Then University of Chicago
CCU Handbook
This handbook is dedicated to The University of Chicago Housestaff. May your CCU experience continue to challenge and inspire you long after your time spent here.

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“I love those who can smile in trouble, who can gather strength from distress, and grow brave by reflection. 'Tis the business of little minds to shrink, but they whose heart is firm, and whose conscience approves their conduct, will pursue their principles unto death.”

~Leonardo da Vinci
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I. Welcome to the Unit!

*Helpful Phone Numbers*

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Working with the non-physician staff

Unit Clerk
- Helpful in setting up clinic appointments for CCU patients that will be discharged directly from the unit

Charge RN
- Helpful in getting patients into the unit proper (i.e. D5)
- Keep him/her in the loop on patients coming in/out of the unit in real time

Policies

ECGs
- ALL ECGs MUST be personally viewed by a member of the CCU housestaff immediately upon acquisition
- Any ECG electronically read as “ACUTE MI” will be handed directly to a physician, and the technician will document the name of this reviewer
- Place a STAT Page to the CCU Fellow for any concerns

CCU-Stocked Meds
(See page 45)

Supplies

CCU Closet always/should have
- Line Kits
- Swan-Ganz Catheters
- Micro-puncture kits
- Ultrasound machine in the neighboring closet (between the supply closet and the pantry)
II. Goals & Objectives

- Maintain real-time knowledge of patients' events
- Know when to ask for help
- Locate and assimilate scientific articles relevant to your patients’ conditions
- Identify adverse events in a timely fashion
- Develop teaching skills
- Develop and complete case presentations and notes that are both comprehensive and succinct
- Hone effective listening skills
- Understand and incorporate principles of humanism
- Carry out professional responsibilities in a timely manner
- Display sensitivity to a diverse patient population
- Serve as patient and family advocate
- Understand rationale for diagnostic studies, pharmacologic and procedural tactics utilized
- Learn to interpret findings from various cardiovascular testing modalities including imaging, angiography, echocardiography and stress test techniques
- Understand indications for various cardiovascular testing modalities
- Develop skills in triage of patients into and out of CCU
- Take advantage of opportunities to practice bedside procedural techniques including insertion of central venous lines, arterial blood pressure monitoring lines, invasive hemodynamic monitoring catheters, temporary transvenous pacemaker wires
III. Delivering CCU-Specific Clinical Presentations

Basic Order
- One-liner (olds) or full HPI (new admit)
- Events overnight
- Vitals (include ranges) +/- vent settings
- I/O’s – have sense of hourly ratio
- Physical Exam
  - Neck
    - JVD
    - Carotids
      - Bruits
      - Murmurs
      - Upstroke
  - Lungs
    - “Clear” lungs does NOT rule out CHF!
  - CV
    - Palpation: Lift, heave, PMI
    - Auscultation
      - S1, S2, clicks
      - Gallops, rubs
      - Murmurs
        - Intensity
        - x/6 for systolic
        - x/4 for diastolic
        - Quality
        - Holosystolic/holodiastolic
        - Crescendo/decrescendo
        - Early/mid/late peaking
        - High/low pitched
        - Radiation
        - Is it new?
  - Abdomen
    - Congested liver
    - Hepatojugular reflex
- Ascites
  - Extremities
    - Edema
    - Cool/warm
    - Pulses; note symmetry

- Objective Data
  - Labs – just read them off, don’t interpret yet
  - ECG
  - CXR
  - ECHO
  - Cath
  - Stress
  - Other

- Assessment and Plan
  - Always start with cardiovascular unless there is another organ system that is primarily active
  - Conceptually divide cardiovascular into the following 3 areas to help you organize your thoughts (it does not need to be stated this way)
    1. Pump:
      - Include what we know about cardiomyopathic conditions in terms of systolic/diastolic function, relevant studies and meds
    2. Rhythm:
      - Known arrhythmias, device management, relevant studies and meds
    3. Ischemia:
      - Status of coronaries/grafts, whether current or prior, relevant meds
IV. Indications for Admission

Acute Coronary Syndrome
- Includes NSTEMI with concerning/dynamic ECG changes, persistent chest pain, hemodynamic compromise

HF exacerbations
- Acute, decompensated
- Patients requiring respiratory support
- Anyone warm & wet (may represent end stage, vasodilatory HF) or anyone that’s cold
- Mental status changes
- Evidence of MSOF/shock

Hypertensive Emergencies
- Particularly with flash pulmonary edema (these pts almost always need coronary angiogram once stabilized)

Severe (particularly if new) valvular lesions including infective endocarditis

Heart transplant recipients (low threshold)

Life threatening/unstable/hemodynamically intolerant arrhythmias
V. Management of Various Conditions

**Acute Coronary Syndrome**

**Coronary Anatomy**

- Sinoatrial node
- Right coronary artery
- Right ventricular
- Acute marginal
- Posterior descending
- Posterior lateral
- Left main
- 1st septal
- Left circumflex
- Intermediate (ramus intermedius)
- Obtuse marginal
- Left anterior descending

**Cath Lab Activation**

- This means that a group page was sent out through x156 to:
  - CCU attg
  - CCU fellow
  - Interventionalist-on-call
  - Interventional fellow (pager 6566)
  - On-call cath team (RN and tech)

- Who sends this page
  - ED attending, Cards fellow or attending

- What automatically happens
  - On-hours:
    - CCU fellow, on-call team and cath lab team go immediately to evaluate patient
- Housestaff helps get patient’s “story” and look up history in chart
- Prompt transport to cath lab is initiated
- If cardiac arrest, patient is resuscitated and brought to cath lab having achieved ROSC

Off-hours:
- CCU fellow and cath lab staff (interventionalist, RN, tech +/- cath fellow) mobilized to come in
- CCU housestaff go immediately to evaluate patient
- CCU resident calls CCU fellow within 10 minutes to provide him/her with the following information:
  - Brief story
  - Known stents/bypass grafts
  - Patient allergies to contrast agent/iodine/shellfish
  - Medications
  - Pertinent labs
    - BUN/Cr, GFR
    - Coagulation labs if relevant
    - H/H, platelet count
  - History of significant contraindications to long-term dual antiplatelet therapy
  - GI bleeding
  - Any significant bleeding history
  - Recent CVA
  - Recent or known upcoming surgeries that cannot be delayed
  - End stage terminal conditions
- CCU resident should obtain serial ECGs (q10min)
- CCU resident works on getting D5 bed
- Must be D5 in event patient exits lab with mechanical support
- Notify primary team if not on CCU service or through ED, most importantly primary care attending ASAP

To deactivate:
o Must be discussed with interventionalist-on-call, who makes this final decision

Cath Lab Alert
- Page goes out to CCU housestaff on call and CCU fellow
  On-hours:
  - CCU team goes immediately to evaluate patient and ECGs
  - CCU fellow presents case to cath lab and makes decision on appropriate management, which may include immediate cath lab evaluation, delayed cath lab evaluation, CCU admission, none of the above
  Off-hours:
  - CCU housestaff evaluates patient immediately
  - Obtains ECG and sends promptly to CCU fellow (Fax: 708-213-9103)
  - CCU resident calls CCU fellow after ECG sent to discuss the case

