MED C Reference Guide / Toolkit

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Resource Also Available on Agile MD (www.agilemd.com), Download free iOS / Android Play Store.

Disclaimer & Acknowledgements

Please note that this is a compilation of information taken from Maine Medical Center Cardiology, Critical Care Surgical and Anesthesiology Attendings, Guidelines from American College of Cardiology ACC, American Heart Association AHA, European Society of Cardiology ESC, peer-reviewed journal articles, and miscellaneous sources including internet/ websites. The management for the individual patient is subject to appropriate clinical scenario. Medication dosing should be confirmed with primary literature source or databases such as uptodate or lexicomp. This reference guide is not a comprehensive source for patient care during cardiology rotation at Maine Medical Center and may not address other important issues of care, which are still essential to your practice.

If there are any concerns about the information contained within this reference guide or recommendations for improvement, please contact Jay Shah (jshah@mmc.org).

Special thank you to assistance from the following folks regarding development of the project:
- MaineHealth Cardiology Attendings, especially Dr. Sanjeev Francis, Dr. Jennifer Hillstrom
- MMC Cardiology Fellows
- CVCC Attendings, especially Dr. Virginia Eddy and Arielle Butterly
- Internal Medicine Chiefs
- Maine Medical Center Institutional Review Board (IRB)
- Maine Medical Center Research Institute Center for Outcomes Research & Evaluation (CORE)

Med C General Orientation

Daily Flow General Format

- 6:30/45-7:00am– Morning CAT (Cardiology – Hem/Onc - Transplant) Sign-out in P1A
- 7:30-8:30am – Morning Report for Senior Resident
- Floor Team:
    - 7:15-8:30am – Discovery rounds with attending and Med C team (rounding on patient's being discharged first)
- ICU Team:
    - 7:15-8:30am – Med student, intern, cardiology fellow bedside round on unit patients
- 8:30am – CICU Rounding w/ attending, CVCC attending (i.e. surgery or anesthesia critical care attending), cardiology fellow, medicine/medicine-pediatrics senior resident, interns (medicine, medicine-pediatrics, emergency medicine, family medicine), and medical students
- Followed by conference room table-side rounding on rest of the floor patient

- Call consults sooner rather than later
- Round with senior in the afternoon ~2-3pm and prior to sign-out especially for CICU pts
- Teaching session as discussed between the Med C senior and attendings
- 6:30pm – Cardiology Attending Day signs out to Cardiology Attending Night on Mon-Thursday (on Friday this happens at 3:30pm)
    - Players include: Day Med C Attending, Night Med C Attending, CVCC, Fellow, Night Resident and Intern

I want to highlight that this a guide/blueprint. This is not a substitution to communications among interns, seniors, fellows and attendings. If an intern is starting a new rotation, please reach out to your senior resident for specifics. Each senior as an individualized style of operating a team! If you do not know who your senior is or who additional details, reach out to medicine chiefs @ imchiefs@mmc.org.

TIPS and RECOMMENDATIONS:

- Always eyeball new overnight patients / new admits / CICU folks
- Update family members everyday
- Stay in touch with social workers for medication assistance and/or discharge planning
- If any patient's acutely deteriorating, contact your senior / fellow / attending (going up the chain, if unable to get hold of each provider)
- Before signing d/c order, review the medication list with senior
    - Go over discharge summary with senior
    - Seniors: Ensure that the place-in orders are signed [Overview tab], so you don't have issues when it comes to signing the discharge orders
- **Night Team:** Always document your actions, especially for the patients in the CICU. For example, starting a pressor agent, adjusting pressors with rationale, dosing diuretics, important overnight family conversation (including code status discussion – update cardiology fellow if unexpected code status change), discussions with fellow, etc.
    - Even though we signout to the day team, without proper documentation there is no way to provide a convincing timeline / depict decision making process
    - Ex: Imagine a complicated case with adverse outcome(s), turning into an M&M. Without proper written documentation, there isn't a convincing method to

document that the assessment and plan was shared with relevant team players and the group census.

- Daily EKG Practice on Med C should be a routine
    - Use EKG Wave Maven for additional practice:
    - https://ecg.bidmc.harvard.edu/maven/mavenmain.asp

Cardiovascular Critical Care (CVCC) Service Line: Communication and role clarification for patients on MED C.

Services involved: ***Cardiology, CVCC, and Neurocritical Care (NCC)***

1) Communication:
   - Resident's first call should always be to the cardiology fellow.
     - After updating the fellow and developing a plan, make sure to discuss the appropriate attending(s) to notify (cardiology, CVCC, NCC) and who will be making this call(s).
   - In emergent situations (Bipap, Intubation, Code) whether in CICU or R7 or R9, residents should follow standard emergency algorithms. ie. Call Code white or Code Blue.
     - update the fellow as soon as able or have a colleague page them while you respond
     - If this necessitates a transfer to CICU, both cardiology and CVCC should be made aware and again this is something for the residents to clarify with the fellow
   - **In situations of uncertainty, the overall consensus from the above services is more communication is better than less**

2) When to notify fellow: (not to be taken as a comprehensive or exhaustive list)
   - Decline in patient condition or status
   - Vasoactive medications: adding or adjusting vasoactive medications
   - Code white or Code blue on MED C patient
   - Equipment concerns: Balloon pump, PA catheter, Artic Sun

3) Therapeutic Hypothermia (TH) Patients:
   - During the day, while rounding on TH patients, NCC will attempt to be available in the CICU to discuss critical care related matters.
   - NCC specifically is available to assist in matters related to targeted temperature management, EEG interpretation, shivering control, vasoactive medications, sedation, and ventilation management.
   - It is not mandatory that the CVCC also be involved in these patients if the aforementioned issues are managed by NCC while the cardiac issues are managed by cardiology
     - There may be specific situations where CVCC is involved with TH patients, but this distinction will be made at the level of Attendings and residents will be made aware.

Presentation Style – Problems based or Systems Based (next page):

Problem Based:

- New Presentations / Structured:
    - CC
    - HPI (can include relevant labs here) / EKG (if relevant)
    - PMH / PSH
    - Medications / Allergies
    - SH (Smoking, Drinking, Drugs, Living/Occupation)
    - Physical Exam including vital signs
        - Important findings to include: Mucous membranes, CVP assessment / JVD, S1, S2 heart sounds, any other additional heart sounds, murmurs, friction rubs, pulses (radial, femoral, dorsalis pedis, posterior tibial), pulsatile liver, ascites, hepatosplenomegaly, edema – pitting vs non pitting
    - EKG/ Labs (*no editorializing, save that for assessment and plan*) / Imaging
    - Assessment and Plan
        - Include contingencies for our overnight colleagues

- Follow-up Patients:
    - Overnight events
    - Subjective
    - Vitals and Physical Exam, new findings or changes
    - Assessment and Plan
    - Disposition / Contingencies / Barriers to discharge (if any)

System Based:
Room # Patient Name:

Quick 1-liner:

Significant Overnight events:

Systems Based Rounding:

1. Cardiovascular
   - #
   - #
   - #
2. Neurology
   - #
3. Pulmonary
   - #
4. Gastrointestinal
   - # Nutrition
   - # Bowel Movements
5. Renal / Electrolytes
   - # Serum creatinine stable / Acute kidney injury / CKD Stage X
   - # Electrolytes: Na, K ~ 4 (goal), Mg ~ 2 (goal), Ca, Phosphorous
6. Endocrine
   - # Blood sugar, TSH
7. Hematology
   - #
8. Infectious Disease
   - #
9. Misc

RN Orders, Concerns:

Checklist:

EXAMPLE
Room # 9XX Patient Name: Jane Doe

Quick 1-liner:
Jane is a 66 year old woman with coronary artery disease, hypertension, diabetes mellitus type 2, who presented to MMC on xx/xx/2017 with chest pain and shortness of breath over the last 1 week. Patient's presenting EKG was notable for ST elevations in inferior leads, cardiac catheterization demonstrated 100% occlusion of RCA s/p DES with minimal residual disease.

Significant Overnight events: Slept well no acute concerns. No CP reported.

Systems Based Rounding:
The goal is to integrate your subjective, physical exam and assessment and plan into the particular system for efficient rounding. I would like people to pay attention throughout the presentation, as opposed to be distracted until we hit Assessment and Plan stage.

1. Cardiovascular
    - # VS: HR, BP
    - # Exam: Audible S1, S2, etc. No signs of volume overload.
    - # Telemetry showed sinus rhythm with NSVT episodes
    - # Inferior STEMI
        - S/p revascularization.
        - Meds: Aspirin, Clopidogrel, Metoprolol, Lisinopril, etc
2. Neurologic
    - # Awake, mentating well / Intubated, responds to stimuli?
3. Pulmonary
    - # For Ventilated Patients:
        - Vent Settings
        - Are they on CMV (Continuous Mandatory Ventilation) or are they on CPAP/PSV (Continuous positive pressure ventilation / Pressure support ventilation)?
            - If on CPAP how long did they CPAP for?
4. Gastrointestinal
    - # Nutrition
    - # Bowel Movements
5. Renal / Electrolytes
    - # Serum creatinine stable / Acute kidney injury / CKD Stage X
    - # Electrolytes: Na, K ~ 4 (goal), Mg ~ 2 (goal), Ca (major cardiac electrolytes), Phosphorous
6. Endocrine
    - # Blood sugar
        - Diabetics BS goal 120-180 / Non-diabetics BS goal 70-140
    - # TSH
7. Hematology
    - # Anemia / Erythrocytosis
    - # Thrombocytopenia / Thrombocytosis
    - # WBC count?
8. Infectious Disease
    - # Infection / Antibiotics. Please state Day X or 7 or 14 or whatever
9. Misc

RN Orders, Concerns:
Checklist:

WORK DISTRIBUTION:

Cardiology Fellow w/ (Computer A, overseeing)
Senior Resident – Running the rounds
Intern #1: Presenter (rotated every week), presenting to the senior resident / fellow
Intern #2: Putting orders (Computer B)
Intern #3: Grabbing RN for rounding, Paper chart, going over the checklist, (Computer C, helping w/ documentation)

CICU Intern Patient Work Load - I suggest intern's cap should be around 5-6 patients. Please be vocal if you cannot handle the work load. I do not wish patient care to be compromised. But I want each intern enough experience to feel comfortable taking care of ICU patients, learn cardiology and develop problem solving analytical skill.

The senior resident and fellow's role is to facilitate the education process.

Yellow Highlight – Indicates MKSAP related questions
Green Highlight – Indicates Best Practice Methods

Didactic Lectures / Topics Assignment

Recommended for senior resident use. Discuss and write down topics you'd like to go over each week together with interns, Cardiology fellow, Cardiology Attendings, CVCC Attending and Pharmacist, at the beginning of the rotation and thereby taking advantage of their expertise. This should reduce the redundancy in lectures and target high yield topics (see Table of contents). On average, there will be 2-4x/week hourly didactic section. You can also download this by visiting, www.bit.ly/medcicu --> Didactic Lectures Topics Assignment.pdf file.

Week 1: Attending: _____ Fellow: _____ CVCC: _____

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-
-
-

Week 2: Attending: _____ Fellow: _____ CVCC: _____

-
-
-
-

Week 3: Attending: _____ Fellow: _____ CVCC: _____

-
-
-
-

Week 4: Attending: _____ Fellow: _____ CVCC: _____

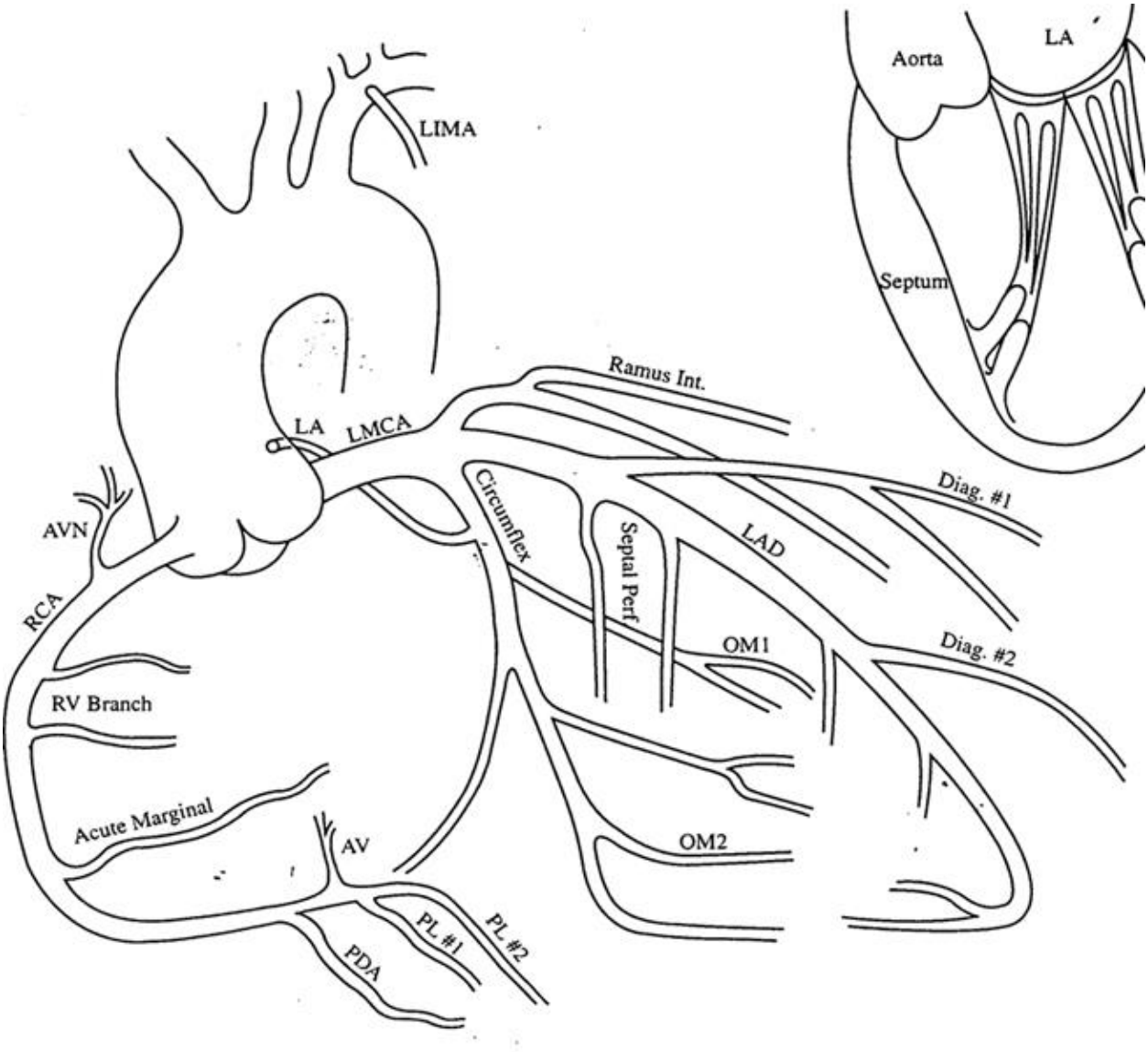
-
-
-
-

Cardiology / CVCC Attendings Interests

Not a comprehensive list of interests, but a starting point.

Dr. Sanjeev Francis – PA Catheter / Hemodynamics discussion, Cardiac Imaging (Echo, MRI), EKG reviews, arrhythmia, NSTEMI
Dr. Marco Diaz – Cardiac imaging, Valvular heart disease, TAVR, MitraClip
Dr. John Lualdi – Cardiac imaging, Valvular heart disease, TAVR, MitraClip
Dr. James Powers - Cardiac imaging, Stress testing
Dr. Edward Teufel – Lipid management, Stress testing
Dr. James Parker - Cardiac imaging, Stress testing
Dr. Jennifer Hillstrom – Chest pain assessment – imaging modalities (Exercise Stress test to Nuclear medicine stress test), Syndrome X, Myocarditis, Mechanical Complications of STEMI
Dr. Jeffrey Rosenblatt – Nuclear cardiology including PET, Cardiac imaging, general cardiology
Dr. Jennifer Monti – Cardiac imaging, PFO closure
Dr. Douglas Sawyer – HF - physical and management, Advanced mechanical support, Cardiomyopathies
Dr. Esther Shao – HF - physical and management, Advanced mechanical support, Cardiomyopathies
Dr. Joseph Wight – HF - physical and management, Cardiomyopathies including hypertrophic cardiomyopathy and sarcoidosis
Dr. Samuel Coffin – HF - physical and management, Advanced mechanical support, Cardiomyopathies
Dr. Christopher Link – HF - physical and management, Advanced mechanical support, Cardiomyopathies
Dr. Andrew Corsello – EP / ICDs / Pacemaker indications, Arrhythmia review, NOACs, Anti-arrhythmics
Dr. Henry Sesselberg – EP / ICDs / Pacemaker indications, Arrhythmia review, VT, Anti-arrhythmics
Dr. Paul Frey – ACS, Mechanical Complications of STEMI, TAVR, MitraClip, Valvular heart disease
Dr. David Butzel – ACS, Mechanical Complications of STEMI, TAVR, MitraClip
Dr. Jeremy Estrada – ACS, Mechanical Complications of STEMI, PFO
Dr. Samip Vasaiwala – ACS, NSTEMI / STEMI guidelines, Mechanical device / IABP, Impella, Hemodynamics
Dr. Thomas Ryan – Thrombolytics, Hemodynamics, Pulmonary HTN, shunts, ACS, STEMI or not?
Dr. Mary Fahrenbach – ACS, Mechanical Complications
Dr. Arielle Butterly – PA Catheter / Hemodynamics, Vent Management, Anesthesiology background
Dr. Virginia Eddy – PA Catheter / Hemodynamics, Vent Management, Surgical background
Dr. Joe Rappold – Hemodynamics, Vent Management, Surgical background
Dr. Anne M. Andrle – Pressors and Inotropes, New medications for HF

Coronary Anatomy

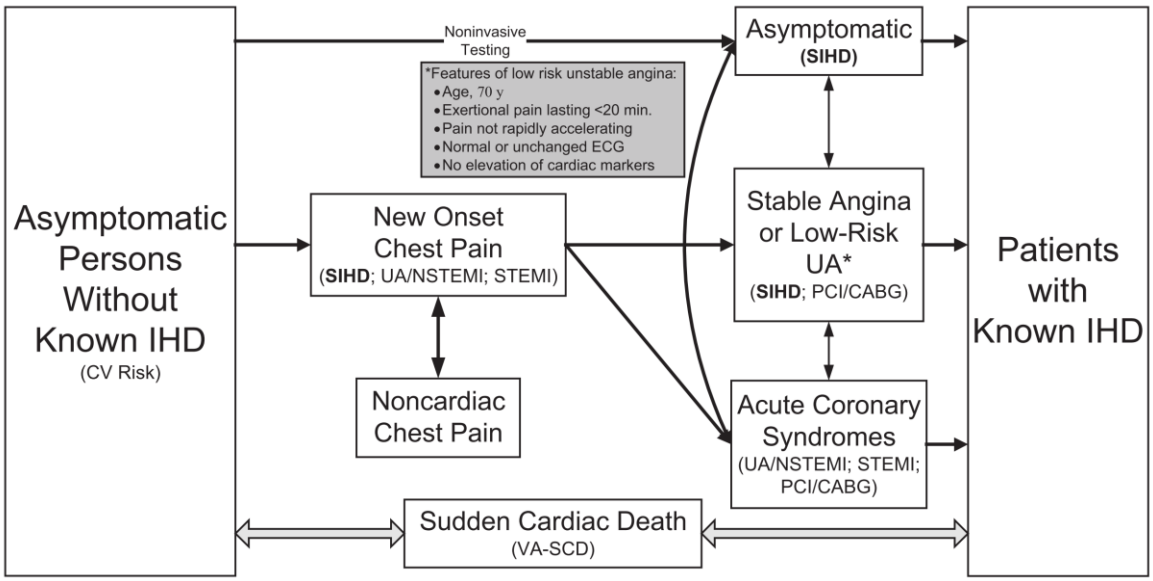


Coronary anatomy orientation during coronary angiography. [1]
IV Contrast injected into LMCA (Left main coronary artery), travels to LAD (left anterior descending) and LCx (left circumflex). You distinguish these from each other by looking at large vessels with multiple branches, i.e. septal perforators and diagonal branches (i.e. LAD). Now, LCx wraps around the back, but you are looking at a 3D object in a 2D plane, so LCx appears medial to LAD (when in fact it's lateral).

