# Suggestions for New Patient Cardiac Evaluation

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Patient Identification: age, gender (man or woman, etc., not male or female), and brief summary of significant demographics.

Chief Complaint (or Reason for Referral)

1) What questions are asked of us - problem and duration in 5-7 words.
2) Source of referral, for feedback to them.
3) If the patient's chief complaint is not the reason for referral, find out what the patient's expectations are.

I) History:
- A) History of Present Illness:
  - 1) Flesh out the chief complaint
  - 2) Don't leave history of present illness until you are pretty sure what is wrong!
  - 3) If things seem confusing, keep asking for clarification
- B) Ischemia symptoms
  - 1) Discomfort (pain) symptoms
    - (a) Location: ischemic pain often poorly localized, may be only in jaw or neck or elbows and skip the chest, often midchest retrosternal, may ascend from lower chest up to upper chest or neck or jaw, one or both sides
    - (b) Quality: usually not sharp, but may range from very mild to quite severe, may not be pain but pressure or squeezing or tightness "Doc, it's not a pain".
      - (i) If a patient has had revascularization, it is usually useful to inquire about the symptoms, especially pain, which occurred before revascularization, and the effect of the procedure on the symptoms, and the similarities and differences between these symptoms and the current symptoms.
      - (ii) If a patient has other sources of pain that have been previously elucidated (GERD, pericarditis, musculoskeletal problems), how does the current symptom compare to those symptoms?
    - (c) If radiation to arms, usually above elbows and usually inner aspect of arms
    - (d) Duration: usually at least a minute, usually less than 20 minutes (MI pain is usually more than 30 minutes),

generally builds up in intensity over time

(e) Provocation: stress or exertion; how much stress or exertion is required to produce symptoms, how consistently are symptoms provoked by a given level of exertion (constant versus variable threshold angina). Use the Canadian Cardiovascular Society classification for angina

**Canadian Cardiovascular Society Functional Classification of Angina Pectoris**

**I.** Ordinary physical activity, such as walking and climbing stairs, does not cause angina. Angina results from strenuous or rapid or prolonged exertion at work or recreation.
**II.** Slight limitation of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, in cold, in wind, or when under emotional stress, or only during the few hours after awakening. Walking more than two blocks on the level and climbing more than one flight or ordinary stairs at a normal pace and under normal conditions.
**III.** Marked limitations of ordinary physical activity. Walking one to two blocks on the level and climbing more than one flight under normal conditions.
**IV.** Inability to carry on any physical activity without discomfort - anginal syndrome may be present at rest.

(f) Relief: rest or Nitroglycerin

(g) Is there more than one type of pain? If so, characterize the other pains as well

(h) Anginal equivalent may be dyspnea or weakness

(i) Levine Sign: patient indicates the location or quality of pain using a clenched fist over the lower sternum, indicates ischemic pain

2) Associated symptoms: feeling of impending doom usually means infarction, diaphoresis often means infarction, nausea may mean infarction, dyspnea or weakness often just are concomitant symptoms of angina

3) "Which bothers you more (or first), chest discomfort or shortness of breath?"

4) It is occasionally helpful to ask the patient what he thinks is the cause of his symptom, and what the reasons are which lead to his conclusion

5) Nonpainful indicators of ischemia ("silent ischemia"), undue weakness or dizziness or dyspnea provoked by stress, relieved by rest

6) Non-ischemic causes of chest pain

(a) Pericarditis: often acute severe chest discomfort, often pleuritic, often worse supine and better sitting, may closely mimic myocardial infarction

(b) Aortic dissection: extreme pain, often maximal at onset, tearing quality, may radiate to the back

C) Cardiac (LV) function or reserve
1) Exercise history - "What's the most strenuous exertion you've done in the last month or so?"
2) Change in exercise tolerance or symptoms
3) Comparison of exercise tolerance to normal age and gender matched population, think in METs (see Appendix 1)
4) Late congestive symptoms ... PND, orthopnea, edema
5) Get good enough exercise history to establish NYHA functional capacity (see Appendix 2) and to form opinion about employment capability
6) Be clear on what limits exercise: cardiac limitation, COPD, musculoskeletal problems, etc.