STEMI
- Serial ECGs (q10-15min) until stabilize
- Repeat ECG every time symptoms recur
- Medications (**must be administered immediately and prior to patient going to cath lab)
  ASA 325mg***
  Clopidogrel 600mg or Ticagrelor 180mg***
  Heparin gtt
  Beta-blocker (carvedilol or metoprolol)
  No NSAIDS
  Nitroglycerin/Morphine for pain relief
- Obtain objective data
- Prior cath/CABG, ECHO
- Medication titration post-procedure
  ASA: per Current OASIS-7^2, can decrease to 81mg daily starting day after PCI. ASA 325mg is given x 1 upon arrival
  Clopidogrel 75mg daily, Prasugrel 10mg daily, or Ticagrelor 90mg BID
Beta-blocker (carvedilol or metoprolol with transition to Toprol-XL before discharge)
ACEI/ARB for LV dysfunction
Eplerenone (EPHESUS³) if ACS patient with EF <35%
- Follow-up
  Ensure proper follow-up arranged pertaining to EF evaluation for possible future ICD (40d post-MI per DINAMIT⁴ trial)

**Unstable Angina/NSTEMI**

- Patients with 2/3 ACS criteria (chest discomfort, ECG changes, +cardiac biomarkers \(\rightarrow\) better off in CCU
- Serial ECGs to assess for evolution (q10-15min) until they stabilize
- Repeat ECG every time symptoms recur
- Medications
  - ASA 325mg
  - Clopidogrel 600mg
  - Heparin gtt
  - Beta-blocker (carvedilol or metoprolol)
  - Discuss GIIb/IIIa inhibitors⁵ with CCU fellow
  - No NSAIDS⁵
- Obtain objective data
- Reasons these patients go to cath lab promptly
  - Refractory chest pain
  - Increasing enzymes
  - Worsening ECG changes
  - Hemodynamic instability
Post-Procedure Care

Bedrest for femoral arterial access
- Manual hold: 6 hours
- Closure device: 2 hours
- Sheath sutured in place: bedrest until 6 hours post-sheath pull
- NB: Other than radial arterial lines, all arterial sheaths are pulled by fellows only
- Sheaths are pulled when the ACT < 180 seconds
- GPIIB/IIIA antagonists are not a contraindication to sheath pull

Femostop
What is it: A compression device that is secured via band placed under the patient to apply compression to the vascular access site
How to use it: After the band is placed, the device is placed with the dome over the access site and the device is cinched into place
The dome is inflated to 20 mmHg above the SBP for 3 minutes, then at the SBP for 3 min, then 20 below SBP for 15 min, then 40 below for 15 min.
This may be adjusted pending anticoagulation and the whim of the Fellow
Monitor distal pulses, if foot turns blue, may need to adjust pressure

What is bedrest?
- Strict immobility of the instrumented leg
- No lifting head, rolling or sitting up
- Pt can have head of bed elevated to 30° by RN/staff only

Troubleshooting
- Hematoma
  o Immediate notification of fellow/attending
  o Check peripheral pulses, listen with diaphragm of stethoscope over femoral access site for bruit
  o Send stat CBC
  o Consider/discuss CT to rule out RP bleed
- Significant bleeding
  - Immediate notification of fellow/attending
- Options for achieving hemostasis include:
  - Manual pressure
  - Application of Femostop device

**Post-MI Complications**

- Reperfusion Injury
  Aggressive electrolyte management (K>4, Mg>2)
  ECG
  Evaluate patient for symptoms
  No indication to revisit cath lab unless patient is hemodynamically unstable or has recurrent symptoms
- Ventricular Aneurysm/Free Wall Rupture*
  Increased risk: Factors that indicate absence of collaterals and larger infarct size
  No prior MI
  STEMI (vs. NSTEMI)
  Peak CK-MB >150
  Anterior MI
  Age >70
  Female gender
  Timeframe: 5-14 days post MI
  Suspect with hemodynamic compromises/shock, acute pericardial effusion/tamponade
- Aneurysm only: persistent STE on ECG w/ Q waves
- Interventricular Septum Rupture*
  Increased risk in patients with STEMI of wrap-around LAD
  Timeframe: 3-7 days post MI
  Suspect with hemodynamic compromise, new biventricular failure, new harsh, loud, holosystolic murmur heard best at left/right lower sternal borders +/- thrill (50% cases)
- Acute Mitral Regurgitation*
  Suspect with hemodynamic compromise and new systolic murmur
  Must evaluate for papillary muscle rupture, chordal rupture, LV aneurysm or acute dilatation
Stat ECHO

*Diagnosis of any of these requires urgent surgical evaluation
**Heart Transplant Patients**

**Limitations to Survival**
- **Up to one year**
  - Nonspecific graft failure
  - MSOF
  - Acute rejection
  - Infection
- **After one year**
  - Malignancy
  - Cardiac allograft vasculopathy (CAV)
    - Significantly increases mortality and nonfatal coronary events
    - Presentation may be “silent” as a result of denervation
    - May present with silent MI, SCD, HF symptoms

**Maintenance therapy**
- Calcineurin inhibitors
  - Cyclosporine or tacrolimus
  - Always check daily trough level
- Antimetabolite
  - Mycophenolate mofetil
- Corticosteroid taper x 1 year

**Rejection**
- Cell-mediated
  - ISHLT Grading System (via biopsy)
    - 0: no rejection
    - 1: mild
    - 2: moderate
    - 3: severe
- Treatment
  - Oral/IV corticosteroids
  - Antithymocyte globulin
  - Murine monoclonal antibody OKT3
- CAV
  - Prophylaxis
    - Annual coronary angiography
- Incidence
  - 2-28% at 1 year
  - 40-70% at 5 years
- May be underestimated angiographically as CAV causes diffuse, concentric narrowing (vs. focal)
- Stress test may be considered after 5 years
- Medications
  - Statins
  - mTOR inhibitors
- Treatment
  - Change/augment immunosuppressive tx
  - PCI
  - CABG
  - Retransplant
- **ECG Interpretation**
  - Low voltage
  - Bradycardia
  - Any level of heart block
- **If infection is suspected**
  - Culture everything
  - Low threshold for broad-spectrum abx
  - Immunocompromised ID consult
## Acute Decompensated Heart Failure

### Evaluation
- Why did the patient decompensate?
  - History
  - Medication compliance
  - Caused by other cardiac conditions
    - ACS
    - Arrhythmia
    - Aortic dissection
    - Acute AI/MR
  - Exacerbation by noncardiac conditions
    - PNA
    - PE
    - COPD/Asthma
    - Missed dialysis sessions
  - Consider invasive hemodynamic monitoring (see below) for the following reasons:
    - Cannot determine filling pressures from exam
    - Guide therapy
  - Diagnostic studies
    - ECG
    - CXR (normal CXR does not rule out ADHF)
    - Cardiac enzymes
    - Electrolytes
    - ABG
    - BNP
    - ECHO
    - Cath for ACS-induced ADHF

