Basics of EKG Interpretation:

A Programmed Study - Barbara Ritter Ed.D, FNP

Description – The course is designed as an elective to give the advanced practice nurse, involved in the care of patients with cardiopulmonary problems, a basic introduction to the principles of EKG interpretation. The course is in a self-programmed format whereby the student reviews EKGs with accompanying case histories and answers. The EKGs selected represent commonly occurring cardiopulmonary problems in the primary care setting and provide additional means by which nurses can correlate their knowledge of pathophysiology and cardiopulmonary physical assessment (theory and skills) with findings demonstrable on an EKG.

Objectives:

Identify structures demonstrable on EKG.

Recognize a normal EKG.

Recognize and name the EKG signs of asystole, atrial fibrillation, atrial flutter, bradycardia, premature atrial contractions, premature ventricular contractions, ventricular fibrillation, angina, myocardial infarction, CHF, and COPD seen with cardiopulmonary disease.

Correlate physical signs and symptoms of cardiopulmonary disease with EKG findings.

Prerequisites:

Graduate standing.

Consent of instructor.

Course Requirements:

Pre-test and Post-test of EKG interpretation administered by instructor.

Grading

Choice of letter grade or satisfactory/unsatisfactory. A satisfactory grade is obtained by achieving 80% or greater on the post-test. The post-test may be retaken as many times as necessary in order to achieve a passing grade.

Required Web Sites:

Basics of EKG Interpretation: A Programmed Study

http://nps.freeservers.com/

Recommended Texts:

Recommended Schedule for EKG Practicum:

This schedule is given to be used as a guideline to the practicum. The order of the EKGS has been selected to build and reinforce prior learning. The material to be read may not follow exactly, but may be utilized as a reference. Basics of EKG Interpretation: A Programmed Study (BEI) is a self-learning module which you may use at your own pace.

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**EKGS: SECTION ONE**

**PART I: THE BASICS**

**ELECTRODE PLACEMENT FOR 12-LEAD ECGs**

**Limb Leads** (I, II, III, aV_R, aV_L, aV_F) are obtained from electrodes labeled as LA or left arm, RA or right arm, LL or left leg, and RL or right leg, which are positioned either on the distal (wrists and ankles) or proximal extremities (shoulders and groins). Left leg electrodes must be placed below the heart (but not on the chest), preferably below the umbilicus, to get an accurate lead II, III, and aV_F. The six limb leads are used to determine frontal plane axis. The sensing electrode for each lead "looks back" at the heart from its positive or sensing electrode. Thus leads II, III, and aV_F "look" at the inferior surface of the heart; leads I and aV_L, "look" at the left or lateral side of the heart; and aV_R, "looks" at the right side of the heart.
Limb leads are usually labeled but also occasionally color coded so that:

Right arm — “White is on the right.”

Right leg — "Green is for go." (Right leg is gas pedal.)

Left leg — "Red is for stop." (Some brake with left leg.)

Left arm — black lead.

**Unipolar Leads** (all have “V” in their names) — $aV_R$, $aV_L$, $aV_F$, and the precordial leads $V_1$-$V_6$.

Require more electrodes on the patient (a minimum of 4-5).

All have an "exploring" electrode which "looks" directly at the heart from its site of placement.

All also require three "indifferent" electrodes (RA, LA, and LL) but which do not contribute toward the tracing.
Addendum: V3 is placed halfway between V2 and V4.

All precordial leads bisect at AV node [point toward the AV node in a horizontal plane]:

Right chest (or anterior) leads — V1, V2; also aVR.

Septal leads — V3 and V4 — located over the interventricular septum.

Left chest (or lateral) leads — V5, V6; also I and aVL.

V₁ and V₂ mirror changes occurring from the posterior side of the heart. None of the usual electrodes are directly adjacent to the posterior surface of the heart.

If additional posterior leads need to be seen, (e.g., to diagnose a true posterior infarction) do another 12 lead ECG but move 3 electrodes to these positions:

V₇ = same horizontal plane as V₄-V₆, PAL (posterior axillary line).

V₈ = same horizontal plane as V₄-V₆, mid-scapula.
If additional right chest leads need to be seen, (e.g., to diagnose a right ventricular infarction) do another 12 lead ECG but move 4 electrodes to these positions:

\[ V_{3R} \] = halfway between \( V_1 \) and \( V_{4R} \).

\[ V_{4R} \] = 5 RICS at MCL.

\[ V_{5R} \] = same horizontal plane as \( V_{4R} \) at AAL (anterior axillary line).

\[ V_{6R} \] = same horizontal plane as \( V_{4R} \) at MAL (mid-axillary line).

**PLACEMENT OF BIPOLAR LEADS (I, II, III, MCL₁, MCL₂) FOR MONITORING**
RA = right arm  LA = left arm

RL = right leg  LL = left leg

**Lead I** — LA is +, RA is -, RL is ground.

**Lead II** — LL is +, RA is -, RL is ground.

**Lead III** — LL is +, LA is -, RL is ground.

*NOTE:* In placing electrodes on the chest, always find the angle of Louis (palpable junction of the manubrium and body of the sternum). Slide to the side and you are on the second rib with the 2nd intercostal space just below it. Count down to the correct interspace from there.

**NOTE:** LL electrode should always be placed below the umbilicus in order to avoid problems in patients with ventricular hypertrophy.

Bipolar leads look from a positive pole toward a negative pole. The positive electrode "looks" directly at the heart from the site where it is placed. Ground electrodes do not contribute to the tracing. "Quick look" defibrillator paddles are also bipolar.

**Lead MCL₁** — use lead I selection on monitor; positive electrode placed in 4ICS (4th intercostal space) at the RSB (right sternal border); negative electrode placed on L shoulder; ground may be placed anywhere.

**V₁** is the single best lead for diagnosing dysrhythmias; MCL₁ is a substitute for V₁ which can be recorded from 3 lead-wire patient cables.

MCL₁ is very similar to, but not identical to, pattern seen in V₁ precordial lead.

Rhythms which can be distinguished in V₁ or MCL₁ but not in other leads include those with a widened QRS complex such as right versus left ventricular rhythms, right and left bundle branch blocks, and differentiation of supraventricular rhythms with aberration from ventricular rhythms. Also, P waves are often visible in V₁ and MCL₁ when they are invisible in other leads because the exploring electrodes is the one closest to the atria.

**Lead MCL₆** — almost the same electrode placement as MCL₁, but the positive electrode is placed in the 5ICS in the left MAL (mid-axillary line). MCL₆ is very similar to, but not identical to, pattern seen in V₁ precordial lead.

**ECG PAPER** — standardized so we can compare patients.

**Time** is measured from the L to the R — **one large box = 0.20 sec and one small one = 0.04 sec.**

The rate of the ECG machine is 25 mm/sec. Marks on the upper or lower border of paper fall every 3 sec or 3 inches.

**Voltage** or current strength is determined from the magnitude or height of the various waveforms and is measured in mV or mm — **one small box** normally = 0.1 mV or 1 mm and **one large box** = 0.5 mV or 5 mm.

Voltage strength can be adjusted when recording the ECG. Thus if the waveform is especially large, as in the precordial leads of a patient with ventricular hypertrophy, or especially small, as in a patient with severe lung disease, the size of the waveforms can be adjusted to fit the paper. A calibration mark is thus made at the beginning of the recording to denote whether it is at full-, half-, or, occasionally, double-amplitude. The normal calibration mark should be a full 10 mm for a 0.1 mV calibration. At half-amplitude each vertical block equals 0.2 mV; at double-amplitude each vertical block equals 0.05 mV.
If an electrical impulse is moving toward the sensing electrode a positive (upright) deflection is recorded; if the impulse is moving away from the sensing electrode, a negative (downward) deflection is recorded; when an impulse travels perpendicular to (90° away from) the sensing electrode, a straight line (isoelectric deflection) or an equiphasic (small amplitude complex with approximately equal height of upward and downward deflections) deflection is recorded.

- **Monophasic** waveform = complex (e.g., P or T) peaks in one direction, either all positive or all negative.
- **Biphasic** = complex has a positive peak and a negative peak (nadir).
- **Triphasic** = three points to the complex, e.g., rsR'.
- **Equiphasic** = negative part of the waveform is equal in size to the positive portion.

