-line drugs for LDL, best data
Ezitimibe	**10-20%**	<10%	-	Best combined with statin
Fibrates	0-20%	**20-50%**	**10-20%**	Mainly for TG and HDL (variable LDL)
Niacin	5-25%	**20-50%**	**15-30%**	Mainly for TG and HDL (also LDL)
Fish oil		**20-50%**	-	Only for TG (may increase LDL)
Resins	**15-25%**	10-30%	<10%	Best combined with statin, may increases TG

Statins (HMG CoAreductase inhibitors):

- Mechanism: increases LDL receptors, anti-inflammatory,pleotrophic effect with plaque, stabilize and reverse endothelial dysfunction. Only class to demonstrate reductions in *overall mortality* for 1° and 2° prevention ~ 30-40% LDL reduction→30-40% CHD risk reduction over 5 yrs
- NNT to prevent 1 event in 1 yr = 63 (high risk), 250 (low risk); 5 yrs = 17 (high risk), 66 (j risk) REVERSAL TRIAL [JAMA 3/2/04], PROVE-IT [NEJM 4/8/04], TNT [NEJM 4/7/05]: goal LDL ~ 70

Transaminitis (persistent) = *0.5-3% occurrence* (dose dependent)

- Monitoring unnecessary?? (0.5-3% transaminitis ~ placebo incidence)
- Baseline transaminitis: < 3x ULN →statin not contraindicated (d/c if persists > 3x ULN)

Myopathy: Myalgias (1-2%) →Myositis (sx + increase CK; 0.5%)→Rhabdo (CK » 10 x ULN; < 0.01%)

- Onset usually w/in first few months, but may occur anytime
- *Routine* CKs not recommended, but monitor if high risk or sxs

Higher risk = elderly/frail, exercise, ETOH/cocaine, renal or liver disease, hypothyroid Teratogenic

Drug interactions:*increaseMyopathy/AEs,*fibrates, niacin, cyclosporine, CYP3A4 inhibitors (amiodarone, macrolides [erythro/clarithro], azoleantifungals, verapamil/diltiazem, anti-retrovirals [Pis], grapefruit juice)

Decrease Statin effects: enzyme inducers (phenytoin, tegretol, rifampin, barbiturates, St. John's wort) Somestatins may increase effects of warfarin (INR) - poorly characterized, case reports

Pravastatin, rosuvastatiin, fluvastatin = less interactions (not metabolized by CYP3A4)

Ezetimibe (Zetia): 10 mg daily **[Vytorin®** = simvastatin/ezetimibe] Cholesterol absorption inhibitor at small intestine brush border. Decreasedabsorbed/excreted in bile; rare angioedema, myopathy

Fibrates (gemfibrozil, fenofibrate): gemfib 600mg BID, fenofib 130mg QD (decrease dose for renal/elderly). Gemfibrozil may cause myopathy-fenofibrate less assoc. with myopathy or statin interactions Can enhance hypoglycemia with glitazones, and enhance effects of warfarin

Niacin (nicotinic acid; not nicotinamide/inositol): Niaspan® 500mg-2gm QHS

Flushing (decrease w/ASA, NSAID), dyspepsia, hepatotox, increase uric acid, hyperglycemia, myopathy

Fish oil (omega-3 FAs): 2-4 gm/day of omega-3 FAs (EPA + DHA content)

Most supplements: 1 gm fish oil = [300J-400 mg omega-3 FAs, therefore dose is ~ 10 caps/dyOmacar® = FDA-approved.1 gm cap = 840 mg EPA/DHA (dose = 4/day) Side effects = Dyspepsia, fishy taste/burp, increase bleeding

Bile acid sequestrants ("resins") cholestyramine, colestipol, colesevelam

Rarely used now but effectively decreasesLDL (esp. w/statin). Powder forms must be mixed with fluid G/I with increase in TG, decreases absorption of many drugs (give 1 hr before or 4 hrs after), constipation

Commonly Cited Cardiology Articles

- **The following articles are some of the common quoted articles in both the CCU and Cardiology in the past decade.**

CAD, ACS:

1. Boden WE, O'Rouke RA, Teo KK, et al. Optimal medical therapy with or without PCI for stable coronary disease. N Eng J Med. 2007 Apr 12;356(15): 1503-16. **COURAGE Trial.**

* Demonstrated that as an initial management strategy in patients with stable coronary artery disease, PCI does not reduce the risk of death, myocardial infarction, or other major cardiovascular events when added to optimal medical therapy. Only noted difference was reduction in symptoms of angina.