### Treatment
- Goals
  - Improve symptoms
  - Optimize volume status
  - Identify and treat precipitating factors
  - Establishing stable oral regimen
  - Patient education
- **Mainstays of therapy**
- Monitor O2 sat, vitals, rhythm
- Supplemental O2
- IV access
- Position patient upright
- Provide respiratory support as needed
  - NIPPV vs. intubation
- If end-organ perfusion adequate, hemodynamically stable
  - IV vasodilator (NTG or Nitroprusside)
  - IV loop diuretic
    - Higher doses needed if pt taking loop diuretic chronically and/or renal dysfunction
- If known systolic HF with severe ADHF/shock
  - IV inotrope +/- mechanical support (IABP)
  - Caution in ACS pts; inotropes may provoke ischemia by increasing myocardial O2 demand
- If known diastolic HF with severe ADHF/shock
  - IV vasopressor preferred over IV inotrope
- If cardiac function unknown but pt has signs/symptoms of ADHF/shock
  - IV inotrope +/- IV vasopressor +/- mechanical support
- Beta-blockers\(^7-8\)
  - If taking chronically
    - Continue if hemodynamically stable
    - Decrease/hold if unstable
  - If naïve
    - Initiate once pt stabilizes
- Oral therapy (transition once stable)
  - ACEI/ARB\(^9-10\)
  - Aldosterone antagonist\(^11\)
    - If EF <30-35%, serum potassium >5 meq/L and GFR >/= 30
0 Antiarrhythmics for concomitant arrhythmia (i.e. Afib), may need DCCV
**Hypertensive Crises**

**Hypertensive urgency**
(Pt is asymptomatic and there is no evidence of end-organ damage)

- Treatment
  - Goal BP ≤ 160/100 over several hours/days
  - Rapid BP reduction ill-advised
  - If already on meds, reinstate
  - If naïve,
    - Initially use short-acting tx (i.e. captopril, metoprolol) and up-titrate
    - CCB (nifedipine XL 30mg QD), Beta-blocker (metoprolol XL 50mg QD), ACEI (ramipril 10mg QD)

**Hypertensive emergency**
(Evidence of end-organ damage, pt is symptomatic)

- Treatment
  - Consider head CT
  - Rapid, cautious lowering is the goal
  - Malignant HTN and hypertensive encephalopathy
    - Nitroprusside IV
    - Nicardipine IV
    - Labetalol IV
    - Esmolol IV
  - Concomitant HF exacerbation
    - IV vasodilators (nitroprusside, NTG)
    - Avoid beta-blockers (decrease contractility) and hydralazine initially (may increase cardiac work)
  - Aortic dissection
    - IV beta-blocker to reduce HR <60bpm, BP to 100-120 mmHg systolic or lowest level tolerated
    - STAT surgical consult
Bradyarrhythmias

Causes
- Hypoxia
- Increased intracranial pressure
- Exaggerated vagal tone
- Acute MI (inferior)
- OSA
- Medications
  - AVN blockers (BB, CCB, digitalis)
  - Amiodarone
  - Methylldopa, clonidine
  - valsalva
  - pressure on carotid sinus (like from neck collar)
  - vomiting/coughing
  - sudden exposure to cold water (typically of face)
  - hypothyroidism
  - hypothermia
  - certain infections
  - Bezold-Jarisch reflex

Medical Therapy
- Atropine
  - 0.5-1mg, repeat q3-5min to a total dose of 3mg
  - Not likely to be helpful if Mobitz II or higher grade block suspected
- Chronotropic Agent Infusion
  - Dopamine (2-10mcg/kg/min)
  - Isoproterenol (2-10mcg/min)
  - Beta-2 agonism can result in vasodilation and decrease in MAP
  - Epinephrine (2-10mcg/min)

Transcutaneous Pacing
- This is painful and not preferred, use sedation

Temporary Transvenous Pacing
Indications
- Hemodynamically significant bradycardia
- Asystole
- Termination of tachycardias (rare)
- Bridge to permanent pacing or recovery
- Severe sinus node dysfunction
- AV block
- V Tach
- AMI
- New bifascicular block
- New LBBB & 1st degree AVB
- Alternating L and R BBB
- Mobitz II
- CHB
- RV Infarct

Insertion
- Sites
  - IJ is preferred
  - Avoid subclavian sites if this is bridge to permanent PPM
  - Femoral often used in emergent settings (i.e. cath lab)
  - Insert at least 5Fr sheath via Seldinger technique
- Insert pacing catheter, inflate balloon, advance toward RV apex (V pacing)/RAA (A pacing),
  - Once in RV apex, rotate so tip points inferiorly
  - Should see LBBB morphology on ECG with pacing
- Set-up
  - Connect cathode to negative terminal, anode to positive terminal
- Capture
  - Start pacing at 10-20 beats faster than native rate at 5mA output
  - If no capture, reposition
  - Once capture, slowly decrease output until lose capture = pacing threshold (good catheter position, threshold usually less than 1A)
  - Output should be 3x threshold (but 5mA is typically where it is set)
- Sensing
- Decrease sensing setting until asynchronous pacing is seen = sensing threshold
- Set at twice sensing threshold
**Tachyarrhythmias**

**DCCV/Defibrillation**

- **Indications**
  - Emergently – unstable arrhythmia
  - Otherwise, these are done electively in the EP lab

- **Anticoagulation**
  - Pertinent for Afib/Flutter cardioversions
  - Emergent
    - Administer therapeutic A/C just prior or immediately after DCCV
  - Elective
    - Therapeutic A/C x 3-4 weeks prior or TEE and therapeutic A/C and DCCV if no thrombus. If thrombus, wait 3-4 weeks with target INRs and then repeat TEE to confirm resolution

- **Protocol**
  - Place pads anterior-posterior
  - Run continuous 12-lead ECG strip
  - Conscious sedation

- **DCCV**
  - Synchronize
  - Charge to 200J
  - Shock

- **Defibrillation**
  - Do not synchronize
  - Charge to 200J
  - Shock
Mechanical Hemodynamic Support

General Complications
- All devices carry risk of significant bleeding due to large sheath size as well as limb ischemia

Intra-Aortic Balloon Counterpulsation
- Indications
  - Hemodynamically unstable cardiogenic shock
  - Class I indication for STEMI pts with cardiogenic shock if not quickly reversed with pharmacologic therapy
  - Class I indication for STEMI and secondary acute MR
  - Refractory ischemia in UA/NSTEMI (Class IIa), STEMI (Class I)
  - High risk PCI
  - Bridge to cardiac transplantation
  - VT Storm
- Contraindications
  - Aortic dissection
  - AAA/TAA
  - Severe PAD, Limb ischemia
  - Descending aorta/peripheral grafts (relative)
  - Moderate/Severe AI
  - Coagulopathy
    - Can anticoagulate with alternative agents if heparin a problem
    - Do not need anticoagulation if IABP is sheathless and on 1:1 CP
- Hemodynamics
  - Afterload reduction
    - Rapid deflation during systole allows for negative pressure in aorta
  - Reduces afterload and improves forward flow from LV
  - Increases CO by 20% decrease in mean PCWP
  - Reduces LV wall stress from decreased filling pressures and decreased afterload → improves SV and CO, decreased O2 demand
  - Augment coronary perfusion
- Inflation during diastole displaces blood to proximal aorta → augments diastolic pressure → augments coronary perfusion pressure
- Will increase peak coronary flow velocity but does not improve flow across critical stenoses
- Does not augment collateral flow

- Monitoring
  - IV heparin if IABP through sheath or inflation is not 1:1
  - Pt must remain supine (i.e. strict bedrest)
  - Serial distal pulse exams
  - Daily CXR to confirm proper placement