D) Arrhythmia Symptoms
1) Sensations
(a) In chest - thump, flutter, flip-flop, racing
(b) In neck - slow-fast AVNRT (atrioventricular nodal reentrant tachycardia) with neck palpitations
(c) Timing - suddenness of onset, and more importantly suddenness of offset
(d) Rate, regularity, particulars
2) Perfusion symptoms
(a) Syncope
(b) Sudden weakness, lightheadedness, near syncope

E) Prior cardiovascular history
1) Prior diagnoses or described abnormalities including hospitalizations (make sure the information makes sense, be satisfied that you have all the important information from all the pertinent volumes of the old chart)
2) Murmur (age first noted, whether any limitations on physical activity were imposed, whether endocarditis prophylaxis was recommended)
3) Rheumatic fever - know the Jones criteria (see Appendix 3)
4) Prior cardiac tests and why they were ordered and trends in abnormal results (Echo, Cath, ETT, Holter, CXR, ECG)
5) Prior cardiac treatment and results of treatment (meds, surgery, PTCA, why changed meds, prior indications for treatment; if prosthetic valve whether for stenosis or regurgitation and etiology of valve lesion, or pacemaker; if valve or pacemaker or stent ask patient for the card which describes the implanted material, such as brand and model number)

F) ASHD risks
1) Chol and HDL and LDL, whether treated and results of therapy
2) Hypertension (duration, therapy, degree of control, complications)

3) DM (duration, therapy, degree of control, complications)
4) Tobacco (duration, amount, when stopped, when tried to stop),
5) Family History
   (a) First order relative
   (b) Sx onset age <55
   (c) Can also be more detailed

G) Past Medical History – usual comprehensive
1) complete on medical allergies and intolerance and iodinated contrast allergy
2) clear and accurate understanding of all the current medications and doses and purpose of medications
   (a) effectiveness of medication – "does this medicine help you?"
   (b) ask about prior medications that are no longer taken if something looks a little strange about their medical regimen. Frequently a medical regimen is strange because of prior minor medication intolerance that might not become known if not specifically sought.

H) Review of Symptoms – usual
1) Especially complete on pulmonary, cerebrovascular, and peripheral vascular
2) Renal dysfunction (nephrotoxicity of contrast material)
3) Ask if the patient is enrolled in any RESEARCH protocols

I) Review of old records ("Thar's GOLD in them thar hills!")
1) Preferably before attending rounds
2) Especially most recent data, catheterization data, surgical data.
3) Obtain primary data whenever possible, such as actual thallium scans, echo tapes, treadmill tracings, Cath films, as well as CXR and ECG
4) Also: prior ECG's from computer system, prior operative reports from CT surgery office, may need to obtain older volumes of medical record, <u>obtain faxed data from other hospitals</u>, can obtain some data from VA computer from other VA hospitals

II) <u>Physical Examination</u>

A) General Appearance (thyroid, cachexia, habitus, movements, level of function, etc.)

B) Vital signs
1) Pulse rate (rate is a number) and rhythm, not "RRR"
2) Blood pressure - RA sitting, LA in any with chest pain (R/O dissection), take it yourself to evaluate alternans and paradox
3) Respiration - rate, pattern and ease
4) Have a low threshold for checking orthostatic blood pressure and pulse rate response

C) HEENT - eye,
1) Conjunctivae to evaluate anemia

2) Retina - the only place we can actually see blood vessels - a/v ratio, nicking (a-v crossing changes), cotton-wool patches, flame or dot hemorrhages, exudates, copper wiring

D) Neck

1) Carotid
   (a) Palpation - upstroke and volume
   (b) Palpation - thrill
   (c) Auscultation - bruit
2) JVP
   (a) External jugular less reliable, do not use unless can well see the a and v waves and respiratory variation
   (b) Internal jugular - movement of relaxed SCM muscle
      (i) Technique: ask the patient to extend the neck by raising the chin, relaxing the SCM (don't ask the patient to turn the head to the other side); position the height of the examination table so that the top of the column of venous oscillations may be best seen
      (ii) Mean level of the venous oscillations, should be less than 3 cm vertically above the angle of Louis (sternal angle)
      (iii) Wave morphology, normally the a wave comes before the carotid pulse and the v wave comes after the carotid pulse, and the a wave is larger than the v wave
      (iv) Respiratory variation, normally the venous pressure falls during inspiration
         (1) failure to fall is Kussmaul's sign and indicates inability of the RV to accept a volume load, as in constrictive pericarditis or severe RV failure (RV myocardial infarction)
         (2) response is reversed if patient is receiving positive pressure ventilation
3) Thyroid - size, bruit

E) Chest

1) Symmetry, skeletal deformations
   (a) Scoliosis vs. curvature from cardiac stress (in severe congenital heart disease)
   (b) Straight back
   (c) Pectus excavatum, carinatum
   (d) Hyperexpansion
2) Motion during respiration

F) Lungs

1) Palpation
   (a) Fremitus - tactile

(b) Expansion

2) Auscultation
   (a) Sounds - bronchial, bronchovesicular, vesicular, intensity
   (b) Adventitious sounds - rales (crackles), rhonchi, wheezes

