Core Curriculum/Goals and Objectives
Cardiovascular Fellowship

UMKC/Saint Luke's Hospital/Mid America Heart Institute/Truman

I. Preface

Welcome to the Cardiovascular Training Program at Saint Luke's Mid America Heart Institute and Truman Medical Center under the administrative control of the Graduate Medical Education of Office at the University of Missouri-Kansas City School of Medicine. Dr. Brent Beasley is the Program Director of Internal Medicine Residency Programs. Dr. George Reisz is the Chairman of Medicine; his office is at Truman Medical Center. Dr. Jill Moormeier is the Associate Dean of Graduate Medical Education. Dr. Spertus is Director of Cardiovascular Education and Outcomes Research.

This core curriculum/goals and objectives guide is to keep you appropriately focused and oriented throughout your three year training program regarding specific rotations and knowledge goals. See the specific rotation goals in part II and the specific knowledge goals through out the remainder of this guide. We will review each rotation's goal and objectives on the 1st week of each month on your new monthly rotation. We also request monthly feedback on your personal evaluation of each rotation.

You should start a Procedure Log that keeps an accurate and constant tabulation of your procedural number goals. Every six months at the time of your semi-annual evaluation, I will request from you an up-to-date tally of your procedure count. After you finish this training program, you will need this information for your medical staff credentials, wherever you choose to practice.

I strongly encourage you to refer to this guide constantly and recurrently throughout each rotation, making sure you are achieving your medical knowledge and procedural goals.

II. Goals and Objections for Specific Rotations

A. CCU-SLH; In-patient consults-TMC; In-patient teaching cardiology-SLH

1. Must spend 8 months in non-laboratory clinical practice activities.
2. Can do a competent, thorough but focused H & P with prioritized differential diagnosis, as expected of a cardiovascular consultant.
3. Demonstrated excellent clinical judgment in utilization of laboratory resources i.e. cost/benefit ratio.
4. Uses evidence-based medicine and up to date clinical practice guidelines (ACC/AHA) for management of patients.
5. Demonstrates excellent communications skills with patients, families, and all health professionals.
6. Demonstrates excellent professional and medical ethics with respect for all ages, gender, races and religions.
7. Understand and uses all resources available for patient care, especially case management concepts.
8. For specific knowledge goals, refer to specific topics in these referred guides.

B. Non-Invasive-TMC/SLH/CC

1. Read and review with faculty 3500 ECG's over the 3 year period of the program.
2. Observe and interpret a minimum of 50 exercise stress tests and preferably 200 (COCATS 2)
3. Interpret a minimum of 75 ambulatory ECG recordings and preferable 150 (COCATS 2).
4. Must perform at least 75 TTE and interpret 150 TTE exams.
5. For specific knowledge goals, see specific topics.

C. Advanced Echo-SLH/CC

1. Completing number of procedures and knowledge goals.
2. Experience in stress echo-cardiography (level 2 training = 100 studies). 3rd year Fellow required to finish only level 1 numbers.

3. Initial experience with TEE, minimum training recommended, 50 supervised studies (COCATS 2).
4. Initial exposure to intraoperative TEE, contract echo, IVUS, and tissue doppler.

D. Cardiac Catheterization-SLH/TMC

1. Must spend a minimum of 4 months in the cath lab.
2. Must participate in minimum of 100 diagnostic procedures.
3. Interventional procedures are expected only for Level 3 or interventional/advanced experience fellows (COCATS 2).
4. For specific knowledge goals, see cardiac heading in this guide and list of core topics in cardiac cath conferences.

E. Nuclear Cardiology

1. Must spend a minimum of 2 months for fundamental knowledge and interpretation skills.
2. A minimum of 80 hours should be spent in study interpretation (COCATS 2).
3. Knowledge and principles of radiation safety is a key goal.
4. See specific knowledge goals under heading in this guide.

F. Electrophysiology-SLH/CC

1. Must spend a minimum of 2 months on EP rotation.
2. Become an expert in ECG interpretation.
3. Further experience with ambulatory ECG recordings.
4. Greater depth in understanding Dx/Rx cardiac arrhythmias.
5. Fundamental knowledge of temporary and permanent pacemakers and ICD's required.
6. Recommended to place 10 temporary pacers and preform 10 elective cardioversions (COCATS 2).
7. For specific knowledge goals, see topic heading in this guide.

G. Research-SLH/TMC

1. Will know how to do a thorough literature search, including use of computers, different search engines, and how to access different databases.

2. Can formulate a research hypothesis and preferably present it at a monthly research conference.
3. Demonstrates the ability to develop a research plan, including study design, how to recruit patients, informed consent, data collection techniques and IRB approval.
4. Understand risk/benefit and cost/benefit analysis, both for patient and society.

III. History of patient's illness skills:

A. Three year observation of Fellow's bedside history taking skills on inpatient rounds and outpatient Fellow Clinic.

IV. Physical examination skills: Goal is to emphasize the 5-fingered approach after Dr. Proctor Harvey, especially with the teaching mannequin, "Harvey".

A. Observation and vital signs.
B. Jugular venous pressure (JVP):
    1. Total height: normal or elevated
    2. Dominant A or V: pressure vs. volume overload
    3. Rapid vs. slow Y descent.
    4. Hepatojugular reflux.
    5. Kussmaul sign.
C. Carotid pulse:
    1. Volume: increased vs. decreased C.O.
    2. Upstroke: valvular AS or not.
    3. Bisferiens/bifid: AI vs. HCM.
    4. Bruit: transmitted or not?
    5. Other pulses: alternans (CHF); paradoxical (tamponade).
D. Precordial palpation:
    1. Thrill.
    2. Heart sounds: □ S_1; □ S_2P; S_3; S_4 (A wave).
    3. Lift: LV, RV, PA, ectopic bulge (dysnergic LV).
E. Auscultation:

1. Abnormal S_1: accentuation in mitral stenosis. Normal splitting, especially tricuspid area. Differentiate from S4 and systolic ejection click (SEC).
2. Abnormalities (S_2):
    a. Normal splitting.
    b. Wide fixed splitting (ASD).
    c. Paradoxical splitting (LBBB).
    d. Increased S_2A (systemic hypertension); increased S_2P (pulmonary hypertension); diminished or absent S_2A (calcified aortic valve).
3. S_3: physiologic vs. abnormal; LV vs. RV
4. S_4: palpable vs. audible; clinical significance.
5. SEC: aortic vs. pulmonic, the latter with respiratory variation; valvular vs. great vessel in origin.
6. Mid-late systolic click/mitral valve prolapse (MVP):
    a. Movement of MSC/LSM with volume change.
7. Opening snap of mitral stenosis: pliability of mitral valve; severity of mitral stenosis relative to A_2-OS interval:
    a. Differentiate O.S. from pericardial knock and S_3.
8. Systolic murmurs: ejection, holosystolic, early systolic, crescendo late systolic, and their differentiation.
9. Diastolic murmurs:
    a. Decrescendo – aortic regurgitation; pulmonic valve insufficiency in pulmonary hypertension (Graham Steele); post-PV valvotomy.
    b. Low frequency rumble – mitral stenosis; flow rumble of □ flow 2^o MR, VSD, PDA; Austin Flint murmur of "pseudo-MS" in AR; tricuspid stenosis.
10. Continuous murmurs: patent ductus arteriosus vs. Other possible causes such as: coronary artery fistula, sinus of Valsalva fistula to the right heart, post-shunt procedure in congenital heart disease, and in collateral vessels:

a. Differentiate from to-and-fro murmurs of combined AR and VSD.
b. Differentiate from pericardial function rub; pleural rub; mediastinal "crunch".
c. Venous hum: normal variant.