Source:
http://www.meddean.luc.edu/lumen/meded/radio/curriculum/vascular/coronary_artery.jpg

Spectrum of Ischemic Heart Disease

- Asymptomatic Ischemic Heart Disease
- Stable Angina / Low Risk Unstable Angina
- Acute Coronary Syndrome
    - Unstable Angina
    - NSTEMI (non-ST elevation myocardial infarction)
    - STEMI (ST elevation myocardial infarction)



Extracted from 2012 ACC Guidelines for Stable Ischemic Heart Disease [2]

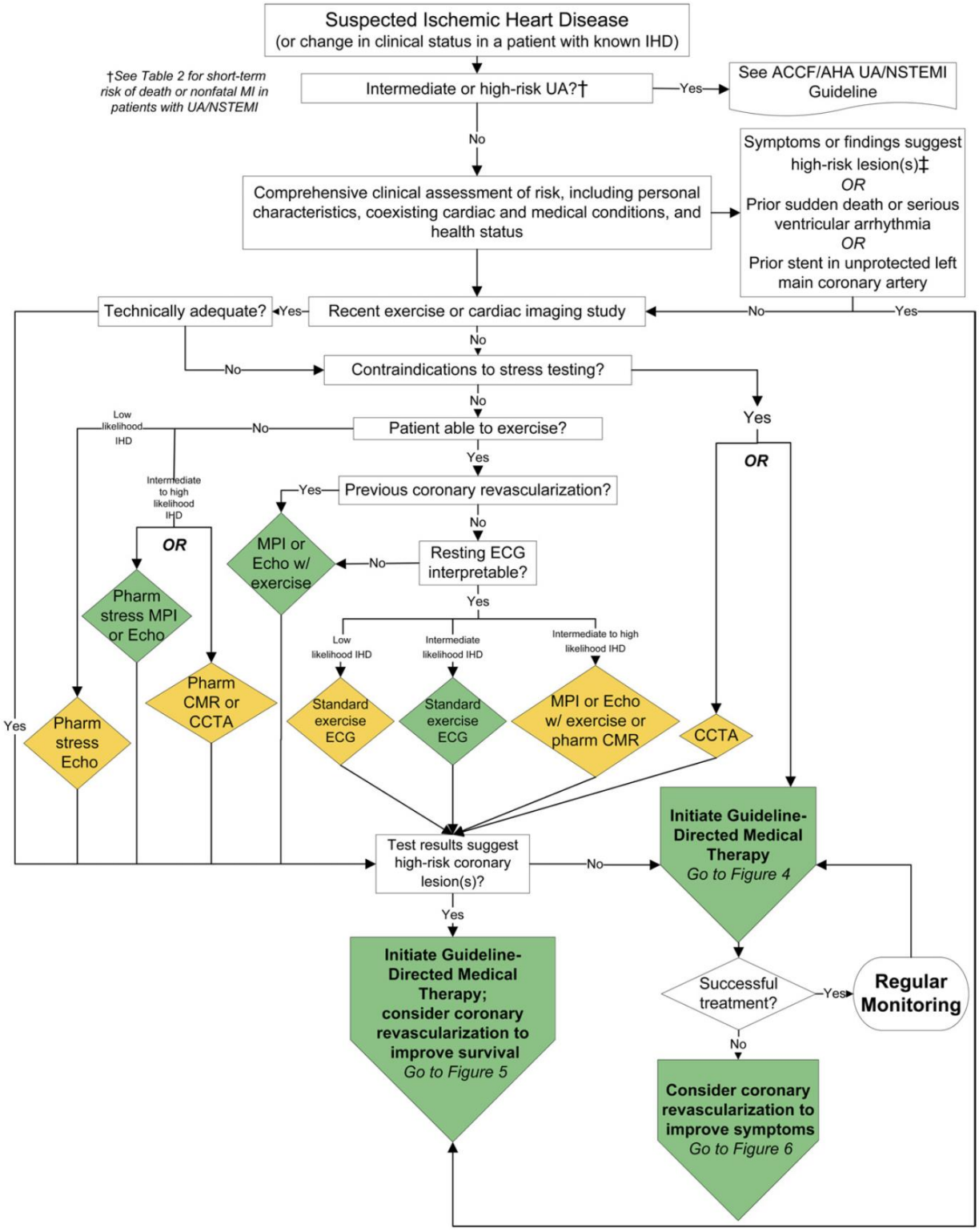
Intermediate Risk for Atherosclerotic Cardiovascular Disease

Additional testing that can help classify high risk vs low risk:

- hsCRP (high sensitivty C-reactive protein) > 2 mg/L
- Coronary artery calcium scoring > 300 or > 75% for age
- ABI < 0.90
- LDL >= 160 mg/dL or genetic hyperlipidemia
- FH of premature ASCVD < 55 in 1st degree male or < 65 in 1st degree female

Suspected Ischemic Heart Disease

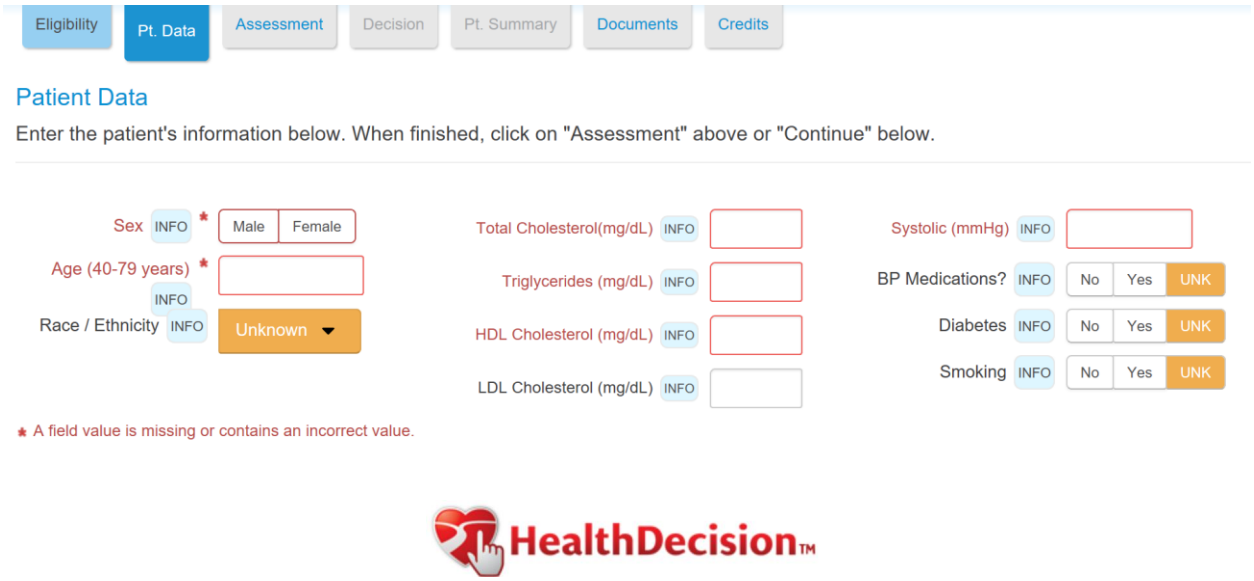
Diagnostic Algorithmic Work up for suspected ischemic heart disease [2-4]



Stable Ischemic Heart Disease Treatment

- Aspirin 75-162mg/d (generally aspirin 81mg/d)
  - (contraindication: desensitization - MMC has a protocol – next page described, clopidogrel 75mg/d)
- Lifestyle modifications: diet, weight loss, physical activity, quit smoking
- Consider addition of statin for moderate / high risk patients (Calculator: http://tools.acc.org/ASCVD-Risk-Estimator/ or .ASCVD dot phrase for Epic)
- Comorbid risk factors control:
  - Hypertension management [5]
    - Normal < 120/80 (Reassess in 1 yr)
    - Elevated BP 120-129/< 80 (Reassess in 3-6 months, nonpharmacologic tx)
    - Stage I Hypertension – 130-139/80-89 (nonpharm tx, meds)
    - Stage II Hypertension – ≥ 140/90 (nonpharm tx, meds)
  - Diabetes control
- Anginal Symptoms
  - Sublingual NTG
  - Consider starting a BB
  - Consider subs/addition of CCB and/or long-acting nitrate (you can do these things prior to considering cath)
  - Consider addition of ranolazine
- Persistent symptoms → consider revascularization

Shared Decision Making Approach to Starting Statin Medication:
https://www.healthdecision.org/tool.html#/



Acute Coronary Syndrome

Spectrum of clinical presentations including Unstable angina (UA), Non ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI) [2, 6, 7].

Simplified distinction:

- Unstable Angina – 1) anginal symptoms at rest, increased in frequency/duration (> 20m), 2) new-onset severe angina 3) crescendo pattern of occurrence; negative cardiac biomarkers
- NSTEMI – Severe ischemia resulting in myocardial damage and release of cardiac biomarkers. Other changes noted ST-T wave changes on EKG (dynamic changes, ST-depression). Subendocardial ischemia.
- STEMI – Occlusion of the coronary artery resulting infarction. Transmural infarction.

Recommended Papers:

- Acute Coronary Syndromes: Diagnosis and Management, Part I and Part II. Amit Kumar, MD and Christopher P. Cannon, MD. Mayo Clin Proc. 2009 Oct; 84(10): 917–938. PMCID: PMC2755812, PMC2770915. [6, 7]

ST-segment elevation myocardial infarction (STEMI)

ACC/AHA STEMI Guidelines

1. Presenting symptoms documentation: Chest pain (CP), arm radiation, jaw pain, GI/ nausea, angina / angina equivalent w/ appropriate timeline
    a. Structured notes recommended for ACS admission (You can use my smartphrase: Go to Smart Phrase Manager; Shah, Jay; open D2BSTEMI; copy paste into new Smartphrase or add yourself to SmartPhrase User under Owners & Users tab):
        i. Time of onset of chest pain / symptoms: ***
        ii. First Medical Contact Time: ***
        iii. ER Arrival Time: ***
        iv. First and Qualifying EKG: ***
        v. Cath Lab Arrival Time: ***
        vi. Open Artery Time: ***
        vii. Medications Provided: ***
2. Coronary territory / Distribution of MI based on EKG [8]:

Anterior	STE V1-V4	LAD
Lateral	STE - 1, aVL, V5, V6	LCx, Diagonal
Inferior	STE - II, III, aVF	RCA, LCX
Suspect in patients with inferior MI		
Posterior	STd - V1-V3 Tall R wave in V1	LCX, RCA Order posterior EKG
RV infarction	STE - V1, V2 STE 1mm RV4 STE in III > II	Proximal RCA Order right sided EKG

Inferior STEMI can spread to 3 regions: Posterior, Lateral and RV infarct [8]
STE – ST elevation; STd – ST depression

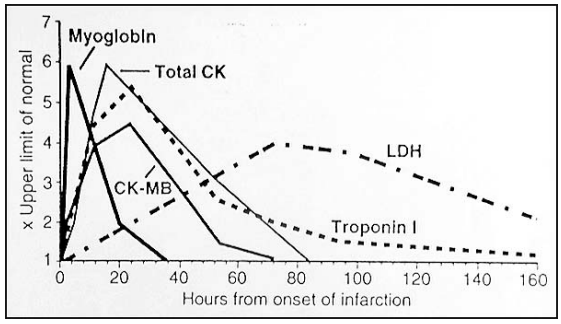
3. TNK yes / no (time and date)
    a. *Every patient post TNK/Lytic undergoes either urgent rescue-PCI (pharmacoinvasive PCI – percutaneous coronary intervention) or PCI later / next day*
    b. *Indications for urgent/rescue PCI s/p lytics include:*
        i. Continued chest pain, hemodynamic instability, lack of ST-elevation resolution < 50%
        ii. If negative for abovementioned criteria, then await 3 hrs prior to intervention due to high bleeding risk (in the setting of TNK)
    c. Accelerated Idioventricular Rhythm (AIVR) / Non-sustained ventricular tachycardia after lytics in 24-48 hours can be a sign for reperfusion
        i. You do not do anything dramatic besides standard care
        ii. AIVR – Wide complex rhythm without discernable atrial activity
4. Cardiac catheterization results:
    a. Single vessel / Residual disease
    b. BMS (Bare metal stent), DES (Drug eluting stent)
        i. BMS – 1 month at least – P2Y12-receptor antagonist
        ii. DES – 12 month at least – P2Y12-receptor antagonist
        iii. Use DAPT score to figure out the duration of dual antiplatelet therapy in patients with high risk CAD profile. Balance risk of bleeding vs risk of stent thrombosis
    c. Ejection fraction - LV gram or Echocardiogram
    d. Document LVEDP (< 12 normal)

e. *FYI: For patients undergoing cath procedure, important information to know/ document include:*
    i. *Any contraindications to taking clopidogrel / P2Y12 inhibitor? For example, any surgeries planned in a year, medical compliance issue, bleeding disorder – GI bleed? Additionally, document in notes whether you are requesting an interventionalist to place a BMS vs DES.*
f. *Senior Residents: Consent for Cardiac catheterization is obtained by Cardiology fellows. Please update the fellows if your patients need consent.*

5. **Nine modifiable risk factors for AMI in order: Dyslipidemia**, Smoking, Psychosocial stressors, DM, HTN, obesity, EtOH, physical activity, and diet low in fruits and vegetables.
6. Post Cath check with progress note documentation in 4 hours for femoral access as opposed to radial access
    a. Pay careful attention to bruits
    b. Hematoma, Retroperitoneal bleed, Acute limb ischemia (emergent vascular consult, CT-A and update fellow/attending/CVCC)
7. Usual Medications pre STEMI [9]
    a. Aspirin
        i. Loading dose: 162-325mg
        ii. Maintenance dose: 81mg/d
    b. $P2Y_{12}$ inhibitors
        i. Loading dose w/ Fibrinolytics (Outside of MMC / ex people who rec'd TNK):
            1. Age ≤ 75: Clopidogrel 300mg
            2. Age > 75: Clopidogrel 75mg
        ii. Loading dose w/o Fibrinolytics (usually MMC Hospital)
            1. Clopidogrel 600mg / Prasugrel 60mg / Ticagrelor 180mg
        iii. Maintenance dose below see 8b
    c. GP IIb/ IIIa
        i. Eptifibatide (Integrilin) 180 mcg/kg bolus x2 (2^{nd} dose 10min after first dose) with 2 mcg/kg/min maintenance; in CrCl < 50% decrease infusion by 50%; Avoid in hemodialysis
    d. Anticoagulation
        i. Heparin
            1. W/ GP IIb/IIIa – 50-70 U/kg IV bolus to achieve ACT 200-250s
            2. W/o GP IIb/IIIa – 70-100 U/kg IV bolus to achieve ACT 250-300s
            3. ACT = activated clotting time
        ii. Bivalirudin
            1. 0.75 mg/kg IV bolus, then 1.75 mg/kg/h infusion w / or w/o prior treatment with UFH.
            2. An additional bolus of 0.3 mg/kg may be given if needed.
            3. Reduce infusion to 1 mg/kg/h with estimated CrCl _30 mL/min
    e. Lytics / TNK
        i. Provided if PCI capable hospital < 120 mins away
8. Usual Medications post STEMI [9]
    a. Aspirin 81mg/d
    b. $P2Y_{12}$-Receptor antagonist (usually clopidogrel 75mg/d, other options: prasugrel 10mg/d, ticagrelor 90mg BID)
    c. High-intensity statin (atorvastatin 80mg/d, rosuvastatin 40mg/d – less lipophilic compared to atorvastatin for patients who can't tolerate atorvastatin)
        i. If unable to tolerate, consider every other day dosing

ii. Additional options include PCSK9i (proprotein convertase subtilisin kexin 9 inhibitor): Repatha™ (evolocumab), Praluent® (alirocumab)
    1. These are q14d, q28d injectable medications
d. BB (titrate gradually for HR 60s)
    i. Usually started w/ metoprolol 6.25mg / 12.5mg q6h, gradually uptitrated and switched to BID dosing (at discharge)
    ii. Caution in patients with inferior MI
    iii. Start slow, and work your way up
    iv. Make sure at discharge BB is at BID or qDay dosing
e. ACEi (ex. Lisinopril) – Reduce ventricular remodeling
    i. Pending renal function
    ii. For patient's w/ LV dysfunction early threshold to start
f. Aldosterone antagonist (ex. Spironolactone) – Reduce ventricular remodeling
    i. Consider for patients with EF < 40%

9. Labs [10]:
    a. Daily BMP to monitor electrolytes and renal function post catheterization
        i. K ~ 4, Mg ~2
    b. Trend cardiac biomarker?
        i. CK and CKMB helpful to trend, to see the size of the infarct (area under the curve)
        ii. Troponin trend
        iii. Fun fact: What other biomarkers were studied in MI?
            1. Leukocytes (1st), AST
        iv. Check with your attending physician / fellow



10. Complications post MI [11]:
    a. Heart failure; Arrhythmias - VT/VF, atrial fibrillation; Inflammatory - Post-MI pericarditis, Dressler syndrome; mechanical complications (described below)
    b. Inferior STEMI (RCA, LCx)
        i. Complete heart block
        ii. Right Ventricle infarction
        iii. Ventricular Septal Defect (VSD) (~3-7d, flash pulm edema, hypotension)
        iv. Papillary posterior muscle rupture with associated mitral regurgitation (agitation feeling doom) (~2-7d)
        v. Free Wall Rupture / Tamponade (late thrombolytics)
    c. Anterior STEMI (LAD)
        i. Ventricular Septal Defect (VSD)
        ii. Free Wall Rupture / Tamponade (late thrombolytics)
11. Chest Pain (CP) post PCI
    a. Mild – inflammation secondary to underlying ischemia / infarct
    b. Mechanical discomfort post PCI
    c. Stent rethrombosis – Symptoms (angia / eq), CP, diaphoresis, vital abn.
        i. GET A STAT EKG
        ii. STE in same distribution would be concerning for stent thrombosis
        iii. Call the fellow / attending for urgent intervention
12. Transfers / Discharge
    a. Transfer

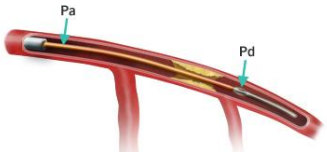
i. From CICU to R9W, 24 hrs post lytic administration
ii. Or post PCI, if patients are stable → discussion with senior/fellow/attending

b. Discharge
i. 2-3 days post adm, further depends on complexity of the individual case
ii. Ensure patients have cardiac rehab scheduled

13. Do you need echocardiogram ~$1200?
a. If V-gram performed, EF (ejection fraction) normal and no concerns for valvular disease or effusion, then unlikely to get an echocardiogram
b. Else depending upon clinical context discussion with fellow / attending

14. Residual disease evaluation options
a. How to evaluate them prior to discharge? / Attending discussion
i. Submaximal exercise stress test (usually prior to discharge), with stress test in 6 wks (Epic Order: "Echocardiogram stress test" → options then include treadmill, bicycle and dobutamine, in comments mention submaximal exercise stress test)
ii. Nuclear Medicine Stress Test (Epic Order: NM Myocardial Perfusion Rest Stress Adenosine or Regadenoson) [12]
iii. Coronary angiography with fraction flow reserve (FFR) < .75 = significant stenosis [13]

$$FFR = \frac{\text{Distal Coronary Pressure (Pd)}}{\text{Proximal Coronary Pressure (Pa)}}$$
(During Maximum Hyperemia)

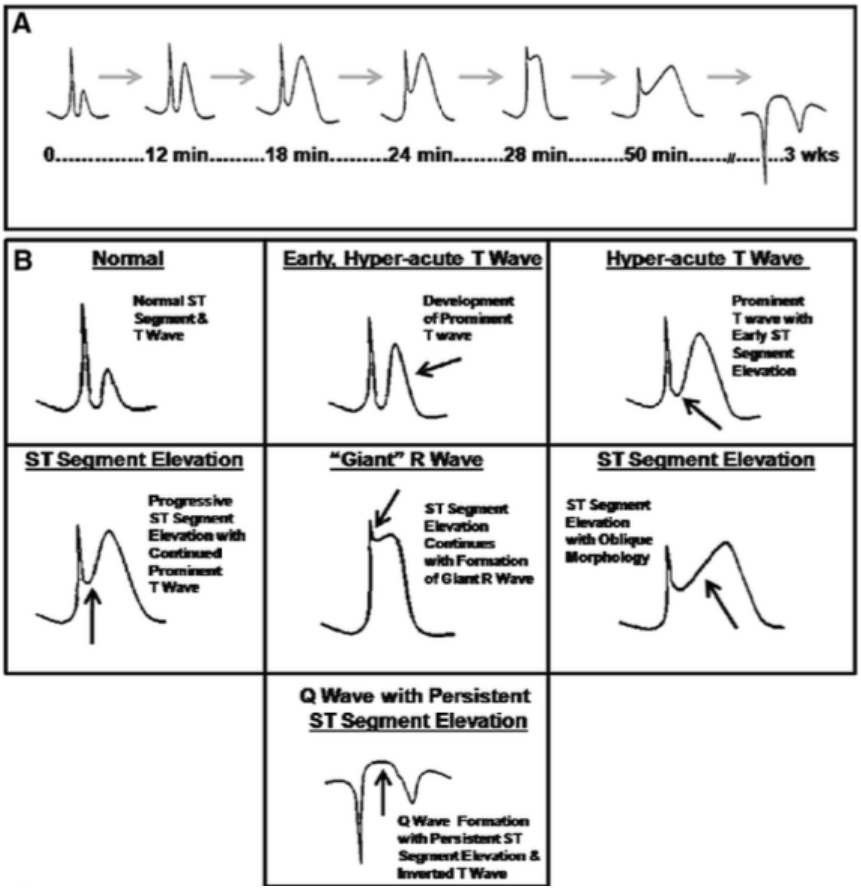


b. If overnight these patients complain of chest pain?
i. EKG, call fellow
ii. Nitro, Morphine
iii. Heparin
iv. ?Eptifibatide

15. Multivessel Coronary Artery Disease (MVD)
a. If diagnostic coronary angiogram was notable for MVD, then there are three treatment options
i. Coronary artery bypass graft (CABG)
1. Consult CT Surgery (discussion with fellow/ attending)
2. Especially if LVEF reduction, DM
ii. Multivessel Percutaneous coronary intervention (PCI)
iii. Medical management
b. Consider continuation of heparin drip post diagnostic catheterization
c. If patient experiences increased chest pain, contact fellow/ attending, next step may include intraaortic balloon pump (to assist with coronary perfusion during diastolic filling) as a bridge to CABG
i. Things to monitor on intraaortic balloon pump (IABP):
1. Peripheral Pulses
2. Platelet count
3. Urine output
4. Daily Chest XR for position of balloon pump and PA catheter
d. Avoid ACEi for subset of MVD patients who are about to undergo CABG procedure
i. Avoid renal perfusion issues, AKI, hypotension /hypoperfusion etc.
ii. Vasoplegia syndrome - postperfusion syndrome characterized by low systemic vascular resistance and a high cardiac output.

16. Example of a succinct 1-liners:
    a. 56 y/o man w/ HTN, HLD p/w CP noted to have an inferior STEMI s/p DES to proximal RCA, single vessel disease, EF 45% by LV gram
    b. 50 y/o woman w/ CAD/MI and smoking p/w CP, nausea and arm numbness found to have an inferior STEMI complicated by VF arrest and acute heart failure s/p TNK and DES to proximal RCA, no residual disease, EF 25% by echocardiogram.