G) Heart
1) Inspection
   (a) Chest wall motion - apex, RV, PA, other
   (b) Other motion - head bob (up and down motion indicates aortic regurgitation) or nod (side to side motion indicates tricuspid regurgitation, with the systolic motion of the head to the left)
2) Palpation of cardiac structures
   (a) Impulse = physical movement in space
   (b) Apex characteristics (size of impulse, presence of presystolic or early diastolic filling waves, duration and character of systolic impulse) and position (standardized only for supine position)
   (c) Other impulses – RV lift, PA tap, Ascending Aortic pulsation, etc
   (d) Thrill = vibratory feeling with some duration, indicates a loud murmur (or bruit if it is an artery)
   (e) Palpable sound = brief vibratory feeling (Palpable S1 is sometimes noted in mitral stenosis, Palpable P2 is sometimes noted in pulmonary hypertension), often called a "tap"
3) Percussion - generally not useful
4) Auscultation described below

III) Auscultation of Sounds
A) S1 and S2 intensity and splitting
1) S1 intensity
   (a) Increased if louder than S2 at the base of the heart, caused by short PR interval, increased contractility from sympathetic or thyroid or similar stimulation, mitral stenosis
   (b) Decreased if softer than S2 at the apex, caused by long PR interval, decreased contractility, LBBB, MR, acute aortic regurgitation (S1 may be absent in acute severe AR)
   (c) Variable S1 intensity from beat to beat, from variable PR interval (AV dissociation or AV block) or atrial fibrillation
2) S1 splitting
   (a) Less important than S1 intensity, frequently encountered
   (b) Increased by RBBB, and in Ebsteins (where the second component of S1 is late and loud, called the Sail sound, could be confused with an ejection sound)
3) S2 intensity

(a) Normally the first part of S2 is A2, aortic closure
(b) A2 is loud with systemic hypertension (look for LV heave)
(c) P2 is loud with pulmonary hypertension (look for RV lift)
(d) P2 is loud if heard at apex, so listen for splitting of S2 at apex

4) S2 splitting
(a) Related to respiratory variation
(i) Accentuated by upright posture - listen first supine, then sitting, then standing if necessary
(ii) Effects reversed in positive pressure ventilation
(b) Normal is physiologic with S2 single during expiration and splitting only during inspiration
(i) A2 comes normally before P2 in inspiration, and they are simultaneous during expiration
(c) Wide physiologic splitting is splitting throughout the respiratory cycle, but increased with inspiration and decreased during expiration
(i) S2 should generally become single during expiration in the standing position
(ii) Caused by RBBB, partial anomalous venous return, pulmonary valve stenosis
(d) Paradoxic splitting is splitting during expiration which closes during inspiration
(i) Caused by reversed split, with P2 preceding A2, sometimes suspected by the split in expiration with the second component (A2) being louder
(ii) Caused by LBBB, significant AS, RV pacemaker, in my experience can be normal variant, especially supine
(e) Single S2, no splitting, usually causes are similar to paradoxic splitting, but also may occur with inaudible P2 or A2, or simultaneous and matched A2 and P2, such as with Tetralogy of Fallot
(f) Wide fixed splitting of S2 is usually due to atrial septal defect with a significant right-to-left shunt, must be confirmed with upright posture

B) S3 and S4
1) Left-sided S3
(a) Best heard at apex during expiration in left lateral recumbent position
(b) Normal in thin young healthy people, in athletes, in pregnant women
(c) In other patients indicates CHF, often but not always systolic dysfunction, or indicates volume load on LV such as with MR or PDA or AR

2) Right-sided S3
   (a) Best heard at LLSB or RLSB during inspiration
   (b) Indicates RV failure or RV volume load, such as TR or PR (rare) or ASD
3) Sounds that can be confused with S3
   (a) Pericardial knock is early diastole, slightly earlier than an S3 (S3 is about 0.14 sec after S2, but a pericardial knock is about 0.10-12 sec after S2), slightly higher pitched than S3, and may increase with inspiration
   (b) Tumor plop of left atrial myxoma is also earlier than an S3, similar to a knock
4) Left-sided S4
   (a) Best heard at apex during expiration in left lateral recumbent position
   (b) Normal in young healthy athletes
   (c) Heard in concentric LVH, ischemic heart disease, hyperkinetic states, acute valvular regurgitation, elderly individuals
5) Right-sided S4
   (a) Best heard at LLSB or RLSB with inspiration
   (b) Heard in RVH from pulmonary hypertension, pulmonic stenosis ("concentric" RVH)

C) Ejection sounds
1) Sound quality is "clicky" – high-pitched, crisp
2) Valvular origin or arterial origin – no difference in sound
3) Pulmonic valvular stenosis or a bicuspid aortic valve produce valvular ejection sounds
4) Systemic arterial hypertension or pulmonary hypertension or idiopathic dilation of the pulmonary artery produce arterial ejection sounds

D) Nonejection systolic clicks – mitral valve prolapse is the most likely cause, but atrial septal aneurysms have also been described to produce a nonejection midsystolic click