11. Use of bedside maneuvers to differentiate origin of murmurs:
    a. Inspiration: augment right heart.
    b. Position: left lateral decubitus, sit, stand, squat: HCM vs. MVP.
    c. Legs up: HCM vs. MVP.
    d. Hand grip: augment AI, MR. S_3 and S_4.
    e. Amyl nitrate inhalation: HCM; Austin Flint vs. MS.

F. Methodology to accomplish history and physical diagnosis skills:
    1. One-to-one review with cardiology staff on teaching rounds and clinic experience.
    2. Intensive reading of Dr. Forker's outline on physical diagnosis plus more in-depth reading with standard articles/books.
    3. Core lectures and Harvey overview at beginning of Fellowship each July-August:
        a. Harvey exam included in Core Curriculum Conference Schedule.

V. Electrocardiography

A. 12 Lead ECG:
    1. Proper placement of limb and precordial leads; identification of limb lead reversal.
    2. Standardization.
    3. Recognition of artifacts.
    4. Frontal axis:
        a. Left axis deviation.
        b. Right axis deviation.

c. Indeterminate axis (S_1, S_2, S_3).

5. P wave abnormalities: right vs. left atrial abnormality/enlargement.
6. LVH and RVH criteria.
7. Differentiate RBBB, LBBB, and nonspecific intraventricular conduction delay (IVCD).
8. Preexcitation/WPW syndrome.
9. Acute myocardial infarction and its mimics:
    a. Early repolarization variant.
    b. Pericarditis.
    c. Hypothermia (Osborn hump).
    d. Pseudo-infarct Q waves.
10. T-wave abnormalities: frequent and multiple causes; not pathognomic of myocardial ischemia:
    a. Broad, splayed T waves of CNS disease.
    b. Hyperkalemia.
11. QT prolongation, and relationship to polymorphic ventricular tachycardia/Torsades:
    a. Congenital long QT syndrome (with/without deafness).
    b. Brugada Syndrome (RBBB with ^ ST in V_1).
12. Miscellaneous:
    a. Dextrocardia.
    b. Electrical alternans.
    c. Low voltage QRS complexes.
13. Pacemakers:
    a. Atrial.
    b. Ventricular.
    c. Rate responsive.

B. Supraventricular arrhythmias:

1. Anatomy of conduction system.
2. Cell membrane action potential: pacemaker (SA node) vs. non-pacemaker cell (muscle).

3. Sinus bradycardia, tachycardia, and arrhythmia.
4. APC's, including non-conducted and aberrant conducted.
5. AV node reentry tachycardia.
6. AV reciprocal tachycardia through accessory bypass tract (WPW):
    a. Orthodromic/narrow QRS.
    b. Antidromic/wide QRS.
7. Atrial flutter:
    a. Drugs: block AV node vs. conversion.
    b. RF ablation.
8. Atrial fibrillation:
    a. Rate control: BB, CaBI, vs. Dig.
    b. Anticoagulation: Heparin, Coumadin, or ASA?
    c. Maintain NSR: Amiodarone or not?
    d. Ablation: AV node; LA ; MAZE procedure.
9. Multifocal atrial tachycardia (MAT): COPD.
10. Atrial tachycardia with AV block: Dig toxicity.
11. Junctional/AV nodal tachycardia.
    a. Accelerated junctional rhythm.
    b. Non-paroxysmal junctional tachy.
    c. AV dissociation.
12. Drug therapy approaches:
    a. Digoxin.
    b. Adenosine.
    c. Verapamil/Diltiazem.
    d. Beta blockers.
    e. Type IA (Quin/Pronestyl/Norpace).
    f. Type IC (Flecainide/Propafenone).
    g. Type III (Amiodarone, Sotalol, Dofetilide, Ibutilide).
    h. When to anticoagulate?
13. Role of radiofrequency ablation:
    a. Fundamental knowledge by EP study of type/location each SVT.

14. Elective cardioversion:
    a. Indications.
    b. Contraindications.
    c. Technique: monophasic vs. biphasic waveform; paddle position; voltage.
    d. Role of TEE.

C. Ventricular arrhythmias.
    1. VPC's: Bigger classification (benign, potentially malignant, malignant).
        a. Parasystole
    2. Ventricular tachycardia.
        a. Non-sustained.
        b. Monomorphic, sustained.
        c. Polymorphic/Torsades.
        d. Recent clinical trials of drug therapy vs. ICD:
            i. MADIT I & II, AVID, CIDS.
            ii. MUSTT, SCD-HEFT
        e. Indications for EP study: inducible or not?
            i. Fundamental knowledge on how to interpret.
        f. Indications for ICD.
        g. Basic programming and troubleshooting ICD.
        h. ACLS Rx protocol.
        i. Accelerated idioventricular rhythm.
        j. Ventricular ectopy vs. aberration.

D. AV block.
    1. 1st degree.
    2. 2nd degree: Mobitz I (Wenckeback) vs. Mobitz II.
    3. 3rd degree: narrow vs. wide QRS complexes.
    4. Indications for temporary pacemaker placement.

    5. Indications for permanent pacemaker placement:
        a. Types of pacemakers available.

b. Why choose ventricular pacing vs. AV sequential (DDD) pacing.
c. Methods of programming.
d. Techniques of follow-up.
e. Possible preliminary experience with placement during EP rotation.

E. Holter monitoring: uses and interpretation.
1. Cardiac arrhythmia recognition and correlation with symptoms.
2. Pacemaker evaluation
3. Post myocardial infarction or ischemic risk stratification.
4. Prolonged monitoring: patient triggered event recorder; implantable.

F. Methodology to accomplish ECG skills.
1. Read standard text books, with emphasis on Marriott and Chou.
a. Review all 80 ECG's in O'Keefe, et al.
2. Noninvasive Cardiology Fellow presents weekly at the Wednesday noon conference 1-2 unknown ECG's for peer review.
3. Daily review with staff on all ECG's. Fellow generally reads 35-40 ECG's each morning at TMC.
4. Bi-Monthly ECG (Monday) Conference.
5. Constant self-evaluation by Fellows/Staff at ECG conference.