Evolution of ST-T wave changes



Source: [14]

Non ST-segment elevation myocardial infarction (NSTEMI)

NSTEMI Guidelines [15]:

Ischemia guided strategy
- Diagnostic cardiac catheterization / revascularization, if objective evidence of myocardial ischemia i.e. recurrent symptoms / stress testing
- Low risk: TIMI 0-1, Grace < 109

Early invasive approach (w/in 24 hrs)
- Coronary angiography early followed by percutaneous intervention or coronary artery bypass graft when appropriate
- In other words non-invasive testing deferred in patients with suspected ACS
- Score: Grace > 140

What does these TIMI Risk and Grace score mean / what factors play a role?
(on the next page)

Usual Medications NSTEMI

1. Aspirin
   a. Loading dose: 162-325mg
   b. Maintenance dose: 81mg/d
2. $P2Y_{12}$ inhibitors
   a. Loading dose: Clopidogrel 600mg or Ticagrelor 180mg
   b. Maintenance dose: Clopidogrel 75mg/d or Ticagrelor 90mg BID
3. GP IIb/ IIIa (Class IIb indication – Only for early invasive + high risk features)
   a. Eptifibatide (Integrilin) 180 mcg/kg bolus x2 (2^{nd} dose 10min after first dose) with 2 mcg/kg/min maintenance; in CrCl < 50% decrease infusion by 50%; Avoid in hemodialysis
4. Anticoagulation (48 hrs / until PCI performed)
   a. Heparin IV - Loading dose 60 U/kg (Max 4000 units) + infusion
   b. Bivalirudin - Loading dose 0.10 mg/kg + 0.25 mg/kg/h infusion
   c. Enoxaparin SC 1mg/kg q12h (or q24h w/ CrCl < 30)
   d. Fondaparinux SC 2.5mg/d
5. Nitrates
   a. SL NTG q5min x3; if unresolved IVNTG for persistent ischemia, HF or hypertension
   b. AVOID: W/ phosphodiesterase inhibitor (sildenafil)
6. Beta-Blockers
   a. AVOID: Heart failure, low output state, risk for cardiogenic shock
7. Calcium-channel blocks (BB first preference; otherwise nondihydropyridine – Verapamil, Diltiazem)
8. Cholesterol
   a. High intensity cholesterol – Atorvastatin 40-80mg/d, Rosuvastatin 20-40mg/d
9. Supplemental Oxygen

Additional Treatment Options Include
- PCI w/ stenting
- CABG

TIMI RISK SCORE for UA/NSTEMI

HISTORICAL	POINTS
Age ≥ 65	1
≥ 3 CAD risk factors (FHx, HTN, ↑ chol, DM, active smoker)	1
Known CAD (stenosis ≥ 50%)	1
ASA use in past 7 days	1
PRESENTATION	
Recent (≤24H) severe angina	1
↑ cardiac markers	1
ST deviation ≥ 0.5 mm	1

RISK SCORE = Total Points (0 - 7)

RISK OF CARDIAC EVENTS (%) BY 14 DAYS IN TIMI 11B*

RISK SCORE	DEATH OR MI	DEATH, MI OR URGENT REVASC
0/1	3	5
2	3	8
3	5	13
4	7	20
5	12	26
6/7	19	41

*Entry criteria:UA or NSTEMII defined as ischemic pain at rest within past 24H, with evidence of CAD (ST segment deviation or +marker)

For more info go to www.timi.org

Antman et al *JAMA 2000*; 284: 835 - 842

A. GRACE Risk Model Nomogram

1. Find Points for Each Predictive Factor:

Killip Class	Points	SBP, mm Hg	Points	Heart Rate, Beats/min	Points	Age, y	Points	Creatinine Level, mg/dL	Points
I	0	≤80	58	≤50	0	≤30	0	0-0.39	1
II	20	80-99	53	50-69	3	30-39	8	0.40-0.79	4
III	39	100-119	43	70-89	9	40-49	25	0.80-1.19	7
IV	59	120-139	34	90-109	15	50-59	41	1.20-1.59	10
		140-159	24	110-149	24	60-69	58	1.60-1.99	13
		160-199	10	150-199	38	70-79	75	2.00-3.99	21
		≥200	0	≥200	46	80-89	91	>4.0	28
						≥90	100		

Other Risk Factors	Points
Cardiac Arrest at Admission	39
ST-Segment Deviation	28
Elevated Cardiac Enzyme Levels	14

2. Sum Points for All Predictive Factors:

Killip Class	+	SBP	+	Heart Rate	+	Age	+	Creatinine Level	+	Cardiac Arrest at Admission	+	ST-Segment Deviation	+	Elevated Cardiac Enzyme Levels	=	Total Points

3. Look Up Risk Corresponding to Total Points:

Total Points	≤60	70	80	90	100	110	120	130	140	150	160	170	180	190	200	210	220	230	240	≥250
Probability of In-Hospital Death, %	≤0.2	0.3	0.4	0.6	0.8	1.1	1.6	2.1	2.9	3.9	5.4	7.3	9.8	13	18	23	29	36	44	≥52

Stress Test

Simplistic Algorithm for Stress Testing

1. Who gets a stress test?
    a. Low risk / Intermediate patients with suspected ischemic heart disease
    b. If high risk / high-intermediate → Follow unstable angina guidelines, with consideration for diagnostic angiography
2. Able to exercise? (Usually, better to exercise patient, to understand functional status)
    a. Yes – Exercise stress testing
        i. EKG Interpretable
            1. YES – Exercise Stress test w/ EKG (low-intermediate likelihood, else choose imaging)
            2. NO – Myocardial perfusion imaging test
    b. No – Chemical Echocardiography or Myocardial perfusion imaging test (Nuclear stress test)

Exercise - Duke Prognostic Treadmill Score

Duke Prognostic Treadmill Score [16] = **Exercise time (min) - (5 x max ST segment deviation in mm) - (4 x exercise angina).**

Exercise angina: 0 = none, 1 = nonlimiting, and 2 = exercise limiting

- Low risk – score ≥+5 (→ if stable angina, work towards medication uptitration rather than additional testing, see stable ischemic heart disease management)
- Moderate risk – score from -10 to +4
- High risk – score ≤-11
- **In Short: Negative is bad**

Contraindications to Exercise Stress Testing:

Absolute

- Acute myocardial infarction (W/in 2 days)
- Unstable angina not previously stabilized by medical therapy
- Uncontrolled cardiac arrhythmias causing symptoms of hemodynamic compromise
- Symptomatic severe aortic stenosis
- Uncontrolled symptomatic heart failure
- Acute pulmonary embolus or pulmonary infarction
- Acute myocarditis or pericarditis
- Acute aortic dissection

Relative

- Left main coronary stenosis
- Moderate stenotic valvular heart disease
- Severe electrolyte abnormalities
- Severe arterial hypertension (>200/110)
- Tachyarrhythmias / Bradyarrhythmias
- Hypertrophic cardiomyopathy / LVOT
- Unable to physically exert
- High degree AV block

Maximum workout heart rate = 220 – Age

Stress Testing Modalities and Options:

Non-Pharmacologic / Exercise-Physical Stress:

1. Standard Exercise Stress Test with EKG
    a. $$
    b. Interpretable EKG
    c. Contraindications/ Caution:
        i. Preexcitation
        ii. LBBB
        iii. Paced rhythm
        iv. ST-depression > 1mm
            1. So ST-depression 0.5 mm can get a stress test, positive test would be > 1mm ST-depression in 2 contiguous leads
2. Treadmill Exercise Echocardiography
    a. Baseline cardiac function
    b. ↑ Specificity, ↓ Sensitivity
    c. Dyspnea
3. Bicycle Supine Exercise Echocardiography
    a. Double product / Pressure rate product = HR x SBP
        i. Hemodynamic response
        > 30,000 – High; 25-30k high intermediate; 20-25k Intermediate; Low intermediate 15-20k; Low 10-15k
4. Exercise Myocardial Perfusion Imaging Test
    a. $$$
    b. ↑↑ Sensitivity, comparative little less specificity
    c. Comparison of rest and stress images

Pharmacologic / Chemical Stress:

Avoid adenosine, regadenoson, or dipyridamole in patients with active bronchospasm (can be used w/ caution in COPD). Dobutamine is an option for these folks.

5. Dobutamine Stress Echocardiography
    a. 5-40 mcg doing
6. Myocardial perfusion imaging, SPECT with vasodilators (dobutamine, regadenoson)
    a. $$$
    b. Avoid caffeine 12hrs
    c. Tracers: Technetium (99mTc) sestamibi
7. Myocardial perfusion imaging, PET
    a. $$$$
    b. Superior diagnostic accuracy
    c. Avoid caffeine 6hrs
    d. Tracers: Rubidium-82, Nitrogen-12-Ammonia

Non-Pharmacologic / Non-Exercise:

8. Cardiac CT – Calcium artery scoring
9. Cardiac MR

Test	N	Sensitivity	Specificity
Exercise treadmill test	24,074	68	77
Exercise echocardiography	2,637	85	77
Exercise nuclear MPI	2,360	**88**	70
Dobutamine echocardiography	6,881	81	82
Dobutamine nuclear MPI	1,359	84	79
Vasodilator nuclear MPI	4,582	**89**	77

Sensitivity - Negative test rules on obstructive CAD
Specificity – Positive test rule in obstructive CAD

Accuracy of stress testing for detecting obstructive coronary artery disease as defined by quantitative coronary angiography [17].

Myocardial Perfusion Imaging Test (MPI) / Nuclear Stress Test

- Non-invasive testing
- Blood flow i.e. perfusion through heart muscle visualized with use of radiotracer and γ-radiation.
- 2 Techniques
  - SPECT (Single Photon Emission Computed Tomography)
  - PET (Positron Emission Tomography)
- Vasodilators: Adenosine (SE: bronchospasms), Dipyridamole (SE: bronchospasms), Regadenoson (Lexiscan), Dobutamine
  - Abnormal coronary arteries will have attenuated vasodilation, i.e. reduced flow / uptake and thereby identifying ischemic foci.
- Radiotracers: Thallium, Technetium (99mTc-sestamibi)
  - MIBI – Technetium-99m (99mTc) labeled methoxy-isobutyl- isonitrile, aka 99mTc-sestamibi. Trade Name: Cardiolite
    - $T_{½}$ - 6 hrs
    - Liphophilic molecule passes myocytes passively
    - Higher photon energy
    - Minimal redistribution
    - Benefits: Greater protocol flexibility due to lack of redistribution
  - Thallium
    - $T_{½}$ - 73 hrs
    - Potassium analogue enters normal myocytes
    - Low photon energy
    - Redistributes
    - Benefits: Better for detecting myocardial viability (hibernating myocardium) because it redistributes

Terms that reports typically include:

- Fixed defect w/ wall motion abnormalities → Infarct
- Fixed defect w/ normal wall motion → Probable artifact / Breast tissue – anterior, Diaphragm - inferior
- Reversible defect → Ischemia
- Partially reversible → Infarct with surrounding ischemia

Whether medications should be held prior to stress testing? Is so what medications

- Depends on the clinical question being inquired. Examples below:
  - If you're making a new diagnosis, might stop the below mentioned medications.
  - If you're assessing whether symptoms are controlled on current medications, then continue the medications.

Typically Held Medications Class 12-18 hrs prior unless otherwise stated:

- BB, Nitrates including patches, paste, isosorbide mononitrate or isosorbide dinitrate, Dipyridamole (48 hrs prior to test)
- NPO 4-6 hrs and avoid caffeine 12 hrs prior

So, at Maine Medical Center when you hear the term R-MIBI it's a pharmacological myocardial perfusion imaging nuclear stress test, SPECT (Single-photon emission computed tomography) study, involving radiotracer 99mTc-sestaMIBI with vasodilator Regadenoson (Lexiscan).

Hemodynamics:

Normal Hemodynamics:

Source: Cardiology Attendings and link below, [18, 19]

Mixed venous saturation (SvO2)	60 - 80%	RULE of 5s
RA pressure / Central Venous Pressure	2 - 6 mmHg	5
RV systolic pressure RV diastolic pressure	15 - 30 mmHg 2 - 8 mmHg	15 5
PA systolic pressure PA diastolic pressure	15 - 30 mmHg 8 - 15mmHg	20 10
PA occlusion pressure (PCWP)/ LA pressure	6 - 12 mmHg	10
Cardiac index	2.5 - 4.0 L/min/m^2	
Systemic vascular resistance	800 - 1200 dyn·s/cm^5	
Pulmonary vascular resistance	< 250 dyn·s/cm^5	

http://ht.edwards.com/scin/edwards/it/sitecollectionimages/edwards/products/presep/ar04313hemodynpocketcard.pdf

Typical Pattern:

i. Right heart failure: ↓ CI, ↑ CVP, ↑ PVR
ii. Left heart failure: ↓ CI, ↑ PCWP, ↑ SVR
iii. Pericardial tamponade: ↑ PCWP, ↑ SVR, CVP = PCWP (Diastolic pressure equalization)
iv. Hypovolemia: ↓ CI, ↓ CVP, ↓ PCWP, ↑ SVR
v. Cardiogenic: ↓ CI, ↑ CVP, ↑ PCWP, ↑ SVR
vi. Sepsis (Distributive): ↑ CI, ↓ CVP, ↓ PCWP, ↓ SVR

Cardiac Index (CI), Central Venous Pressure (CVP), Peripheral Vascular Resistance (PVR), Pulmonary capillary wedge pressure (PCWP), Systemic Vascular Resistance (SVR).

PA catheter complications:

1. Early
   Complication of central line, Arrhythmia, Heart block (6% RBBB – caution if pt already has LBBB), infection, knotting, pulmonary infarction, hypotension, hypoxia, PA rupture, air embolism, valve damage or incompetence
2. Late
   Thrombosis, pulmonary artery rupture (see pulmonary hemorrhage after PAOP measurement, Line sepsis, endocarditis, inability to remove (due to knotting)

WHAT THE FICK?

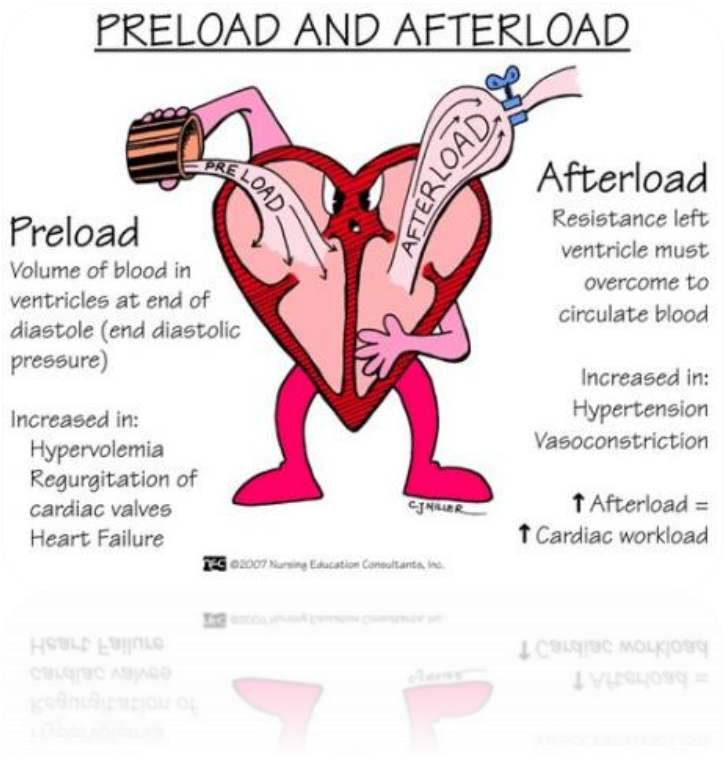
Cardiac Output:

2 methods of assessment: Fick equation and Thermodilution

In patients with acute decompensated heart failure, our goal is to optimize their hemodynamic function. Three principles that we focus on include **preload**, **contractility** and **afterload reduction**.

1. **Preload** assessment is based on physical exam (JVD, crackles, edema) and pulmonary artery (PA) catheter (Swann Ganz) can further guide your volume status (Looking at CVP, PA diastolic or wedge pressure PCWP).
2. **Contractility**: Contractility is assessed by cardiac output, i.e. how much blood flow is delivered to the rest of the body / tissues. Normal cardiac output is roughly 5-8L/min or cardiac index > 2.2. Another way to think about cardiac output standardized to body surface area is cardiac index. Cardiac output, cardiac index and cardiac contractility – all these terms will be used interchangeably in this document.
    i. Right heart catheterization or PA catheter can provide you, this piece of information. If the cardiac index is low (+ clinical findings/ presentation) [20], you can start patients on inotropic support (ex. milrinone, dobutamine, digoxin) to improve their heart contractility. You can titrate your agents for goal CI (cardiac index) > 2.2.
    ii. Consider leaving the PA catheter during hemodynamics optimization period. Although, have a low threshold to discontinue due to potential for grave complications such as pulmonary artery rupture. Always get a daily Chest XR to monitor pulmonary catheter placement position. Low threshold to obtain CXR if poor wave tracing. You'll need cardiology fellow or CVCC attending to adjust / pull back PA catheter.
    iii. 2 methods to access cardiac contractility: Fick methods and Thermodilution.
        ① Fick: Oxygen consumption by tissues can be calculated by amount of blood delivered (= Cardiac output) x amount of oxygen extracted (arterial – venous oxygen). When you rearrange this equation, you can calculate for cardiac output, by assuming a constant for oxygen consumption. I've described this in more detail in next section.
            1. Goto www.bit.ly/medcicu, download "**001. Fick Calculator.xlsx**" or there's Fick Calculator google sheet. This sheet includes a calculator for CI, CO, SVR and **ability to track over time**. Most apps such as Qx calculate, MedCalc won't have this option. It also uses the same VO2 consumption assumption as our MMC Cath lab. Thereby the day team and the night team use the same equation.
            2. Or ONLINE simplistic option: www.bit.ly/fickcalc
        ② Thermodilution: This method uses the principle of temperature variation / dilution over time and transit, remember area-under-the-curve (AUC) / calculus-integral? Here's the simplistic explanation: You inject cold/ room temperature saline in right atrium (using PA catheter ports) and then graph the change in temperature over time (temperature is measured at distal end of PA catheter sitting in the pulmonary artery). Area-under-the-curve is inversely proportional to cardiac output [21].
            1. If cardiac output is low, the area under the curve will be longer
            2. If cardiac output is high, the area under the curve will be short
            3. Recommend reading: Thermodilution pdf (Med C Must Read Article collection), especially seniors who are interested in cardiology, critical care and interested in lines.

3. **Afterload** assessment is based on patient's blood pressure, renal function and systemic vascular resistance. Systemic vascular resistance looks at pressure on the left side of the heart (MAP – Mean arterial pressure) and pressure on the right side of the heart (central venous pressure or right atrial pressure) over amount of blood being delivered by cardiac contraction (Cardiac output).



Picture 1: http://www.newhealthadvisor.com/images/1HT00036/preload%20and%20afterload.jpg

Fick Equation:

Cardiac Output CO (Q = flow) $= \frac{VO2}{(CaO2 - CvO2)x10}$

$= \frac{VO2 (= 135\ x\ BSA)}{Hb\ x\ 1.39\ x\ 10\ (SaO2 - SvO2\ \%)}$

Cardiac index (CI) $= \frac{Cardiac\ Output}{Body\ surface\ area}$

$= \frac{\frac{VO2 (= 135\ x\ BSA)}{Hb\ x\ 1.39\ x\ 10\ (SaO2 - SvO2\ \%)}}{BSA}$

$= \frac{135\ x\ \cancel{BSA}}{\cancel{BSA}\ x\ Hb\ x\ 1.39\ x\ 10\ (SaO2 - SvO2\ \%)}$

$= \frac{135}{\boldsymbol{Hb\ x\ 1.39\ x\ 10\ (SaO2 - SvO2\ \%)}}$

Please see next page for additional information

CO – Cardiac Output (L/min) – Amount of blood pumped by heart in 1 min. 5-8L/min
CI – Cardiac output indexed to patient's body surface area
VO_2 – Oxygen Consumption (mL O_2/min, assumed to be 135 mL O_2/min) x BSA (more information below)
CaO_2 – Oxygen content of arterial blood (mL O_2/100 mL blood)
→ (1.39 x Hemoglobin x O_2 Sat) + (Partial pressure of dissolved O_2 aka. PaO_2 x 0.031)
CvO_2 – Oxygen content of mixed venous blood (mL O_2/100 mL blood) or MvO_2 or SvO_2
BSA – Body surface area (more information below)

Maine Medical Center Equations (Constant 1.39, VO_2 135):

$$Cardiac\ Index = \frac{VO_2\ (135)}{(1.39\ x\ 10\ x\ (SaO_2\% - SvO_2\%))}$$

$$Cardiac\ Output = Cardiac\ Index\ x\ BSA$$

BSA Formula Used w/ this equation on certain websites:

$$BSA = 0.007184\ x\ Height\ (cm)^{0.725}\ x\ Weight\ (kg)^{0.425}$$

LaFarge CG, Miettinen OS Formula (Constant 1.36 w/ VO2 described below) Q-Calculate:

$$VO_2(males, \frac{mL}{\min\ x\ m^2}) = 138.1 - (11.49\ x \ln age) + (0.378\ x\ HeartRate)$$
$$VO_2(females, \frac{mL}{\min\ x\ m^2}) = 138.1 - (17.04\ x \ln age) + (0.378\ x\ HeartRate)$$

$$Cardiac\ Index = \frac{VO_2\ (male/female\ formula)}{(1.36\ x\ 10\ x\ (SaO_2\% - SvO_2\%))}$$

$$Cardiac\ Output = Cardiac\ Index\ x\ BSA$$

BSA Formula Used w/ this equation on certain websites:

$$BSA = \sqrt{\frac{Height\ x\ Weight}{3600}}$$

Other Simple Equations (Constant 1.3, VO_2 135):

$$Cardiac\ Index = \frac{VO_2\ (135)}{(1.3\ x\ 10\ x\ (SaO_2\% - SvO_2\%))}$$

$$Cardiac\ Output = Cardiac\ Index\ x\ BSA$$

What's cardiac index? What's the normal cardiac index (CI) and cardiac output (CO)?
CI Cardiac index - Cardiac output standardize to patient's body surface area.
Normal CI > 2.2; CO ~ 5-8L/min

What is VO2 consumption and how is it calculated?
VO2 – Oxygen consumption, is the difference between inspired and expired O2. It can be measured by measuring inspired and exhaled gas, in a collection bag. Conventionally, it's estimated by resting metabolic consumption of oxygen. For example:

1. **135mL O2 per square meter of BSA per min**
2. 125mL O2 per square meter of BSA per min
3. 3mL of O2 per kg per minute
4. 3.5mL of O2 per kg per minute

Our current MMC Cath lab formula uses 135 (constant) x BSA.
Use caution when assuming the resting metabolic consumption of oxygen in an hypermetabolic state ex hyperthyroidism.

How do you obtain Mixed venous O_2? Or in other words SvO_2? What's normal?
You need a PA catheter or central venous line to get this piece of information
Normal 65-70%.

Why is PA catheter information more accurate than Central venous line?
Central venous line (IJ/Subclavian) provides you with venous O2 saturation from SVC
PA catheter provides you with data from SVC + IVC + coronary sinus, therefore more accurate representation of oxygen extraction throughout the system.

SvO2 from coronary sinus will be lowest # (most O2 extracted), followed by SVC, and then IVC.

Reasons for Low SvO2 or High SvO2
Low SvO2 = High tissue demand/consumption for O_2 **OR** low O2 delivery to begin with

High SvO2 = Low tissue demand/consumption for O_2 **OR** Increased O2 delivery
Examples: Sepsis (Microvascular shunting), cyanide poisoning, hyperthyroidism, liver disease

How is body surface area calculated?
BSA is calculated using many different formulas, all-dependent on **weight** and **height.**

1) DuBois D, DuBois DF. A formula to estimate the approximate surface area if height and weight be known. Arch Int Med 1916;17:863-71.
Equation: BSA (m2) = 0.007184 x Height(cm)$^{0.725}$ x Weight(kg)$^{0.425}$
This is our current MMC Cath Lab formula

2) Mosteller RD. Simplified calculation of body-surface area. N Engl J Med 1987;317:1098.
Equation: BSA (m2) = SQR RT ([Height(cm) x Weight(kg)]/ 3600)

3) Gehan EA, George SL. Estimation of human body surface area from height and weight. Cancer Chemother Rep 1970;54:225-35.
Equation: BSA (m2) = 0.0235 x Height(cm)$^{0.42246}$ x Weight(kg)$^{0.51456}$

What are the parameters that affect cardiac output/ index, when calculated during Fick method?

→ Hemoglobin, O_2 saturation, Mixed venous O_2 saturation (MvO2 or SvO2) and Oxygen consumption.

→ Relating these terms to clinical scenarios: acute severe blood loss (Hb), hypoxemia/ pulmonary pathology (O_2 saturation), Sepsis (affecting oxygen consumption and Mixed venous return

→ These are some of the medical scenarios were Fick equation data should be interpreted with caution / clinical picture.