- Triggering and Timing
  - Trigger
    - ECG – allows for appropriate delay after R wave at dicrotic notch
    - Pressure – if ECG tracings are poor
    - Internal – if pt has arrested or ECG/Pressure triggers are unreliable
  - Timing
    - Ideal inflation before dicrotic notch and deflates before onset of next systolic waveform

- Sample Waveforms
Early Inflation

Late Inflation
Early Deflation

Late Deflation
Percutaneous Ventricular-Assist Device

- **Indications**
  - Cardiogenic shock
  - Backup support during high-risk percutaneous/surgical procedures in patient with poor cardiac function

- **Contraindications**
  - AI, prosthetic aortic valve
  - Aortic aneurysm/dissection
  - Severe aortic or peripheral vascular disease
  - Left ventricular/atrial thrombi
  - Bleeding diatheses
  - Overwhelming sepsis

- **Complications**
  - Infection
  - Thromboembolism
  - Arrhythmias due to mechanical irritation
  - Thrombocytopenia
  - Hemolysis
  - Effusion/tamponade

- **Types**
  - Axial Flow Pump (Impella 2.5)
    - Percutaneously placed into femoral artery
    - Inflow cannula advanced retrograde across aortic valve and seated in left ventricle
    - Revolving pump draws blood out of LV and ejects it into ascending aorta
    - Nonpulsatile flow
Tandem-Heart
- Percutaneously placed via femoral artery
- Venous catheter inserted into LA via transeptal puncture
- Arterial cannula inserted into iliac artery

ECMO
- Removes CO2 from and adds O2 to venous blood via artificial membrane
- Pulmonary circulation is bypassed
- Requires systemic anticoagulation
- Venovenous (severe respiratory failure)
Venoarterial (cardiac failure)

What to watch for:
There is a big cannulae in the artery (13Fr, 15Fr, or 17Fr) and distal perfusion is provided by a 5Fr antegrade sheath, need to assess limb perfusion
Groin complications should be noted
If device flows drop (the ECMO oxygenates and circulates blood) transfuse and page Perfusion 3984
**Invasive Hemodynamics**

**Indications**
- Heart Failure
- Cardiogenic vs. noncardiogenic pulmonary edema/shock
- Guide therapy
- Pre-transplant – evaluate for reversible pulmonary HTN
- AMI
- Guide therapy in cardiogenic shock from AMI
- Manage acute pulmonary edema or RV infarct refractory to medical therapy
- Perioperative Use
- Determine cause of low cardiac output (hypovolemia vs. ventricular dysfunction)
- Pericardial Disease
- Constriction vs. Restriction vs. Tamponade (if echocardiography is unclear)
- Primary Pulmonary HTN
- To exclude postcapillary causes (elevated PCWP)
- Diagnose and establish severity of precapillary (normal PCWP) pulmonary HTN
- Select and establish safety and efficacy of long-term vasodilator therapy
- Pre-lung transplant assessment
- Assess for Shunt

**The Procedure**
- Test all ports/balloon before inserting
- Choose Site
  - Typically use IJ or femoral approach
- Sterile preparation and technique
- Seldinger technique to introduce sheath (typically 7Fr)
- Insert PA catheter
- Inflate balloon once catheter is out of the sheath
- Avoid over-wedging (increased risk of PA rupture)
- Post-procedural CXR to rule out PTX

**Calculations**
- CO (normal = 4-8L/min)
  - Fick: \([Wtx3 \text{ mL/kg}]/[(\text{Ao sat} – \text{ V sat}) \times 1.36 \times \text{Hgb} \times 10]\)
  - Thermodilution method
  - Performed by injecting 10cc of saline into RA port (balloon deflated)
  - CI (normal = 2.8-4.2 L/min/m2)
- SV (normal = 40-120 cm^3/beat)
- SV = CO/HR
- SVR (normal = 770-1500 dynes*s/cm^-5)
- \(\text{SVR} = [(\text{MAP-CVP}) \times 80]/\text{CO}\)
- PVR (normal = 20-120 dynes*s/cm^-5)
- \(\text{PVR} = [(\text{PAP-PCWP}) \times 80]/\text{CO}\)
- Convert to Wood Units by dividing by 80

**Clinical Scenarios**

<table>
<thead>
<tr>
<th></th>
<th>RA</th>
<th>PCWP</th>
<th>CO</th>
<th>SVR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiogenic</strong></td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>RV Infarct</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
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<tr>
<td>MR</td>
<td>↑</td>
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<td>↓</td>
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<tr>
<td>PE</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Tamponade</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Sepsis</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
</tbody>
</table>
- VSD
  - Step up in O2 sat from RA to PA
- Pulmonary HTN
  - ↑RAP, ↑RVP, ↑PAP, normal PCWP, PAP and RV
- Tamponade
  - Diastolic equalization of pressures
  - RAP = RV diastolic pressure = PCWP
  - ↑RAP, ↑RVP, ↑PCWP
  - Prominent x descent
  - Constrictive pericarditis
Waveform Characteristics and Normal Values

RA (normal = 0-8mmHg)

RV (normal = 15-30mmHg/0-8mmHg)
PA (normal = 15-30mmHg/3-12mmHg)

PCWP (normal = 6-12mmHg)
**Therapeutic Hypothermia**

**Background**
- Risk of death increases for each degree over 37° during first 48h post-arrest
- Lowering brain temp to 32-34° during first few hrs post-arrest reduces risk of neurological injury

**Indications**
- Not following commands/lack of purposeful movements post-ROSC

**Contraindications**
- Active, non-compressible bleeding
- DNR
- Not contraindicated in pregnancy, hemodynamic instability, or pts receiving thrombolytics

**Timing and Duration**
- Achieve goal temp within 6 hrs post-ROSC
- No benefit if placed mid-arrest
- Maintain for 12-24hrs

**Methods**
- Intravascular\(^{13}\)
  - IV infusion of cold (4°C) isotonic saline, 30mL/kg via pressure bag will reduce temp by >2°C/hr
  - 1L of pressure-infused cold saline/15min drops temp by 1°C
  - Placement of endovascular catheter
    - Can be done bedside or in cath lab
- Surface
  - Ice packs, cooling blankets, cooling vests, cold water immersion reduce body temp by 0.5-1°C/hr
  - Apply ice packs to groin, axillae, neck

**Sedation and Shivering**
- Titrate sedation to shivering suppression
- Shivering increases body temp and can delay time to achieve goal temp
- Continuous gtt (propofol, fentanyl)
- Can use benzo gtts but may interfere with assessment of neurological function during re-warming phase
- Intermittent meperidine to suppress shivering, but primary metabolite is pro-convulsive
- Can use paralytics but this necessitates continuous EEG monitoring as these agents may mask seizures

**Monitoring**
- Continuous temp monitoring via central venous, esophageal, bladder or rectal probes

**Rewarming**
- Rate of increase should not exceed 0.5°C/hr (0.2-0.25°C/hr recommended)
- May induce electrolyte abnormalities (hypokalemia), cerebral edema, seizures
- Can be passive or active