VI. Chest X-ray

A. Cardiac size with measurement of cardiac thoracic (C/T) ratio.
B. Which chamber enlarged.
C. Aortic root/size.
D. Pulmonary artery: Left-right shunt, pulmonary hypertension, prominent left pulmonary artery (pulmonic valve stenosis); absent MPA (Tetralogy).
E. Signs of pulmonary congestion, including Kerley B lines.
F. Calcification: valves, patent ductus, myocardial, pericardial.
G. Rib notching: coarctation of the aorta.

H. Dextrocardia with situs inversus/situs solitus.
I. Method-

1. Basic lecture by Dr. Forker.
2. Daily in-patient rounds and out-patient clinic.

VII. Echocardiography

A. Basic principles.

1. M-Mode, 2-D, Doppler

B. M-mode/single crystal:

1. Basic positions/views.
2. Normal anatomy.
3. Current diagnostic application/uses:
    a. LV Chamber size and function
        i. systolic
        ii. diastolic
        iii. regional wall abnormalities
        iv. LV thickness
        v. LV volumes, estimated vs. Simpson calculations
    b. Valvular Heart Disease
    c. Pericardial effusion and Tamponade

C. Sector scan.

1. How different from M-mode; how technically obtain sector views.
2. Normal anatomy and views:
    a. Parasternal long axis.
    b. Parasternal short axis.
    c. Apical 4 chambers, 2 chamber and long axis.
    d. Subcostal.
    e. Suprasternal.
3. Normal measurements and values.
4. Valvular abnormalities, including all 4 cardiac valves:
    a. Stenosis.
    b. Insufficiency.

5. Myocardial disease:

a. Differentiate global (dilated CM) vs. focal wall motion abnormalities (CAD).
b. Utilization of wall motion score.
c. Differentiate different segments of involvement in relationship to specific coronary artery.
d. Importance of quality images to see endocardium.
e. Calculate ejection fraction:
    i. Simpson: computerized (how derived).
    ii. Visually estimating ejection fraction.
f. Hypertrophic cardiomyopathy:
    i. ASH (asymmetric septal hypertrophic) vs. hypertrophic in other areas (apical).
    ii. Differentiate obstructive (SAM) vs. nonobstructive forms.
g. Restrictive CM:
    i. "Sparkling" of myocardium and differential diagnosis.
    ii. Amyloidosis: value of Doppler.
    iii. Obliterative/Endocardial fibroelastosis.

6. Pericardial disease:
    a. Effusion:
        i. Size estimation.
        ii. Hints to cardiac tamponade (RA collapse, RV collapse, IVC dilation and non-changing size).
    b. Constriction: importance of Doppler information.
7. Cardiac masses:
    a. Tumors.
    b. Vegetations.
    c. Clot; mural; transient pulmonary emboli in right heart chambers.

    d. Pseudo-tumors:

i. Eustachian valve.
ii. Chiari network.

e. Masses attached to pericardium.

8. Adult congenital heart disease: See individual lesions.
    a. Structural abnormalities require knowledge of congenital heart disease pathology.
    b. Doppler flow with emphasis on color flow Doppler abnormalities to detect left-right shunts.

D. Doppler.

1. Differentiate pulsed wave (PW), continuous wave (CW), and color flow Doppler (CFD), and how they are obtained.
2. How to measure valvular gradients: peak velocity, mean velocity, pressure half time and valve area calculation.
3. How to estimate valvular regurgitation:
    a. Regurgitant jet area.
    b. Appropriate chamber dilatation.
    c. Width of regurgitant jet.
    d. PISA.
4. Estimate pulmonary artery pressure: tricuspid regurgitant jet derivation.
5. Recognize left-right shunts:
    a. PDA: Parasternal short axis view.
    b. ASD: Subcostal view best.
    c. VSD: Parasternal short axis and apical 4 chamber views best.
6. Diastolic LV function:
    a. E velocity.
    b. A velocity.
    c. E/A ratio.
    d. IVRT (isovolumic relaxation time).

e. Deceleration time (DT) on anterior mitral leaflet and tricuspid valve.
f. Typical pattern of noncompliant left ventricle vs. restrictive pattern.
g. Differentiate restrictive cardiomyopathy vs. constrictive pericarditis.
h. Pulmonary vein identification and value, especially in mitral regurgitation.
i. Hepatic vein/IVC identification.
j. Tissue Doppler

E. Harmonic imaging.
F. Contrast imaging.
    1. Viability
    2. Endocardium visualization.
G. Transesophageal echo (TEE).
    1. Compare single plane, biplane, multiplane techniques.
    2. Technique:
        a. Patient selection.
        b. Sedation/anesthesia.
        c. Antibiotic prophylaxis
        d. Probe insertion.
        e. Imaging planes: correlate with anatomy:
            i. Base.
            ii. Mid-esophageal.
            iii. Transgastric.
            iv. Aorta.
    3. American Society of Echocardiography (ASE) criteria for appropriates experience and training.
    4. Specific diagnostic usefulness:
        a. Cardiac masses.
        b. Source of systemic embolus:

i. Tumor-atypical location.
ii. LA clot.
iii. Spontaneous contrast.
iv. Vegetations.
v. Atrial septal aneurysm.
vi. PFO with contrast injection.
vii. Aortic plaque.

c. Endocarditis:
i. Value/sensitivity vs. TTE.
ii. Complications: abscess, ruptured chordae.

d. Aortic dissection.
e. Intraoperative monitoring, such as with mitral regurgitation severity post repair.
f. Differentiate forms of left ventricular outflow tract obstruction.
g. Congenital disease.
h. Unable to visualize cardiac structure with transthoracic echo.

5. Contraindications for use.
6. Complications.

H. Methods to evaluate:

1. **After your 1st Echo month**: The fellows should make sure to get enough "hand-on" experience so he/she will be able to perform and standard 2D echocardiogram with routine Doppler for assessment of presence or absence of aortic and mitral stenosis, valvular regurgitation, presence of wall motion abnormalities, LV function, RV function and pericardial effusion. The fellow should also begin to familiar with the basic of performance and evaluation of stress echocardiograms. Start 'pre-reading' echocardiograms as soon as you are comfortable with the computers and have the very basic understanding of echocardiography.

2. **After your 2nd Echo month:** The fellow should learn different types of valvular heart disease, complications of CAD/MI, hypertrophic cardiomyopathy, diastolic function, etc. The fellow should feel comfortable to access patients prior to stress testing and be able to perform exercise and Dobutamine stress studies independently. The fellow can start to generate a preliminary report by 'pre-reading' studies that have entered by the sonographers before the attendings interpret them.