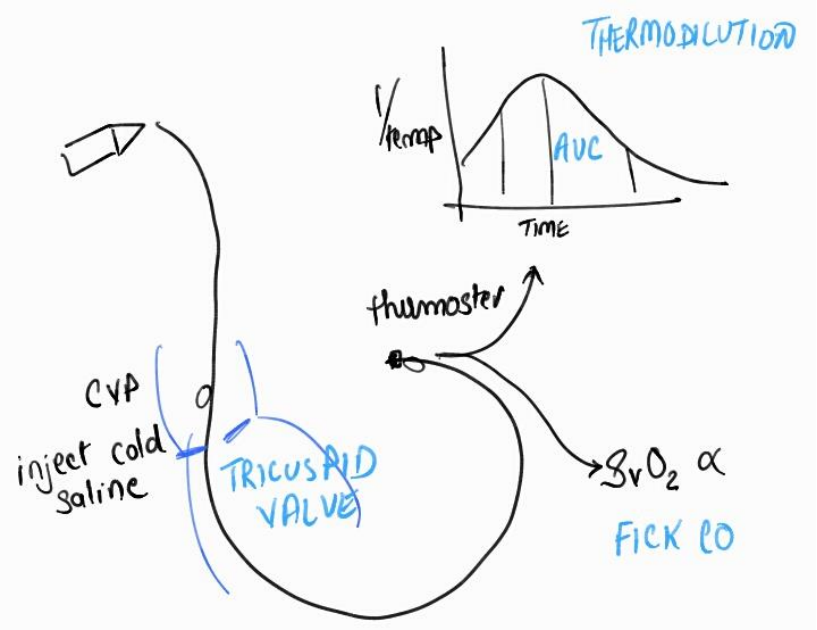
→ Looking at the trend rather than a single # is another way to track patient status / progress

How is oxygen content calculated?

CaO2 = (1.39 - constant x Hemoglobin x SaO2%) + (PaO2 x 0.0031)
SaO2 = Arterial O2 Saturation
PaO2 = partial pressure of dissolved O2

Different constant's have been utilized for O2 content calculation including 1.34, 1.36 or 1.39. Our cath lab currently uses, 1.39 constant. Thereby, current website www.fickcalc.com are based on 1.39 constant.

Thermodilution:



Some of the issues to consider when interpreting thermodilution results:

- Tricuspid regurgitation
- Error with performing the test, e.g. administered saline too fast/slow

Check out this paper: [21].

- There are YouTube videos, demonstrating how cardiac output/index calculations work using thermodilution and Fick method via Swan-Ganz Pulmonary Artery Catheter. Here's an example video: https://www.youtube.com/watch?v=rdyUjTB4vaU

Pulmonary Artery Catheter / Swan Ganz:

- Here's a reference article [22]
- Get Daily Chest XR to document PA catheter position
- Save it as quick preference in Epic EMR to optimize your time!
- Pulmonary-Artery Catheterization: https://www.youtube.com/watch?v=6D_ihQZ2e-M
- Swan Ganz Physiology: https://www.youtube.com/watch?v=7putxZN7ij4

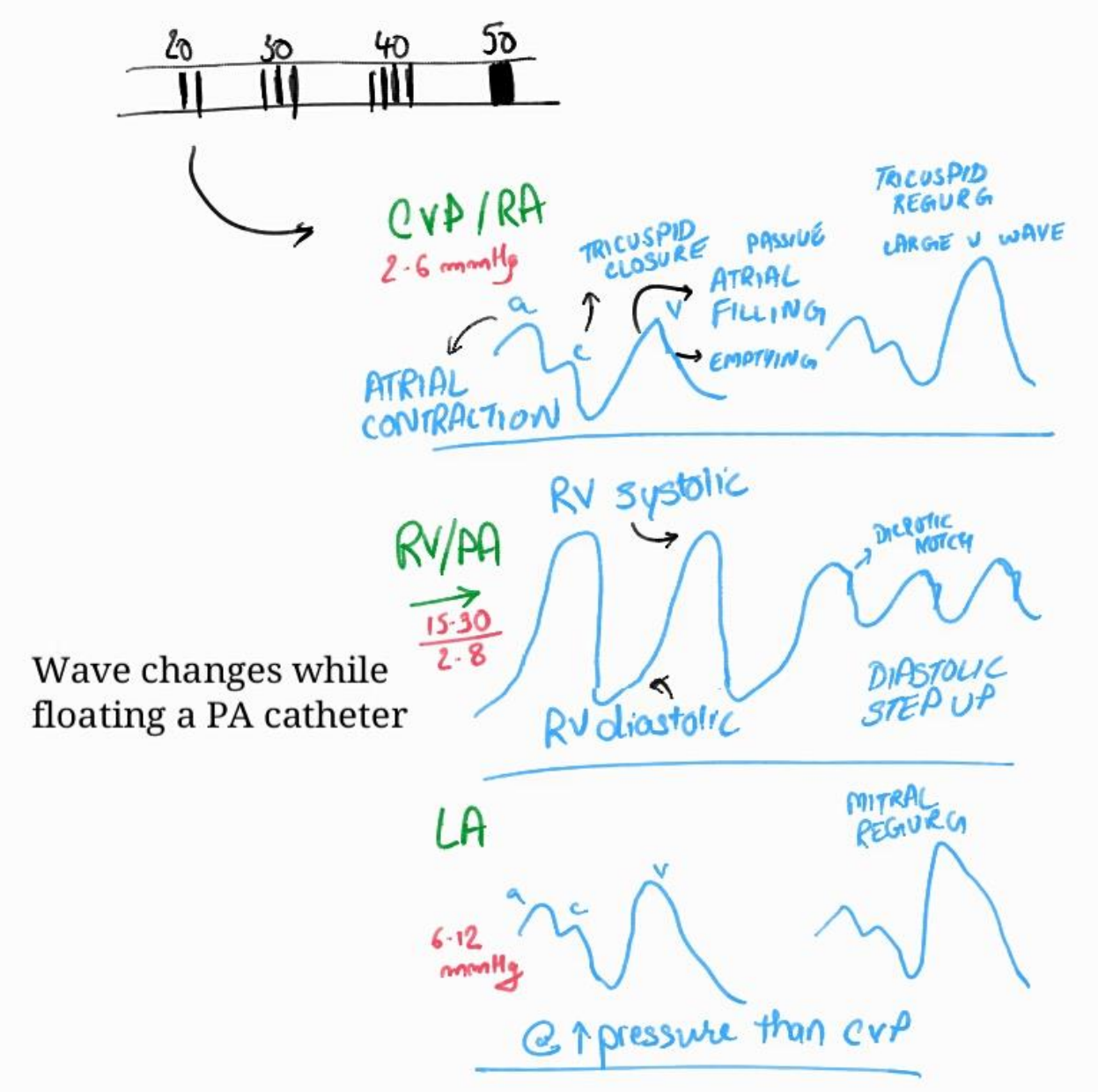
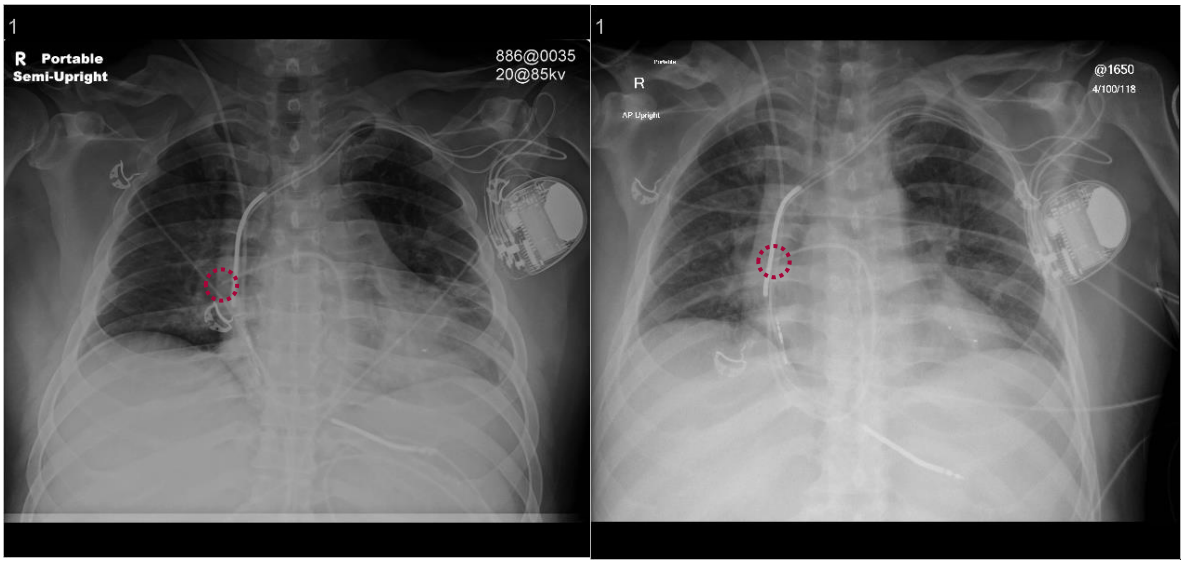
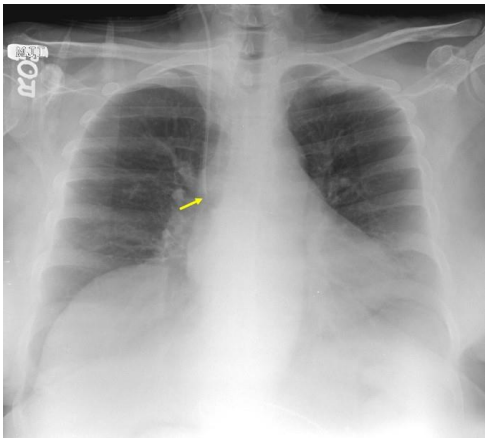


Figure: Reference for senior residents, to assist interns and medical students through the pulmonary artery catheter wave tracings.



PA catheter placement: Tip should be located in the right or left main pulmonary arteries, and not extend beyond the proximal interlobar pulmonary artery. Tip should be within 2cm of hilum. Above our 2 Chest XR pictures of the same patient, left demonstrates PA catheter tip farther than it should be.



Central venous line placement: Tip should be located in the SVC, ideally above the right atrium (cavo-atrial junction), at the level of the right mainstem bronchus.

Resistance:

Resistance = $\frac{\Delta P}{Q}$

Change in pressure across the circulation over flow.

Systemic Vascular Resistance:

Systemic Vascular Resistance (SVR) = $\frac{80\ x\ (MAP - CVP)}{CO}$

Normal: ~800-1200 dyn·s/cm^5

MAP – Mean Arterial Pressure
CVP – Central Venous Pressure
CO – Cardiac Output

Pulmonary Vascular Resistance:

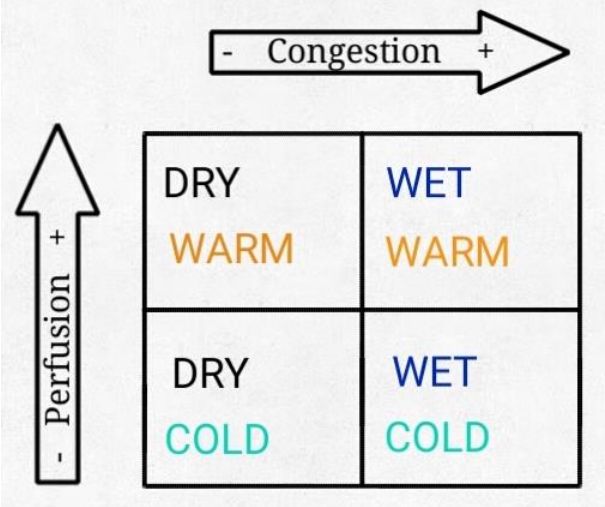
Pulmonary Vascular Resistance (SVR) = $\frac{80\ x\ (Mean\ PA\ pressure\ - PCWP/LVDEP)}{CO}$

Normal: < 250 dyn·s/cm^5
Mean PA pressure – Mean pulmonary artery pressure
PCWP – Pulmonary Capillary Wedge Pressure
LVEDP – Left ventricular end diastolic pressure

Heart Failure (HF)

Many types: Left sided HF, Right sided HF, Systolic HF (HFrEF), Diastolic HF (HFpEF)
Here's, the condensed version of clinical assessment.

Signs of Low Perfusion:
Cool extremities
Low urine output
Pre renal azotemia
Inadequate response to IV diuretic
Altered mental status



Signs of Congestion:
Jugular Venous Distention
Hepatojugular reflux
Peripheral Edema
S3
Dyspnea on Exertion
Orthopnea / Paroxysmal nocturnal dyspnea
Crackles / Rales
Weight Gain

In decompensated state (wet x warm/cold box), diuresis is key!

NYHA Classes / ACC Stages:

NEW YORK HEART ASSOCIATION (NYHA) CLASSES[1]

NYHA class I	NYHA class II	NYHA class III	NYHA class IV
• No limitation on physical activity • No overt symptoms	• Slight limitation on physical activities • Comfortable at rest, but ordinary physical activity causes symptoms of heart failure	• Marked limitation on physical activities • Comfortable at rest, but less than ordinary activity causes symptoms of heart failure	• Inability to carry on any activity without symptoms • Presence of symptoms even at rest

AMERICAN COLLEGE OF CARDIOLOGY AND AMERICAN HEART ASSOCIATION (ACC/AHA) STAGES[1]

AT RISK FOR HEART FAILURE		HEART FAILURE	
Stage A	Stage B	Stage C	Stage D
At high risk for heart failure but without structural changes or symptoms	Structural heart disease but without signs or symptoms of heart failure	Structural heart disease with prior or current symptoms of heart failure	Refractory heart failure including specialized interventions

NYHA Class / ACC Stage and management are well described in Heart Failure Guideline. [23, 24]

Guideline Directed Medical Therapy for Heart Failure with reduced Ejection Fraction (GDMT for HFrEF) / Systolic Heart Failure

In your notes: Consider documenting etiology of heart failure, last echocardiogram, and below mentioned medical therapy breakdown.

Etiology: Ischemic or Non-Ischemic Cardiomyopathy (coronary angiography evaluation or Myocardial perfusion scan required). Last echocardiogram demonstrated EF of *** % (brief pertinent positive echo findings).

You need to know the underlying cause behind heart failure to prevent exacerbation or understand the prognostic outcomes. When it comes to management, understand 3 components to optimization of heart failure, 1st what's coming into the heart = preload (Diuresis to optimize the Frank-Starling curve), 2nd heart's strength = contractility (BB to allow more filling or inotropes if acute decompensation and the heart needs a kick) and 3rd how much force you're working against = Afterload (Arterial BP reduction) [24-28].

- Guideline directed medical therapy for HFrEF (ACC 2013-2016 Guidelines):
    - Afterload reduction / Renin angiotensin aldosterone system inhibition
        - ACEi – Angiotensin converting enzyme inhibitors
        - ARBs – Angiotensin receptor blockers
        - ARNi – Angiotensin Receptor-Neprilysin Inhibitors (Ex. Entresto sacubitril/valsartan)
        - Arteriovenous dilator – Hydralazine and/or isosorbide dinitrate (TID) or mononitrate (qD)
            - Use hydralazine-nitrates (For patient's that can't tolerated ACEi/ARB or African Americans, NYHA Class III-IV)
        - Severe Decompensation consider short term: IV Nitroprusside or IV clevidipine
        - Systemic Vascular Resistance (SVR) goal: ~800-1100 dyn·s/cm^5)
            - Titrate these medications to maximum tolerated dose, as blood pressure allow; studies have document better benefit with higher dose of medications
    - Mineralocorticoid receptor antagonist for reverse LV remodeling
        - Pre-Req: NYHA Class II-IV, ~eCr > 30mL/min and K+ < 5 mEq/dL
        - Spironolactone 12.5-25mg/d, max dose 50mg/d
        - Eplerenone 25mg/d titrate to 50mg/d in 4 weeks
    - Preload Reduction w/ diuresis (NYHA Class II-IV)
        - Loops: Furosemide PO / IV push / gtt, Bumetanide PO / IV push / gtt, Torsemide PO
            - Bumetanide gtt at high doses can cause allodynia
            - Furosemide works for 6 hrs and albumin dependent
            - Torsemide is longer acting (12 hrs)
        - Thiazide additions: Metolazone PO, Chlorthalidone IV
    - Reduce Catecholamine Stimulation with BB (metoprolol succinate, carvedilol or bisoprolol)
        - Avoid during acute decompensation
    - Positive Inotropic support:
        - PO Digoxin

            - Reduces hospitalization rate, does not affect mortality
            - Narrow therapeutic index
            - Monitor renal function, trough digoxin levels
        - IV milrinone (provides a little peripheral vasodilation) → these are usually titrated to goal cardiac index > 2.2
            - Usually starting dose: 0.250 or 0.375 mcg/kg/min
            - Uptitrated by: 0.125 mcg/kg/min
            - Max: 0.5-0.75 mcg/kg/min
            - **Renally cleared caution use in renal injury/failure pts**
        - IV dobutamine → these are usually titrated to goal cardiac index > 2.2
            - Usually starting dose: 2.5-5 mcg/kg/min
            - Uptitrated by: 1 mcg/kg/min
            - Max: 20 mcg/kg/min
        - Home inotropy support, mortality rate 90% at 1 year
    - IV Iron therapy calculating deficit
        - Ganzoni Equation - Total Iron Deficit = Weight (kg) x (Target Hb – Actual Hb) (g/l) x 2.4 + Iron stores (mg → assume 500mg)
        - http://www.cosmofer.com/product/dose-calculation/dose-calculator.aspx

- Mechanical Support:
    - Devices
        - Cardiac Resynchronization Therapy
        - Implantable Cardiac Defibrillator
        - (Indications listed under "Mechanical Devices II: Pacemaker / ICD / BiV ICD")
    - Ventricular Assist Device / Extracorporeal Membrane Oxygenation
        - Left Ventricular Assist Device (LVAD)
        - Right Ventricular Assist Device (RVAD)
        - BiVentricular Assist Device (BiVAD)
        - Extracorporeal Membrane Oxygenation (ECMO)
        - Impella
    - Heart transplant
- Target (Dry) weight: ***
- Daily weights - preferably standing, Monitor I&Os (make sure ordered in Epic)
- Diet: 2gm Na restrict
- Replete electrolytes PRN (Goal: K ~ 4, Mg ~ 2)

My (Jay Shah) typical HF documentation format, so I don't miss major therapeutic interventions, it's under Epic SmartPhrase: JSGDMT

#Acute * on Chronic *** Systolic Congestive Heart Failure, NYHA Class ***, ACC Stage *** - Warm/cold *** & Dry/Wet *** Box**
-Guideline directed medical therapy for HFrEF
    -Afterload reduction /RAAS inhibition: ***
    -Mineralocorticoid receptor antagonist: ***
    -Preload/Diuretic(s): ***
    -Reduce Catecholamine Stimulation/BB: ***
    -Positive Inotrope: ***
-Target weight: ***
-Daily weights - preferably standing, Monitor I&Os
-Diet: 2gm Na restrict
-Replete electrolytes PRN (Goal: K ~ 4, Mg ~ 2)

Cardiac Transplantation

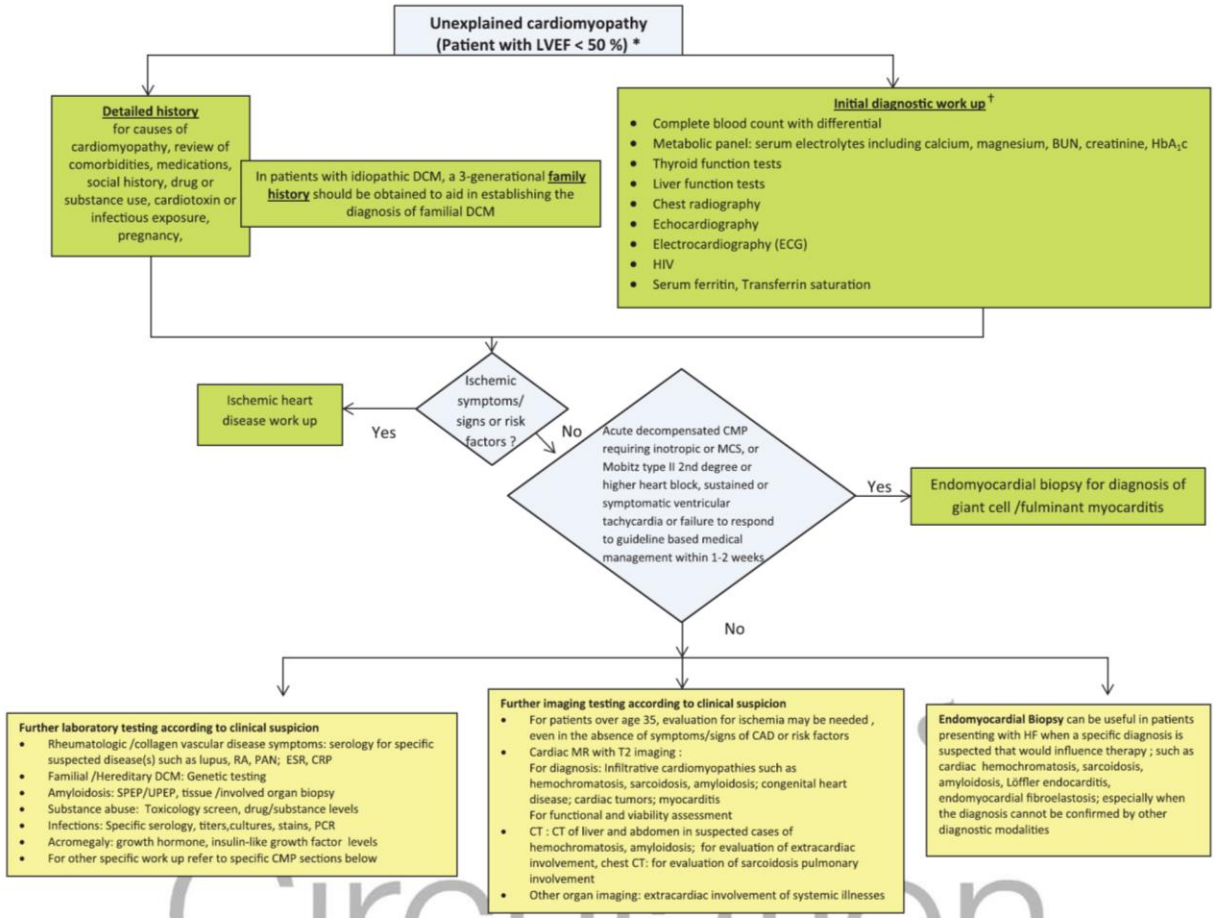
Key Points

Post Cardiac Transplantation

1. Common causes of dyspnea
    a. Rejection
        i. Endomyocardial biopsy for evaluation
    b. Cardiac allograft vasculopathy
        i. Stats:
            1. 50% incidence by 5 years, most common problem year 1 after transplant
            2. Avg HR 90-110
        ii. These patients are denervated and thereby no classic anginal pain symptoms
        iii. Next step: Coronary angiography
        iv. Dobutamine stress echocardiography would be a reasonable option in lower-risk patients

Unexplained New Cardiomyopathy Work-Up:

- CBC, BMP, Mg, Ca, LFT, A1c, TSH, CXR, ECG, Echocardiogram (i.e. look for treatable causes)
- HIV, Fasting iron studies (Fe, TIBC, Ferritin)
    - Hemochromatosis work up: transferrin saturation > 55% in men, 45% in women; HFE gene analysis – done every fortnight on Thursdays at MMC)
- If ischemic signs or symptoms:
    - Troponins, Stress Echocardiogram, NM Myocardial Perfusion Testing, Consider coronary angiogram, etc.
- Situational Testing per Clinical Scenario:
    - Amyloidosis: SPEP, UPEP, light chains
    - Rheumatologic: ESR, CRP, ?RF
    - Substance use disorder: Alcohol, Drug tox screen
    - Acromegaly: IGF-1 (Insulin like growth factor), Growth hormone
    - Infection
    - Familial: Genetic testing but I'd rec'd ref to cardiologist specializing in genetics.
        - Maine Health Cardiology / Dr. Jennifer Monti
    - Cardiac MR consideration
    - Endomyocardial biopsy consideration



Extracted from AHA Paper Current Diagnostic and Treatment Strategies for Specific Dilated Cardiomyopathies [29]:

Cardiomyopathy and MyoPericardial Disease

Hypertrophic Cardiomyopathy

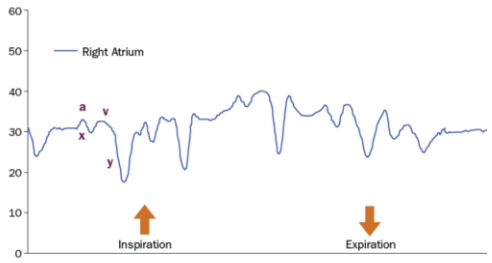
- Screening
    - < 12 years: optional except competitive athlete, symptoms, LVH, family hx of malignant ventricular tachyarrhythmia
    - 12-18 years: q12-18months
    - 18-21 years: Symptoms onset / q5y
- Hold off diuretics in HCM pts
- ICD recommended for ≥ risk factors for SCD (Sudden cardiac death)
    - Massive myocardial hypertrophy (wall thickness ≥30 mm)
    - Previous cardiac arrest due to ventricular arrhythmia
    - Blunted blood pressure response or hypotension during exercise
    - Unexplained syncope
    - NSVT on ambulatory electrocardiography
    - FH of SCD due to HCM
- Rate of ICD discharge:
    - Secondary prevention can be 11%/year
    - Primary prevention can be 4%/year

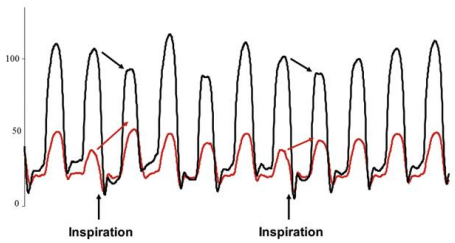
Restrictive vs Constrictive Pericarditis

http://www.learnonly.com/2012/03/constrictive-pericarditis-cp.html

	CONSTRICTIVE PERICARDITIS	RESTRICTIVE CARDIOMYOPATHY
Paradoxical Pulse	⅓ of the patients	Absent
JVP	Prominent Y descent	
Equalization of left and right heart filling pressures	Yes / √Sign	No / Variable
Respiratory variation of left and right heart	Discordance (Ventricular interdependence)	Concordance
Filling Pressure > 25 mmHg	Rare	Present
PA Sys Pressure > 60 mmHg	No	Yes
Atrial Size	Possible increase in LA size	BiAtrial enlargement
Ventricular Wall Thickness	Normal	Increased
Pericardial Thickness	Increased	Normal
Echocardiogram Data: *Septal bounce* *Medial E' 8 ≥ cm/s (higher)* *Hepatic vein diastolic reversal w/ expiration*	Yes	No

Examples of constrictive pericarditis [30],
1st Figure: RA pressure pressure tracing with prominent y-descent,
2nd Figure: LVEDP and RVEDP equalization of pressure, √sign and discordance with with respirophasic variation due to ventricular interdependence.





Pericardial Disease

Acute Pericarditis

- Treatment:
    - Aspirin high-dose (esp in Acute MI situation)
    - NSAIDs
    - Colchicine (0.5-1.2 mg/d)
        - GI side effect, liver toxicity, bone marrow suppression

Constrictive Pericarditis

- See above
- Can present as liver dysfunction / chronic hepatic congestion and hepatopathy
- Additional diagnosis can be confirmed w/ cardiac catheterization
- Transient vs Chronic
    - During initial diagnosis chronicity cannot be determined, therefore try aspirin/NSAIDs/colchicine rather than jump to pericardiectomy

Effusive Constrictive Pericarditis

- Symptoms: Dyspnea, ascites, weight gain/loss, peripheral edema, chest pain/pressure
- Pericardial effusion ± tamponade
    - Despite drainage of pericardial effusion, normalization of intrapericardial pressure, persistent elevation of RA pressure
- Hemodynamics: Diminished y-descent (tamponade)
- Visceral pericardium is affected!
- Tx: Visceral pericardiectomy in certain cases

Cardiotoxicity due to Chemotherapy

- Anthracyclines such as doxorubicin, daunorubicin, can cause cardiotoxicity. Dose dependent.
    - Prevalence:
        - 1% - 400mg/m2
        - 26% - 550 mg/m2
- Meds for CVD protection: ACEi (1st), consider BB (carvedilol)

Maine Medical Center Shock Team

A-HF = Advanced Heart Failure

Shock A - Moderate or severe cariogenic shock resistant to initial therapy

Consult: Advanced-Heart Failure Attending (A-HF) in-person consult
Players Involved: A-HF attending, Med C Attending (secondary role).
Other players involved if pathway activated: The A-HF physician then activates the shock international team which will include interventional cardiologist, CardioVascular Critical Care Attending (CVCC), CICU/On Call Fellow, Cardiac Access Coordinator (CAC), CICU RN for patients best treated with percutaneous revascularization or support options.

In the case of Impella placement the imaging doctor of the day (or on call will be asked to confirm the proper placement). If A-HF determines that surgical revascularization, treatment of structural heart disease, or surgically based mechanical circulatory support is required, the A-HF physician activates the shock surgical team which will include: A-HF, CT Surgeon, CVCC, Pump Team, Cardiology Fellow, CAC, CTICU Charge RN, CT Surgery PA, OR Team and unit anger.

Shock B - Impending or mild cariogenic shock

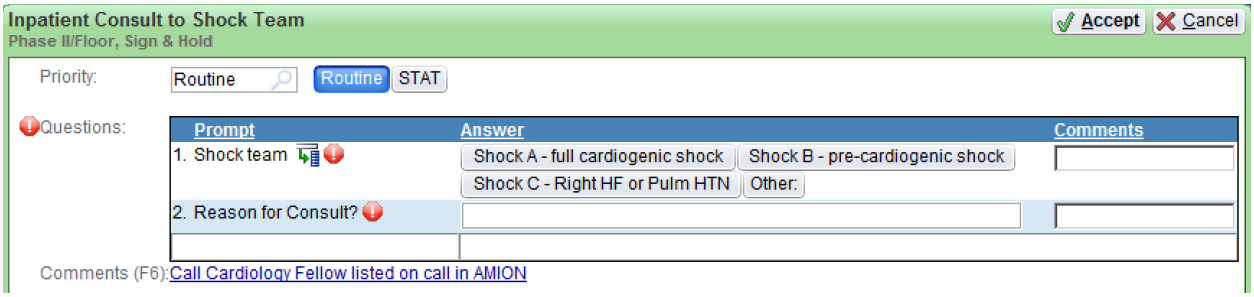
Consult: Attending-HF in-person or phone consult
Players Involved: A-HF attending, Attending cardiologist
The case will be presented. Milestones will be established. If milestones not met in an agreed upon time frame, case will be escalated to Shock A.

Shock C - Right heart failure shock

Payers Involved: Shock C team, pulmonary critical care team.
Patient population: Pulmonary HTN, acute right heart failure (PE, RV infarct).
Under development / Led by Dr. Patricia Lerwick

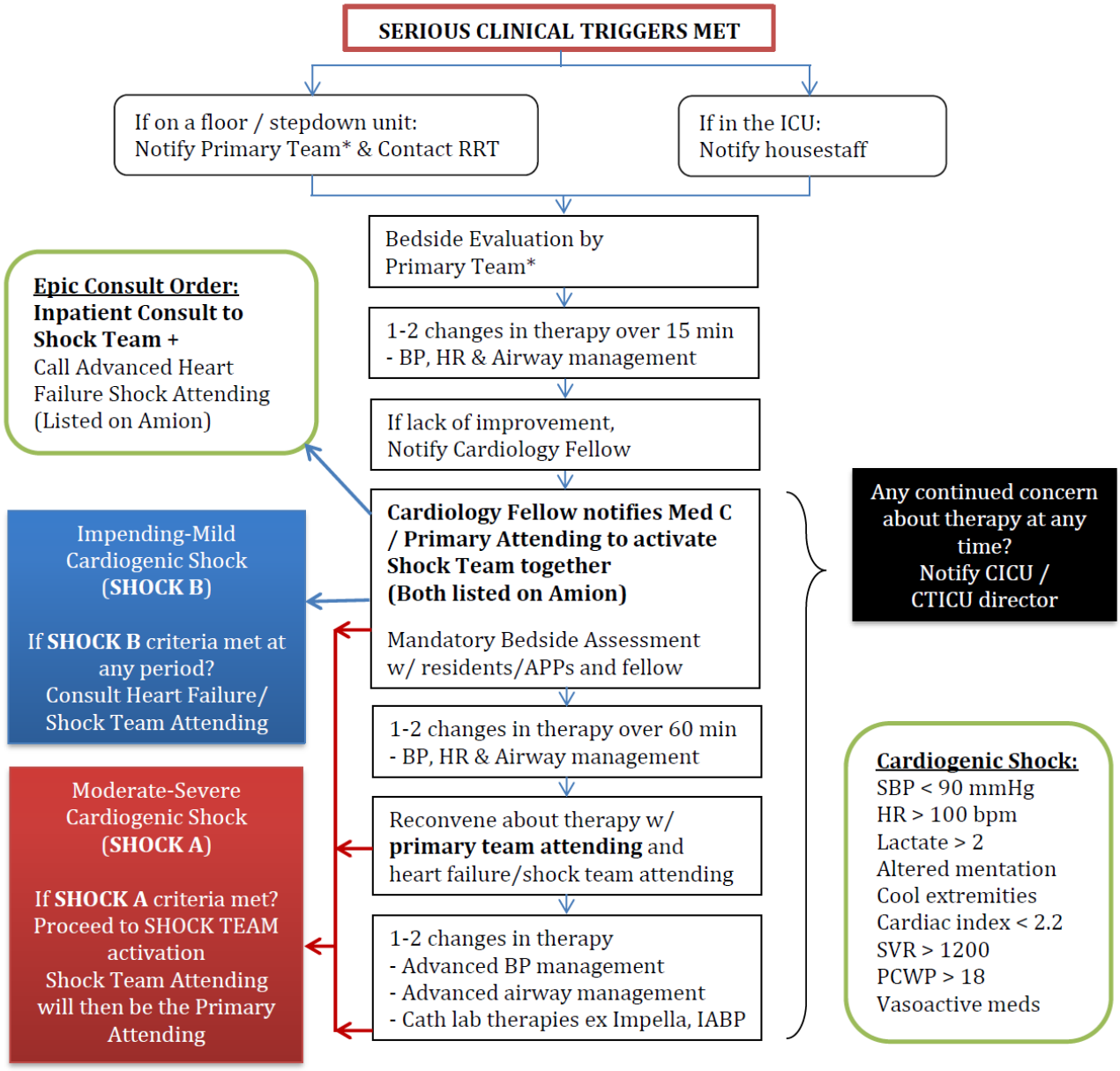
Shock D - Distributive / Sepsis

Payers Involved: Medicine Team / SCU Team
Under development / Led by Dr. Dan Meyers



Visit: www.fickcalc.com (Fick Calculator, Online Articles, Shock protocol)

Maine Medical Center
SHOCK TEAM Protocol

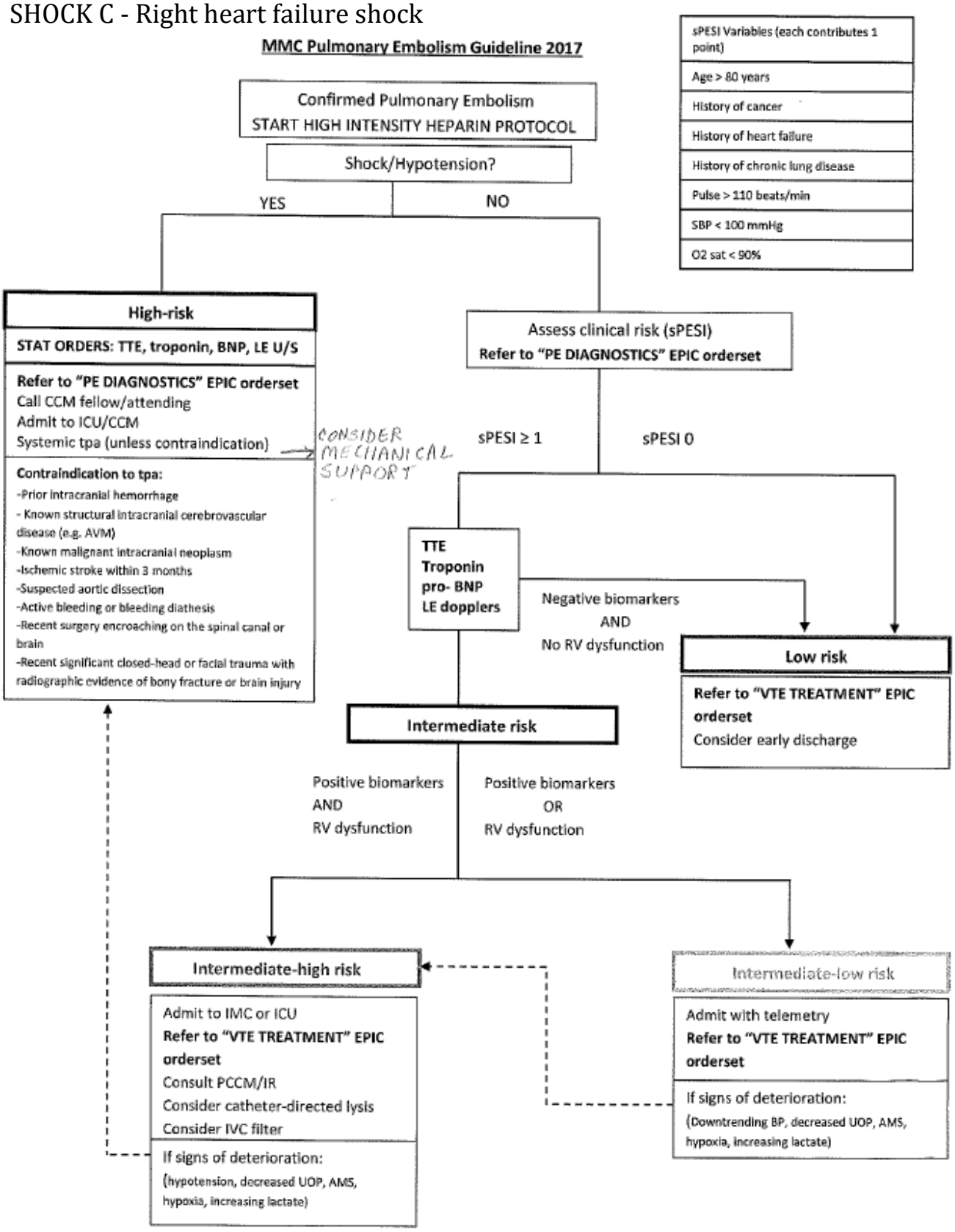


SERIOUS CLINICAL TRIGGERS

- Acidemia – pH < 7.35
- **Acute change in mentation**
- Addition of 2nd IV Pressor
- Brady/Tachycardic - HR ↓/↑ by 40
- Brady/Tachypnea – RR < 8 / RR > 24
- ECG – ST elevations
- Experienced RN "knowing" without rationale
- **Hyper/Hypotensive – BP < 90 / BP > 160**
- Hyper/Hypothermia – rigors, fever
- Hypoxemia – Sat < 90% refractory to additional O2
- **Lactate > 4**
- Pain – Refractory to treatment
- Patient feeling impending doom
- Threatened limb ischemia – Pain, Pallor, Pulselessness, Paraesthesia, Paralysis, Perishing Cold
- **Urine output < 50 cc/hr for 2 hrs**

*Primary Team includes but not limited to Cardiology, Medicine, Surgery APP and resident service

SHOCK C - Right heart failure shock



Indications for catheter directed thrombolysis:

- Massive PE with increased risk of bleeding
- Intermediate risk patients with evidence of clinical deterioration which includes:
    - Downtrending blood pressure
    - Change in mental status
    - Decrease in UOP
    - Increased lactate level
    - Worsening hypoxia

Indication for IVC filter placement:

- Contraindication to anticoagulation
- Recurrent PE despite therapeutic levels of anticoagulation
- Presence of acute DVT in high-risk patients
- Consider in the presence of acute DVT in intermediate risk patients

Indications for surgical embolectomy:

- Hypotension with contraindication to thrombolytics
- Hypotension with failed response to thrombolytics
- Consider in intermediate-risk or massive PE with clot-in-transit +/- PFO

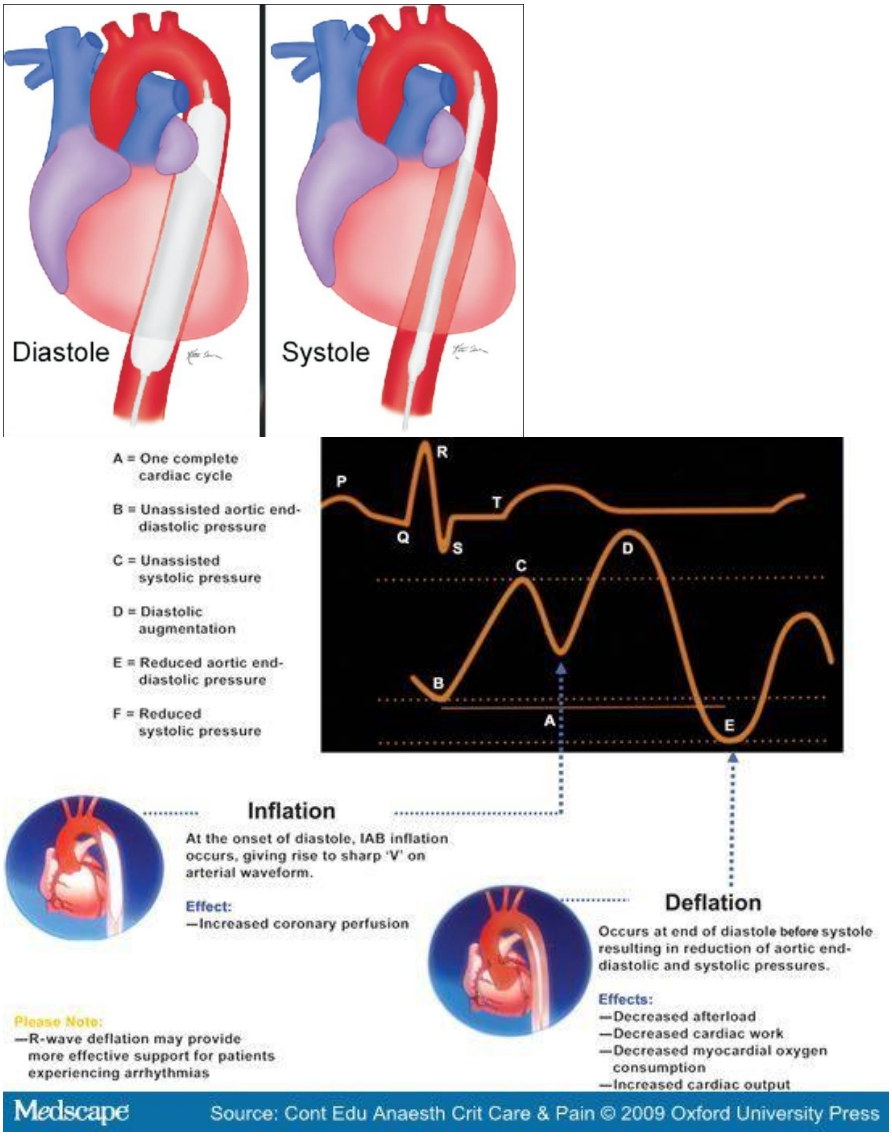
Mechanical Devices Part I – Cardiogenic Shock

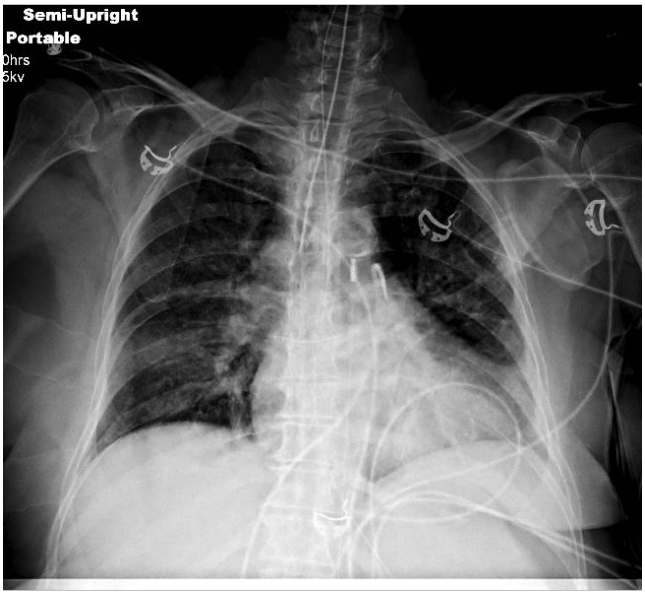
Good resource [26]:

Intra-aortic Balloon Pump

- ↓ LV afterload
- ↑ Coronary blood flow
- Catheter that sits in the aorta, inflates during diastole and deflates during systole
- **Get a daily CXR for these patients, monitor position**

Good q/a and resource: http://lifeinthefastlane.com/cardiovascular-curveball-005/ [31]



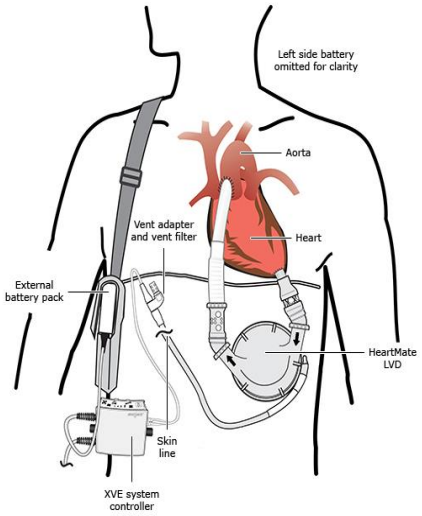


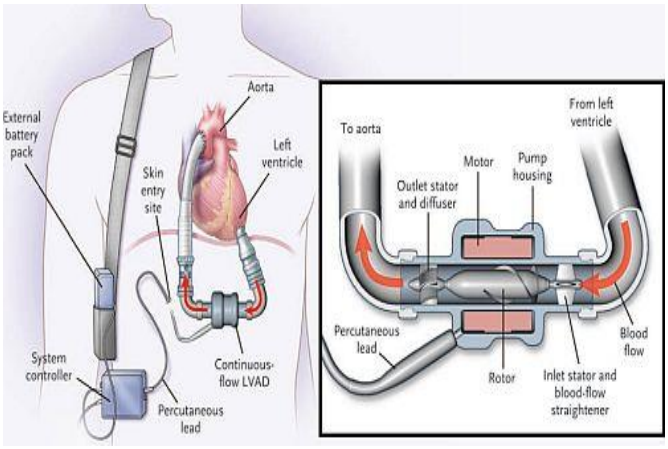
Intra-aortic balloon pump (IABP): Tip should be located in the descending aorta, adjacent to carina OR 2cm distal to the origin of the left subclavian artery. Remember, the balloon moves about 1-2cms during systole / diastole.