**Adverse Effects**
- Mild coagulopathy
  - Slows clotting enzymes and decreases platelet function
  - TH should be stopped if significant bleeding occurs
- Increased risk of infection
  - Impairs leukocyte function, particularly >24h
- Cardiac arrhythmias
  - Bradycardia
  - QT prolongation
- Hyperglycemia due to insulin resistance
- “Cold diuresis”
  - hypovolemia, hypokalemia, hypomagnesemia, hypophosphatemia may result
- May interfere with metabolism of various drugs, thus prolonging their effects

**Benefit in VT/VF**
- TH shown to decrease mortality and improve neurological outcomes

**Benefit in Non-shockable Rhythms**
- Not demonstrated in randomized trials, but still thought to incur benefit in these patients
**Transcatheter Aortic Valve Replacement**

### Anticoagulation
- ASA 81mg and clopidogrel 75mg daily x 6 months, followed by ASA 81mg daily for life

### Access-site complications
- Access sites potentially utilized
  - Direct femoral access (most common)
  - Subclavian artery
  - Transapical
  - Direct aortic cannulation
  - Iliac conduit (particularly for patients with severe peripheral vascular disease that precludes femoral access)

### Periprocedural CVA
- Low threshold to suspect, periprocedural risk of CVA is 10-20%
- Patients are often elderly with heavily calcified aortic arch and valve, hence significant risk of embolization/showering

### Other post-TAVR issues
- Stat ECHO with any new signs of HF, SOB, hemodynamic compromise
- Rupture
  - Pericardial effusion/tamponade
- Heart block
  - Will need temporary transvenous pacing
- Ventricular rupture (with transapical access)
Figure 1. Transcatheter Aortic-Valve Replacement.

The transcatheter valve is positioned at the level of the native aortic valve during the final step of valve replacement, when the balloon is inflated within the native valve during a brief period of rapid ventricular pacing. The delivery system is shown after it has traversed the aorta retrograde over a guidewire from its point of insertion in the femoral artery (transfemoral placement). Before balloon inflation, the valve and balloon are collapsed on the catheter (dark blue) and fit within the sheath (blue). After balloon inflation, the calcified native valve (upper panel) is replaced by the expanded transcatheter valve (lower panel, shown in short-axis view from the aortic side of the valve).
2010 AHA Algorithm for BLS

Adult BLS Healthcare Providers

1. Unresponsive
   No breathing or no normal breathing
   (i.e., only gasping)

2. Activate emergency response system
   Get AED/defibrillator
   or send second rescuer (if available) to do this

3. Check pulse:
   DEFINITE pulse
   within 10 seconds?
   Definite Pulse

3A. High-Quality CPR
   - Rate at least 100/min
   - Compression depth at least 2 inches (5 cm)
   - Allow complete chest recoil after each compression
   - Minimize interruptions in chest compressions
   - Avoid excessive ventilation

4. No Pulse
   Begin cycles of 30 COMPRESSIONS and 2 BREATHS

5. AED/defibrillator ARRIVES

6. Check rhythm
   Shockable rhythm?

7. Shockable
   Give 1 shock
   Resume CPR immediately
   for 2 minutes

8. Not Shockable
   Resume CPR immediately
   for 2 minutes
   Check rhythm every
   2 minutes; continue until
   ALS providers take over or
   victim starts to move
AHA Algorithm for PEA Arrest
AHA Algorithm for Tachyarrhythmias
AHA Algorithm for Bradyarrhythmias

**Adult Bradycardia (With Pulse)**

1. **Assess appropriateness for clinical condition. Heart rate typically <50/min if bradyarrhythmia.**

2. **Identify and treat underlying cause**
   - Maintain patent airway; assist breathing as necessary
   - Oxygen (if hypoxemic)
   - Cardiac monitor to identify rhythm; monitor blood pressure and oximetry
   - IV access
   - 12-Lead ECG if available; don’t delay therapy

3. **Persistent bradyarrhythmia causing:**
   - Hypotension?
   - Acutely altered mental status?
   - Signs of shock?
   - Ischemic chest discomfort?
   - Acute heart failure?

4. **Monitor and observe**
   - **No**
   - **Yes**

5. **Atropine**
   - If atropine ineffective:
     - Transcutaneous pacing
     - **Dopamine** infusion
     - **Epinephrine** infusion