IV) Murmurs

A) Systolic (midsystolic, regurgitant)
1) Midsystolic (ejection) murmur occurs after isovolumic contraction time, so there is a brief silent period after S1, then crescendo of the murmur, and then decrescendo, usually with a brief silent period before the appropriate component of S2 (A2 for left-sided regurgitant murmurs, and P2 for right-sided regurgitant murmurs)
   (a) Left-sided midsystolic murmurs are aortic or LV outflow, and will end before A2, and will be best heard during expiration
   (b) Right-sided midsystolic murmurs are pulmonic or RV outflow, will end before P2 (perhaps after A2), and will be

augmented during inspiration

2) Regurgitant systolic murmur occurs immediately with S1 before isovolumic contraction time, so there is no silent period after S1, and the murmurs are usually holosystolic (plateau-shaped) or early systolic, but may also be late systolic, being ended by the appropriate component of S2 (A2 for left-sided regurgitant murmurs, and P2 for right-sided regurgitant murmurs), so there is generally no silent period before S2
   (a) Left-sided regurgitant murmurs are mitral or VSD, and are best heard in expiration, and are increased by maneuvers that increase blood pressure or LV afterload, such as squat or handgrip
   (b) Right-sided regurgitant murmurs are tricuspid murmurs, and are best heard in inspiration or Trendelenburg or supine posture (maneuvers which increase venous return to the right heart); the increase of a TR murmur with inspiration is called a positive Carvallo sign
      (i) In pulmonary hypertension, the murmur sounds holosystolic like mitral regurgitation
      (ii) In the setting of normal pulmonary pressures, the murmur may be shorter, softer, and lower pitched, and can be confused with an innocent pulmonic flow murmur

B) Diastolic (stenosis, regurgitation)
   1) Diastolic stenosis murmurs generally are low-pitched and rumbling in quality (due to the low pressure gradient across these valves), and begin after the isovolumic relaxation time of the cardiac cycle with the opening of the AV valve (mitral or tricuspid)
      (a) The mitral stenosis murmur is best heard at the point where the apex is best felt in the left lateral recumbent position in held expiration, and is augmented by maneuvers which increase heart rate or cardiac output, such as handgrip, exercise, or amyl nitrite inhalation. An exception to the increase with amyl nitrite inhalation is with a diastolic mitral murmur caused by turbulent flow in the absence of stenosis, such as with the Austin Flint murmur of mitral flow in the setting of aortic regurgitation, where amyl nitrite inhalation transiently decreases the aortic regurgitation and consequently decreases the associated mitral flow rumble.
      (b) The tricuspid stenosis murmur is best heard at the LLSB or RLSB, is augmented by inspiration, as well as by maneuvers which increase heart rate or cardiac output
   2) Diastolic regurgitant murmurs begin immediately with the appropriate component of S2, occurring during the isovolumic

relaxation period

(a) Left-sided diastolic regurgitant murmurs (aortic regurgitation) are decrescendo, and high pitched, best heard at the lower sternal border

(i) If the aortic regurgitation is from leaflet pathology, the murmur is generally best heard at the LLSB ("L, left, leaflet")

(ii) If the aortic regurgitation is from pathology of the aortic root, the murmur is generally best heard at the RLSB ("R, right, root")

(iii) Murmurs are increased with maneuvers that increase blood pressure or afterload (like left-sided systolic regurgitant murmurs such as MR), such as handgrip or squat

(b) Right-sided diastolic regurgitant murmurs (pulmonic regurgitation) depend on pulmonary pressure for auscultatory character

(i) Pulmonary hypertensive pulmonic regurgitation is generally a high-pitched diastolic decrescendo murmur beginning with P2

(ii) Pulmonary normotensive pulmonic regurgitation is generally a low or mid-pitched diastolic murmur, which may be crescendo-decrescendo in character, and might be confused with the murmur of tricuspid stenosis

C) Continuous (PDA, venous hum, mammary souffle)

D) Rub (pericardial, pleuropericardial): if a rub is noted, it is wise to have the finding confirmed by a colleague or supervisor quickly, since this finding is frequently evanescent – classically it has 3 components which occur due to rapid changes in heart volume (systole, rapid filling, and atrial contraction), but very often there will be only one or 2 components, and the most common component is systolic, so might be confused with a flow murmur

V) Maneuvers

A) Posture

1) Supine is the standard position for auscultation

2) Trendelenburg, to increase LV size to move the click of MVP later in systole, or to decrease the murmur of HCM, also increases all right-sided murmurs and gallops, due to the increase in venous return

3) Left lateral recumbent, necessary in every patient, may be the only position where S4, S3, MR murmur or MS rumble may be heard at the apex. If the patient is not examined in the left lateral recumbent position, a complete cardiac auscultation has not been performed

4) Sitting or standing, and especially standing after squat
   (a) Upright posture causes decrease in venous return, and results in decreased intensity of right-sided murmurs and gallops
   (b) Upright posture causes an increased dependence of right heart filling on the augmentation by inspiration, so increases the respiratory variation of heart sounds, murmurs and gallops
5) Squat
   (a) Contraction of leg muscles and abdominal musculature causes increased venous return
   (b) The dramatic increase in bends of the arterial tree causes increased afterload of the left heart