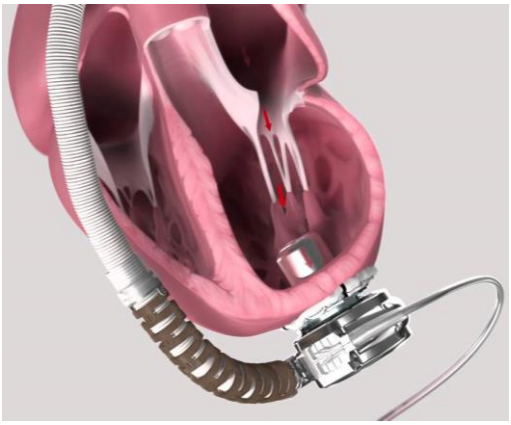
Monitor BMP/ especially renal function, to make sure that the renal artery is not blocked by the balloon.

Ventricular Assist Device (VAD)

- Blood pumps
- Left VAD pumps blood from LV to Aorta
- Right VAD pumps blood from RV to pulmonary artery
- 3 generations:
    - 1st pulsatile flow (HeartMate) (Top Right)
    - 2nd axial pump (HeartMate II) (Bottom Left)
    - 3rd centripetal pump (HeartMate III, VentrAssist, HeartWare) (Bottom Right)
- Some of them are implantable and others external
    - Implantable: Pump inside the body, cable is hooked to control unit, which is then booked to battery







Indications

- Bridge to cardiac transplantation
- Bridge to decision regarding transplant eligibility
- Destination (or permanent) therapy
- Bridge to recovery of heart function.

VAD Emergencies
(very brief overview per Dr. Shao)

- Most patients **do not have a palpable pulse**
- Standard measures for BP are not reliable
- Pulse oximetry not reliable
- Pump failure is incredibly rare
- **NO CPR without contacting VAD cardiologist or surgeon first**
- Check for alarms and confirm driveline is attached

Code 99 (VAD Patient)

- Airway
- Breathing
- Circulation—almost always guaranteed
- Call VAD MD on call (Amion) under cardiology:
    - VAD/Transplant call
- Obtain MAP (normal is 65-90)
- Ask VAD trained nurse if pump is functioning normally
- If MAP<60, give a lot of volume, norepi and vasopressin if needed, place arterial and central line
- Consider urgent neuro work up
- Check STAT INR, CBC, CMP, Bili, LDH, haptoglobin, plasma free hemoglobin

As a senior resident holding code pager, you should not receive VAD pages.
But in case, the code pager does goes off for VAD patient, follow above instructions.

Impella

- Blood from LV through an inlet area near the tip and expels blood from the catheter into the ascending aorta
- Goes from femoral artery up
- 2.5L/min
- Three versions of impella

Indications
- Need for LV support during high risk PCI ≤ 6 hr
- Cardiogenic shock post MI or open heart surgery due to isolated LV failure ≤ 4 days

Echocardiogram
- 3-4.5cms between inlet to outlet
- Ideal 3.5 cms

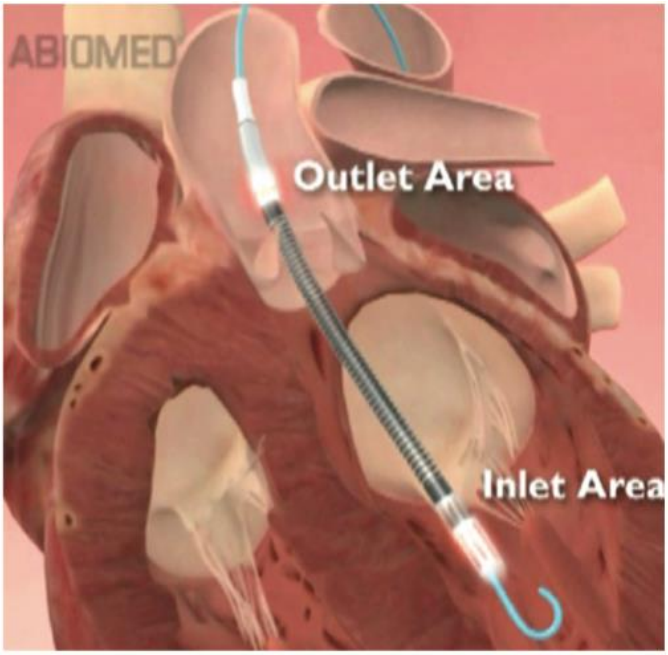
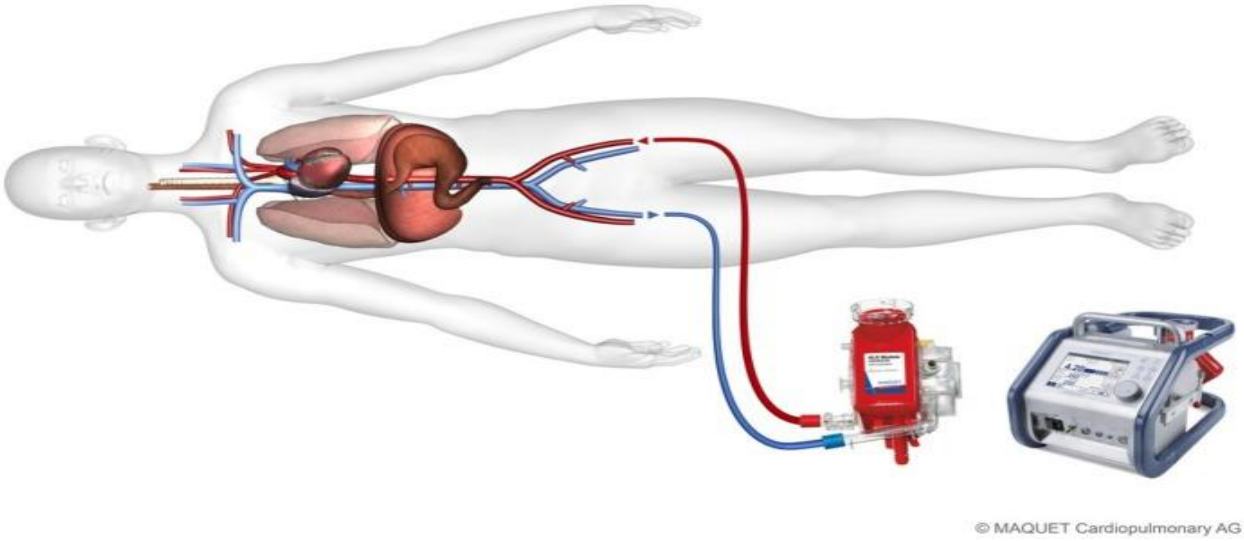


Figure 2. The Impella device: a motor drive in the device rotates at a maximum speed of 51,000 rpm, drawing blood out of the left ventricle through an inlet area and ejecting it into the ascending aorta beyond the end of the pump via the outlet. Courtesy of Abiomed.

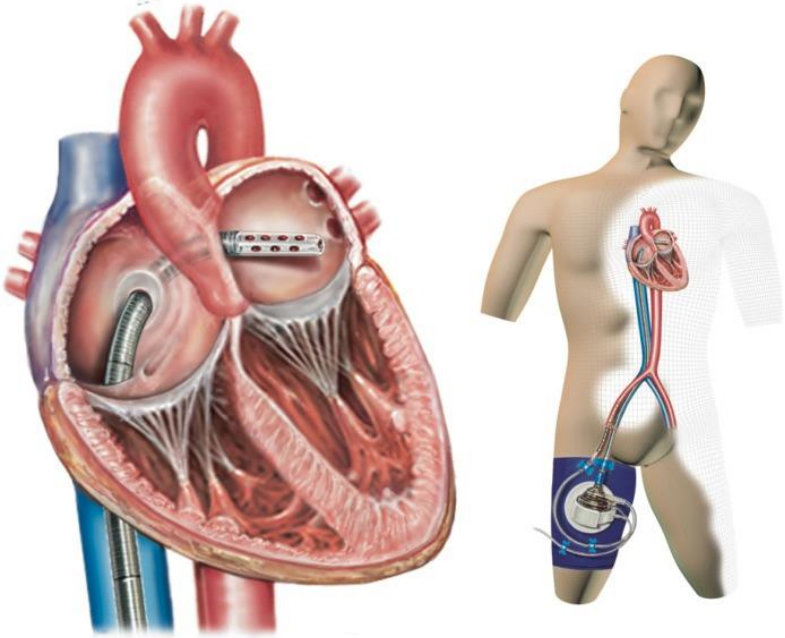
Extracorporeal Membrane Oxygenation (ECMO)

- Blood drained from venous side is oxygenated outside the body
- 2 Types
    - V-A (Veno-arterial)
    - V-V (Veno-venous)



TandemHeart

- Transseptal cannula – takes oxygenated blood from LA to femoral artery
- 5L/min

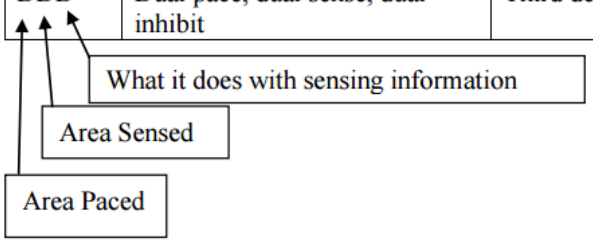


Mechanical Devices Part II: Pacemaker / ICD / BiV ICD

Terminology:

Area paced, area sense, what it does with sensing
Terms: O No, A Atrial, V Ventricular, D Dual, I Inhibit

Code	What is it?	Who gets it?
AOO	Atrial pace, no sense, no inhibitions	Sick sinus syndrome with intact conduction in the operating room with bovie. Ie Cardiac case, in OR, with bovie, with heart rate low from narcotics.
AAI	Atrial pace, atrial sense, inhibited by atrium	Sick sinus syndrome with intact conduction system.
VOO	Ventricular pace, no sense, no inhibit	Third degree heart block in OR with atrial fibrillation. Why atrial fibrillation? Because you can't effectively pace the atrium if it is fibrillating.
VVI	Ventricular pace, ventricular sense, ventricular inhibit	Third degree heart block with atrial fibrillation.
DOO	Dual pace, no sense, no inhibitions	Third degree heart block in OR with bovie.
DVI	Dual pace, ventricular sense, ventricular inhibit	Third degree heart block with supraventricular tachycardias
DDD	Dual pace, dual sense, dual inhibit	Third degree heart block.



http://www.cardiacengineering.com/pacemakers-wallace.pdf

Pacemaker Indications:

1. Bradycardia
   A) Symptomatic bradycardia without reversible cause
   B) Asymptomatic bradycardia with significant pauses (>3 seconds in sinus rhythm) or persistent heart rate <40/min
   C) Atrial fibrillation with >5-second pauses
   D) Asymptomatic complete heart block or Mobitz type 2 second-degree atrioventricular block
   E) Alternating bundle branch block

When you come across advanced heart block, consider possibility of infiltrative heart disease.

Implantable Cardioverter Defibrillator (ICD):

Primary Prevention (Class I):

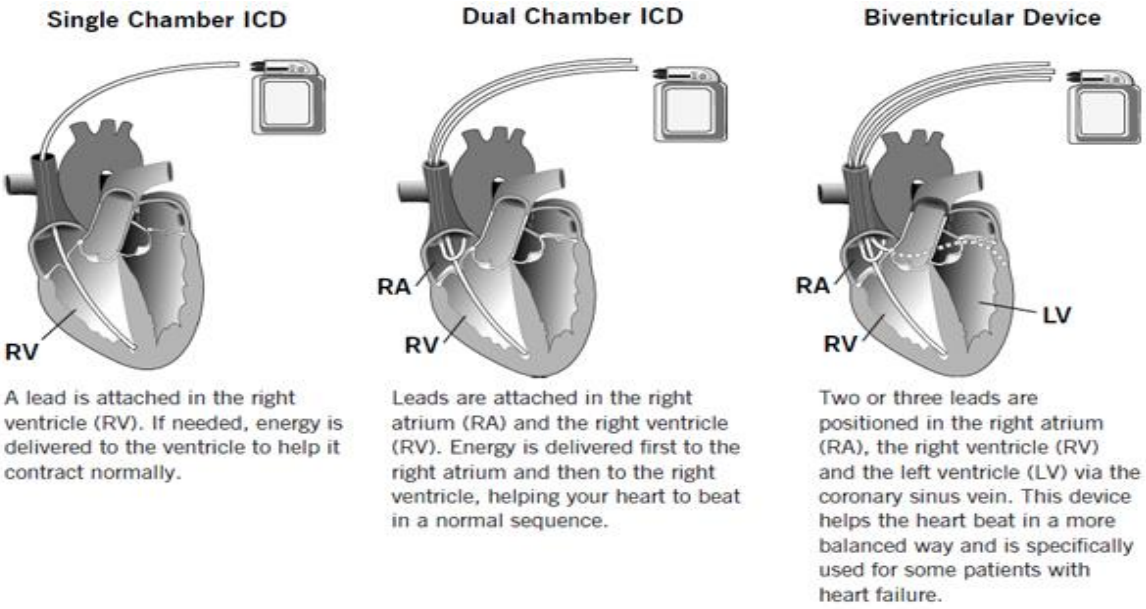
1. MI history, EF < **35%** on GDMT, **NYHA Class II or III**, ≥ 40d from MI, > 3months from revascularization
2. NICMP, EF ≤ **35%** on GDMT, **NYHA Class II or III**, high risk for SCD
3. MI history, EF < **30%** on GDMT, **NYHA Class I**, ≥ 40d from MI, > 3months from revascularization
4. MI history, w/ inducible VF / sustained VT at EPS

Other indications (Class II):
Hypertrophic CMP w/ ≥ 1 major risk factors for SCD, ARVC/D w/ ≥ 1 major risk factors for SCD, NICMP w/ EF ≤ 35% w/ NYHA Class I, Long QT syndrome w/ ≥ 1 major risk factors for SCD, Infiltrative (Cardiac sarcoidosis, giant cell myocarditis, Chagas disease), familial CMP, LV noncompaction, nonhospitalized inidivudals awaiting heart transplantation

Secondary Prevention:
Sudden cardiac arrest due to ventricular arrhythmia

Cardiac Resynchronization Therapy (CRT-P or CRT-D)

1. **NYHA Class III – IV, EF ≤ 35%, GDMT, Ventricular dyssynchrony (LBBB w/ QRSd ≥ 150 msec)**

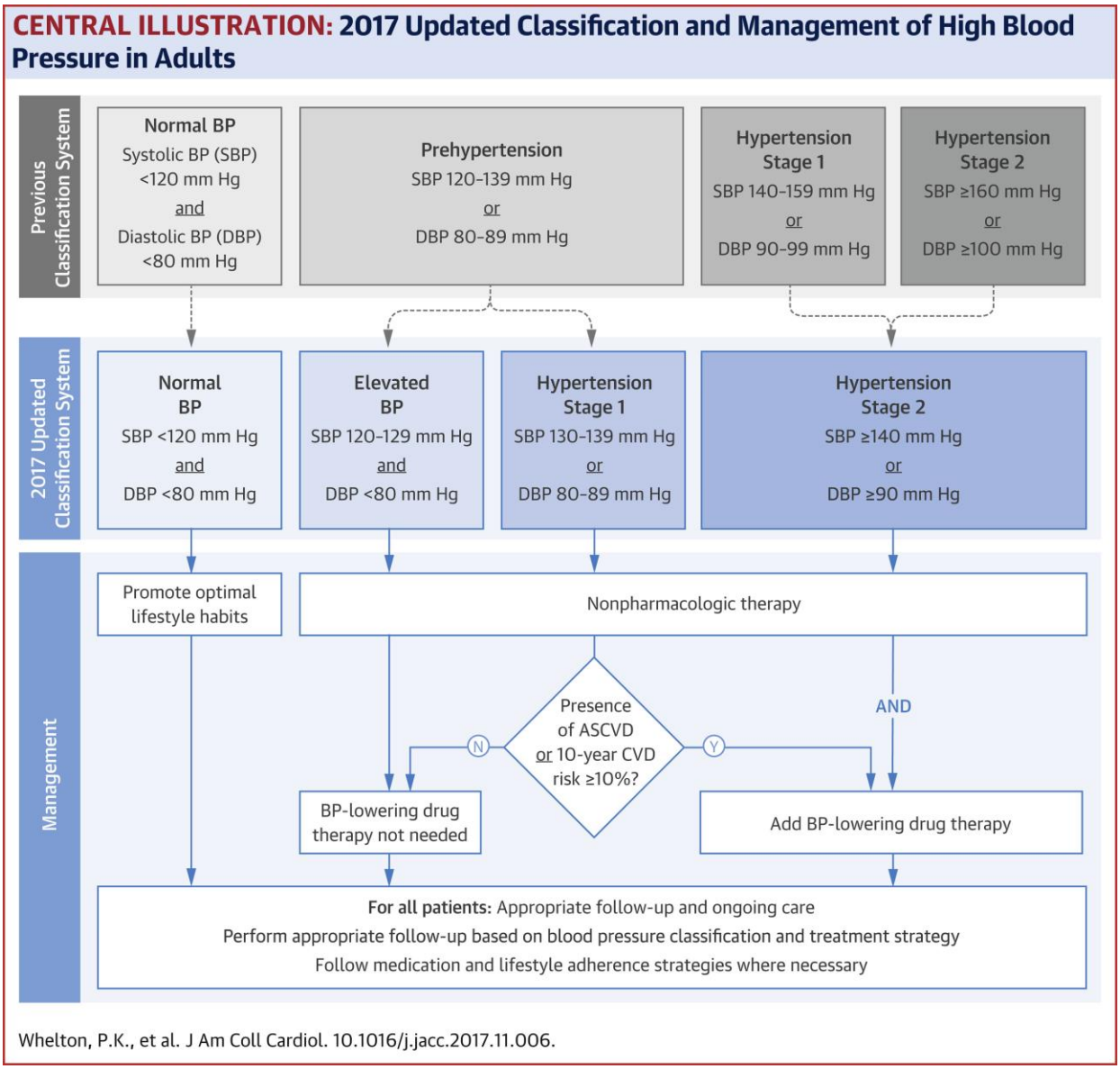


Similar concept when you are thinking about ICD or pacemaker, with regards to lead placement.

Abbreviation: GDMT guideline directed medial therapy, NICMP nonischemic cardiomyopathy, EF ejection fraction, SCD sudden cardiac death, VF ventricular fibrillation, VT ventricular tachycardia, EPS electrophysiologic study, CMP cardiomyopathy. Source: [32, 33]

Hypertension

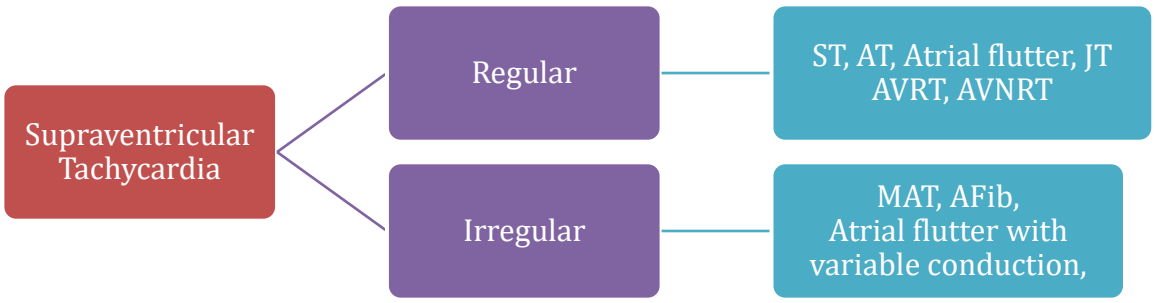
Source [34]:



Tachycardia Management

Narrow Complex Tachycardia

Source [35, 36]:



- Get an EKG (better than tele)
- Hemodynamically Stable (HR, BP, RR, Mentation)
  - NO – Synchronized Cardioversion
  - YES →
    - Supraventricular tachycardia (Regular)
    - Atrial Fibrillation
    - Sinus tachycardia
      - Common type of tachycardia. Identify underlying cause before considering treatment.