**NORMAL ECG WAVEFORMS AND INTERVALS:**

**P Waves** — represents depolarization of the atrial myocardium. (Sinus node depolarization is too small in amplitude to be recorded from the body surface so it is not seen.)

The normal P wave is:

- Not wider than 0.11 sec (under 3 little boxes on the ECG paper).
- Not taller than 3 mm.
- Not notched or peaked; does not have an excessive trough if biphasic.
- Positive and rounded in leads I, II, and aVF in 94% of normals; usually upright in V₁-V₆.

Inverted P waves in these leads are either abnormal or due to improper lead placement.

- Negative in aVR.
- Positive, negative, or biphasic in lead III, aVL, and V₃.
- P wave axis = + 60°.
- Normally has 1:1 ratio with the QRS and should be regular.

Initial portion of the P is largely a reflection of R atrial depolarization and the terminal portion reflects depolarization of the L atrium. The P waves should all look alike.
ECG WAVEFORMS

**PR Interval** — represents atrial depolarization plus the normal delay at the AV node.

- Normally = 0.12-0.20 sec. (No longer than one large box.)
- Increased in length if AV conduction is prolonged (first-degree AV block).

**PR Segment** — begins at the end of the P wave and ends with the onset of the QRS complex.

- Should be isoelectric (flat).
- Can be elevated with atrial infarction or pericarditis.
- Can be depressed if there is a large repolarization wave (T_p) following the P wave.

**QRS Complex** — represents depolarization of the ventricular myocardium. (Depolarization of the AV node, His bundle, bundle branches, and Purkinje fibers are too small in amplitude to be detected by electrodes on the body surface.)

- All positive waves of the QRS complex are labeled R waves. If there are more than one, the second one is labeled R'. An upper case capital letter describes a sizable R wave (> 5 mm); a lower case letter describes a tiny r wave (<4 mm).
- Negative waves of the QRS are labeled with Q waves (preceding the R wave) or S waves (following the R wave). Subsequent negative waves are labeled S' waves.

Relative size is denoted by upper or lower case letters.

- Although termed the "QRS" complex, many complexes do not contain all three waves.
• **Monomorphic** = one shape; refers to a cardiac rhythm in which each QRS complex has a consistent pattern as, for example, monomorphic ventricular tachycardia which arises from one specific location. Older term, "unifocal" means the same thing.

• **Polymorphic** = multiple shapes such as polymorphic ventricular tachycardia which arises from multiple sites in the ventricles. Older term, "multifocal" means the same thing.

**Normal QRS Characteristics:**

• 0.07-0.11 sec in width. QRS widths often vary in different leads. The *widest* QRS measurement on the 12-lead ECG is the *correct* one. Best leads to look at are usually leads I and V1.

• Should not be smaller than 6 mm in leads I, II, and III and nor should it be taller than 25-30 mm in the precordial leads.

• **R Wave Progression** — in the precordial leads, the QRS starts off primarily negative (rS) in V1 and gradually becomes primarily positive (qRs) with the tallest R wave in V5 or V6.

The transition from mostly negative to mostly positive normally occurs between V3 and V4. Normally the R wave in V6 is always less than the R wave in V5. Precordial R waves are very sensitive to lead placement and this must be considered in interpreting R wave progression.

![NORMAL R WAVE PROGRESSION](image)

**Early R waves** — R waves in leads V1 and V2 as large as those in the next several leads can reflect *posterior infarction*, lateral MI, right ventricular hypertrophy (RVH), or septal hypertrophy.

Tall R wave In V1 — consider RVH, posterior MI, or Wolff-Parkinson-White (W-P-W).

"Low" R waves in the right precordial leads — most likely due to left ventricular hypertrophy (LVH) but also consider left anterior fascicular block (LAFB), COPD, or MI. R wave < 2-3 mm in V1 is abnormal unless there is LVH. LVH causes loss of R height from V2-V5 without MI. Loss of R height between V1-2 or V2-3, in the absence of LVH suggests *anterior MI*.

**Poor R Wave Progression** — R waves do not begin to dominate QRS until V5 or V6. *This may represent infarction or injury of the anterior LV and carries almost as much significance as Q waves.*

**Q Wave** — a negative wave preceding the R wave. Not all leads normally record a Q wave. Normal Q waves represent *septal depolarization* and they must be distinguished from *pathologic* Q waves which indicate myocardial infarction.

**Normal Q wave:**

• *Present only in leads I, aVL, V5, and V6 (left lateral leads).*

Small in aVF and V3 — normal variant.

If there is no Q where there should be one — septal fibrosis is present.

If large — myocardial damage. Large, diagnostic Q waves represent altered electrical activity in the myocardium due to transmural myocardial damage.

• *Less than 0.04 sec.*

• *Not deeper than one-third of the QRS complex.*
• "Diagnostic" Q wave in V₁, aV₅, and III may be present without indicating myocardial damage.

**ST Segment** — represents the time when ventricular cells are in the plateau phase (phase 2) of the action potential in which there is no current flow and thus little, if any transmembrane gradient (transmembrane potential hovers around zero). QRS and ST segment also represent a time when the ventricles are in their absolute refractory period and will not respond to stimulation.

• ST segment starts at the J point (junction of the end of the QRS complex with the ST segment) and ends at the beginning of the T wave.

• ST segment (as well as the PR and TP segments) should be isoelectric (flat).

• ST segment always has a smooth contour unless something else is added to it.

• Clinical importance is related to its level relative to the isoelectric line rather than to its duration.

**Primary ST-T Wave Changes** — ST deviation and T wave abnormalities are seen with myocardial ischemia.

• Vessel occlusion causes S-T elevation.

Problem usually transmural.

See localized ECG changes and one can predict which artery is involved.

*Criteria for thrombolytic therapy = ST elevation in two electrically contiguous leads.*

Adjacent precordial leads are "contiguous." To figure out which limb leads are "electrically contiguous," use a hexaxial diagram. (The diagram used to figure out numerical electrical axis - see page 29.) Start thrombolytic therapy by giving aspirin as soon as possible.

• ST deviation due to total coronary occlusion does NOT start with ST depression and progress to ST elevation. Rather, **ST elevation is the first and only ECG indication of total occlusion.** If the ST elevation is transient, it is then termed a "pattern of injury."

**Secondary ST-T Wave Changes:**

*Secondary means there is an explainable cause.*

**Secondary ischemia causes S-T depression:** associated with increased oxygen demand with limited blood flow; usually due to subendocardial ischemia as the endocardium is at the tail end of the blood supply.

ECG changes are more global; therefore one can't predict what coronary artery is involved.

ST deviation and T wave abnormalities are seen with conditions other than myocardial ischemia such as a wide QRS complex or secondary to effects of medications.

It is possible to have both primary and secondary changes (e.g., bundle branch block plus ischemia).

In this case, the ST segment may appear to normalize because both ST depression and elevation are occurring simultaneously.

**QT Interval** — measurement of the refractory period or the time during which the myocardium would not respond to a second impulse; measured from the beginning of the QRS complex to the end of the T wave.

• Best leads to measure the QT are V₂ or V₃.

• If there is a U wave visible, the measurement is made to the end of the U wave and is called the Q-T_U interval.

• **Q-T interval should be roughly less than half the preceding R-R interval.**

• It is longer with slower rates and shorter with faster rates. Normals also vary with age and gender.

• If a Q-T table is not available, the Q-T interval can be corrected for heart rate using Basset's formula:
- If a patient develops a wide QRS complex (a problem with depolarization) such as a bundle branch block, the QT interval will be increased. Thus, a long QT interval is not thought of as abnormal in patients with a wide QRS complex unless you have subtracted the extra width of the QRS from the QT interval and still found it prolonged.

- If the rhythm is irregular, measure the QT relative to the rate of the prior R-R interval.

**QT Dispersion** — QT is measured on the same beat in all 12 ECG leads and the shortest QT interval is subtracted from the longest QT interval. Recent evidence indicates that, if there is much of a difference, heterogeneous refractoriness exists in the heart muscle and the patient may be at higher risk of cardiac death from development of ventricular tachycardia/fibrillation, especially from any proarrhythmic effects of antiarrhythmic drugs.

**JT Intervals** — JT interval reflects repolarization alone, not both depolarization and repolarization.

Sometimes used to measure the refractory period in patients who have been started on a Na+ channel blocker antiarrhythmic drugs (e.g., Quinidine, Pronestyl, and other class I agents). This is because such drugs slow depolarization, slightly prolonging the QRS complex.