3. **After your 3rd-4th Echo month:** The fellow should now feel comfortable with the basic echocardiography and Doppler examination of most common cardiac diseases. The fellow should be able to recognize all common pathologic entities. Some exposure to congenital heart disease is expected. The fellow should now be able to generate a fairly accurate preliminary report by 'pre-reading' studies before the attending interprets them.

4. **After your 5th-6th Echo month:** The fellow should now have a comprehensive understanding of what constitutes a high quality and complete study. The fellow should understand the correlation with clinical results in a broad range of problems. A fellow that has completed 6 months of echocardiography training should be able to independently perform and interpret an echocardographic study and Doppler that is diagnostic, complete and accurate.

VIII. Stress testing

A. Treadmill.

1. Physiologic responses to exercise, especially as related to sex and age; myocardial oxygen demand and VO_2 max.
2. Indications:
    a. Chest pain: ischemic.
    b. Prognosis and severity of disease: exercise tolerance.
    c. Post MI risk stratification.
    d. Evaluation of therapy

e. Arrhythmias: Provocable

f. Evaluation of functional capacity.

3. Protocols:

a. Bruce.

b. Modified Bruce.

c. Naughton.

4. Contraindications, risks, and safety precautions:

a. Absolute contra-indications.

b. Relative contra-indications.

c. When to terminate exercise test.

5. Test interpretation:

a. Functional capacity.

b. Clinical response

c. ECG response

d. Arrhythmias.

e. Heart rate recovery.

f. BP response.

g. Duke prognostic score and risk assessment/summary.

6. Computer analysis and reporting.

B. Stress echocardiography.

1. Basic principles:

a. Determinants of myocardial oxygen demand.

b. Ischemic cascade (sequence of events in ischemia).

c. Coronary flow reserve.

d. Relationship between coronary artery anatomy and segmental wall motion.

2. Technical and interpretative aspects:

a. Echo views for evaluation of LV wall motion.

b. Types of exercise (pros and cons):

i. Treadmill.

ii. Bicycle:

1. Upright.
2. Supine.

iii. Pacing.

iv. Miscellaneous:

1. Handgrip.
2. Cold presser.

c. Pharmacologic:

i. Dobutamine.

ii. Arbutamine.

iii. Dipyridamole.

iv. Adenosine.

d. End-points:

i. Target heart rate.

ii. Clinical ischemia (intolerable chest pain).

iii. Hypotension (BP < 90 or fall in systolic pressure > 30 mmHg from baseline).

iv. Hypertension (BP > 220 mmHg systolic).

v. Sustained V-tach or SVT.

vi. Obvious new WMA involving $\geq$ 2 segments.

e. Relative contraindications:

i. Unstable angina/acute MI.

ii. Uncontrolled hypertension.

iii. Serious, uncontrolled arrhythmias.

iv. Mobile LV thrombus.

v. Severe aortic stenosis.

vi. Decompensated CHF.

f. Wall motion score index:

i. Evaluate pre- and post-stress.

g. Interpretation:

i. Bayesian analysis.

ii. Criteria for positive test.

iii. Interobserver variability.
iv. Reproducibility.
v. Causes for false positive tests:
1. Nonischemic cardiomyopathy.
2. Septal motion abnormalities (LBBB, s/p CABG).
3. Basal inferior wall.
4. Excessive hypertensive response.
5. Poor image quality.
6. Interpreter bias.
vi. Causes of false negative tests:
1. Single vessel disease.
2. "Mild" coronary stenosis.
3. Left circumflex disease.
4. Inadequate stress.
5. Rapid recovery.
6. Poor image quality.

h. Limitations:
i. Image quality.
ii. Respiratory interference.
iii. Translation, rotation.
iv. Baseline abnormal global or regional function.
v. Arrhythmias.

i. Safety/complications.

3. Accuracy:
a. Sensitivity and specificity:
i. General population.
ii. Coronary disease populations.
iii. By individual vessels.
iv. Single vs. multivessel disease.

b. Comparisons:
i. With exercise ECG.

ii. With nuclear tests.
iii. Stress echo:
1. Treadmill vs. bicycle.
2. Exercise vs. pharmacologic.
3. Comparison of various pharmacologic agents.

4. Dobutamine stress echo:
a. Basic principles.
b. Special topics:
i. Role of atropine.
ii. Hypotension.
iii. LV outflow tract obstruction.
iv. "Optimal" infusion protocol.

5. Prognostic role of stress echo:
a. Various populations:
i. General referral.
ii. Known CAD.
iii. Post myocardial infarction.
iv. Post revascularization (thrombolysis, PTCA, CABG).
b. Pre-op evaluation prior to noncardiac surgery.

6. Myocardial viability:
a. Basic principles:
i. Myocardial stunning.
ii. Hibernating myocardium.
b. Dobutamine stress echo:
i. Interpretation:
1. Biphasic response:
a. Viable myocardium.
b. Predicts recovery of function.

2. Improvement of both low and high dose:
    a. Viable myocardium.
    b. Less predictive of recovery function.
3. No improvement:
    a. Myocardial infarct (scar).
    b. Non-viable myocardium.

ii. Accuracy.

iii. Comparison with other tests (post-revascularization recovery of function):
1. Thallium.
2. PET.

7. TEE-Stress Echo:
    a. Pharmacologic.
    b. Pacing.
8. Stress echo for hemodynamics in valve disease:
    a. Aortic stenosis.
    b. Mitral stenosis.
    c. Valve regurgitation.
    d. Prosthetic valves.
    e. Pulmonary hypertension.
    f. Hypertrophic cardiomyopathy.
    g. Diastolic function.
9. Special topics:
    a. Stress echo in women.
    b. Beta blockers.
    c. LVH, LBBB.
    d. Transplant CAD.

C. Methods of evaluation
1. Included in monthly ECG/Echo self evaluation materials.

IX. Nuclear cardiology

A. Basic principles of nuclear cardiology:

1. Normal perfusion physiology during rest and stress.
2. Abnormal perfusion physiology during rest and stress.
3. Myocardial uptake of radiopharmaceuticals during rest and stress in normal and abnormal myocardial perfusion.

B. Radiopharmacology:
   1. Introduction to various radiopharmaceuticals.
   2. Pharmacokinetics of Thallium 201, 99m Tc Technetium sestamibi, tetrofosmin, Rb 82 (Rubidium).
   3. Advantages and disadvantages of each one.

C. Imaging procedures:
   1. Patient preparation.
   2. Test procedure.
   3. Imaging protocols:
      a. Rest/exercise.
      b. Same day or 2 days.
      c. Dual isotope.
   4. Test procedures.
   5. Image acquisition and equipment.

D. Selecting optimal procedure and radioisotope in patients with:
   1. Poor functional capacity.
   2. Left bundle branch block.
   3. Obesity.
   4. Cardiomyopathy.