Supraventricular tachycardia

- Treatment:
  - Vagal Maneuvers – Modified Valsalva (blow through 10cc syringe upto 40mmHg pressure for 15sec and raise legs 45° for 15 sec), Bear down / push belly against hand on the abdomen, Carotid massage
  - Medications:
    - Adenosine 6mg IV followed by 10cc NS push, raise arm
    - Adenosine 12mg IV followed by 10cc NS push, raise arm
    - OR Adenosine 3mg IV via central line
    - Other options include:
      - BB, CCB, Digoxin

Atrial Fibrillation

✧ Atrial fibrillation / flutter (Irregular)
- Identify reversible cause: Volume overload/too dry (consider CXR), medications side-effect (ex steroids), comorbidities, etc.
- Treatment:
  - Vagal Maneuvers – Modified Valsalva
  - Medications:
    - BB: Metoprolol 5mg IV q5min (Max dose 15mg), flexible range 2.5-10mg IV push
    - CCB: Diltiazem 0.25 mg/kg (1st bolus), 0.35 mg/kg (2nd bolus), next step gtt
    - Digoxin
    - Rhythm control agents:
    - Amiodarone – although make sure patient's on anticoagulation, bc if they are in paroxysmal A-fib, and then they are converted to SR, there is a risk of ischemic stroke
    - Other options:
      - Class Ic: Flecainide, Propafenone
        - Avoid in pts w/ coronary artery disease
      - Class III: Dofetilide, Sotalol, Dronedarone
        - Watch QTc
        - Hemodynamically unstable individuals, TEE/CV (Transesophageal echocardiogram followed by cardioversion)
  - Anticoagulation:
    - Medications:
      - PO Warfarin – Class: Vitamin K antagonist
        - Goal INR 2-3 usually
      - SQ LMWH
      - IV UFH
      - PO Dabigatran – Class: Direct thrombin inhibitor
      - Class Xa inhibitor
        - PO Apixaban 5mg BID or 2.5mg BID
          Reduced dose for 2/3 conditions: Age ≥ 80, sCr ≥ 1.5 mg/dL, weight ≤ 60kg
        - Rivaroxaban w/ food 20mg/d or 15mg/d (for GFR 15-30 mL/min)
        - PO Edoxaban
    - Risk assessment below
      - When you see a patient with atrial fibrillation, it's helpful to document their CHA2DS2VASc [37-39] score along with how you calculated the score
        - Ex: CHADS2VASc 4 (HTN, Age x2, F)
    - Depending upon the situation okay to use triple tx if especially after stent placement
  - Other Advanced Options
    - RFA (Radiofrequency ablation) of AV Node & PPM
    - Pulmonary vein isolation / catheter ablation

    - ✧ SE: Pulmonary vein stenosis
- MAZE
- Modified MAZE
    - ✧ Scarring the tissue using radio frequency ablation or freezing, rendering it unable to conduct signal, and thereby reducing aberrant conduction
- Left atrial appendage closure / WATCHMAN Device placement http://www.bostonscientific.com/en-US/products/laac-system/watchman-device/overview/laac-implant-procedure.html

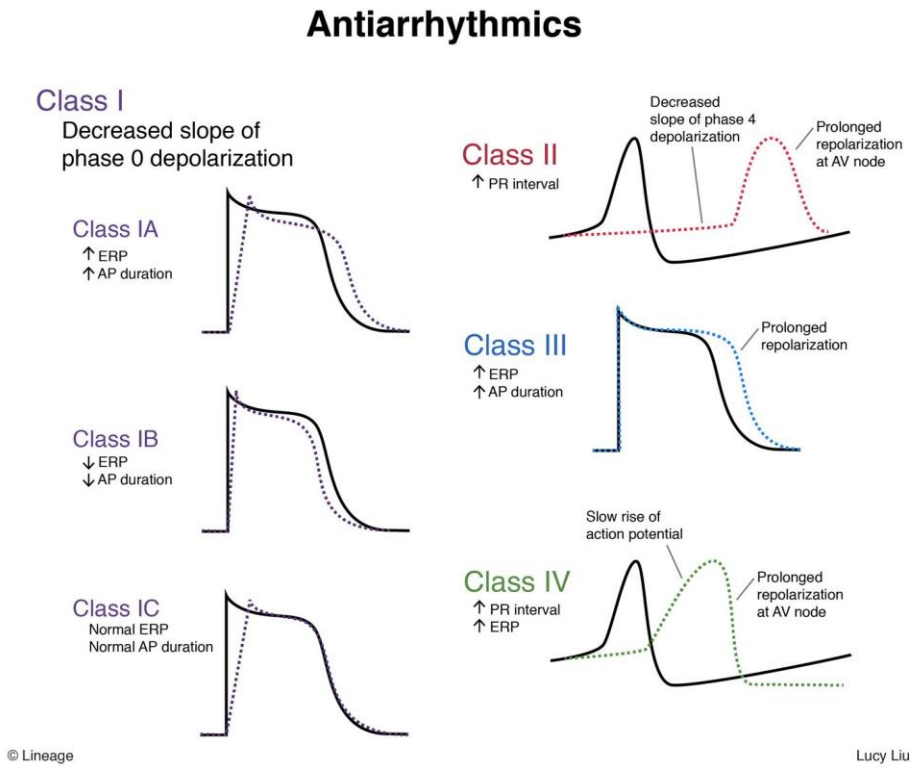
Atrial Fibrillation Risk Assessment Tool
https://www.healthdecision.org/tool.html#/

➔ I recommend using this shared decision-making tool with your patients for improved patient education and making a wise decision. Your patients will be more inclined to be compliant with medications if they understand risks better.

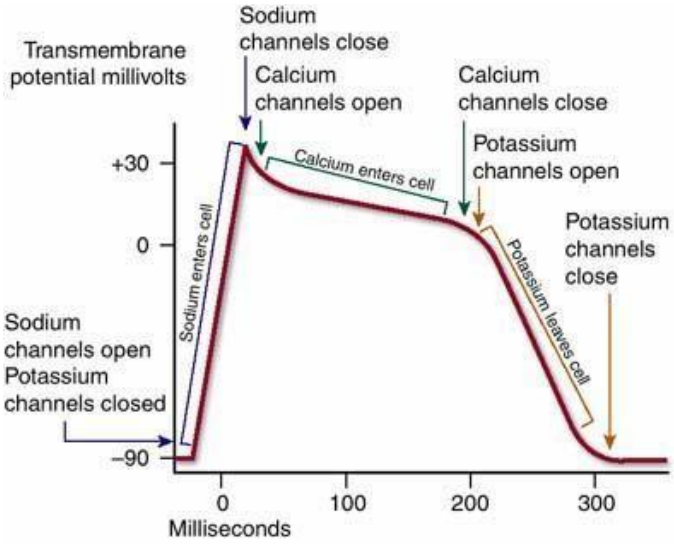
CHADS2VASc	Points	HAS-BLED	Points
C CHF	1	H HTN	1
H HTN	1	A Abn renal fnc Abn liver fnc	1 1
A Age > 65	1	S Stroke	1
D Diabetes	1	B Bleeding	1
S Stroke, TIA,	2	L Labile INR	1
V Vasc (MI hx, PAD, aortic plaque)	1	E Elderly	1
A Age > 75	1	D Drugs/ Alcohol * Drugs – NSAIDs or antiplatelet agent	1
Sex Category Female	1		

CHADS2VASc	Annual Stroke Risk %	HAS-BLED	Bleeds per 100 Pt-years
0	0	0	1.13
1	1.3	1	1.02
2	2.2	2	1.88
3	3.2	3	3.74
4	4.0	4	8.70
5	6.7	5	12.50
6	9.8		
7	9.6		
8	6.7		
9	15.2		

Antiarrhythmic Properties



Source: http://upload.medbullets.com/topic/108097/images/01222017llstep12antiarrhythmics.jpg



Source: https://www.pinterest.com/pin/491596115556053047/

Wide Complex Tachycardia (Is it VT or not?)

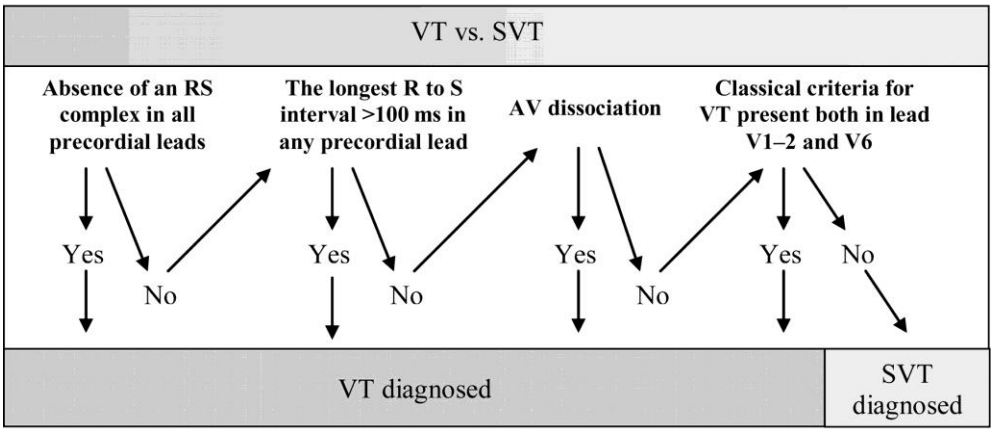
1. Hemodynamically stable
   (a) NO – Shock
   (b) YES
      ① Consider sedation (fent 50mcg/ midazolam 2mg) – shock
      ② Antiarrhythmic agents
         1. Amiodarone 300mg IV push followed by 150mg

Again, when in doubt, shock!
>80% of wide complex tachycardia, is likely VT!

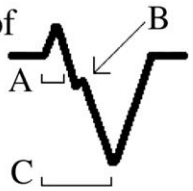
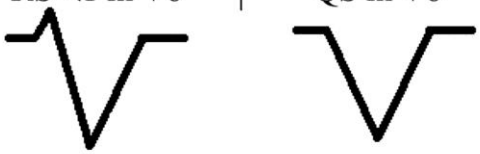
Several features increase the likelihood of Ventricular Tachycardia (Although, beware, things are lot more complicated, and that's why there are cardiologist who specialize in electrophysiology) [40, 41]:

- Absence of typical RBBB or LBBB morphology
- Extreme axis deviation ("northwest axis")
- Very broad complexes (>160ms)
- AV dissociation (P and QRS complexes at different rates)
- Capture beats — occur when the sinoatrial node transiently 'captures' the ventricles, in the midst of AV dissociation, to produce a QRS complex of normal duration.
- Fusion beats —sinus & ventricular beat coincides to produce a hybrid complex.
- Positive or negative concordance throughout the chest leads, i.e. leads V1-6 show entirely positive (R) or entirely negative (QS) complexes, with no RS complexes seen.
- Brugada's sign – The distance from the onset of the QRS complex to the nadir of the S-wave is > 100ms
- Josephson's sign – Notching near the nadir of the S-wave
- RSR' complexes with a taller left rabbit ear. This is the most specific finding in favour of VT. This is in contrast to RBBB, where the right rabbit ear is taller.

Brugada Criteria



Wellens Criteria

Classical, Wellens, criteria favouring VT	
AV dissociation, capture or fusion beats, negative or positive concordance, tachycardia QRS more narrow than sinus QRS	
RBBB configuration	LBBB configuration
QRS width >140 ms, left axis	QRS width >160 ms, right axis
QR, R, RSr' complex in V1  qR in V1 R in V1 Rabbit ear in V1	(A) Initial R in V1 >30 ms (B) Slurring or notching of the downstroke of the S-wave in V1–2 (C) Begin QRS-nadir S-wave >70 ms in V1–2 
RS <1 in V6 QS in V6 	Any Q V6  Any Q in V6

Ventricular tachycardia with fusion beats and capture beats

The QRS complex is wide (grid not shown) and appears as something between the ventricular beats and the normal beats (seen to the right). This is a **fusion beat**.

These beats are presumably supraventricular beats that managed to get through and depolarize the ventricles. They are called **capture beats**. Note that these beats display large Q-waves (which indicates prior myocardial infarction).

Frequent Symptomatic PVCs causing cardiomyopathy – Catheter ablation can be considered

Causes of Ventricular Fibrillation Cardiac Arrest

- Automaticity
- Re-entry
- Triggered Activity

- Structural heart disease
    - Coronary artery disease / ischemia
    - Dilated CMP
    - Hypertrophic CMP
    - Valvular heart disease
    - Infiltrative heart disease (sarcoid, amyloid, hemochromatosis)
    - Familial
        - Arrhythmogenic RV dysplasia
- Electrolyte abnormalities
- Channelopathies
    - Brugada syndrome SCN5a
        - 3 Patterns
        - If symptomatic / syncope, need an ICD
    - Wolff-Parkinson White syndrome
    - Long QT syndrome
        - Congenital
        - Acquired
            - Drugs
            - Electrolyte abnormalities
    - Short QT syndrome
    - Catecholaminergic polymorphic ventricular tachycardia

Valvular Heart Disease

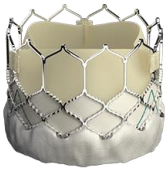
Good Resources [30, 42, 43]:

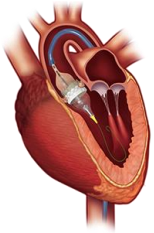
Aortic Valve Disease – Aortic Stenosis / Regurgitation

Aortic Stenosis

Clinical Features, Physical Exam, Diagnostic Criteria, Treatment → Well described in the Pocket Medicine Book. I've included a brief summary.

- Etiology,
  - Calcific, Congenital, Rheumatic heart disease
- Clinical Manifestations
  - Angina, Syncope, Heart Failure, Fatigue, Malaise
- Physical Exam
  - Midsystolic crescendo-decrescendo murmur at RUSB, radiated to *carotids or apex*, increases w/ leg raise (↑ Preload) and decreases with standing / Valsalva (↓ Preload)
  - Audible click after S1 – Bicuspid AV
  - Pulsus parvus et tardus – small and delayed carotid pulse
  - Aortic valve incompetence w/ regurgitation causes diastolic murmur in bicuspid valves
- Diagnostic
  - ECG: LVH< LAE, LBBB, AF
  - CXR: Cardiomegaly, AV calcification, Pulmonary congestion
  - Echo: Valve morphology and gradients
  - Cath: Pressure gradient across AV





- Treatment
  - Treatment indicated for symptomatic AV disease or asymptomatic severe AV disease
  - Careful diuresis, control HTN
  - Caution / Avoid vigorous exercise, nitrates, (negative inotropic) BB and CCB, in severe aortic stenosis → preload and inotropic dependent. If cardiogenic shock situations, afterload reduction will be your friend → consider using nitroprusside
  - SAVR (Surgical Aortic Valve Replacement)
  - TAVR (Transcatheter aortic valve replacement)
    - Crush the native valve and replace with a bovine Edwards Sapien 3 Valve via minimally invasive catheter based procedure
    - Indications: High risk / prohibitive candidate for surgical AVR
    - Not tested for bicuspid aortic valve, rheumatic
    - Approaches: Transfemoral. Transapical, Transsubclavian and Transortic
    - Pre-Op: CT Scan, PFTs, TTE, TEE, Angiogram, etc.
    - SE: Stroke, Paravalvular leak, Coronary obstruction, Arrhythmia (AF)
    - http://www.edwards.com/therapies/transcatheter-aortic-valve-replacement-tavr

Aortic Regurgitation

Clinical Features:
Diastolic decrescendo murmur heard best @ left third intercostal space
Austin Flint Murmur (premature closure of the mitral leaflets due to regurgitant flow)
Wide pulse pressure
Bounding carotid and peripheral pulses
Diffuse and lateral displacement of PMI

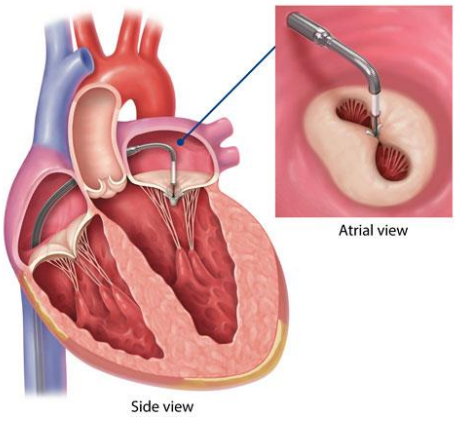
See MKSAP/uptodate/ACC Valvular Heart Disease Guidelines for treatment

Mitral Valve Disease

Mitral Regurgitation

Clinical Features, Physical Exam, Diagnostic Criteria, Treatment → Well described in the Pocket Medicine Book. I've included a brief summary.

- Etiology
  - Primary mitral regurgitation is also referred to as structural MR and is related abnormalities of mitral leaflets or subvalvular apparatus. Examples of primary MR include myxomatous, prolapse, flail mitral leaflet, ruptured chordae tendineae, or calcific degeneration.
  - Secondary MR is also referred to as functional MR, and it is related to disease states that affect the papillary muscle or left ventricular geometry
- Clinical Manifestations
  - Pulmonary edema, hypotension, cardiogenic shock
  - Dyspnea on exertion, fatigue, atrial fibrillation, pulmonary hypertension
  - 5yr survival w/ medical therapy 85% if asymptomatic, 45% if symptomatic
- Physical Exam
  - High pitched blowing holosystolic murmur at apex radiated to axilla, increases murmur Handgrip (↑ afterload), decrease murmur Valsalva (↓ preload)
- Diagnostic
  - EKG: LAE, LVH, Atrial fibrillation
  - CXR: Cardiomegaly, dilated LA, pulmonary congestion
  - ECHO: MV anatomy
  - Cath: LVgram for MR severity
- Treatment
  - Surgical treatment -repair vs replacement
  - Percutaneous mitral valve repair
    - Examples: Edge-to-edge repair, Annuloplasty, Chordal reapir, Valve spacer, etc.
    - MitraClip – Percutaneous trans-atrial-septal catheter based approach (femoral vein starting point) for mitral valve repair using a V-shaped clip for creating double-orifice mitral valve (connecting the anterior and posterior MV leaflets in the middle → a crude explanation).
    - Indications: Primary. If secondary/ functional component, then enrollment into COAPT trial



Mitral valve prolapse

Clinical Features:

Mid-Late systolic, click (due to tensing of the chordae tendineae or valve leaflets)

Mitral stenosis

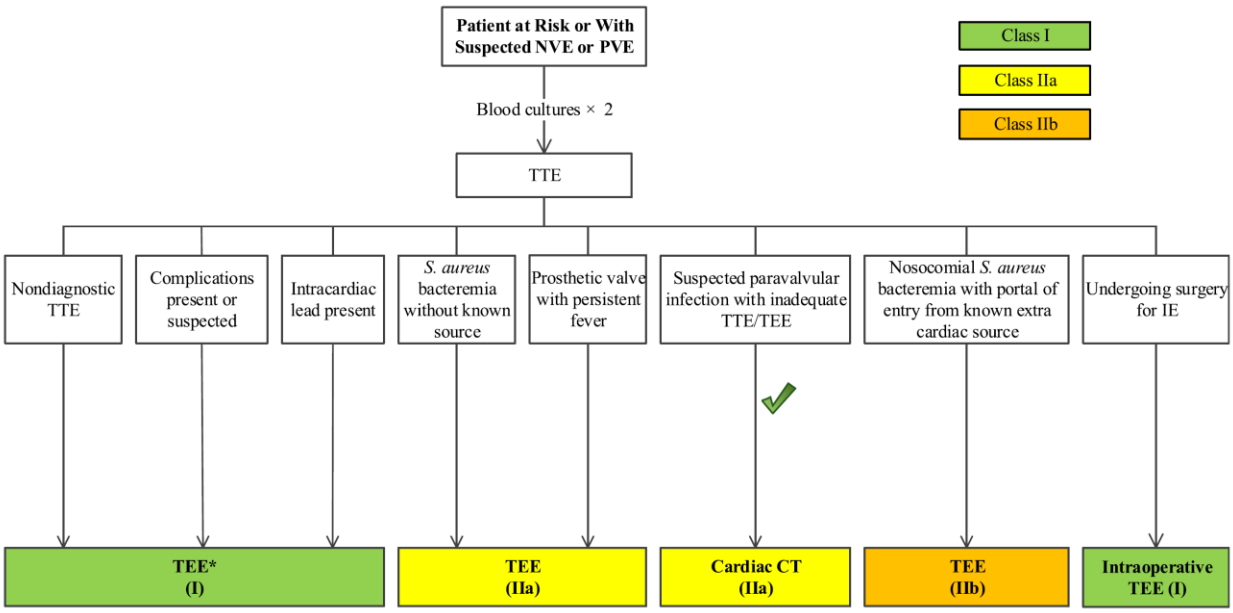
Clinical Features:

S1 increased intensity, S2 normal
Opening snap
Pressure in the LA increases, mitral valve opens earlier in ventricular diastole

Great Additional Resource:

1. Pocket Medicine: The Massachusetts General Hospital Handbook of Internal Medicine. Pocket Notebook. [43] = Green / Purple Book ~$50-60 Amazon
2. ACC 2014 – Valvular Heart Disease Guidelines

Infective Endocarditis (IE)



Why TTE?

- NVE: Sensitivity: 50 – 90%, Specificity > 90%
- PVE: Sensitivity: 36 – 69%
- Superior images: For anterior aspect of prosthetic AV commonly shadowed by the valve on TEE. TTE also allows measurement of aortic transvalvular velocity/gradient, which is not always possible on TEE.
- Although TTE will not definitely exclude vegetations or abscesses in IE, it can identify very high-risk patients and establish the diagnosis as well as guide early treatment decisions

Definite IE:

Pathologic Criteria: Vegetation, intracardiac abscess

Clinical Criteria: 2 major, 1 major and 3 minor, 5 minor

Possible IE: 1 major and 1 minor / 3 minor

Rejected: Alternative dx, resolution of IE syndrome w/ abx tx for < 4d, no pathologic evidence at surgery or autopsy w/ abx tx for < 4 d, not meet criteria

Table 25. Major and Minor Criteria in the Modified Duke Criteria for the Diagnosis of IE

Major Criteria

1. Blood culture positive for IE

Typical microorganisms consistent with IE from 2 separate blood cultures:

- *Viridans streptococci*, *Streptococcus bovis*, HACEK group (*Haemophilus* spp., *Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella* spp., and *Kingella kingae), Staphylococcus aureus*; or community-acquired enterococci, in the absence of a primary focus; or

Microorganisms consistent with IE from persistently positive blood cultures, defined as follows:

- At least 2 positive cultures of blood samples drawn 12 h apart; or
- All of 3 or a majority of $\geq$4 separate cultures of blood (with first and last samples drawn at least 1 h apart)
- Single positive blood culture for *Coxiella burnetii* or antiphase I IgG antibody titer $>$1:800

2. Evidence of endocardial involvement

- Echocardiogram positive for IE defined as follows:
  - Oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomic explanation;
  - Abscess; or
  - New partial dehiscence of prosthetic valve
- New valvular regurgitation (worsening or changing of pre-existing murmur not sufficient)

Minor Criteria

1. Predisposition, predisposing heart condition, or injection drug use

2. Fever, temperature >38° C (100.4° F)

3. Vascular phenomena, major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, and Janeway lesions

4. Immunologic phenomena: glomerulonephritis, Osler's nodes, Roth's spots, and rheumatoid factor

5. Microbiological evidence: positive blood culture but does not meet a major criterion as noted above* or serological evidence of active infection with organism consistent with IE

*Excludes single positive cultures for coagulase-negative staphylococci and organisms that do not cause IE.