B) Respiratory maneuvers
1) Quiet respiration
2) Held expiration
   (a) Good for eliminating the interference of breath sounds which might obscure soft murmurs, such as mitral stenosis
   (b) Decreases the distance between the cardiac structures and the stethoscope head, improving audibility of left heart murmurs
   (c) Especially important for left heart gallops and diastolic murmurs at the apex in the left lateral recumbent position
3) Valsalva
   (a) Maneuver must be performed properly - forced expiration against a closed glottis
      (i) Patient should inspire about half a breath
      (ii) Patient is to bear down as if constipated
      (iii) Patient should cause his face to become red
      (iv) The internal and external jugular veins must distend
   (b) Normal physiologic response
      (i) First few seconds: the increase in intrathoracic pressure causes immediate marked reduction in venous return into the right atrium, and an almost immediate marked reduction in right ventricular stroke volume and output into the lungs. Also there is augmented LV and aortic systolic pressure as the increase in intrathoracic pressure is transmitted to the arterial system
      (ii) After a few seconds, the decrease in venous return results in an emptying of pulmonary capacitance vessels and a consequent fall in left heart filling, with resultant decrease in left ventricular stroke volume and aortic systolic pressure and pulse pressure
      (iii) The decrease in aortic pressure results in a decrease

in carotid sinus stimulation with resultant vagal withdrawal and sympathetic stimulation and then tachycardia. This hypotensive, tachycardic phase is called phase 2, or the strain phase of the Valsalva, and continues as long as the patient continues the forced expiration; arterial vasoconstriction also occurs to help maintain blood pressure

(iv) After the patient releases pressure and returns to quiet breathing there is a brief but immediate further fall in blood pressure due to the withdrawal of intrathoracic pressure, and venous return is brisk into a sympathetically stimulated cardiovascular system. First the right ventricular stroke volume increases, and after 5 to 10 beats, the left ventricular stroke volume increases.

(v) The now adequately filled, sympathetically stimulated LV ejects blood into an aortic system which is somewhat clamped down resulting in an overshoot of aortic pressure above baseline. This elevation of aortic pressure stimulates the carotid baroreceptors that results in vagal stimulation and reflex bradycardia. This bradycardic hypertensive phase is called phase 4, or the overshoot phase.

(c) The physiologic response of the four phases depends on the cardiovascular status and the responsiveness of the autonomic nervous system

(i) If the patient is in a state of excessive sympathetic tone, the heart rate changes may be attenuated

(ii) If the patient has autonomic insufficiency from a primary neurologic disorder or as a complication of diabetes mellitus, the heart rate changes may be attenuated

(iii) If the patient has congestive heart failure there will be a state of excessive sympathetic tone, and the cardiac dynamics will be altered so that the LV stroke volume is not nearly as affected by the decrease in venous return because of the decreased baseline stroke volume and the increased pulmonary blood volume. In severe congestive failure, there will be no tachycardia or bradycardia, and in phase 2 there will be no hypotension

(d) All murmurs decrease during the strain phase except mitral valve prolapse (MVP) and hypertrophic cardiomyopathy (HCM)

(i) HCM generally increases

(ii) MVP often begins earlier and lasts longer, and may become either louder or softer

(e) The rapidity of return to normal intensity of murmurs softened during the strain phase is useful to differentiate left-sided from right-sided murmurs. Right heart murmurs return rapidly, within 2 to 5 beats after release of Valsalva, but left heart murmurs return more slowly, after about 10 beats.

4) Mueller, not very useful

5) All respiratory variation is accentuated by upright posture, as stated above

C) Exercise

1) Handgrip causes increase in afterload, and increases left-sided regurgitant murmurs and gallops

2) Cough may increase cardiac output and heart rate in debilitated individuals, eliciting gallops or MS rumble

3) Situps increase cardiac output and may evoke gallops or MS rumble

D) Carotid Sinus Massage (after safety established by history and exam of carotid)

1) Levine test: carotid sinus massage is performed during chest discomfort with slowing of the heart rate and the patient is asked if the chest pain is worsened. If the patient says the pain is improved, the test is indicative of ischemic pain

2) Rhythm analysis: useful to distinguish sinus tachycardia from other supraventricular tachyarrhythmias

E) Spontaneous arrhythmia

1) Atrial fibrillation

2) PVC

3) The beat after a PVC or after a long R-R interval in atrial fibrillation shows an increase in intensity of the systolic murmur of aortic flow or stenosis or HCM, but no change in the murmur of MR

F) Drugs

1) Amyl nitrite

2) Other

VI) Proving that a murmur is innocent

A) The patient should be free of cardiac symptoms

B) There should be no significant pathology on precordial palpation (no LV heave or RV lift)