E. Interpretation of myocardial perfusion images:
   1. Planar images and interpretation.
   2. Single Photon Emission Computed Tomography (SPECT) and interpretation.
   3. Gated SPECT and interpretation: EF.
   4. Quality control.
   5. Normal myocardial perfusion imaging.
   6. Abnormal myocardial perfusion imaging:

a. Artifacts.
b. Tracer distribution suggestive of CAD.
c. Ischemic (reversible) vs. infarcted (fixed) defects.
d. Specific coronary artery disease vessel.
e. Left main and multiple vessel disease.
f. Lung uptake of thallium.
g. LV cavity size: dilatation, e.g. TID
h. Myocardial viability: stunned vs. hibernating vs. scar.
i. Reverse redistribution.

7. Right ventricular myocardial tracer activity.
8. Extracardiac uptake.
9. Quantification of myocardial tomograms.
10. Bulls-eye imaging.

F. First pass imaging.

G. Radionuclide ventriculography:
   1. Principles and interpretation.

H. Test accuracy, sensitivity, and specificity in relation to:
   1. Degree of CAD and specific vessel.
   2. Female patients.
   3. Baseline EKG abnormalities.
   4. Limited exercise capacity.
   5. Non-diagnostic EKG changes during exercise or non-exercise protocol.
   6. Cardiac drugs: beta blockers.
   7. Collateral vessels.

I. Abnormal results related to:
   1. Subcritical CAD.
   2. Coronary spasm.
   3. LBBB.
   4. COPD.
   5. Cardiomyopathy (ischemic or non-ischemic).

J. Prognostic role:
    1. CAD.
    2. Preoperative evaluation.
K. Cost-effectiveness/benefit ratio.
L. Other imaging modalities:
    1. EBCT.
    2. MRI.
    3. PET.
    4. Coronary CT
M. Final interpretation:
    1. High vs. low risk.
    2. How much LV at risk.
    3. Summed stress score
N. Methods of Evaluation: per Dr. Bateman

X. Cardiac catheterization

A. Basic concepts: Can be found in ACC Guidelines
    1. Indications.
    2. Contraindications.
    3. Leadership and team approach.
    4. Radiation: radiation safety, conservation of fluoro time, can be found in ACC guidelines.
    5. Arterial and venous access:
        a. Needles, sheath, and guidewires.
        b. Femoral, arm approach.
    6. Left heart catheterization technique: how to move patient table, move manifold/stop cock, etc.
    7. Right heart catheterization technique: access, catheters, oximetry, pressures, etc.
B. Basic hemodynamics:
    1. Recognize and measure basic pressures and pressure wave forms (A, V, mean); normal values.
    2. Measurement of cardiac output: thermodilution and Fick techniques.

3. Measurement and interpretation of gradients: peak, mean, and end diastolic gradients, at rest and exercise.
4. Calculation of mitral and aortic valve areas: Gorlin formula.
5. Measurement and calculation of left-right shunts on the basis of oxygen saturation data:
    a. Calculation of pulmonary/systemic flow ratio (Qp/Qs).
    b. Familiarity with other methods of left-right shunts: angiography, hydrogen inhalation, dye dilution.
6. Calculation of pulmonary/systemic resistance ratio (Rp/Rs):
    a. Recognition of inoperability (Eisenmenger's).
    b. Transplant evaluation.
7. Value of rest vs. exercise data: gradients, PA pressure.

C. Angiography:

1. Different types of contrast media:
    a. Hemodynamics.
    b. Complications.
    c. Cost.
2. Types of catheters:
    a. Sizes 5-8 french.
    b. Shapes: Judkins, Amplatz, Vein grafts, Internal Mammary, Pigtail, Cournard, Multipurpose, Swan-Ganz.
3. Coronary arteriography different views (LAO, RAP, AP, cranial, caudal, lateral).
4. Left ventriculography (RAP, LAO):
    a. Volume injected? PSI?
5. Peripheral angiography.
6. Interpretation:
    a. Normal coronary anatomy.
    b. Quantitate stenosis of native coronary vessels, veins, and arterial grafts.
    c. Left ventricle: size, wall motion, EF, degree of mitral regurgitation.

d. Ascending aortography: aortic regurgitation.
e. Congenital heart disease: see individual diseases.

D. Special procedures:
    1. Temporary pacer placement: indications, techniques.
    2. Pericardiocentesis: indications, techniques.
    3. Myocardial biopsy: indications, technique.
    4. Intravascular ultrasound (IVUS):
        a. Indications.
        b. Basic procedure.
        c. Image interpretation.
        d. Quantitative assessment.
    5. Myocardial blood flow: angioscopy, intracoronary Doppler, fractional flow reserve.
    6. Insertion of intra-aortic balloon counter pulsation: risks and post-placement follow-up.
    7. Coronary angioplasty:
        a. Indications.
        b. Post-procedure management.
        c. Understanding of stents, intracoronary radiation: prevent thrombosis; prevent re-stenosis.

E. Complication in catheterization laboratory: prevention, recognition, and treatment:
    1. Local: bleeding, hematoma, retroperitoneal bleeding, dissection, pseudo aneurysm.
    2. Hemodynamic: systemic hypertension, hypotension and shock, elevated left ventricular end diastolic pressure and threat for pulmonary edema.
    3. Cardiac arrhythmias.
    4. Perforation: artery, vein, cardiac chambers, tamponade.
    5. Systemic emboli: stroke, cholesterol emboli (livido reticularis).

F. Methodology:
    1. Read standard text.

2. Case-by-case experience and critique.
3. Review of hemodynamics and angiograms on each case with staff; frequent presentation at cardiac cath conference for peer review.
4. Lectures by senior fellows and faculty on core curriculum material.
5. Included in self evaluation material monthly

XI. **Pathophysiology, diagnosis, and treatment of individual diseases:**

Identify the key symptoms and physical exam findings; how to interpret the 12 lead ECG's and chest x-ray findings; when to order and how to interpret noninvasive tests (echocardiograms, exercise and drug stress tests, nuclear imaging studies); when invasive testing is appropriate and how to interpret the key findings; and how to construct an appropriate differential diagnosis and therapeutic plan.

A. Coronary artery disease:

1. Pathogenesis of atherosclerosis:
    a. Arterial wall structure: elastic arteries, muscular arteries, endothelium, intima, media, adventitia.
    b. Type I – Type III vascular injury on angiography.
    c. Role of endothelium in regulation of vascular tone: nitric oxide.
    d. Endothelial dysfunction with risk factors and in atherosclerotic vascular disease.
    e. Atherosclerotic plaque: fatty streak, fibrous cap, platelets, macrophages, lymphocytes, intimal cellular proliferation.
    f. Hypothesis of atherosclerosis: lipid oxidation, platelet aggregation, inflammation, smooth muscle cells and macrophage activity, metalloproteinases.
    g. Key lipids: LDL, HDL, Triglycerides.
    h. Infectious etiologies: chlamydia, H. pylori, herpes, CMV.
        1. Use of CRP
    i. Homocysteine metabolism.