**T Wave** — represents repolarization of the ventricles.

- Earliest time ventricles can respond to another stimulus usually coincides with the apex of the T wave.

- *T wave should have the same polarity as the QRS complex. Thus if the QRS complex is primarily negative, the T wave should be negative.*

- There are literally dozens of conditions that cause abnormal-looking T wave in leads with positive QRS waveforms.

**T waves are very fickle; not as reliable as ST depression or elevation in diagnosis of ischemia.**

- Myocardial ischemia/non-Q waves.

- Normal variants (juvenile T wave pattern; early repolarization).

- Cerebrovascular accidents (especially intracranial bleeds) and related neurogenic patterns (e.g., radical neck dissection, Stokes-Adams syndrome).

- Post-tachycardia or post-pacemaker T wave pattern.

- Intermittent left bundle branch block (LBBB).

- Left or right ventricular overload (e.g., classic “strain” patterns or apical hypertrophic cardiomyopathy.

- Secondary T wave alterations due to bundle branch blocks or Wolff-Parkinson-White patterns.

- Respiratory alkalosis.

- *It is no longer believed that the first sign of infarction is T wave inversion.*

Textbooks stating that are old and need revision.

**U Wave** — A shallow, gently curved wave (in the same direction as the T wave but smaller) following the T wave. May not be visible at all.

- It is not clear what the U wave represents. May represent repolarization of intramural Purkinje conduction system.

- Conditions which may cause a pronounced U wave are antiarrhythmic drug effects, especially when the patient is prone to proarrhythmia (drug-induced arrhythmias such as polymorphic ventricular tachycardia or "torsades de pointes").

- Prominent U wave — usually suggests digitalis toxicity or hypokalemia. Also seen in bradycardias.
Evolution of ECG Changes in Myocardial Infarction:

• J point elevation and ST elevation in leads facing the damaged wall; represent total occlusion of the coronary artery supplying that area — begins in first minutes.

• Start getting Q waves which are $>0.04$ sec wide (in the precordial leads this is manifested by loss of R waves); presence of pathologic Q wave tells you some cardiac cells have died; other cells can still be salvaged with prompt initiation of thrombolytic therapy. This Q wave reflects the zone of necrosis. These are abnormal (pathologic) Q waves.

• Get T wave inversion. The ST elevation reflects the zone of injury; the T wave changes reflect the zone of ischemia.

• Long-term — ST segment returns to the isoelectric line approximately 2-6 weeks after the MI (unless the patient develops a ventricular aneurysm) and the T waves normalize although they sometimes remain inverted for months. The Q waves remain and permanently alter the 12-lead ECG.
Besides these diagnostic changes there are reciprocal changes in leads on opposite side of the heart.

Reciprocal changes:

• No Q wave.

• Increased height of R wave.

• ST segment depression.

Upright T wave.

Controversy currently whether or not reciprocal changes must be in the same plane (horizontal or frontal) as the ST elevation. Unresolved.

INFARCT LOCATION — infarcted tissue is electrically silent.

A MI may be described as subendocardial, endocardial, subepicardial, epicardial, intramural or transmural depending on the location and the extent of damage.

• A transmural MI involves the full thickness of the wall. Most MIs are transmural.

Usually Q waves are present or R waves are lost.

Indicative changes (IC) = Q wave (QS), ST elevation, and T wave inversion noted in leads facing area of damage.

Reciprocal changes (RC) = absent Q wave, ST depression, and tall, upright T waves noted in leads opposite area of damage.

"Q-wave infarction" = better term. Pathologic Q waves are seen best in leads with big R waves.

Pathologic Q wave defined as:

Any Q wave in V₁-V₃.

Q wave > 20 mm in V₄.

Q wave > 30 mm in V₅.

Q wave > 30 mm in V₆.

Q wave > 30 mm in I, II, aV₅, or aV₆.
A subendocardial MI is limited to the inner half of the myocardium; may extend transmurally or recur within 6 months.

Subendocardial MIs occur because coronary flow is compromised by systole and by high filling pressures during diastole. (Normally subendocardial flow is greater during diastole while subepicardial flow is greater during systole so overall perfusion is similar.) With coronary occlusion and no reperfusion, subendocardial cells die in approximately 15-30 min.

Transient ST segment depression and sustained T wave changes; QRS changes depend on depth of infarct.

Shallow — depressed ST without alteration of QRS.

Somewhat deeper — depressed ST; R waves lowered.

Somewhat deeper yet — initial R wave may be replaced by QS in lead overlying infarct.

Better terms = "non-Q wave" infarction.

Significant Q waves are not seen. Mainly see T wave inversion where they shouldn't be inverted.

ST changes may or may not be present.

Not treated with thrombolytics. Probably occur as result of different mechanism than thrombosis.

A MI is also usually classified according to the wall involved — inferior, anterior, and posterior, lateral.
Individual of average build with normally positioned heart and correctly placed precordial leads:

$V_1$ and $V_2$ lie over RV, $V_3$ lies over anterior ventricular septum, and $V_4$-$V_6$ lie over LV.

Because of variations of the chest in various individuals, exact localization of an infarct may be impossible; criteria below give approximate locations.

Infarct revealed only in $V_1$ = anteroseptal.

$V_4$ ordinarily overlies anterolateral LV; displays lateral infarcts.

$V_6$ faces posterolateral LV because apex of heart is tipped toward front of body displays posterolateral infarcts.

**Localization of the Infarct**

**Leads With**

**ST Segment Reciprocal**

**Category Elevation Changes Coronary Artery**

Infarct II, III, aVF I, aVl, RCA or LAD of LCA

Anterior $V_1$, $V_2$, $V_3$ II, III, aV1, LAD
Extensive I, aVl, V1- V6, II, III, aVf L main or LAD + Cx

Anterior

Apical V5, V6 (? V3, V4) None Terminal LAD or RCA

Anterolateral I, aVl, V3, V5, V6 II, III, aVf Cx

Anteroseptal V2 and V3 or in V1 and V4 None 1st division of LAD only

Anterobasal I, V5 Cx

Lateral I, aVl, V5, V6 V2, OM or Cx of LCA

Posterolateral V4, V5, V6 aVF Cx of LCA (Apical)

Inferior II, III, aVf I, aVf PDA of RCA (85%) or LCA (15%)

Posterior None V1, V2 Cx; ? RCA; variable

True Posterior V1, V5, V6 V1, V2 Variable — LCx or RPL (posteroseptal)

Posteroinferior II, III, aVF; V3-V4 PDA of RCA

Posterobasal V6: aVF Cx

Right Ventricular V4, V6 RCA

LAD = left anterior descending Cx - circumflex

LCA = left coronary artery PDA = posterior descending artery

OM = obtuse marginal RCA = right coronary artery.

RPL= right posterolateral

Note: LAD supplies majority of left ventricle.

**Distinguishing Between Infarct of RCA and LCx Arteries Using V4R**

**V4R Morphology of ST Segment Artery Involved Sensitivity Specificity**

- 1 mm ST elevation Proximal RCA 93% 88%

No ST elevation with Circumflex 85% 97% down sloping ST segment

Up sloping ST segment Distal RCA 74% 92% with no ST elevation

**QRS Criteria for the Diagnosis of Myocardial Infarction**: Criteria are valid in the absence of LVH, LAFB, RVH, LBBB, RBBB, COPD, or W-P-W patterns.

See Appendix B for location of coronary arteries.

**Anterior MI — Leads V1-V4, especially V1** Involves the anterior wall of LV which is responsible for most of the cardiac output (CO). Lesion is in left anterior descending artery (LAD). Involves greatest muscle mass.

• V2 — any Q wave; OR R< 1 mm & < 10 msec.
• V₃ — any Q wave; OR R< 2 mm & < 20 msec.

Indicative changes in I, aV₆, V₁-V₄

If conus branch of RCA is especially well-developed, V₁ will be normal in anterior MIs as V₁ is caused by depolarization of septum. (This fact has not been published yet.)

Reciprocal changes in II, III, and aV₆.

Loss of R wave progression (see page 6). Has same meaning and significance as the presence of a significant Q wave. Loss of R height between V₁-V₂ or V₂-V₃ in the absence of LVH suggests anterior MI.

May be accompanied by CHF, tachycardias, atrial rhythms.