C) There should be no significant pathological sounds, such as S3 or S4 in settings other than normal

D) The murmur should be an early-peaking midsystolic murmur

E) The murmur should decrease with the strain phase of theValsalva maneuver (if it increases, then HCM or MVP are likely, and the murmur is

not innocent)

F) There should be no pulmonary or aortic ejection sound (if there is, then bicuspid aortic valve or pulmonic stenosis or pulmonary or systemic hypertension may be present, and further workup is needed)

G) There should be physiologic splitting of S2 (if there is fixed splitting of S2, then ASD is likely, or if there is wide split S2 which moves physiologically in the absence of RBBB, then partial anomalous pulmonary venous return is possible)

H) Soft diastolic aortic or pulmonic murmurs should be carefully excluded (by handgrip or squat for aortic regurgitation and by careful auscultation during inspiration supine for pulmonic regurgitation)

I) Consideration for hyperkinetic circulation should be entertained (AV fistula or malformation, thyrotoxicosis, pregnancy, anemia, etc.)

VII) The effects of electrocardiographic abnormalities on auscultatory findings

A) Changes in ventricular depolarization

1) Left bundle branch block

(a) Delay in left ventricular depolarization and contraction produces paradoxic S2, with about 70% of patients exhibiting this abnormality, more likely in wider QRS

(b) Discoordinate LV contraction produces low rate of rise in LV pressure during isovolumic contraction, leading to soft S1

2) Right bundle branch block

(a) Delay in right ventricular depolarization and contraction produces widely split S2 which moves physiologically

(b) Delay in right ventricular depolarization and contraction produces widely split S1

3) Paced QRS - generally same abnormality as in LBBB

4) WPW

(a) no change in S1 intensity - even though the PR interval is short, leading to the likelihood of increased intensity, the S1 is not increased (not universally accepted), but published since at least 1949 with phonocardiograms

(b) S2 is affected, so that if the delta wave comes from a RV free wall pathway, the S2 is paradoxic, and if the delta wave comes from an LV free wall pathway, the S2 will be widely but physiologically split, like a RBBB

B) Changes in electrical atrioventricular relationships

1) Short PR interval - S1 is loud and constant from beat to beat

2) First degree AV block - S1 is soft and constant from beat to beat

3) AV dissociation from AV block or from other causes

(a) When PR interval is variable, S1 is variable in intensity from beat to beat, with the louder S1 occurring with the shorter PR interval down to a PR interval of about 0.10 sec

(b) Causes

(i) Wenckebach type second degree AV block
(ii) Complete AV block
(iii) AV dissociation
(1) From usurpation
(2) From default

(c) During a beat when the atrial contraction is in mid-diastole with a prolonged PR interval, there may be a diastolic sound (similar in mechanism of genesis of an S4) associated with the atrial contraction, or a soft murmur (the Rytand murmur) coincident with the end of the antegrade flow across the mitral valve caused by the P wave

4) Atrial fibrillation
(a) Irregular RR interval
(i) Most common situation
(ii) S1 is louder on beats after short RR interval
(iii) In mitral stenosis, S1 doesn't vary with RR interval
(b) Regular RR interval
(i) Ventricular paced rhythm
(ii) Junctional or ventricular escape or acceleration
(iii) S1 is constant from beat to beat

VIII) The remainder of the examination
A) Abdomen
1) Liver
(a) Span
(b) Pulsatile (tricuspid regurgitation): necessary to differentiate the pulse of the abdominal aorta from the pulsatile liver of tricuspid regurgitation, which is different in quality and location, and distribution from the abdominal aorta. Additionally, in patients with COPD and hyperinflation, palpation of the epigastrium can reveal the RV impulse with the tips of the fingers (as distinguished from the abdominal aorta which is felt with the pads of the fingers)
(c) Bruit
2) Spleen - size
3) Aortic aneurysm, width of abdominal aorta - bruit

B) Peripheral vascular examination
1) Cyanosis
2) Clubbing
3) Edema (also presacral, especially in bedridden patients)
4) Pulses - for acute chest pain patients, check BP in both arms and frequently check all peripheral pulses in search for dissection of the aorta; for younger hypertensive patients, check for radial-femoral lag seen in aortic coarctation. If an intermittent pulse loss is noted, it is best to have the finding confirmed by a colleague or supervisor quickly

5) Distal hair pattern, nail deformities
6) Capillary refill
7) Varicosities (examined standing)

C) Rectal if none recent

IX) Laboratory Tests

A) ECG
1) Rate - a number, not "regular rate"
2) Rhythm
3) Morphology
(a) Ventricular Depolarization (hypertrophy, infarct, conduction delay)
(b) Ventricular Repolarization (primary or secondary)
(c) Atrial depolarization and repolarization
(d) Other (pacing spikes)