2. Stable angina pectoris:

    a. Knowledge of ACC/AHA guidelines.
    b. Stress test for risk stratification.

c. Low risk: medical therapy:
    i. ASA.
    ii. Beta blockers.
    iii. Statins.
    iv. ACE-I.
d. High risk: medical therapy plus coronary angiography (PTCA vs. CABG).

3. Unstable angina and NSTEMI :

a. ACC/AHA Guidelines.
b. Pathophysiology: plaque rupture and thrombus formation vs. spasm.
c. Diagnosis: r/o other causes than CAD; r/o atypical chest pain.
d. Initial risk stratification: rest pain, abnormal echo, positive troponin (Braunwald classification).
e. ECG evaluation: normal; □ ST; □ ST.
f. Therapy: ASA, Heparin, IV nitroglycerin, GPIIb/IIIa inhibitor, beta blockers, statins
g. Role of intra-aortic balloon pump
h. Indications for cardiac catheterization and myocardial revascularization.
i. Killip class and relation to Swan-Ganz parameters

4. Acute ST MI:

a. ACC/AHA Guidelines
b. ECG evaluation: anterior vs. inferior vs. lateral.
c. Markers of myocardial necrosis: CK-MB, troponin, myoglobin.
d. Principles of thrombolysis: TIMI classification of coronary flow; thrombolytic agents; absolute vs. relative contraindications; clinical detection of reperfusion.
e. Clinical trials of thrombolytics.

f. Conjunctive therapy with thrombolytic agents: ASA, GPIIb/IIIa inhibitors, IV heparin.
g. Adjunctive therapy: beta blockers, ACE-1, statins, nitrates, calcium blockers.
h. Complications of thrombolytic therapy and conjunctive therapy.
i. Primary PTCA: advantages vs. thrombolysis:
    i. Clinical trials comparing.
j. Hemodynamic complications: CHF, shock, MR, VSD.
k. Rhythm complications: supraventricular and ventricular; heart block.
l. Pre-dismissal priorities:
    i. Risk stratification.
    ii. Risk factor modification:
        1. Emphasis on ACC/AHA and ATPIII Guidelines.
    iii. Which drugs and why?
    iv. Cardiac rehabilitation program?
    v. Follow-up plans.

5. PTCA/Stent vs. CABG:
    a. ACC/AHA Guidelines.
    b. Clinical trials comparing the two.
    c. Post-op management CABG.
6. Prevention of CAD:
    a. Primary vs. secondary.
    b. Knowledge of lipid-lowering trials.
    c. Know ACC/AHA and ATP Guidelines.

B. Congestive heart failure/shock:

1. Pathophysiology:
    a. Systolic vs. diastolic LV dysfunction.
        i. HF with normal EF
            1. high cardiac output

2. Restrictive cm
3. Corpulmonale

b. Acute vs. chronic.

c. Etiology:

i. Ischemic.

ii. Viral.

iii. Toxins.

iv. Tachycardia-induced.

v. Infiltrative disease.

vi. Myocarditis.

2. Initial Evaluation:

a. Clinical:

i. History: dyspnea, PND, fatigue, weight gain, edema,

ii. Physical examination: fluid retention, S3, BMI, Orthostatic BP's, daily weights

iii. Functional class, I >IV

iv. Progression: stages A>D

b. Noninvasive:

i. Laboratory studies: UA CBC, BMP, cardiac enzymes, ABG, thyroid studies, serum iron and ferritin, FLP, LFT's

ii. ECG

iii. Chest radiograph, heart size, pulmonary congestion

iv. Echocardiography: wall motion, EF, valves, effusion & R/O CAD.

v. Exercise testing for functional capacity.

vi. BNP

vii. Nuclear studies: assess CAD and myocardial viability

c. Invasive:

a. Right heart catheterization.

b. Left heart catheterization.

c. Endomyocardial biopsy.

3. Current approach to treatment: knowledge of current published clinical guidelines:

   a. Basic Rx
      i. optimize BP
      ii. Optimize Lipids
      iii. Optimize Glucose, HbAC
      iv. Rate/rhythm control
   b. Oxygen
   c. Beta Beta blockers:
      i. When to use.
      ii. How to use: go low, go slow.
      iii. Trials: MOCHA, CIBIS-II, MERIT-HF, COPERNICUS, BEST, CAPRICORN, COMET.
   d. Diuretics:
      i. Acute IV – Furosemide.
      ii. Chronic/Oral – Furosemide, Metolazone.
      iii. Spironolactone (RALES trial)
      iv. Ultrafiltration
   e. Digoxin indications/contraindications; toxicity.
      i. DIG trial.
   f. Vasodilators:
      i. ACE inhibitors (classic trials: VHEFT, CONCENSUS, SOLVD, SAVE).
      ii. ARB's (CHARM, VALHEFT).
      iii. Hydralazine and nitrates (VHEFT; AHEFT)
   g. Inotropes:
      i. Acute IV Dobutamine/phosphodiesterase inhibitors; Dopamine.
      ii. Chronic oral: lack of support in literature.

   h. Calcium channel blockers:

i. PRAISE-I & II, VHEFT-III, DIDI

i. B-type Natruretic peptide role

j. Anti-coagulation-Warfarin

k. Newer drug approaches (under investigation)

i. Neutral endopeptidase inhibitors.

ii. Tumor necrosis factor receptor blockers.

iii. Endothelin receptor blockers.

l. Resynchronization pacemaker therapy (CRT)

m. ICD to prevent SCD.

n. Surgical interventions:

i. IABP.

ii. LV assist device (LVAD, BiVAD)

iii. ECMO

iv. Total Artificial heart

o. Cardiac transplantation:

i. Indications, contra indications

ii. Postop management: immunotherapy, infections, CAD.

C. Systemic hypertension:

1. Pathophysiology:
   a. Neurohormonal.
   b. Renal.
   c. Vascular remodeling.
2. Causes of secondary HTN: When to screen?
   a. Renal HTN.
   b. Pheochromocytoma.
   c. Primary Aldo.
3. Risk stratification: Target organ damage (JNC VII Guidelines).
4. Treatment:
   a. JNC VII
   b. Systolic HTN only (SHEP, SYST-EUR).
   c. Diabetics:

i. Lower goal (125/80)

ii. UKPDS trials.

d. Renal protection: ACE-1 vs. ARB.

e. More aggressive goals: HOT, UKPDS.

f. LVH regression?

g. Peripheral alpha blockers: ALLHAT.

h. Monotherapy vs. combination drugs.

i. ASCOT trial

D. Valvular heart disease:

1. Aortic valvular stenosis; differentiation from supravalvular and membranous subvalvular stenosis, and aortic sclerosis.
2. Aortic insufficiency:
    a. Acute vs. chronic.
    b. Aortic root vs. valvular etiology.
3. Mitral valve stenosis:
    a. Differential diagnosis: left atrial myxoma, cor triatriatum.
4. Mitral regurgitation:
    a. Acute vs. chronic.
    b. Ruptured chordae tendineae.
    c. Mitral valve prolapse.
    d. Papillary muscle dysfunction/rupture.
    e. Rheumatic.
5. Tricuspid valve disease, with emphasis on secondary to pulmonary hypertension:
    a. Intrinsic: Ebstein's; Endocarditis; Carcinoid.
6. Indications for surgery: valve repair vs. replacement; annuloplasty.
7. Prosthetic valves: current usage, complications, and follow-up.
8. Acute rheumatic fever recognition/prophylaxis.