6. **Consider:**
   - Expert consultation
   - Transcutaneous pacing

**Doses/Details**
- **Atropine IV Dose:**
  - First dose: 0.5 mg bolus
  - Repeat every 3-5 minutes
  - Maximum: 3 mg
- **Dopamine IV Infusion:**
  - 2-10 mcg/kg per minute
- **Epinephrine IV Infusion:**
  - 2-10 mcg per minute
<table>
<thead>
<tr>
<th>Meds</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACETAMINOPHEN (UDC)</td>
<td>20.3mL cup 160MG/5ML 650 MG LIQ</td>
</tr>
<tr>
<td>ACETAMINOPHEN 325 MG TAB</td>
<td></td>
</tr>
<tr>
<td>ACETAMINOPHEN/CODEINE 30MG TAB</td>
<td></td>
</tr>
<tr>
<td>ACETAMINOPHEN/OXYCODONE 325mg/5mg TAB</td>
<td></td>
</tr>
<tr>
<td>ADENOSINE (3MG/ML)</td>
<td>2 ML INJ</td>
</tr>
<tr>
<td>ALBUMIN 5% 250 ML INJ</td>
<td></td>
</tr>
<tr>
<td>ALBUTEROL NEBULIZING SOLN O.083% 3 ML SOLN</td>
<td></td>
</tr>
<tr>
<td>ALBUTEROL/IPRATROPIUM (DUO-NEB) 2.5-0.5/3 3 ML SOLN</td>
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<tr>
<td>ALUMINUM-MAGNESIUM HYDROX 1200-1200 30 ML SUSP</td>
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<tr>
<td>AMIODARONE HCL 50MG/ML 3 ML INJ</td>
<td></td>
</tr>
<tr>
<td>ASPIRIN 325 MG TAB</td>
<td></td>
</tr>
<tr>
<td>ASPIRIN EC 325 MG ECTA</td>
<td></td>
</tr>
<tr>
<td>ATROPINE SULFATE (0.1MG/ML 1 MG SYRIN</td>
<td></td>
</tr>
<tr>
<td>CADD PUMP 1 EA MISC</td>
<td></td>
</tr>
<tr>
<td>CALCIUM CHLORIDE (100MG/ML) 10 ML SYRIN</td>
<td></td>
</tr>
<tr>
<td>CODEINE PHOS/GUAIFENESIN 10 ML ELIX</td>
<td></td>
</tr>
<tr>
<td>D5/0.45% NAACL-KCL 20MEQ 1000 ML INJ</td>
<td></td>
</tr>
<tr>
<td>DEXAMETHASONE 10MG/ML 1 ML VIAL</td>
<td></td>
</tr>
<tr>
<td>DEXTROSE 5% IN WATER 250 ML INJ</td>
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</tr>
<tr>
<td>DEXTROSE 5% IN WATER 5% 100 ML INJ</td>
<td></td>
</tr>
<tr>
<td>DEXTROSE 5% IN WATER 5% 1000 ML INJ</td>
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<tr>
<td>DEXTROSE 5%/0.45% NAACL 1000 ML INJ</td>
<td></td>
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<tr>
<td>DEXTROSE 50% 50 ML SYRIN</td>
<td></td>
</tr>
<tr>
<td>DIAZEPAM 2 MG TAB</td>
<td></td>
</tr>
<tr>
<td>DIAZEPAM 5 MG TAB</td>
<td></td>
</tr>
<tr>
<td>DIAZEPAM 5MG/ML 10 MG SYRINGE</td>
<td></td>
</tr>
<tr>
<td>DIGOXIN 0.25MG/ML 2 ML AMPUL</td>
<td></td>
</tr>
<tr>
<td>DILTIAZEM HCL (5MG/ML) 50 MG VIAL</td>
<td></td>
</tr>
<tr>
<td>DILTIAZEM HCL 30 MG TAB</td>
<td></td>
</tr>
<tr>
<td>diphenhydrAMINE HCL 25 MG CAPSULE</td>
<td></td>
</tr>
<tr>
<td>diphenhydrAMINE HCL 50MG/ML 50 MG INJ</td>
<td></td>
</tr>
<tr>
<td>DOBUTAMINE/D5W 250ML 1 GM IV SOLN.</td>
<td></td>
</tr>
<tr>
<td>DOCUSATE SODIUM 100 MG CAP</td>
<td></td>
</tr>
<tr>
<td>Dopamine HCL 800 mg/D5W 250 ML IV SOLN.</td>
<td></td>
</tr>
<tr>
<td>DRONABINOL C-III 2.5 MG CAP</td>
<td></td>
</tr>
</tbody>
</table>
EPINEPHRINE 1:1000 1MG/ML 1 MG AMPUL
EPINEPHRINE 1:10000 0.1 MG SYRIN
ESMOLOL HCL 2.5G 250 ML PIGGYBACK
FENTANYL (12MCG/HR) PATCH 12 MCG/H PATCH TD72
FENTANYL (25MCG/HR) 10 CM PTCH
FENTANYL (50MCG/HR) 20 CM PTCH
FENTANYL /0.9% NAACL 10 MCG/ML 100 ML IV SOLN.
FENTANYL /0.9% NAACL 10 MCG/ML 100 ML IV SOLN.
FILTER 1 EA NEEDLE
FLUMAZENIL (0.1MG/ML) 5 ML INJ
FUROSEMIDE 10MG/ML 40 MG INJ
GLUCAGON 1 MG INJ
HALOPERIDOL 5MG/ML 5 MG INJ
HEPARIN 25,000U/D5W 500 ML INJ
HEPARIN SODIUM 10,000U/ML 5000 UNIT SYRIN
HEPARIN SODIUM 10ML VIAL, 1000 U/ML VIAL
HURRICANE 1 SPRY AERO
hydrALAZINE HCL 20MG/ML 20 MG INJ
HYDROCODONE/ACETAMINOPHEN 5MG/500MG 1 EA TAB
HYDROCODONE/ACETAMINOPHEN 7.5MG/500MG 15 ML LIQUID
HYDROMORPHONE (PCA) 1MG/ML 30 ML IV
HYDROMORPHONE HCL 1MG/ML 1 MG DISP SYRIN
HYDROMORPHONE HCL 2 MG TAB
INSULIN ASPART 100U/ML 1 IU VIAL
INSULIN GLARGINE, HUMAN (LANTUS) 100U/ML 1 UNIT VIAL
INSULIN HUMAN NPH 100U/ML 1 IU INJ
INSULIN HUMAN REGULAR 100U/ML 1 IU INJ
INSULIN NOVOLIN 70/30 1 IU INJ
IPRATROPIUM NEBULIZING SOLN 0.5MG/2.5ML 2.5 ML SOLN
ISOPROTERENOL HCL 200 MCG INJ
LABETALOL (5MG/ML) 100 MG INJ
LACTATED RINGERS 1000 ML INJ
LIDOCAINE 2% (PF) 100 MG 20MG/ML 5 ML SYRIN
LIDOCAINE 2GM/D5W 250 ML INJ
LIDOCAINE HCL (10MG/ML) 20 ML INJ
LORAZEPAM 0.5 MG TAB
LORAZEPAM 1 MG TAB
LORAZEPAM 2MG/ML 2 MG VIAL
MEPERIDINE HCL 50MG/ML 50 MG SYRIN
METHADONE HCL 10 MG TAB
METHADONE HCL 10MG/ML 10 MG INJ
METHADONE HCL 5 MG TAB
METOCLOPRAMIDE HCL 5MG/ML 10 MG INJ
METOPROLOL TARTRATE 1MG/ML 5 MG INJ
MIDAZOLAM / 0.9% NAACL 1 MG/ML 100 ML IV SOLN.
MIDAZOLAM HCL 1MG/ML 2 MG VIAL
MIDAZOLAM HCL 5MG/ML * 5 MG VIAL
MILRINONE 20MG/D5W 0.2MG/ML 100 ML INJ
MINI MED PUMP 1 EA MISC
MORPHINE /0.9% NAACL 1 MG/ML 100 ML IV SOLN.
MORPHINE SULFATE (2MG/ML) 10 MG ELIX
MORPHINE SULFATE 10MG/ML 10 MG SYRIN
MORPHINE SULFATE 2MG/ML 2 MG SYRIN
MORPHINE SULFATE 60 MG SRTA
MORPHINE SULFATE IR 15 MG TAB
NAACL (EXCEL) 0.9% 250 ML IV SOLN.
NAACL 0.45% 1000 ML INJ
NAACL 0.9% 100 ML INJ
NAACL 0.9% 1000 ML IV SOLN.
NAACL 0.9% 250 ML INJ
NAACL 0.9% 50 ML INJ
NAACL 0.9% 500 ML INJ
NAACL MINI-BAG PLUS 0.9% 50 ML IV
NALOXONE HCL 0.4MG/ML 1 ML AMPUL
NARCOTIC BOX KEY 1 EA MISC
NITROGLYCERIN #25 0.4 MG SLTB
NITROGLYCERIN (OR) 50MG/D5W 250 ML INJ
NITROGLYCERIN 2% 1 GM OINTMENT
NOREPINEPHRINE BITARTRATE (1MG/ML) 4 MG INJ
ONDANSETRON HCL 4 MG TAB
OPIUM/BELLADONNA 15° 1 SUPP SUPP
OXYCODONE 10 MG SRTA
OXYCODONE 20 MG SRTA
OXYCODONE HCL 5 MG TAB
PHENYLEPHRINE HCL (10MG/ML) 10 MG VIAL
POTASSIUM CHLORIDE (KLOR-CON) 20 MEQ SRTA
POTASSIUM CHLORIDE 20MEQ/15ML 20 MEQ LIQ
POTASSIUM CHLORIDE 20MEQ/mL 100 ML IV SOLN.
PREGABALIN 100 MG CAPSULE
PREGABALIN 150 MG CAPSULE
PREGABALIN 50 MG CAPSULE
PREGABALIN 75 MG CAPSULE
PROPOFOL (OR) 1% 20 ML VIAL
PROPOFOL 1% 100 ML VIAL
PROPOFOL 1% 100 ML VIAL
PROTAMINE SULFATE (10MG/ML) 50 MG INJ
SODIUM BICARBONATE (1MEQ/ML) 50 MEQ VIAL
TRANSPORT 1 EA KIT
VASOPRESSIN 20 UNIT VIAL
ZOLPIDEM TARTRATE 5 MG TAB