B) Chest X-ray
1) Heart size and contour and great vessels
2) Calcification (coronary, LV, aorta)
3) Pulmonary vasculature
4) Localized lung field abnormalities
5) Skeletal system
6) Extrathoracic abnormalities

C) Other Tests
1) Stress tests - for ischemia, functional capacity, viability, assessing symptoms
(a) Stress
(i) Exercise
(1) Treadmill (ETT, BETT)
(2) Arm crank
(3) Stationary bike
(ii) Pharmacology (Vasodilator, Inotrope)
(b) Test
(i) ECG
(ii) Perfusion (Thallium, MIBI)
(iii) Function (Exercise MUGA - rarely done, or Stress Echo)
2) Extended rhythm tests
(a) Holter
(b) Event monitor
3) Pacemaker evaluation in ECG clinic
4) ?Vectorcardiogram
5) Signal averaged ECG
6) Ultrasound - TTE or TEE, can tailor the exam
7) Radionuclide
(a) Rest perfusion
(b) Gated blood pool (MUGA)

8) More complex tests
   (a) Tilt test
   (b) Electrophysiologic study
   (c) Cath for hemodynamic information (congenital, valvular, myocardial or pericardial disease, primary pulmonary hypertension)
   (d) Coronary angiography and ventriculography

X) Assessment:
   A) Use CCS classification for angina and NYHA Functional classification for CHF
   B) Remember risk factors and etiology (etiology, anatomy, physiology)
   C) Remember predisposing and precipitating factors
   D) Every medicine the patient takes should have an appropriate problem noted on the problem list

XI) Plan: every problem has a plan, and every plan has 3 parts
   A) Diagnostic
   B) Therapeutic
   C) Patient Education - often the key to preventing readmissions

XII) Management notes:
   A) Know how the result of any test ordered will affect management, and know every test result
   B) Write the orders routinely for the am labs and CXR, especially in the CCU
   C) For each patient every day:
      1) See the patient
      2) Identify yourself to the patient
      3) Interim history, change in symptoms, appearance of new symptoms
      4) Directed exam - touch the patient, generally auscultation daily
      5) Check exams ordered, including telemetry results for the last 24 hours, learn to assess trends and events stored in the monitoring system
      6) Check response to therapeutic maneuvers, both improvement and adverse effects
      7) Ask the patient if he/she needs anything
      8) Check for smile
      9) Explain status to patient, including prognosis, expected response to therapy, recommendations for physical and occupational and sexual activity
   D) To make things go smoothly
      1) Clearly communicate with the heart station on the specific needs for timing of noninvasive studies, with appropriately annotated request forms (for TTE, TEE or noninvasive stress tests)
      2) Ensure NPO status for patients who need exams requiring NPO status
      3) Follow-up test results on patients near discharge on the afternoon of the same day as the test, especially echocardiography or stress

tests

4) For patients referred to CT surgery, be sure the appropriate pre-operative battery of tests is accomplished

E) Chart Notes

1) Note on every patient every day.
2) Sign all verbal orders.
3) Note attending concurrence with transfer out of intensive care.
4) Date and sign all entries, especially consent forms.

F) Discharge summary reminders

1) Send a copy to the referring provider/institution (good idea to call the provider as well)
2) For CHF patients, be clear on discharge weight and vital signs and medication doses
3) For patients with atherosclerosis, be sure plans for risk factor modification are well underway and that the goals are clearly stated in the summary, including dietary prescription, exercise prescription and activity and employment recommendations, lipid lowering therapy, blood pressure control regimen, documentation of advice to stop tobacco product use, and diabetes control measures if needed
4) Include thought processes for why the diagnostic and therapeutic options were chosen as they were, as well as the results of tests and therapies

XIII) Disclaimer: this set of suggestions is not meant to substitute for textbook reading

# Appendix 1: Exercise Capacity Assessment in MET level

I) Physiology of exercise

A) Oxygen delivery to the body increases (mainly muscle blood flow)

1) Metabolic equivalent (MET) definition: Approximately 3.5 ml $O_2$/kg body wt/min, Resting sitting $O_2$ consumption

2) Increase in cardiac output at maximal exertion (normal resting is 5.8 L/min or 3.4 L/min/$m^2$)- five or six fold (up to 25-30 L/min)

(a) Stroke volume (resting normal is 80 ml or 47 ml/$m^2$ with diastolic volume of 120 ml and ejection fraction of 0.67)

(i) Increase maximally of 10-15% during supine exercise

(ii) Increase maximally of 30-100% during upright exercise

(b) Heart rate (normal resting is about 72)- maximally can increase up to perhaps three fold, depending on age and conditioning

3) Increase in peripheral $O_2$ extraction - up to three fold, from 25% to 75%

4) Increase metabolic activity up to about 18 fold (6-fold cardiac output times 3-fold extraction), or 18 METs, with some athletes able to increase even to 24 METs