E. Cardiomyopathies:

1. Dilated Cardiomyopathy (DCM):
    a. Definition.

b. Etiology: important causes: ischemic, valvular, hypertensive, inflammatory, metabolic, muscular dystrophies, peripartum, HIV/AIDS.
c. Prognostic factors in DCM that predict poor survival.
d. Arrhythmogenic Right Ventricular Dysplasia.
e. Role of myocardial biopsy.

2. Hypertrophic Cardiomyopathy (HCM):
   a. Pathology.
   b. Genetic abnormalities.
   c. Variants of HCM – hypertensive, apical, mid-ventricular.
   d. Pathophysiology: LVOT gradient, diastolic dysfunction, arrhythmias.
   e. Histology.
   f. Clinical presentation.
   g. Physical examination.
   h. Echocardiography: 2 D-Echo, Doppler, ASH, SAM.
   i. Natural history.
   j. Management: medical vs. surgical; dual chamber pacing; alcohol septal ablation; ICD.
3. Restrictive Cardiomyopathy (RCM):
   a. Definition and etiology.
   b. Clinical presentation.
   c. Physical exam.
   d. Echocardiography: dilated atria, normal sized ventricles with thick walls, severe diastolic dysfunction, primary amyloidosis.

F. **Pericardial disease**:
1. Acute pericarditis:
   a. Etiology, physical exam.
   b. EKG, CXR, echo, inflammatory markers.
   c. Treatment: NSAIDs, steroids, colchicine, pericardectomy.
2. Pericarditis in AMI:

a. Hemopericardium risk.
b. Dressler syndrome.
c. Postcardiotomy syndrome.

3. Pericardial effusion / Tamponade:
    a. Etiology.
    b. Physical exam.
    c. Hemodynamics.
    d. Pulsus paradoxus (> 10 mmHg ↓ SBP with inspiration).
    e. Echocardiographic diagnosis of pericardial effusion / tamponade (MV inflow interrogation, RA and RV collapse in diastole, lack IVC response to inspiration, abnormal VS motion).
    f. Treatment: when tap; by whom; echo guided.
4. Constrictive pericarditis:
    a. Etiology.
    b. Clinical features.
    c. Physical exam (Kussmaul sign, rapid Y collapse, pericardial knock).
    d. Pericardial calcification (CXR – PA and Lat).
    e. Echo.
    f. Pericardial thickness (MRI).
    g. Hemodynamic findings (RA pressure with prominent "y" decent dip and plateau configuration diastolic pressures.
5. Restriction vs. constriction:
    a. When biopsy indicated? What tissue?
    b. Surgical results..

G. Pulmonary heart disease with emphasis on Pulmonary hypertension (PH):

1. Diagnosis.
2. Etiology:
    a. Pulmonary venous/LA hypertension.

b. Chronic hypoxia with secondary vasoconstriction (lung disease, cor pulmonale).
c. Pulmonary artery obstruction (pulmonary emboli).
d. L-R shunts with increased flow across the pulmonary bed vs. Eisenmenger syndrome.
e. Primary.

3. Clinical profile of each of the above mentioned types.
4. Diagnostic procedures:
    a. Physical exam: RV lift, P_2, PI, TR.
    b. CXR: big PA.
    c. EKG: RAD, RAE, RVH.
    d. Echo: large RV/RA; flat ventricular septum; estimate PA pressure (TR jet, IVC).
    e. Radionuclide studies: r/o pulmonary emboli.
    f. Hemodynamic studies: r/o correctible cause; reversible PH?
    g. When CT-Post angio?
5. Clinical course:
    a. Predictors of survival.
6. Treatment:
    a. High dose CCB.
    b. Prostacyclin continuous infusion.
    c. Endothélium receptor blockers
    d. Phosphodiestersas inhibitors
    e. Anti-coagulation
    f. Lung transplantation.

H. Infective Endocarditis (IE) :

1. Native valve endocarditis :
    a. Men > women 2:1.
    b. Most commonly infected: MV > AV > TV > PV.
    c. Increased risk of endocarditis in congenital heart disease (except secundum ASD).

d. Pathogenesis: vascular endothelium, mucous membranes or other colonized tissues.
e. Pathologic changes/findings: CNS (emboli, mycotic aneurysm), lungs (emboli), spleen (infarct, abscess), skin (petechia, Osler nodes, Janeway lesions), eye (Roth spots), renal (GN).
f. Clinical manifestations – symptoms and physical findings (prolonged fever, new or changing murmur, development of CHF, embolic phenomena, septic complications).
g. IE in IVDA – 5% risk/patient/year; changing/new murmur are important findings but rare; right-sided IE is predominant.
h. Laboratory findings: false positive VDRL and Lyme may occur.
i. Blood cultures: most important finding, at least 3 should be obtained during the first 24 h.
j. Echocardiography: TEE more sensitive than TTE in detection of vegetations and perivalvular abscess.
k. Duke criteria for diagnosis of IE.
l. Microbiology: Streptococcal IE (most common, high cure rate); Staphylococcal IE (2nd most common, frequent cause of IE in IVDA); gram negative IE (narcotics addicts, prosthetic valves, cirrhosis); HACEK (patients with dental infections, IVDA; require 2-3 weeks for isolation); fungal (C. Albicans, Aspergillus); culture negative IE (< 5%, HACEK, prior antibiotics treatment, fungal, intracellular parasites – Bartonella species, Chlamydia species, Coxiella, Q fever); enterococcal IE (relative or absolute resistance to antimicrobial therapy).
m. Antimicrobial therapy.
n. Cardiac surgery – indications: CHF, unresponsive infection, systemic emboli, relapse, abscess.

2. Prosthetic valve endocarditis:
    a. 10% of patients during the lifetime of prosthesis.
    b. Early (< 60 days after implantation), late (> 60 days).
    c. Staph epidermidis: early.
    d. TEE is mandatory.
    e. Treatment: antibiotics; surgical reintervention, hemodynamic status – important in timing the surgery).