**Determination of Extent of Myocardial Involvement in Anterior MI:**

**Occlusion proximal to 1st diagonal branch** — ST elevation >1 mm in either I or aV₆.

Without thrombolytic therapy, patient will lose much of the pumping force of the LV.

**New left anterior fascicular block** — occlusion is proximal to the 1st diagonal branch.

**New RBBB** — occlusion is proximal to the 1st septal perforator; infarct penetrates deep into septum.

**ST elevation in V₁-V₃ — septum is involved.** Leads V₂-V₃ lie directly over the septum, so you may see a Q wave or poor R progression in V₂. You also see poor R progression with LV hypertrophy. Anteroseptal infarcts predispose the patient to tachy- and brady- dysrhythmias and septal rupture.

**Anterior (anteroseptal) infarct** — only one division (R) of LAD occluded

• Loss of R waves in leads V₂ and V₃; often not in leads V₄-V₆ or IC in V₁ and V₄ only

Limbo leads normal

**ST elevation in V₅-V₆** — apex is involved.

**ST elevation in aV₆** — high lateral anterior wall is involved.

Patient prone to dysrhythmias (bundle branch and AV blocks, supraventricular tach.), ventricular aneurysms, CHF, pulmonary edema, shock, L ventricular thrombi, and apical akinesis.
Ventricular dysrhythmias are frequently seen immediately after the MI; usually due to a reentry type mechanism (2° to ischemia).

Dysrhythmias seen several hours later tend to be due to increased automaticity.

Anterior (and multiple site) MIs are associated with the greatest degree of ventricular impairment and the greatest mortality; they tend to be larger than inferior MIs, which predisposes both to stasis and clot formation.

**Anterolateral MI — Leads I and aVL**

- Q > 30 msec in either I or aVL.

**Anterolateral Infarct** — due to occlusion of LAD.

- Indicative changes in precordial leads (V2-V5 or V1-V6) overlying infarct.

Loss of R waves plus deep Q waves in V2-V5.

Lead I — Q wave common; ST segments elevated; T waves inverted.

Reciprocal changes in lead III (upright T waves and ST elevation).

**Anterobasal Infarct** — occlusion of a branch of the LCx.

- Small Q wave in I and enlarged Q wave in V6.

ST and T waves initially may be elevated in V5 and I if infarct is acute; followed later by inverted T waves.

**Inferior MI — Leads II, III, and aVF.** Criteria in lead III are non-specific, however.

Infarcts on inferior surface of the heart where it rests against the diaphragm.

Degree of ST segment elevation in descending order = leads III, aVF, and II.

- Q > 30 msec in either II or aVF.

- aVF — Depth of Q wave > 1/3 the height of the R.

Normally, smaller portion of LV involved than in anterior MI unless concomitant areas are involved.

Therefore, usually has a better outcome than anterior MI.

**Inferior infarct** — occlusion of RCA.

- Indicative changes in leads II, III, aVF.

Reciprocal changes in leads I, aVL.

May be accompanied by ventricular rhythms, bradycardia, 1° AV block.

Acute inferior wall ischemia or infarction may cause increased parasympathetic activity manifested as sinus bradycardia and, on occasion, an inappropriate decrease in systemic vascular resistance; these changes may be associated with significant systemic hypotension.
**Determination of Extent of Myocardial Involvement in Inferior MI:**

**ST Elevation ≥ 1 mm in V_{4R} — right ventricle is involved.**

ST segment elevation in V_{1} or ST elevation > in lead III than lead II — be sure to get right precordial leads to confirm RV infarction. Because RV branch is one of the 1st branches off the RCA, RV infarction is seen only with very proximal RCA occlusions.

Important to diagnose RV infarction as they often get jugular venous distention and volume responsive systemic hypotension. R infarcts are not associated with S_{3} sounds or pulmonary congestion. The R filling pressure increases but the L decreases, so these patients usually need fluid challenges. Treatment of R failure with diuresis or nitroglycerine may produce hypotension and death.

**ST elevation in II, III, V_{5}-V_{6} — apex is involved.**

**ST depression in V_{1}-V_{3} (esp. V_{2}-V_{3}) or ST elevation in V_{7}-V_{9} — true posterior MI.**

Prone to papillary muscle dysfunction and valvular insufficiency.

Associated with lesions of RCA; therefore, you expect to see ischemia of the SA node and sinus bradycardia.

Frequently manifested by sinus bradycardia, AV block, RV infarction, or LV infarction of modest extent AV node also often affected; patient may present with a 1° AV block that progresses to 3° block or to brady dysrhythmias; may be transient or permanent. Atropine should be kept at bedside. Be cautious about vagal stimulation (rectal temperature, etc.). Vagal stimulation causes decreased oxygen demand but increased PVCs and hypotension with a resulting decrease in coronary flow.

**Posterior Infarctions — Leads V_{1}-V_{3}.**

Posterior MIs are rare. The posterior surface lies closer to atria than to the inferior surface.

Posterior MIs are associated with lesions of the circumflex or right coronary arteries.

No electrodes "look" directly at the posterior part of the heart, thus we look at the leads that are directly opposite or reciprocal (V_{1,2}). Then hold a mirror over the ECG and look for Q waves and an elevated ST.

• See only reciprocal changes in V_{1}-V_{2} (? V_{3}) unless posterior leads (V_{7}-V_{9}) are obtained.

With a posterior MI, there are large R waves and ST depressions in leads V_{1,2}.

Reciprocal changes may also be seen in leads I and aV_{L}.

• No ST _ is seen in standard 12-lead ECG; therefore the patient may not receive needed thrombolytic therapy in the emergency department.
True Posterior = Indicative Changes in Leads V7-V9.

Posterolateral Infarct — occlusion of LCxA

• Q waves in V6; also elevated ST segments and inverted T waves.

aVF — resembles V6 as it also faces infarct.

Area of injury often considerably wider than area of necrosis; therefore elevated ST segments and inverted T waves also seen in leads II, III, and aVF.

Posterobasal Infarct — occlusion of LCxA

• Q waves in V6; also elevated ST segments and inverted T waves.

aVF — resembles V6.

Posterior (postero septal) Infarct — true posterior = occlusion of LCxA or of RCA or its posterior descending branch, often situated over interventricular septum.

• Reciprocal changes (ST depression; tall R waves) in anterior leads, especially V1 and V2; small R waves in V3 and V4.

ST depression in V1 and V2; persist for at least a few days.

• This infarct is in a blind spot; must look for reciprocal Δs.

Posteroinferior (diaphragmatic) Infarct — occlusion of posterior descending branch of RCA.

• Large Q waves in leads II, III, and aVF.

ST initially negative in leads I, V5, and V6; ST elevated in lead III.

• aVF— large Q wave, ST elevation, and inverted T waves; probably most revealing lead.

Diagnosis — Q wave in aVF = at least 25% of R wave esp. when this difference in amplitude exists with breath held in deep inspiration (brings heart to a more vertical position).

Markedly impaired LV function with pulmonary congestion or edema indicative of extensive injury, intraventricular conduction defects such as hemiblock — more typically seen with LCA occlusion.

Apical MI — Leads V5-V6 (or V4-V6) + criteria for infarction in one of the areas above.
Apex involvement reflected by ST elevation in leads V₄-V₆.

No reciprocal changes seen in standard 12-lead ECG. RC are seen in V₅R and V₆R if recorded.

• Loss of R wave progression (see page 6).

Apical Infarct — terminal portion of LAD occluded.

• Q = initial wave in leads I; deep Q in V₃ and V₄.

Lead I — ST segments elevated; T waves inverted.

Lead III — ST segment depression; upright T waves.

Lateral MI — Leads I and aVL.

• Occlusion of LCxA.

• Indicative changes in leads I, aVL.

• Lateral MIs are associated with lesions of left circumflex and sometimes the LAD.

ST/T changes and Q waves are seen best in leads I and aVL. Leads V₄₅ may also be used, but reflect the lower lateral rather than the upper lateral ventricle wall and usually indicate involvement of apex.

Commonest sites for thrombosis are LAD, RCA, and LCxA, in approximately a 3:2:1 ratio LAD (40%) — anterior LV free wall, anterior septum.

RCA (27%) — posterior LV free wall, posterior septum.

LCxA (11%) — lateral LV free wall.