5) Pulmonary and hematologic considerations are normally insignificant: When pulmonary disease limits exercise tolerance, the peak heart rate achieved at exhaustion is generally submaximal, i.e. there is some "heart rate reserve" remaining

6) Submaximal exercise results in partial changes in heart rate, stroke volume and extraction, generally in a relatively linear fashion

7) Examples of activities and their MET level are shown on next page

8) Clinical significance of maximal comfortable MET level

(a) <5 METs: Poor prognosis: usual limit immediately after myocardial infarction, peak cost of basic activities of daily living

(b) 10 METs: Prognosis with medical therapy is as good as CABG

(c) 13 METs: Excellent prognosis regardless of other exercise responses

B) Oxygen consumption by the myocardium increases

1) Demand

(a) Estimated by the product of HR * contractility * wall tension

(b) LaPlace relation: Tension = pressure * radius/(2* wall thickness), T=Pr/2h

(c) Clinically can estimate oxygen demand by the product: HR * SysBP; called the double product or the rate-pressure product

(d) Resting double product is about 8,000 (HR 70 and SysBP 120)

(e) Can increase to about 40,000 at peak exercise (HR 200 and SysBP 200)

2) Supply

(a) Coronary blood flow - can increase with exercise up to a maximum of four or even 5-fold greater than rest

(b) Myocardial oxygen extraction - (near) max at rest, no increase with exercise; coronary sinus $O_2$ saturation is about 25% at rest

| MET LEVEL, estimated | ACTIVITY Examples |
|---|---|
| 0.7 | Resting supine |
| 1.0 | Resting sitting |
| 2.0 | Walking 2 mph level ground |
| 3.0 | Walking 3 mph level ground |
| 3.0 | Bicycling 5 mph |
| 3.0 | Housework |
| 3.0 | Mowing lawn (power mower) |
| 3.0 | Walking 2 mph up 3% grade |
| 3.5 | Walking 1.7 mph up 5% grade (Stage 2 Modified Bruce) |
| 4.0 | Walking 4 mph level ground |
| 4.0 | Walking 3 mph up 2.5% grade |
| 4.0 | Walking 2 mph up 5% grade |
| 4.0 | Gardening (weeding, raking leaves, no lifting), manual labor (light carpentry, painting) |
| 4.0 | Dancing (rhumba) |
| 4.0 | Bicycling 9.5 mph |
| 5.0 | Walking 1.7 mph up 10% grade (Stage 1 Bruce 3 min) |
| 5.0 | Walking 3 mph up 5% grade |
| 5.0 | Chopping wood, washing the car |
| 5.5 | Badminton |
| 5.5 | Ice skating |
| 6.0 | Dancing (fast) |
| 6.5 | Practicing karate or judo |
| 6.5 | Roller skating |
| 6.0 | Tennis (doubles) |
| 6.8 | Skiing (water or downhill) |
| 7.0 | Climbing hills, digging, spading soil, singles tennis, carrying 60 lbs. |
| 7.0 | Swimming (fast) |
| 7.7 | Field hockey |
| 8.0 | Moving heavy furniture, jogging slowly, climbing stairs quickly, carrying 20 lb. upstairs |
| 10.0 | Jogging 6 mph, walking briskly uphill |
| 11.0 | Cross country skiing, full court basketball |
| 12.0 | Rope skipping, running 8 minutes/mile |
| 12.0 | Playing squash |

METs = 1.11 + 0.016*(duration in seconds) for the Bruce protocol

## Appendix 2:New York Heart Association (NYHA) Classification for CHF

The NYHA functional capacity:

Class 1. No limitation of physical activity; no undue symptoms with ordinary physical activity.
Class 2. Slight limitation of activity. Symptoms with ordinary activity.
Class 3. Marked limitation of activity. Symptoms with less than ordinary activity.
Class 4. Symptoms with any physical activity or even at rest.

From NYHA, 1994.

## Appendix 3: Modified Jones Criteria for Rheumatic Fever

Diagnosis: T. Duckett Jones criteria, update 1992, Needed are 2 major, or 1 major and 2 minor, in setting of evidence of prior Group A Strep infection

<table>
<tr><th>Major Manifestations</th><th>Minor Manifestations</th><th>Supporting Evidence for Strep</th></tr>
<tr><td>Carditis</td><td rowspan="2">Clinical - Arthralgia<br>Clinical - Fever</td><td>Positive throat culture</td></tr>
<tr><td>Polyarthritis</td><td rowspan="2">Positive rapid streptococcal antigen test</td></tr>
<tr><td>Chorea</td><td rowspan="2">Laboratory: Elevated ESR<br>Laboratory: Elevated C-reactive protein</td></tr>
<tr><td>Erythema marginatum</td><td rowspan="2">Elevated or rising streptococcal antibody titer</td></tr>
<tr><td>Subcutaneous nodules</td><td>ECG: Prolonged PR interval</td></tr>
</table>