I. Congenital heart disease in the adult:

1. Diagnostic approach: TTE, TEE, MRI, cardiac catheterization.
2. Left-Right shunts:
    a. Atrial septal defect: Primum, secundum, sinus venosus.
    b. Ventricular septal defect AV canal: membranous; supracristal; muscular.
    c. Patent ductus arteriosus.
3. Pulmonary hypertension: flow vs. resistance (Eisenmenger).
4. Obstructive lesions:
    a. Pulmonary stenosis: valvular, infundibular, peripheral.
    b. Aortic stenosis: bicuspid aortic valve; associated with other diseases.
    c. Subaortic stenosis: fixed vs. muscular.
    d. Aortic coarctation.
5. Complex lesions:
    a. Tetralogy of Fallot.
    b. Transposition of great vessels: complete or d-TGV; congenital corrected or L-TGV.
    c. Tricuspid atresia.
    d. Single ventricle.
    e. Truncus arteriosus.
    f. Ebstein's anomaly.
6. Common surgical procedures:
    a. Blalock Taussig shunt.
    b. Glenn procedure.

c. Potts procedure.
d. Damus-Kaye-Stansel (DKS) procedure.
e. Mustard/Senning repair.
f. Arterial switch procedure.
g. Fontan procedure.
h. Ross procedure.
i. Management of late complications: CHF, arrhythmias.

7. Postop complications:
a. SVT (Mustard, Fontan, ASD).
b. VT (Tetralogy).
c. PI, AI, MR.
d. Polycythemia: phlebotomy only for hyperviscosity symptoms.
e. RV failure (Mustard).

J. Peripheral vascular disease:

1. Lower Extremity PAD

A. Epidemiology
1. Risk factors
2. Prevalence

B. Prevalence
1. Coprevalence with CAD and carotid disease
2. CAD Risk
3. Limb prognosis

C. Presentation
1. Asymptomatic
2. Claudication
3. Critical limb ischemia
4. Acute limb ischemia
5. Prior revasc.

D. Diagnostics
1. Ankle- and Toe-Brachial Indices, segmental pressure exam
2. Pulse volume recording

3. Continuous-wave Doppler US
4. Treadmill exercise test
5. Duplex US
6. CTA
7. MRA
8. Contrast angiography

E. Treatment

1. Risk reduction
    a. Lipid management
    b. BP control
    c. T2DM control
    d. Smoking cessation
    e. Antiplatelet and antithrombotic drugs
2. Claudication
    a. Exercise
    b. Medications
    c. Role of revasc.
    d. Endovascular repair
    e. Surgery
3. Critical limb ischemia and salvage
    a. Medical Tx
    b. Surgical Tx
    c. Thrombolysis
    d. Surgery

2. Renal Arterial Disease

A. Prevalence and natural History
    a. Clinical endpoints

B. Clues to the diagnosis
    a. HTN @ < 30yo
    b. Severe HTN
    c. Accelerated HTN
    d. Resistant HTN

e. Malignant HTN
f. Azotemia or increased creatinine after initiating ACE-I/ARB
g. Kidney size discrepancy
h. Sudden, unexplained pulmonary edema
i. Refractory anemia

C. Pathophysiology
a. Atherosclerosis
b. FMD
c. Other causes

D. Diagnosis
a. Duplex US
b. CTA
c. MRA
d. Angiography

E. Medical Tx
a. ACE-I
b. ARB
c. CCB
d. Beta-blocker

F. Indications for revasc.
a. Asymptomatic stenosis
b. HTN
c. Preservation of renal fxn
d. CHF
e. USA

G. PTCA

H. Surgery
a. FMD
b. Atherosclerotic

3. Mesenteric Arterial Disease

A. Acute Intestinal Ischemia by Arterial Obstruction

a. Etiology
b. Dx
c. Natural Hx
d. Surgery
e. Endovascular Tx

B. Acute Nonocclusive Intestinal Ischemia
a. Etiology
b. Dx
c. Tx

C. Chronic Intestinal Ischemia
a. Etiology
b. Natural Hx
c. Dx
d. Interventional Tx
e. Surgery

4. Aortic aneurysms, including dissections:
a. Abdominal.
b. Thoracic.
c. Marfan's Syndrome
5. Raynaud's phenomenon/disease/Vasospasm.
6. Venous thrombosis/insufficiency.
7. Lymphedema
8. Acute arterial occlusion
9. Vasculitis

K. Cerebrovascular Disease

1. Types of CVA
a. Sources of CNS emboli
2. Non-Invasive vs. Invasive Evaluation
3. Medical Rx
4. Angioplasty/Stents
5. Carotid Endarterectomy

L. Heart Disease in Pregnancy

1. Pathophysiology
2. High risk adult congential heart and valvular disease
3. Peripartum cardiomyopathy

M. CV Trauma: Penetrating and Non-Penetrating

N. Miscellaneous Diseases:

1. Tumors: primary, metastatic.
2. Neuromuscular disorders:
    a. Primary skeletal muscle disease and associated myocardial involvement.
3. Metabolic disorders, especially thyroid.
4. Hematology/Oncology disease and chemotherapeutic drugs effects on the heart; radiation effect on heart.
5. Collagen vascular diseases/vasculitis.
6. Chronic renal failure : influence in choice of drugs, CA, HTN and interventions for vascular disease.

O. Molecular cardiology :

1. Understand basic terminology in order to interpret the recent literature and future applications.
    a. Example: hypertrophic cardiomyopathy.

P. Methodology**:**

1. Daily teaching rounds: inpatient rotations.
2. Review of each clinic patient workup.
3. Read majority of one textbook, such as Braunwald.
4. Weekly conference, especially ones prepared/given by Fellows.
5. Mayo clinic Board Review

XII. Critical assessment and decision sciences.

A. Critical evaluation of medical literature.

B. Clinical epidemiology.

C. Clinical study design for medical statistics: Biannual course for all subspecialty fellows.

D. Methodology: monthly Journal Club; noon conference presentations; Department of Medicine clinical study design and statistics course; monthly research conference.

XIII. Continuous Quality Improvement.

A. Quality assessment: daily with staff; monthly written evaluation.

B. Clinical rounds: analysis of risk/benefit ratios daily on rounds.

C. Methodology: daily rounds with patients analyzing the individual specific risk/benefit ratio; review history and physicals, progress notes, consults, noninvasive and invasive reports, with special emphasis on invasive procedures.

D. Patient Safety Conference.

XIV. Psychosocial, economic, ethical issues.

A. Constant daily cost/benefit analysis of decisions.

B. Understand principles of:

1. Patient autonomy.
2. Paternalism/beneficence of physician.
3. Justice and allocation of limited resources.
4. Informed consent and truth telling.

C. Methodology: clinical rounds, noon conferences, and individual case conferences, especially on Critical Care Unit patients.