CLINICAL AND HEMODYNAMIC MANIFESTATIONS OF LEFT VENTRICULAR MUSCLE LOSS

LV Muscle Loss Clinical or Hemodynamic Manifestation

Greater than 8% Decreased compliance

Greater than 10% Decreased ejection fraction

Greater than 15% Increased ventricular end-diastolic pressure

Greater than 20% Increased ventricular end-diastolic volume (systolic failure)

Greater than 25% Clinical evidence of heart failure

Greater than 40% Cardiogenic shock and/or death

Is there evidence of electrolyte or drug abnormalities?

Hypokalemia — prominent U waves; may have camel hump effect. It is never normal for the U wave to be larger than the T wave.

Normal serum K⁺ 3.5-5.0 — normal ECG; T wave is much higher than the U wave.
Serum K⁺ 3.0-3.5 — ECG may be normal. If ECG changes are present, they are most prominent in the anterior precordial leads (V₂ and V₃).

- Appearance of U waves. (U wave also seen with digitalis, quinidine, epinephrine, hypercalcemia, exercise, hyperthyroid.)
- T wave may be flat, inverted and ST may be depressed.

Serum K⁺ 2.7-3.0

- U waves become taller and T waves become smaller.
- Prolongs repolarization as indicated by U wave and flat T which may merge (T-U fusion). The ratio of the amplitude of the U wave to the amplitude of the T wave frequently exceeds 1.0 in V₂ or V₃.

Serum K⁺ <2.6

- ST segment depression associated with tall U waves and low amplitude TR waves.
- May produce PVCs, tachycardia, ventricular fibrillation because necessary for polarized state

**Hypokalemia**
- Flat T, prolonged QT due to prominent “U” wave
- Very important, potentially life saving diagnosis
- May result from diuretic misuse, IV fluid administration, Rx during diabetic acidosis, etc.

**Hyperkalemia:**

When interstitial K⁺ is elevated you lose the gradient to excite the cells and open the Na⁺ gate.
Early hyperkalemia ($K^+ 5.5-7.5$) — sharp, pointed, tall T waves with narrow base; seen best in leads $V_2, V_4$. Deep S wave in lead I and $V_6$. The P wave flattens due to an intra-atrial block, which may progress to an AV block with a prolonged PR interval.

Absence of P waves — sinoventricular rhythm or atrial paralysis.

Always think of hyperkalemia when you lose the P waves.

Serum $K^+ 7.0-8.0$ — QRS widening; slurring of both the initial and terminal portions of the QRS; ST segment elevation; low, wide P waves; 1° and 2° AV block; atrial arrest; bradycardia.

Late hyperkalemia ($K^+ > 9.0$) — marked widening of QRS — probably sinoventricular rhythm which mimics an idioventricular rhythm; distinct ST-T wave may not be noted; high risk of ventricular fibrillation or asystole.

In patients with hyperkalemia and ventricular fibrillation, electrically depressed cells repolarize fastest but to a more positive (less negative) level.

**Hypocalcemia**

Slight decrease in QRS duration.

ST segment lengthened and corrected QT interval prolonged.

PR interval may be shortened.

T waves may become flat or inverted in severe hypocalcemia.

Signs and symptoms include tetany, spasms, cramps, numbness, and tingling.

Decreased $Ca^{++}$ will cause increased $Na^+$ entry into cells with repetitive firing of nerves; skeletal muscle contraction is unaffected. Hypocalcemia leads to decreased cardiac contractility and arrhythmias.

**Hypercalcemia**

Slight increase in QRS duration.

ST segment short or absent.

PR interval may be prolonged.
Short Q-T interval. abnormal Q; uncommon in the ICU.

Signs and symptoms include irritation, somnolence, muscle weakness, peripheral neuropathies, anorexia, constipation, and N/V.

*Hypercalcemia promotes digitalis toxicity.*

![Hypercalcemia]

**Hypomagnesemia and Hypermagnesemia**

- Marked decreased Mg²⁺ is usually associated with K⁺ depletion and the ECG demonstrates the characteristic changes of hypokalemia. Ventricular arrhythmias may be present.

- Hypermagnesemia is uncommon clinically; usually encountered in patients with uremia who often have other electrolyte disturbances.

- It is uncertain if changes in body magnesium alone affect the surface ECG.

**Digitalis Effect**

- S-T segment depression and sagging (looks like an inverted check mark or inverted bowl, especially in lead V₄).

- Low amplitude T wave.

- Shortened Q-T interval.

- Shortened P-R interval or, occasionally, P-R prolongation (1° AV block).

- Digitalis toxicity may cause the following:
  - Bradycardia when previously normal or fast (due to SA or AV block).
  - Tachycardia when previously normal (due to atrial tachycardia, junctional tachycardia, or fascicular ventricular tachycardia).
  - Unexpected regularity (due to complete AV block with a regular AV junctional rhythm in a patient with prior atrial fibrillation or flutter).
  - Regular irregularity (due to group beating of ventricular bigeminy, SA, or Wenckebach, or a combination of these).

- Increased sympathetic tone, hypokalemia, hypercalcemia, hypomagnesemia, diuretics, ischemia and reperfusion, increased wall tension, and CHF promote digitalis dysrhythmias.

- In monitoring patients taking digitalis, use lead II if P waves are present and lead V₁ (MCL₁) if the patient is in atrial fibrillation.

Atrial tachycardia caused by digitalis toxicity has upright P waves in lead II, very similar in shape to the sinus P wave. Once the atrial rhythm has been evaluated, look for AV dissociation.
Lead V₁ or MCL₁ is useful to discern junctional and fascicular rhythms. The shape of the QRS in a junctional rhythm will be that of the normal QRS (rS); a fascicular rhythm will look like RBBB (eSR).

**Quinidine Effect (and Other Class Ia Antiarrhythmics)**

- Prolonged Q-T interval.

- Slightly widened QRS complex; widening is an early sign of toxicity.

- Quinidine and procainamide may also produce U waves and flattened or inverted T waves.

**Phenothiazines** — ECG changes seen in approximately 50% of patients receiving “therapeutic” doses.

- Mimics hypokalemia.

- Prominent U waves.

- Low amplitude T waves or T wave inversion.

- ST segment depression.

- Prolonged QT interval.

- Toxic doses — prolonged QT interval and QRS duration; AV and intraventricular conduction delays; increased automaticity; ventricular arrhythmias due to reentry.

**Tricyclic Antidepressants**

- Usually do not produce ECG Δs except sinus tachycardia during early phase of therapy.

- QRS and QT prolongation.

- Toxic doses — ECG Δs similar to those noted with toxic doses of a phenothiazine; due to direct effect of drug on myocardium; also see rightward deviation of terminal QRS forces and anterior rotation of the ST segment vector.

- **Is there evidence of pulmonary embolus?**
New signs of tachycardia, RBBB (complete or incomplete), large S wave in lead I, Q wave in lead III with and inverted T wave, right axis shift.

May get inferior or RV injury pattern.

Possibly see a right atrial abnormality?

**Is there evidence of chronic pulmonary disease?**

May get S waves in leads I, II, and III.

Associated with right axis deviation or RVH.

Associated with poor R wave progression in precordial leads.

Low voltage.

Right atrial abnormality.

**Is there evidence of pericarditis?**

Widespread S-T segment elevation *without T wave inversion* in both anterior and inferior leads lasting 5-10 days. See it globally in all 12 leads.

Widespread T wave inversion 10-15 days after onset of acute pericarditis.

Electrical alternans and low voltage if a large pericardial effusion is present. Can get P wave alternans or QRS wave alternans or both (total alternans). Tamponade can produce T wave alternans.

PR segment in aV_{R} sticks up like a "knuckle."

Fairly common after transmural infarctions.

**Is there evidence of congestive Heart failure?**

May get atrial fibrillation/flutter, left atrial abnormality, LV hypertrophy with strain, bundle branch block, low voltage, and Q waves, especially in the anterior and/or lateral leads.