2. Cannon CP; Braunwald E; McCabe CH; Rader DJ; Rouleau JL; Belder R; Joyal SV; Hill KA; Pfeffer MA; Skene AM. Intensive versus moderate lipid lowering with statins after acute coronary syndromes. N Engl J Med 2004 Apr 8;350(15):1495-504. **PROVE IT-TIMI 22 Trial.**

* This study demonstrated that among patients who have recently had an acute coronary syndrome, an intensive lipid-lowering statin regimen with atorvastatin 80mg/day vs. simvastatin 40mg/qday provided greater protection against death or major cardiovascular events. It revealed that intensive lipid-lowering regimen had added benefits compared to standard therapy.

3. Ray KK, Cannon CP, Cairns R, Morrow DA, Ridker PM, Braunwald E. Prognostic Utility of ApoB/AI, Total Cholesterol/HDL, Non-HDL Cholesterol, or hs-CRP as Predictors of Clinical Risk in Patients Receiving Statin Therapy After Acute Coronary Syndromes. **Results From PROVE IT-TIMI 22**. Arterioscler Thromb Vasc Biol.

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* In ACS patients receiving statin therapy, apoB/AI, TC/HDL, and non-HDL-C offered similar prognostic information to LDL-C. However, it revealed that the addition of hs-CRP to lipid-based measurements significantly improved risk prediction. Thus, CRP measurement in patients on treatment with statins may offer additive prognostic information to lipids in ACS patients.

4. Giuseppe Patti, MD; Giuseppe Colonna, MD; Vincenzo Pasceri, MD, PhD et al. Randomized Trial of High Loading Dose of Clopidogrel for Reduction of Periprocedural Myocardial Infarction in Patients Undergoing Coronary Intervention. Results From the **ARMYDA-2 Study** . Circ 2005;111:2099-2106

*Pretreatment with high loading dose of clopidogrel 600mg when given 4 to 8hrs prior to PCI is safe and superior compared to 300mg loading dose. This dose significantly reduced periprocedural MI in patients undergoing PCI; it was associated with a 50% risk reduction of MI.

5. Salim Yusuf, D.Phil., F.R.C.P.C., Feng Zhao, M.Sc., et al. Effects of Clopidogrel in Addition to Aspirin in Patients with Acute Coronary Syndromes without ST-Segment Elevation. *NEJM* 2001;345:494. **CURE Trial**

***** Clopidogrel has beneficial effects in patients with acute coronary syndromes without ST-segment elevation in addition to aspiring. . However, the risk of major bleeding is increased among patients treated with clopidogrel, but risk of fatal bleeding is not increased.

6. Ridker PM, Danielson E, Fonseca FA. Rosuvastatin to prevent vascular events in men and women with elevated C-reactive protein. *NEJM* 2008; 359: 2195-207 **JUPITER Study**

*In apparently healthy people without hyperlipidemia (LDL <130) but with elevated high-sensitivity C-reactive protein levels (hsCRP >2), rosuvastatin 20mg reduced both LDL and hsCRP levels and significantly reduced mortality and the incidence of major cardiovascular.

Heart Failure:

1. Poole-Wilson PA, Swedberg K, Cleland JG. Comparison of carvedilol and metoprolol on clinical outcomes in patients with chronic heart failure in the Carvedilol Or Metoprolol European Trial **(COMET):** randomized controlled trial. Lancet 2003 Jul 5;362(9377):7-13

*This study demonstrated that in subgroup of CHF patients with EF of <35%, Carvedilol more significantly reduced all-cause mortality and conferred a survival benefit when compared to Metoprolol.