VII. References

51
1. Visit the following website for links to updated ACC/AHA joint guidelines on various cardiovascular disease processes.
http://my.americanheart.org/professional/StatementsGuidelines/ACCAHA-Joint-Guidelines_UCM_321694_Article.jsp


### Atrial Fibrillation

<table>
<thead>
<tr>
<th>Study</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AFFIRM</strong></td>
<td>No difference in mortality between rate and rhythm control, however, increased mortality in rhythm control in older pts, those with CAD, those without CHF; 2002</td>
</tr>
<tr>
<td><strong>ARISTOTLE</strong></td>
<td>Apixaban was superior to warfarin in preventing stroke or systemic embolism, caused less bleeding, and resulted in lower mortality.; 2011</td>
</tr>
<tr>
<td><strong>RELY</strong></td>
<td>Dabigatran is non-inferior to warfarin in preventing stroke and systemic embolism with lower major bleeding profile; slight increase in GI bleeding; 2009</td>
</tr>
<tr>
<td><strong>SPAF I, SPAF II, SPAF III</strong></td>
<td>Warfarin &gt; ASA &gt; placebo in reducing stroke events in Afib. For high risk patients with Afib, Warfarin INR 2-3 is more effective. Low risk patients, ASA 325 has acceptable low risk of stroke &lt; 3%. Sub-study of SPAF III established high risk factors of the CHADS2 risk score; 1991, 1994, 1996</td>
</tr>
</tbody>
</table>

### Pacemaker

<table>
<thead>
<tr>
<th>Study</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AVID</strong></td>
<td>ICD is more effective than antiarrythmic drugs in reducing arrhythmia related cardiac deaths. 1999</td>
</tr>
<tr>
<td><strong>MADIT</strong></td>
<td>Defibrillator along with BiV ICD (CRT-ICD therapy ) is associated with improved EF and HF. Most</td>
</tr>
<tr>
<td>Study</td>
<td>Description</td>
</tr>
<tr>
<td>-------</td>
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</tr>
<tr>
<td><strong>REVERSE</strong></td>
<td>CRT reverses remodeling in systolic LV dysfunction, pts with asymptomatic to mild HF or wide QRS, EF &lt; 40% - significant improvement in reverse LV remodeling seen by measures of LVESV and LVEDV along with EF after 6 months in pts with CRT with further improvement overtime; there was significant decrease in <em>morbidity and mortality</em>; 2009</td>
</tr>
<tr>
<td><strong>SCDHeFT</strong></td>
<td>Amio vs. placebo, ICD vs. placebo for CHF - In pts with mild-moderate CHF, EF &lt; 35, shock only ICD reduced risk of death (ARR 7.2% at 5 years), main effects in pts with Class II symptoms, minimal effect in Class III; Amio showed no benefit in Class II, but reduced survival in Class III; 2005</td>
</tr>
<tr>
<td><strong>Lipids</strong></td>
<td><strong>AtoZ</strong> NSTEMI, STEMI Reduction in CV death, MI and readmission for ACS reduced in pt receiving <em>zocor</em>. Significant decrease in CV death and CHF; 2004</td>
</tr>
<tr>
<td><strong>JUPITER</strong></td>
<td>Rosouvastatin reduced primary endpoint (CV death, MI, CVA, unstable angina, <em>revascularization</em>) in women &gt; 60 and men &gt; 50, LDL &lt; 130 – low normal, elevated hsCRP &gt; 2 by</td>
</tr>
<tr>
<td>Study</td>
<td>Description</td>
</tr>
<tr>
<td>-------</td>
<td>-------------</td>
</tr>
<tr>
<td><strong>LIPID</strong></td>
<td>Pravastatin reduced mortality from all causes and CV events in pts with ACS and cholesterol 155-271; 1998</td>
</tr>
<tr>
<td><strong>PRINCESS/MIRACL</strong></td>
<td>Early &lt;96hr initiation of atorvastatin improved MI and revasc in AMI patients</td>
</tr>
<tr>
<td><strong>TNT</strong></td>
<td>Pts with CAD (prior MI +/- revascularization, stable angina) - Lipitor - high dose has significantly lower LDL and total cholesterol levels, and reduced risk of major CV event 2005</td>
</tr>
<tr>
<td><strong>WOSCOPS</strong></td>
<td>Pts hyperlipidemia and no hx of MI, pravastatin reduced CV deaths (RRR 30%) and need for revascularization (RRR 37%); 1995</td>
</tr>
</tbody>
</table>

**Preventative Cardiology**

<p>| ALLHAT | Thiazide vs. CCB vs. ACEI - Thiazide type diuretics (chlorthalidone) are superior at preventing 1 or more forms of CVD and should be first line of therapy; amlodipine higher 6 yr rate of HF and lisinopril had higher 6 yr rates of CHD, stroke, and HF; 2002 |
| FRAMINGHAM | High levels of LDL, Hypertension, cigarette smoking, obesity, elevated blood sugar levels, stress, lack of exercise, menopause, ECG abnormalities increase risk of coronary heart disease; 1984 |</p>
<table>
<thead>
<tr>
<th>Study</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>HOPE</td>
<td>Ramipril reduced risk of death, MI, stroke, and revascularization. <em>Vitamin E</em> did not lower the risk of CAD; 2000</td>
</tr>
<tr>
<td>SHIFT</td>
<td>Ivabradine reduction in hospitalization or CV death from heart failure. 2010</td>
</tr>
<tr>
<td>UKPDS</td>
<td>(HTN in Diabetes study) - BP control &lt; 150/85 in pts with HTN and Diabetes with ACEI or BB, plus additional meds if needed, reduces risk of diabetic related complications and death related to diabetes (MI, PV0D, renal disease, CVA, sudden death) along with decrease in progression of <em>neuropathy</em> and <em>retinopathy</em>; 1998</td>
</tr>
<tr>
<td>WHI</td>
<td>(Women’s Health Initiative) - post menopausal women on combined hormonal therapy is associated with increased risk of CAD, PE, CVA and invasive <em>breast cancer</em> but decreased risk of <em>hip fractures</em> and <em>colorectal cancer</em>; absolute risk excess was 19 per 100,000 person-years; 2002</td>
</tr>
</tbody>
</table>