**ESTIMATE OF LEFT VENTRICULAR FUNCTION FROM ECG**

Normal ECG — 95% probability of normal ejection fraction (EF > 45%)

Subtract the following points from 60 for each abnormality present to estimate EF %:

LBBB — 30 points

ST Depression — 10 points

Inferior Q Waves (leads II, II, aV_{F}) — 10 points

Anterior Q Waves (leads V_{2} - V_{4}) — 30 points

Lateral Q Waves (leads I, aV_{L}, V_{5}) — 15 points
Septal Q waves (leads V₁, V₂) — 10 points

**QRS MORPHOLOGY (R > S in V₁ or V₂)**

**Differential Specific ECG Characteristics**

**RVH** • Right axis deviation

• S wave (terminal slowing) in V₆

• QRS Duration < 120 msec

**RBBB** • "Rabbit ears" in V₁; S wave in V₆

• QRS Duration > 120 msec

**Post Myocardial Infarction** • Associated with inferior MI (Q waves in II, III, aV₅)

• QRS duration < 120 msec

**Wolff-Parkinson-White** • Short PR with delta wave

• QRS duration > 120 msec

**Normal variant**

• Normal ECG
• PART III: DETERMINATION OF PRECISE ELECTRICAL AXIS

Normal Limits of QRS Axis in Adults: -30° to +90°.

Quadrant Method (I and aV_F; can also be approximated using I and III).

- Both leads have primarily positive QRS deflections = normal axis.
- QRS in I is primarily positive and QRS in aV_F is primarily negative = LAD.
- QRS in I is primarily negative and QRS in aV_F is primarily positive = RAD.
- Both leads are primarily negative = extreme axis; equivocal.

Perpendicular Rule (all frontal leads)

- Mean QRS vector is perpendicular (⊥) to the axis of the lead with the most equiphasic complex in the pre-selected quadrant using quadrant method.
- Lead ⊥ to the most equiphasic QRS lead = axis.
- Numerical value of axis is determined by following ⊥ lead toward pole that is in the preselected quadrant.

E.g., quadrant method shows LAD [0°-90°].
Lead II is most equiphasic.

aV_{L} is \perp to II.

Therefore a LAD of -30° is present.

**Parallel Rule** (look at all frontal leads).

- Used to confirm quadrant and/or perpendicular rule.
- Find lead with largest (+ or -) QRS deflection.
- If the QRS is positive, follow that lead to its positive, pole thus giving you the numerical axis.
- If the QRS is negative, follow that lead to its negative pole to obtain numerical axis.

**LAD — Etiology:**

- May occur normally in obese person [fat pushes diaphragm up] also in elderly.

**Abnormal LAD Etiology (in descending order of occurrence):**

- Left anterior fascicular block (LAFB).
  
  Axis -45° to -90° (possibly < -30°).

  Criteria: qR in aV_{L}, with onset of Q to peak of R 45 msec (R-peak time).

  *Clinical correlations:* seen in hypertensive heart disease, coronary artery disease, or idiopathic conducting system disease.

- Acute MI — inferior (q 30 msec in aV_{E}).


- COPD (uncommon - 10%).

**RAD — Etiology**

- May occur normally in slender person and in infants.

**Abnormal LAD Etiology (in descending order of occurrence):**

- R ventricular hypertrophy (RVH) [more muscle \pm more electrical activity so axis moves toward area of hypertrophy]; the MOST COMMON CAUSE of RAD; however, *one must first exclude* inferior or posterior MI or acute inferior injury causing LPFB in addition to #s 2 and 3 below.

- Acute MI (no electrical activity in area of necrosis; therefore the axis moves away from MI site.)

  Anterior

  Anterolateral

  Lateral

- Left ventricular free wall accessory pathway (W-P-W pattern)

- L posterior fascicular block
Criteria include: axis > +90° to +150° with qR complex in III with onset of Q to peak of R 45 msec (R-peak time)

ETIOLOGY OF AXIS DEVIATIONS

Left anterior fascicular block (LAFB)
Right ventricular hypertrophy
Left bundle branch block Acute MI:
Hypertensive heart disease
Coronary artery disease
Idiopathic conducting system disease
Acute MI — inferior LV free wall accessory pathway (W-P-W)
Posteroseptal accessory pathway (WPW)
L posterior fascicular block
COPD (uncommon - 10%)
Other conduction defects:
L ventricular hypertrophy
RBBB
Elevated diaphragm: R anterior hemiblock
Pregnancy
Pacing of R ventricle
Abdominal mass
Pulmonary conditions
Ascites
Pulmonary hypertension
Tumor
COPD
Conduction defects: Emphysema/bronchitis
R ventricular (apical) pacing
Pulmonary emboli/infarcts
Systemic hypertension, esp. chronic
Congenital defects
Valvular lesions

Rheumatic heart disease

Pulmonic stenosis

Aortic regurgitation

Mitral regurgitation

Mitral stenosis

Coarctation of the aorta

Tricuspid regurgitation

Hyperkalemia

Pulmonic stenosis

Normal variant in obese and in elderly

Pulmonic regurgitation

FASCIULAR BLOCKS (HEMIBLOCKS)

Anterior Hemiblock — a block of the anterior superior division of the left bundle branch.

More common and less serious than posterior hemiblock.

Anterior fascicle of the LBB is long, thin, and has only one blood supply as does the RBB.

Located in the outflow tract of the LV and is, therefore, subjected to mechanical stresses.

Impulse activates the ventricles via the posterior inferior fascicle producing LAD.

Rate, rhythm, and PR interval are normal.

QRS complex — normal. Lengthens by only 0.02 sec.

Distinguishing features — LAD of > 45°; increased QRS voltage in the limb leads; a terminal R wave in aV_R and aV_L; R wave in aV_R is later than the R wave in aV_L; q wave in I and aV_L, r wave in II, III, and aV_F.

Causes — aortic valve calcification, cardiomyopathy, ischemic heart disease, acute MI, cardiac catheterization, hyperkalemia, otherwise normal hearts.

Prognosis — depends on underlying cause.

Treatment — usually none. A pacemaker may be indicated if LAFB develops after an acute (anteroseptal) MI.

Posterior Hemiblock — a block of the posterior inferior division of the LBB.

Implies compromise of the right and left coronary arteries and damage to a broad inferior conduction system in the LV.

Impulse gets into the ventricles via the anterior superior fascicle producing a RAD.

Rate — may be normal, accelerated, or slow.
Rhythm and PR interval — normal.

QRS complex — normal. Lengthens by only 0.02 sec.

Distinguishing features — RAD of > + 120°; increased QRS voltage in the limb leads; q wave in II, III, and aVf; r wave in I and aVL.

Causes — aortic valve calcification, cardiomyopathy, ischemic heart disease, acute MI, cardiac catheterization, hyperkalemia.

Notify M. D. immediately if posterior hemiblock develop in the setting of acute anteroseptal MI in which case it is usually associated with RBBB (possibly intermittent) and a poor prognosis.

Complete subnodal AV block develops in approximately 90% of cases.

Treatment — a pacemaker is usually indicated if LPFB develops after an acute anteroseptal MI.

**Trifascicular Block** — complete or incomplete pathologic conduction impairment in the RBB and both divisions of the LBB.

Implies compromise of the left anterior descending branch (LAD) of the left coronary artery (LCA) and the right posterior descending artery (PDA).

If all three fascicles are simultaneously blocked a complete AV block with a ventricular escape rhythm will result.

Rate — < 40 bpm.

QRS complex — broad.

Incomplete trifascicular blocks may manifest as:

- RBBB plus left anterior hemiblock plus 1° or 2° AV block.
- RBBB plus left posterior hemiblock plus 1° or 2° AV block.
- LBBB plus 1° or 2° AV block.

Various combinations of the above.
PART II: 12-LEAD ECG INTERPRETATION

To ensure consistency, develop a systematic method of assessing 12-lead ECGs and rhythm strips.

• Read name, date, and time on ECG.

Make sure you have the correct ECG.

Use the computer interpretation as a learning tool rather than a substitute for thinking.

• Get old ECG for comparison.

Are those Q waves, if present, new? Is it LVH with stain or lateral ischemia? A prior ECG is needed to tell with certainty.

Temporal change is more significant than constant, isolated findings.

Comparison of serial changes is necessary for proper ECG interpretation.

• Is the ECG recorded at full-amplitude?

Is the calibration mark a full 10 mm in height?

• Ventricular Rate — slow, normal, or fast?

An ECG record is 10 seconds long; therefore if there are 17 or more complexes across the page the rate is fast and if there are 6 or less the rate is slow.

Know the 300, 150, 100, 75, 60 rule:

If the QRS complexes (R-R intervals) are 1 large box (thick line) apart the ventricular rate is 300.

2 large boxes = 150. 5 large boxes = 60. 8 large boxes = 37.

3 large boxes = 100. 6 large boxes = 50. 9 large boxes = 33.