2. JoAnne Micale Foody; Michael H. Farrell; Harlan M. Krumholz. Beta Blocker Therapy in Heart Failure: Scientific Review. *JAMA* 2002; 287:883.

*This study demonstrated that in NHA class II-IV patients, beta blockers decrease mortality by 35%, and rehospitalization by 40%.

3. Anne L. Taylor, M.D., Susan Ziesche, R.N., Clyde Yancy, M.D. Combination of Isosorbide Dinitrate and Hydralazine in Blacks with Heart Failure. *NEJM* 2004; 351:2049. **A-HeFT Trail**.

* Addition of a fixed dose of isosorbide dinitrate plus hydralazine to standard therapy for heart failure demonstrated a 40% decrease in mortality in blacks.

4. Bertram Pitt, M.D., Faiez Zannad, M.D., Willem J. Remme, M.D. The Effect of Spironolactone on Morbidity and Mortality in Patients with Severe Heart Failure. *NEJM* 1999; 341: 709. **RALES Trial**.

*Among patients with NYHA class III or IV heart failure and EF<35%, the addition of Spironolactone to standard therapy decreased mortality by 30% and hospitalization by 35%. Standard therapy included an ACEI, loop diuretic and in most cases digoxin.

5. The ESCAPE Investigators and ESCAPE Study Coordinators. Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness. *JAMA* 2005; 294:1625 **ESCAPE Trial**.

*Addition of PAC to careful clinical assessment increased anticipated adverse events, but did not affect overall mortality and hospitalization; however, it was untested in cardiogenic shock. No benefit was demonstrated in decompensated heart failure.

Electrophysicology/ Arrhythmias:

1. John G.F. Cleland, M.D., Jean-Claude Daubert, M.D., Erland Erdmann, M.D et al.

The Effect of Cardiac Resynchronization on Morbidity and Mortality in Heart Failure. NEJM 2005;352:1539-49. **CARE-HF Trial**.

*In subgroup of patients with HF due to left ventricular systolic dysfunction with NHA class III-IV and cardiac dyssynchrony, cardiac resynchronizations (biventricular pacing) along with medical therapy illustrated 36% decrease in mortality, improved EF, and improved quality of life compared to medical therapy alone. It should be considered if patients have refractory HF, EF </= 35% and QRS >/= 120ms, and if there is evidence of dyssynchrony on echocardiography.

2. Gust H. Bardy, M.D., Kerry L. Lee, Ph.D., Daniel B. Mark, M.D. et al. Amiodarone or an Implantable Cardioverter–Defibrillator for Congestive Heart Failure.. *NEJM* 2005;352:225**.** *Sudden Cardiac Death in Heart Failure Trial* **(SCD-HeFT Trial)**

***** This study demonstrated that In patients with NYHA class II or III CHF and LVEF of 35 percent or less, amiodarone had no favorable effect on

survival, but single-lead shock-only ICD reduced overall mortality by 23 percent.

3. D.G. Wyse, A.L. Waldo, J.P. DiMarco et al. A Comparison of Rate Control and Rhythm Control in Patients with Atrial Fibrillation. NEJM 2002; 347:1825. **AFFIRM Trial**.

*Rhythm-control strategies in atrial fibrillation offer no survival advantage over the rate-control strategies. Moreover, there are additional advantages of lower risk of adverse drug effects with rate-control.

4. Li-Fern Hsu, M.B., B.S., Pierre Jaïs, M.D., Prashanthan Sanders, M.B., B.S., Ph.D et al. Catheter Ablation for Atrial Fibrillation in Congestive Heart Failure. NEJM 2004; 351:2373.

*Restoration and maintenance of sinus rhythm by catheter ablation in patients with AF and CHF increased EF by 21% and significantly improved symptoms, exercised capacity and quality of life.