**Heart Failure**

<table>
<thead>
<tr>
<th>Study</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIRE</td>
<td>Ramipril started 3–10 days after MI, benefit noticeable as early as 30 days, reduction in progression to heart failure; no reduction in reinfarction or stroke; 1993</td>
</tr>
<tr>
<td>CAPRICORN</td>
<td><em>Carvedilol</em> decreases cardiovascular and all cause mortality in post- infarction pts</td>
</tr>
<tr>
<td>Study</td>
<td>Treatment</td>
</tr>
<tr>
<td>-------------</td>
<td>---------------------------------------------------------------------------</td>
</tr>
<tr>
<td>CHARM</td>
<td>Candesartan addition to concurrent BB and/or ACEI therapy –significant reduction in CV death or hospital admission for CHF (16%); NNT is 23 in 1 year</td>
</tr>
<tr>
<td>CIBIS</td>
<td>EF &lt; 35% and NYHA class III or IV, B1 blocker bisoprolol significantly reduced all cause mortality, sudden death, and all cause hospitalizations from CHF; 1999</td>
</tr>
<tr>
<td>COPERNICUS</td>
<td>Coreg in addition to diuretic plus ACE/ARB reduces all cause mortality and hospitalization; 2001</td>
</tr>
<tr>
<td>EPHESUS</td>
<td>Eplerenone in addition to standard therapy reduces mortality in pts with severe CHF; 2001</td>
</tr>
<tr>
<td>MERIT-HF</td>
<td>Toprol CR/XL when used on top of ACE-I reversed ventricular remodeling as shown by decreased LV- EDV and ESV by cardiac MRI and decreases all cause mortality when started in pts with CHF; 2000</td>
</tr>
<tr>
<td>RALES</td>
<td>Aldactone 25 mg in addition to standard therapy reduces mortality and risk of sudden death in pts with severe CHF (EF &lt; 35, Class 3-4); RRR 30%; 1999</td>
</tr>
<tr>
<td>RESOLVD</td>
<td>Enalapril plus candesartan combination was more beneficial in preventing LV dysfunction (reduced ESV and EDV), compared to either drug alone ; 1999</td>
</tr>
<tr>
<td>SAVE, TRACE, SOLVD</td>
<td>ACEI reduces all cause mortality remodeling, and decreased risk of</td>
</tr>
</tbody>
</table>
worsening heart failure when started 2–10 days after MI, and in pts with CHF (EF < 35%)

**VALIANT**
ARBS have mortality equivalent to ACEI; side note: 1999

**V-HEFT**
**Vasodilator** (enalapril vs. hydralazine/nitrate)-Heart Failure Trial) - pts with CHF on digoxin and diuretic; enalapril has greater reduction mortality 1991

### Coronary Artery Disease

<table>
<thead>
<tr>
<th>Study</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td><strong>ACUITY</strong></td>
<td>Enoxaparin equivalent to heparin upstream during ACS 2004</td>
</tr>
<tr>
<td><strong>CAPRIE</strong></td>
<td>Plavix was slightly better than aspirin in reducing primary endpoints of MI, CVA, and vascular death; conferred benefit for CVA and PAD; no benefit in pts with previous MI; 1996</td>
</tr>
<tr>
<td><strong>CAST</strong></td>
<td>Flecanide and encanide increased mortality in pts with post-MI asymptomatic or mildly symptomatic supraventricular arrhythmias; 1989</td>
</tr>
<tr>
<td><strong>COMMITT</strong></td>
<td>Plavix plus ASA reduces 30 day mortality (0.6% ARR), BB good after MI if pts do not have heart failure/cardiogenic shock; 2005</td>
</tr>
<tr>
<td><strong>CURE</strong></td>
<td>Plavix in addition to aspirin in patients with non-STEMI ACS reduces risk of CV death, MI, and CVAs by 20%; 2002</td>
</tr>
<tr>
<td><strong>GISSI-3</strong></td>
<td>Lisinopril, when given &lt; 24 hrs in pts with acute MI, reduced</td>
</tr>
<tr>
<td>Study</td>
<td>Details</td>
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<tr>
<td>GUSTO-1</td>
<td>Accelerated t-PA plus IV Heparin has lower mortality although higher bleeding than streptokinase and standard therapy. 1995</td>
</tr>
<tr>
<td>HORIZONS-AMI</td>
<td>Bivalirudin reduced bleeding and death compared to Heparin plus Glycoprotein IIb/IIIa inhibitor in 30 days; increased &lt;24 hr in stent thrombosis with bivalirudin but offsetted between 24 hrs to 30 days; 2009</td>
</tr>
<tr>
<td>ISIS-2</td>
<td>ASA lowers CV death, Recurrent MI, CVA when given to patients with acute MI; 1988</td>
</tr>
<tr>
<td>NORWEGIAN TIMOLOL</td>
<td>In pts who survive acute MI, Beta blockers reduce all cause mortality, sudden death, and reinfarction; 1981</td>
</tr>
<tr>
<td>PLATO</td>
<td>Pts with ACS, with or without ST elevation, ticagrelor reduces death from vascular, MI, and CVAs; slight increase non-procedural related, i.e. fatal intracranial bleeding, 2009</td>
</tr>
<tr>
<td>TACTICS-TIMI 18</td>
<td>GpIIb/IIIa inhibitor plus invasive strategy in pts with moderate-high risk UA/NSTEMI is better than conservative management; 2001</td>
</tr>
<tr>
<td>TRITON-TIMI 38</td>
<td>Prasugrel reduced CV death, nonfatal MI, nonfatal stroke compared to clopidogrel 19% reduction in CV death, MI or stroke compared with clopidogrel in patients undergoing PCI for</td>
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### Intervventional

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<tbody>
<tr>
<td><strong>CARP</strong></td>
<td>In patients with stable CAD, <em>coronary artery</em> revascularization prior to elective major vascular surgery, s.a. expanding AAA, PVOD of legs, does not improve outcomes; 2004</td>
</tr>
<tr>
<td><strong>ERACI, GABI, BARI, CABRI, RITA, EAST</strong></td>
<td>PTCA and CABG have similar rates of survival and avoidance of MI and similar long term health care costs; PTCA group had increased rates of recurrent angina and revascularization; nearly ¼ of PTCA patients required CABG; At 10 year follow up some studies showed that Diabetics and pts &gt; 65 yrs have slightly decreased mortality with CABG; Subset of CABRI trial - pts with multi-vessel or chronically occluded major vessel disease had better outcomes with CABG; 1994, 1994, 1996, 1995, 1998, 1999</td>
</tr>
<tr>
<td><strong>FRISC-II, RITA-3</strong></td>
<td>At 5 year follow up, in moderate-high risk pts with ACS without ST elevation, early invasive intervention strategy has improved outcomes in terms of death/MI; 2005, 2006</td>
</tr>
<tr>
<td><strong>GUSTO-2B</strong></td>
<td>PTCA has better outcomes than thrombolysis in pts with AMI. Lowest 30 day mortality when D2B time &lt; 60 minutes (1%), 60–90 minutes (4%), &gt; 90 minutes</td>
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<tr>
<th><strong>NORDISTEMI</strong></th>
<th>(6.5%) 1997</th>
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<tbody>
<tr>
<td>Early invasive strategy in pts with STEMI has significant reductions in primary outcomes (death, stroke, reinfarction) at 30 days, but at 12 months, reductions were nonsignificant, but trended towards significance as invasive group had less incidence of death, strokes, reinfarction at 12 months; 2010</td>
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<tr>
<th><strong>PARTNER TRIALS</strong></th>
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<td>TAVR superior to medical therapy and equivalent to surgical therapy of high risk patients. 2011</td>
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<tr>
<th><strong>SHOCK</strong></th>
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<tr>
<td>In pts with cardiogenic shock due to acute MI, early revascularization vs. medical stabilization does not improve 30 day mortality but does improve 6-month and 12 month survival 1995</td>
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<th><strong>SYNTAX</strong></th>
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<tr>
<td>PCI vs. CABG in pts with severe CAD - At 1 yr, CABG group had lower rates of major cardiac or cerebrovascular events (12% vs. 18%) and repeat revascularization (6% vs. 14%); however, there was increase in rate of strokes (2.2% vs. 0.6%). Conclusion - CABG should be standard of care in pts with severe 3 vessel or left main disease; 2009</td>
<td></td>
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</tbody>
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