<table>
<thead>
<tr>
<th>Type of Block</th>
<th>Axis</th>
<th>QRS Shape</th>
<th>Other ECG Changes</th>
<th>QRS Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBBB</td>
<td>Normal</td>
<td>[ \rightarrow ][ \downarrow ]</td>
<td>Large S wave in V6</td>
<td>\geq 120 msec</td>
</tr>
<tr>
<td>RBBB/Left Anterior Hemiblock</td>
<td>LAD</td>
<td>[ \rightarrow ][ \downarrow ]</td>
<td>Large S wave in V6</td>
<td>\geq 120 msec</td>
</tr>
<tr>
<td>RBBB/Left Posterior Hemiblock</td>
<td>RAD</td>
<td>[ \rightarrow ][ \downarrow ]</td>
<td>Large S wave in V6</td>
<td>\geq 120 msec</td>
</tr>
<tr>
<td>LBBB</td>
<td>Normal</td>
<td>[ \downarrow ][ \rightarrow ][ \rightarrow ]</td>
<td>↓ ST; unable to read ischemia or infarction</td>
<td>\geq 120 msec</td>
</tr>
<tr>
<td>Left Anterior Hemiblock</td>
<td>LAD &gt;45°</td>
<td>Normal</td>
<td>Small Q in aVL</td>
<td>&lt;120 msec</td>
</tr>
<tr>
<td>Left Posterior Hemiblock</td>
<td>RAD</td>
<td>Normal</td>
<td>None</td>
<td>&lt;120 msec</td>
</tr>
</tbody>
</table>
4 large boxes = 75. 7 large boxes = 43. 10 large boxes = 30.

As the marks on the upper or lower border of the ECG or monitor paper fall every 3 sec, with regular rhythms the complexes occurring in a six second interval can be counted and multiplied by 10.

**QRS Duration (of dominant QRS complex) — is QRS narrow or wide?**

- Best leads to look at = I and V₁. Should be less than 3 small boxes wide.

Narrow QRS means the impulse originated from a supraventricular focus (sinus, atrial, AV nodal, or AV reciprocating).

**Causes of wide QRS:**

- Rhythm supraventricular in origin with a bundle branch block (BBB).
- Rhythm supraventricular in origin with aberrant conduction.
- Rhythm supraventricular in origin with pre-excitation (in patients with an accessory AV pathway, termed Wolff-Parkinson-White or W-P-W syndrome).
- Ventricular in origin (the wider the complex, the more likely rhythm is to be ventricular in origin).

If QRS is wide (_120 msec or 0.12 sec or 3 small boxes) see the criteria for diagnosis of bundle branch blocks below.

**Bundle Branch Blocks (BBB)**

- If QRS duration 120 msec, but typical waveforms of either RBBB or LBBB are not present, diagnosis is intraventricular conduction delay or IVCD.
- Incomplete BBB = waveform typical of RBBB or LBBB but QRS duration is < 120 msec.

Incomplete LBBB — septal Q wave normally present in leads I and V₆ is absent.

- **Right and left bundle branch blocks cannot be diagnosed from lead II.** Much of the wide QRS complex during BBB is isoelectric in lead II, often producing a narrow, normal-looking complex.

**Right Bundle Branch Block** — characteristic triphasic QRS morphology; best observed from a right precordial lead (MCL₁ or V₁) because the exploring electrode is located near the RV free wall. Consequently, delayed RV activation which occurs in the right bundle branch produces a primarily positive, wide QRS complex with a large R’ in these leads.

**ECG Characteristics in RBBB:**

- Wide QRS complex (> 0.11 sec.); often 0.12-0.14.
- Triphasic rS’ or rR’ in MCL₁/V₁; triphasic qRS in MCL₆/V₆.
- S-T segment and T wave slope away from major deflection (i.e., negative in MCL₁/V₁ and positive in MCL₆/V₆).
- If the QRS complex has the typical RBBB contour, but measures 0.09-0.11 sec., the diagnosis of incomplete RBBB is made. (We don't know what else to call it and are not sure exactly what is happening in heart.)

**Left Bundle Branch Block** — characteristic monophasic QRS morphology can be observed in a right or left precordial lead; delayed LV activation which occurs in the LBB produces a negative, wide QRS complex in the right precordial leads (MCL₁ or V₁) and a positive, wide complex in the left precordial leads (MCL₆, V₆, or lead I).

The left bundle branch splits into two divisions or fascicles. Blocked conduction in a fascicle (a partial block in the left bundle branch or hemiblock), however, alters the electrical axis in the frontal plane rather than widening the QRS complex. Fascicular blocks are discussed further in the sections on axis deviation. (See pages 14 and 29-31.)
QRS Rhythmicity (R-R Intervals) — regular? or irregular?

"Irregular" is defined as any change in R-R intervals.

Irregularly irregular suggests atrial fibrillation.

Regularly irregular suggests a bigeminal or trigeminal rhythm or Wenckebach cycles.

• Is the rhythm a sinus rhythm (does it originate in the SA node)?

  Can you see upright P waves in I, II, and aVf? If so, the rhythm is sinus in origin.

  A "rhythm" = 3 or more of anything in a row.

  Rate of normal sinus rhythm = 60-100 bpm.

  Sinus bradycardia = rate < 60.

  Sinus tachycardia = rate > 100.

  Sinus arrhythmia = P-P interval varies > 10%. (See page 32 for discussion of sinus arrhythmia and other arrhythmias originating in the SA node.)

Normal Sinus Rhythm (NSR):

  • SA node is the pacemaker.

  • P wave is rounded, symmetrical, identical and a QRS follows each P.

  • PR interval is 0.12-0.20 sec and constant.

  • QRS complex will be positive or negative (depending on lead) and 0.10 sec or less.

  • ST segment is slightly concave, sloping into T wave.

  • T wave is rounded, slightly asymmetrical.

  • Rate = 60-100 bpm.
Rhythm not of diagnostic significance unless the rate is inappropriate for the clinical setting.

Sinus rhythm is *physiologic* in that it tracks the body's requirements.

**Sinus Tachycardia:**

Due to increased automaticity of the SA node.

Rate is 100-180 beats per minute (bpm). *Diffs from NSR only in that the rate is >100 bpm.*

(Some books say > 90 bpm = tachycardia.)

Rhythm regular.

PR interval normal.

One QRS for each P.

Onset and termination of the rhythm is gradual and regular, but may vary from minute to minute.

Not paroxysmal; it doesn't start and stop abruptly except for a rare type called sinus nodal reentrant tachycardia.

Almost always a *secondary* arrhythmia so the underlying cause should be determined and treated if necessary.

Usually harmless except in patients with heart disease — increased oxygen demand causes symptoms.

Often a poor prognostic sign in patients with heart disease.

**Causes of sinus tachycardia in normal situations** — exercise, emotional stress, stimulants.

Abnormal, non-cardiac causes of sinus tachycardia — fever, hemorrhage, infection, hypoglycemia, anxiety, pain, thyrotoxicosis, shock, hypoxemia, hypovolemia.

**Causes of sinus tachycardia in cardiac patients** — any of the above; poor LV function (cardiogenic shock, CHF, pulmonary edema); sympathomimetic drugs (Isuprel, dopamine, dobutamine, epinephrine); vagolytic drugs (atropine); vasodilators (nitroglycerine), especially if the patient is volume-depleted.

Management of sinus tachycardia includes — none; treating the underlying cause, sedation, and possibly digitalis (if the patient has CHF).
Sinus Bradycardia:

Due to depressed SA node automaticity.

Rate < 50-60 bpm. Differs from NSR only in that the rate is below 60 bpm.

Rhythm is regular but may vary minute to minute.

PR interval is normal.

One QRS for each P (1:1 ratio).

Decreased HR may decrease cardiac output (CO) and cause ischemia.

Bradyarrhythmia is usually harmless unless it is associated with a low CO that is followed by sensorium changes, diaphoresis, skin color and temperature changes, chest pain, or dysrhythmias.

Causes of sinus bradycardia in normal situations — athletic heart, vasovagal reactions (Valsalva maneuver results in first bradycardia, then tachycardia), sleep.

Non-cardiac causes of sinus bradycardia — ocular pressure (glaucoma and drugs to treat glaucoma which have β-blockers in them like Timoptic which contains timolol); increased intracranial pressure; obstructive jaundice (bile salts effect on SA node); vagal stimulation from nausea and vomiting; hypothermia; and hyperkalemia.