C indicates Celsius; F, Fahrenheit; IE, infective endocarditis; spp, species; TEE, transesophageal echocardiography; and TTE, transthoracic echocardiography. (642,644)

Infective Endocarditis Treatment Algorithm

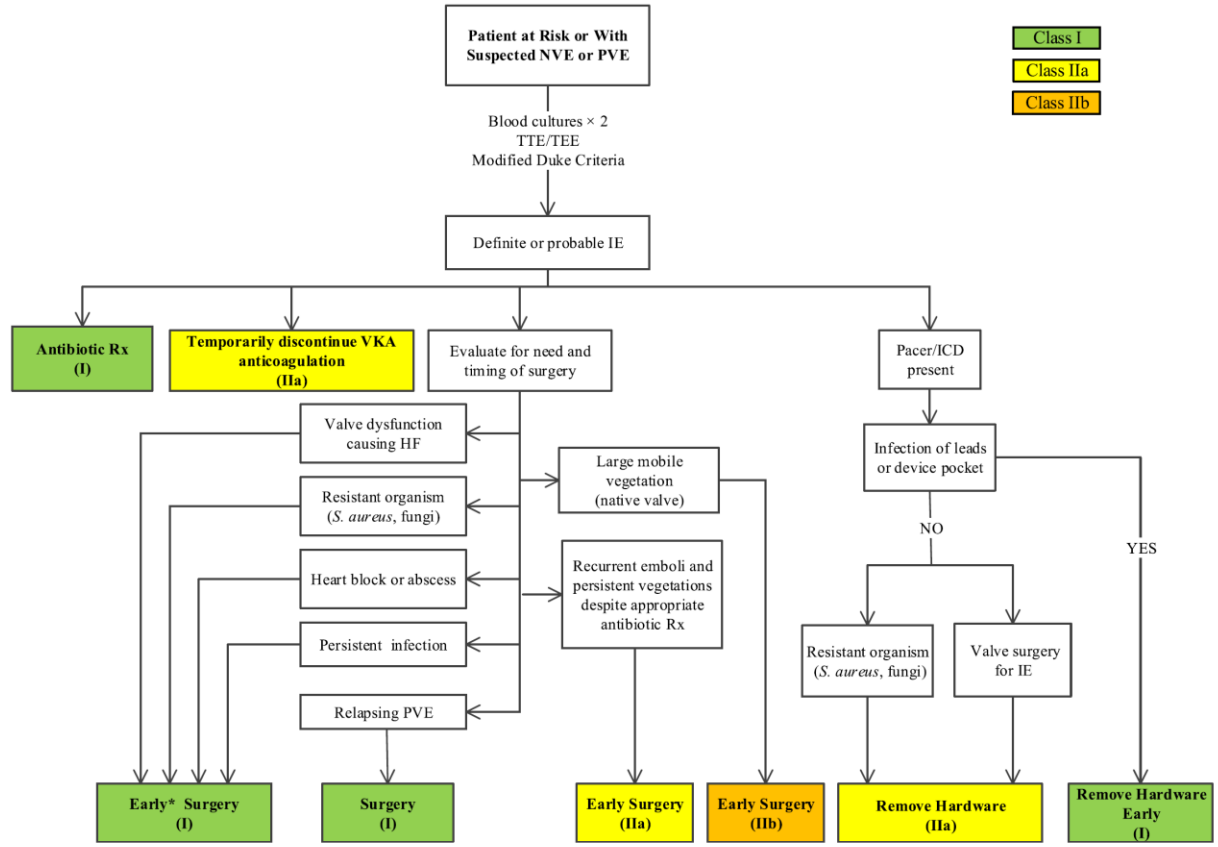
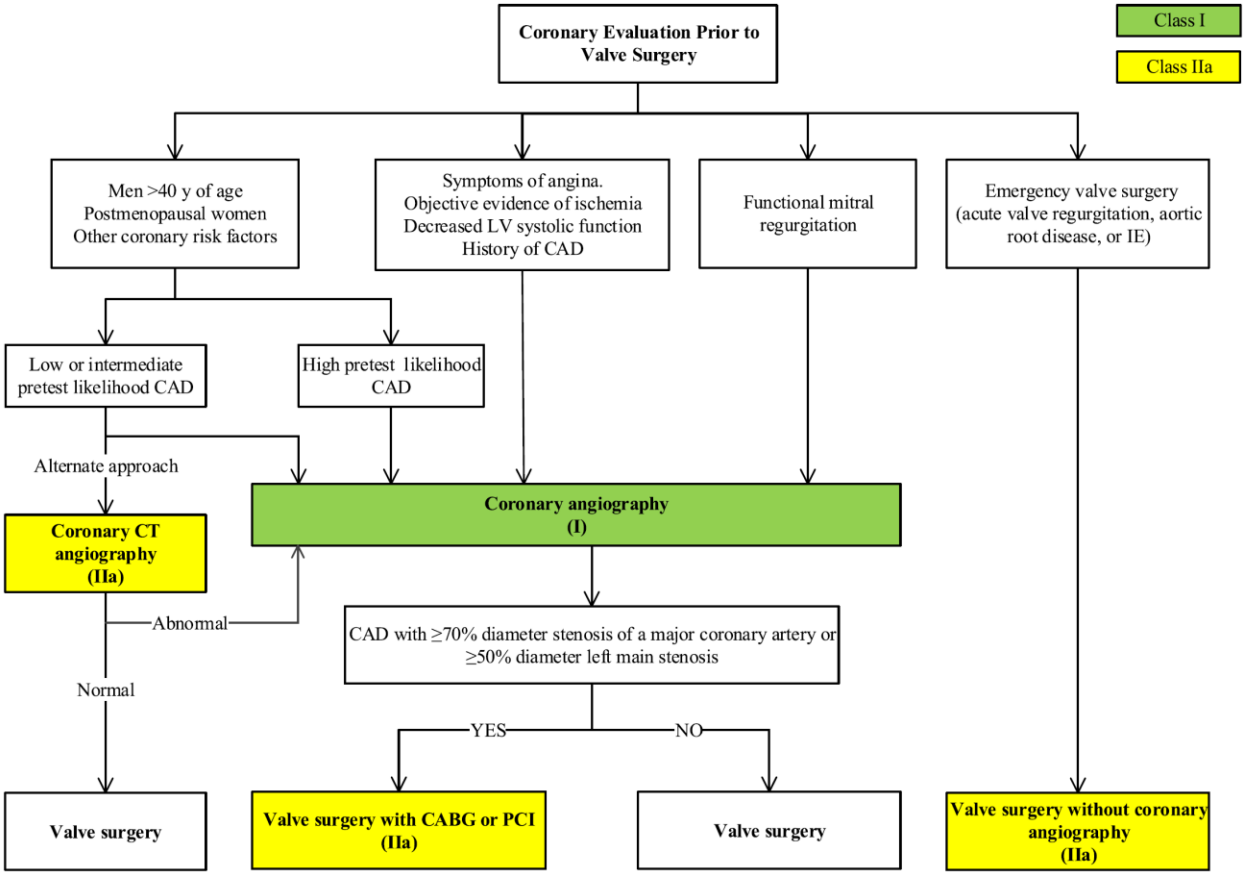


Figure 9. Diagnosis and Treatment of IE

Coronary Assessment Prior to Valve Surgery



Source Valvular Heart Disease [42]:

Pregnancy and Cardiovascular Disease

Important Points

- Anticoagulation in patients with mechanical valve prosthesis
    - Warfarin
        - Less risk for maternal thromboembolism
        - Increased risk for fetal embryopathy, teratogenicity and fetal loss
        - Risk esp reduced w/ warfarin dosing 4mg/d
    - Dose-adjusted UFH
    - LMWH (2nd preference)
        - adjusted to anti–factor Xa activity in mechanical prosthesis
- Low-dosed aspirin can be continued
- Mitral valve intervention, Class I, in prengnacy
    - Severe mitral stenosis despite asymptomatic

Lines CheckList (Triple lumen, Swann/Introducer, Arterial line)

Triple Lumen Central line placement 7.0 Fr equipment:

- Gloves, Masks, Gowns, Buffon caps (at least 3 sets, self, attending and RN)
- Tape
- Ultrasound Sterile probe cover (Aquasonic)
- Chlorhexidine Scrub x2 total (x1 in the kit)
- 3M Tegaderm CHG Medium Sized
- **Sterile** sodium chloride syringe x1-2
- MicroClave clear valve x3 (for 3 ports)
- 4x4 Sterile Gauze x2
- Needle 22ga / 25ga w/ lidocaine
- Introducer Needle 5cm and 7cm (18ga)
- Guide wire, Dilator
- Safety Scalpel
- Catheter over Needle 7cm
- 3-Prefilled Sodium Chloride syringes
- Braided Silk Suture
- Fenestrated Drape
- Make sure hemo contacted for setup/hook up

Blue'd out items are included in the kit.
All of the non-grayed items are usually found in the Central line blue cart

Introducer 9Fr Central line placement equipment + PA Cath Eq:

- Gloves, Masks, Gowns, Buffon caps (at least 3 sets, self, attending and RN)
- Tape
- Ultrasound Sterile probe cover (Aquasonic)
- Chlorhexidine Scrub (another usually provided in the kit)
- 3M Tegaderm CHG Medium Sized
- **Sterile** sodium chloride syringe x4
- MicroClave clear valve x2 (1 for introducer, 1 for Swann line dedicated port)
- 4x4 Sterile Gauze x2
- Needle 22ga / 25ga w/ lidocaine
- Introducer Needle 5cm and 7cm (18ga)
- Guide wire, Dilator
- Safety Scalpel
- Introducer
- Needle + Suture
- Swandom
- Fenestrated Drape (not included in the kit)
- Pentalumen™ pulmonary artery Swann-Ganz catheter w/ syringe (not included in the kit)
- Make sure hemo contacted for setup/hook up

Blue'd out items are included in the kit.
All the non-grayed items are usually found in the Central line blue cart

Radial Arterial Line:

- Gloves, Masks, Buffon caps (at least 3 sets, self, attending and RN)
- Tape, Support – Green Pad, Kerlex
- Ultrasound Sterile probe cover (Aquasonic)
- Chlorhexidine Scrub x1
- 3M Tegaderm CHG Small Sized
- **Sterile** sodium chloride syringe x1-2
- MicroClave clear valve x1
- 4x4 Sterile Gauze x2-3
- Sterile drape or 3 sterile towels
- Radial Art Line Kit (over the wire technique) and/or Arrow kit (over the needle technique)
- 3 way stop cock w/ extension
- Arterial line- hookup setup

Brief Drug Dosing Guide

Dose Equivalents

Diuretics:

Drug Name	Drug PO dosing	Drug IV dosing	Furosemide PO dosing	Furosemide IV dosing
Bumetanide	1 mg	1 mg	40 mg	20 mg
Torsemide	20 mg	20 mg	40 mg	20 mg
Furosemide	40 mg	20 mg	40 mg	20 mg

Beta Blockers:

Drug Name	Approximate Dose Equivalence	Max Total Daily Dose
Metoprolol tartrate IV	5mg BID	
Metoprolol tartrate PO	12.5 mg BID	400 mg PO
Metoprolol succinate	25 mg PO qD	

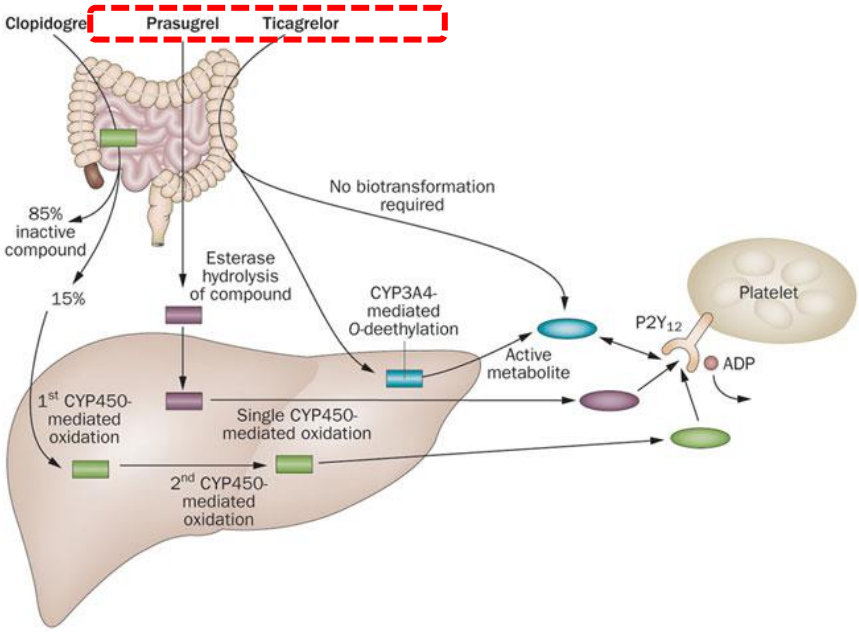
ACEi:

Drug Name	Approximate Dose Equivalence	Max Daily Dose	Brand Name
Captopril	12.5 mg TID	150 mg	Capoten
Lisinopril	10 mg qD	40 mg *	Prinivil, Zestril
Enalapril	5 mg qD	40 mg	Vasotec
Ramipril	2.5 mg qD	20 mg	Altace
Benzepril	10 mg qD	40 mg	Lotensin

*Lisinopril 80 mg have been used without significant clinical benefit compared to 40mg

P2Y1,2 receptor antagonist (*TRITON-TIMI-38*):

Options include: Clopidogrel [Plavix], Ticagrelor [Brilinta], Prasugrel [Effient]



Most potent antiplatelet agent in decreasing order?
1. Ticagrelor– no biotransformation required, thereby it's an active metabolite
2. Prasugrel [black box warning against stroke] goes through esterification
3. Clopidogrel 2-step process to convert to active metabolite.
Alas, clopidogrel is the cheapest and not ticagrelor!

Plavix [Clopidogrel] to Brilinta [Ticagrelor] conversion (especially for pts who cannot afford ticagelor)
- Cont Ticagelor 90mg BID
- Give Clopidogrel 600mg 6 hrs prior (e.g. afternoon) to last Ticagelor dosing (if evening's last dose)
- And then start Clopidogrel 75mg/d (next day)
- *Uptodate can provide you with other regimens, always check with primary source / pharmacist*

Amiodarone

It's a 10gm load

Bolus during a code:	Amiodarone IV 300mg bolus, followed by IV 150mg pushes, when stable start maintenance gtt
Bolus during stable VT/VF/SVT:	Amiodarone IV 150mg bolus, and then maintenance

Loading IV formulation:
Amiodarone 900mg in 500mL NaCl 0.9% or D5 (Consider appropriate solution) @ 1mg/min (33mL/hr) for 6 hrs and then 0.5 mg/min (17mL/hr)
→ Painstaking but calculate how much amount was administered. Here's an ex:
→ 33mL/hr for 6 hrs & then 17mL/hr for 18 hrs = 198mL + 306 mL = 500mL in 24 hrs
→ 500mL → turns out to be 900mg in 1 day
Remember: Central access / PICC line needed for IV Amiodarone

Loading PO formulation: Amiodarone 400mg TID

Maintenance dosing post 10gm load:
VT/VF - 400mg/d
SVT (A-fib) – 200 mg/d

Side Effects: TFT, LFT and PFTs

Lidocaine

2nd agent if amiodarone fails

Cardiac Arrest from VT/VF:
Loading dose: 1-1.5 mg/kg slow IV/IO bolus over 2-3 min
May repeat doses of 0.5-0.75 mg/kg in 5-10 min up to 3 mg/kg total

For stable VT, wide-complex tachycardia of uncertain type and significant ectopy:
Loading dose: 0.5-0.75 mg/kg and up to 1-1.5mg/kg
Repeat 0.5 to 0.75 mg/kg every 5-10 min with maximum total dose of 3 mg/kg

Lidocaine 1-2mg/min, mg in 500mL D5W

Therapeutic Level: 1.5 to 5.0 mcg/mL
Toxicity: Neuro, Oral numbness

PO equivalent: Mexilitine ~ 150mg TID (dosing usually)

Pressors and Inotropes and Dosing:

Norepinephrine - a1, B1

- 0.03-0.6 mcg/kg/min - cardiogenic, septic

Vasopressin - ↑ intracellular Ca2+

- 0 .03 U/min - septic, cardiogenic

Epinephrine - a1, b1, b2

- 0.05-0.6 mcg/kg/min - cardiogenic, septic, anaphylactic

Phenylephrine - a1 - norepinephrine-resistant septic

- 0.2- 5 mcg/kg/min

Dopamine - a1 (at higher doses), b1

- 0.5-20 mcg/kg/min - cardiogenic, septic, neurogenic

Dobutamine - a1, b1, b2

1. 2.5-20 mcg/kg/min - cardiogenic, septic

Milrinone - PDE3 inhibitor

- 0.2-.0.5 mcg/kg/min – cardiogenic (mild peripheral vasodilator effect)

Aspirin Desensitization Protocol

OrderSet: CARD Aspirin Desensitization Protocol for IgE Mediated Reactions
Management of ASA Desensitization Adverse Reactions

1. Mild Reaction – Repeat PROVOKING (same) aspirin dose after treating reaction with below agent, AND patient is stable (e.g., pretreatment vital sign values)
    a. Adverse Effect – Tx w/
       Cutaneous, ocular, nasal pruritis, congestion – PO/IV Diphenhydramine
2. Moderate Reaction – Repeat PROVOKING (same) aspirin dose after treating reaction with below agent, AND patient is stable (e.g., pretreatment vital sign values)
    a. Adverse Effect – Tx w/
       Isolated pulm symptoms: cough, wheeze, chest tightness, SOB – Albuterol Nebs
       Isolated laryngeal symptoms: stridor, throat tightness, throat swelling – IM Epi
       Laryngeal symptoms with hypotension (SBP < 100 mm Hg) – IM Epi
       Isolated hypotension (SBP < 100 mm Hg) – IM Epi
3. Severe Reaction – Notify provider and contact Allergist
    a. Adverse Effect – Tx w/
       Refractory hypotension (SBP < 100mm Hg) – IM Epi amd IV Fluids

Doses:
1mg, 3mg, 10mg, 20.25mg, 40.5mg, 81mg and another 81mg advanced q20minutes

Contraindicated for below mentioned conditions:

- Aspirin Exacerbated Respiratory Disease (AERD)
- History Consistent with Urticaria/Angioedema Triggered by NSAIDs/ASA
- Symptoms consistent with known pharmacologic properties of ASA, including dyspepsia, nausea/vomiting, abdominal pain, tinnitus, dizziness, constipation, and diarrhea
- A history of exfoliative dermatoses (e.g., Stevens-Johnson Syndrome or toxic epidermal necrolysis)

Common Cardiac Abbreviations / Acronym

3VD – Three Vessel Disease
ACEi – Angiotensin-converting-enzyme inhibitor
ARB – Angiotensin receptor blockers
ARNi – Angiotensin receptor-neprilysin inhibitors
ASA – Aspirin
BB – Beta Blocker
CABG – Coronary Artery Bypass Graft
CCB – Calcium Channel Blocker
CMP – cardiomyopathy
CRT - Cardiac Resynchronization Therapy
DM – Diabetes Mellitus
DOAC – Direct Oral Anticoagulation Agent
EF – ejection fraction
EPS –electrophysiologic study
GDMT – guideline directed medial therapy
HFpEF – Heart failure with preserved ejection fraction
HFrEF - Heart failure with reduced ejection fraction
ICD – implantable cardioverter defibrillator
LMWH – Low-molecular weight heparin
MVD – Multivessel Disease
NICMP – nonischemic cardiomyopathy
NOAC – New Oral Anticoagulation Agent
PCI – Percutaneous coronary intervention
POBA – Plain old balloon angioplasty
PPM – Permanent Pacemaker
SCD – sudden cardiac death
UFH – UnFractionated Heparin
VF – ventricular fibrillation
VT – ventricular tachycardia

Applications Website or Apps (iOS / Android)

1. Cardiac Index, Cardiac Output, Systemic Vascular Resistance
    1. www.fickcalc.com
2. CHADS2VASc score for atrial fibrillation
3. HASBLEED SCORE
    1. www.afib.ca
4. ASCVD – 10 year ASCVD risk calculator
    1. http://tools.acc.org/ASCVD-Risk-Estimator-Plus/#!/calculate/estimate/
5. STS Score (To access mortality and morbidity post open heart surgery (includes CABG, MV replacement, etc.) Great tool to provide #s and stats to patients and their family members)
    1. http://riskcalc.sts.org/stswebriskcalc/
6. DAPT score
7. EKG QTc correction
8. TIMI / GRACE
    1. QX calculate

Day and Night service teams, you are encouraged to use www.fickcalc.com, for universal calculation.

Cardiology Key Trials

Some of the key cardiology trails are listed, although not a substitute for actual first hand review of the literature (http://cardiologytrials.org/) or a comprehensive list [44]. You can use app "Journal Club" to further assist you with reviewing articles.

- Stable Coronary Disease
    - COURAGE - PCI w/ bare-metal stents in stable CAD non-superior to medical therapy
    - FREEDOM – CABG Superior to PCI for MVD in DM
    - ***Triple therapy (ASA, P2Y12 and NOAC or warfarin) requires additional thought/ clinical consideration.***
- Acute Coronary Syndrome
    - CURE – Proved benefit of clopidogrel added to aspirin in NSTEMI/UA
        - CREDO, ARMYDA-2, CURRENT-OASIS – 600mg clopidogrel load
    - TRITON-TIMI-38 - Prasugrel superior to clopidogrel in ACS scheduled for PCI
    - PLATO Trial - Ticagrelor superior to clopidogrel in ACS, including mortality
    - EPHESUS - First trial to show benefits of aldosterone antagonists after acute MI with CHF
- CABG vs PCI
    - SYNTAX trial – CABG better than PCI in patients with LMS/3VD. Involved 1st gen DES (revascularization rates and CV deaths).
    - Freedom trial – CABG superior to PCI for MVD in T2DM
- Heart Failure
    - Guideline directed medial therapy
        - ACEi / Hydralazine/Imdur
            - SOLVD, CONSENSUS – Looked at ACEi
        - ARNI- Entresto (sacubitril/valsartan)
            - PARADIGM HF – Reduction in primary endport: cardiovascular death / hospitalization, in patients compared to ACEi
        - Spironolactone
            - RALES - Spironolactone reduced mortality and symptoms in NYHA 3+
            - EMPHASIS-HF TRIAL - Eplereonone reduced mortality and symptoms in NYHA 2+
        - BB (Metoprolol succinate, Carvedilol or Bisoprolol)
            - COMET - Carvedilol superior to metoprolol tartrate reducing mortality in NYHA II+ & EF <35%
                - Avg dose: 25mg BID
                - Other relevant trials: COPERNICUS
            - MERIT-HF - Metoprolol succinate better in HF patient
                - Target dose: 200mg/d
            - CIBIS-II - Bisoprolol improved mortality in NYHA 3-4
            - MDC - metoprolol tartrate not better than placebo
        - Digoxin
            - DIG - Digoxin reduced HF hospitalization but not mortality
    - IV Iron treatment
        - IV Fe therapy in iron-deficient patients with systolic HF improves outcomes, exercise capacity, and quality of life, and alleviates HF symptoms. Effects of intravenous iron therapy in iron-deficient patients with systolic heart

failure: a meta-analysis of randomized controlled trials. European Journal of Heart Failure (2016) doi:10.1002/ejhf.473

- Atrial Fibrillation
    - AFFIRM - Rate control non-inferior to rhythm control and possibly superior in elderly and co-morbid patients
    - BRIDGE – Low-intermediate risk AF receiving AC and undergoing invasive procedure, periprocedural bridging anticoagulation with LMWH (Dalteparin, used in the study) did not reduce risk of arterial thromboembolism/ischemic stroke when compared to no bridging, but did increase risk of major bleeding.
    - NOAC
        - ARISTOTLE – Apixaban [Eliquis] improved mortality, bleeding and stroke rates
        - ROCKET-AF – Rivaroxaban [Xarelto] non-inferior to warfarin
        - RE-LY – Dabigatran [Pradaxa] compared to warfarin lower rates of strokes / embolism, similar bleeding events
    - Reversal Agent
        - Idarucizumab [Praxbind] – Idarucizumab for Dabigatran Reversal (Pollack et al., NEJM 2015)
    - ***Triple therapy (ASA, P2Y12 and NOAC or warfarin) requires additional thought/ clinical consideration.***

All these cardiology key trails paper along with reference articles will be incorporated into Google Drive folder, a free shareware folder for instant article access (rather than jumping through hoops to access articles). Link at the bottom of every page.

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Not a comprehensive list.

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