Causes of sinus bradycardia in cardiac patients — any of the above; β-blockers; vagal stimulation from carotid sinus massage; digitalis; morphine; some Ca++-channel blockers such as verapamil and diltiazem; SA node ischemia (inferior or posterior MI).

Acute sinus bradycardia may be extremely dangerous in a patient with an acute MI or poor LV function because stroke volume (SV) cannot be increased to maintain cardiac output (CO = HR x SV).

Other more malignant arrhythmias may surface.

Treat only if symptomatic (syncope, decreased BP, chest pain, or SOB); don't treat for "dizziness" alone.

Treatment should be prompt — atropine 0.5 mg IV push; put head of bed flat or Trendelenburg to increase blood to SA node and increase BP.

• Is there one P wave for every QRS complex (a 1:1 ratio)?

Lack of a 1:1 ratio implies A-V dissociation, either complete or incomplete (presence of capture or fusion complexes). A-V dissociation refers to a group of three categories of rhythms in which the atrial and ventricular rhythms are unrelated to each other. Four major causes:

• Slowing of the primary pacemaker (e.g., sinus block [page 33] with junctional escape rhythm).

Speeding of a subsidiary pacemaker (e.g., ventricular [page 53] or junctional tachycardia [page 49]).

• Third degree A-V block. (See page 46.)

• Combinations of the first two above (e.g., some sinus slowing with accelerated junctional rhythm or junctional tachycardia.)
• **What is the PR interval and is it constant?**

  Use Lead II to measure the PR interval.

  *If it is less than 1 large box it is not prolonged.* (Normal range = 0.12~0.20 sec.)

  \[ \text{PR > 0.20 sec with 1:1 A-V ratio = first degree (1°) A-V block.} \]

  **First Degree Block** — This refers to an excessively long PR interval only.

  Not really a block; conduction is just delayed at the AV junctional area. Every impulse eventually reaches ventricle.

  Whether the rate and rhythm are regular depends upon the underlying rhythm.

  P is upright, uniform and has a 1:1 ratio with the QRS.

  PR interval is greater than 0.20, but constant.

  May result from disease in the AV node, high vagal tone, or medication that increase conduction through the AV node. Seen with digitalis, quinidine, and procainamide toxicity; hyperthyroidism, anterior MIs, cardiac surgery, coronary artery disease, and myocarditis.

  Usually no treatment is necessary, although the medications may be discontinued if they are causing the problem. The patient should be observed for 2nd or 3rd degree block.

  All P waves are conducted through the AV node to the ventricle.

  By itself, it is a benign condition.

  **Short PR Intervals**

  A short PR interval, less than 0.12 seconds, is associated with accessory pathways between the atria and ventricles — the Wolff-Parkinson-White syndrome (see page 42) and Lown-Ganong-Levine syndrome.

  A short PR interval may also result from junctional or low atrial ectopic rhythms.

  • **Is there evidence of atrial enlargement?** Look at leads II, V₁, and V₂.

  Thicker muscle mass in atria increase distance traveled by the depolarizing impulse and increase current flow in the areas of hypertrophy.

  **Criteria for Left Atrial Enlargement (LAE) or P Mitrale** = any one of the following:

  P wave takes on appearance of an "m" in limb leads in LAE because of difference in depolarization of the two sides of the heart. Look at leads V₁ and II.

    - Negative portion of the P wave is wider and/or deeper than normal in V₁. Depth (of negative portion of P) in mm x width (of negative portion) = _0.04._ (Depolarization moves away from lead V₁ creating a terminal negative deflection.)

    - The space between the peaks of the P wave is more than 0.04 sec in *any* lead.
The P wave in lead II is taller than 3 mm or wider than 0.12 sec.

The P wave is 1.6 or more times wider than the P-R segment.

Often 2° to LV hypertrophy, coronary artery disease, or mitral valve disease.

Criteria for Right Atrial Enlargement (RAE) or P Pulmonale = any one of the 3 criteria below:

- QRS axis > +90°.
- R/S ratio ≥ 1 in V1. (In RVH, but not in RBBB or post MI.)
- Upright P wave in V1-2 ≥ 1.5 mm. (V2 is best lead to look at.)
- Pronounced positive portion of P wave in V1.
- Tall, peaked P waves in II, III, and aVf.
- ?? negative P wave in aVL. (P wave axis is to the right of +60°.)

Tall, peaked P waves are indicative of possible RAE, but more commonly are due to COPD and/or increased sympathetic tone, not RAE.

See in pulmonic stenosis in children and in tricuspid stenosis and tricuspid regurgitation.

Diseases affecting the left atrium produce P waves which look like P mitrale and diseases affecting the right atrium produce P waves which look like P pulmonale.

• What is the electrical axis of the QRS complexes?

Axis represents the major direction of the total electrical forces of the heart, a summation of all vectors.

The main thing to determine is whether or not it is normal. This determination is much more important than calculation of the numerical axis.

- Look at leads I and II.

• If both I and II have upright QRS complexes, the axis is normal.

• If I is upright and II is negative, left axis deviation (LAD), the most common axis deviation, is present.
• If I is negative (and the P wave in I is upright) with an upright QRS in II, right axis deviation (RAD) is present. If both the P wave and the QRS are negative in lead I, the right arm and left arm electrodes are reversed — they were attached in the wrong places.

Other leads that are commonly reversed are V₁ with V₃ and V₆ with V₄.

• If both I and II have negative QRS complexes, an extreme axis deviation is present (left upper quadrant).

• If the QRS is equiphasic in both leads, the axis cannot be determined. i.e., an indeterminate axis is present.

![](image)

• Is the QT interval normal, prolonged, or short?

  _ Q-T interval should be roughly less than half the preceding R-R interval.

  If a patient develops a wide QRS complex (which is a problem with depolarization) such as a bundle branch block, the QT interval will be increased. Thus, a long QT interval is not thought of as abnormal in patients with a wide QRS complex unless you have subtracted the extra width of the QRS from the QT interval and still found it prolonged.

  _ If the rhythm is irregular, measure the QT relative to the rate of the prior R-R interval.

![](image)

  _ Causes of prolonged QT (> 50% of R-R interval) — congenital, hypomagnesemia, hypocalcemia, type IA anti-arrhythmic agents such as quinidine, ischemia, myocarditis, phenothiazines, tricyclics, subarachnoid hemorrhage, torsades de pointes.

  _ Causes of short QT (< 50% of R-R interval) — hypercalcemia, digoxin, thyrotoxicosis, hyperkalemia, and hypermagnesemia.
• Is there evidence of ventricular hypertrophy?

Ventricular enlargement alters the QRS and T waves. The thicker muscle mass increases the distance traveled by the wave of depolarization as well as the amount of current that flows from hypertrophied cells (amplitude is therefore increased). LVH causes eightfold increase in mortality.

_Especially important to look for evidence of left ventricular hypertrophy (LVH) in preoperative screening ECG as there is increased incidence of perioperative cardiac events._

**Criteria for Left Ventricular Hypertrophy (LVH) in Adults:**

Do not diagnose LVH in the presence of W-P-W pattern or LBBB. **LVH is assumed to be present with LBBB.**

The most important ECG feature is _enormously tall QRS complexes_ in leads situated over the left ventricle — I, aVl, V6, V5, and V6.

_ Height of R wave in aVl > 9 mm (females) or > 11 (males); OR
_ Height of R in aVl + S in V1 > 20 mm (females), or > 25 mm (males); OR
_ Height of R in aVl + S in V1 times the QRS duration (msec) > 1847 (females) or > 2530 (males); OR
_ S in V1 + R V1 or V6 > 35 mm (age > 35); OR
_ R wave in lead I plus S wave in lead III > 25 mm.
_ Left atrial enlargement and ST segment “strain” pattern in a patient not taking digitalis.

LVH may show repolarization abnormalities with evidence of “strain” — the ST and T wave are directed opposite the dominant QRS waveform.

LVH is seen with systemic hypertension, aortic stenosis, aortic regurgitation, and coarctation of the aorta. Left atrial hypertrophy often occurs concomitant with LVH.

The QRS voltage is decreased, there is ST depression and T inversion.

In leads I, V5, V6, the T wave is inverted and the ST is depressed.
There may be a LAD. LVH is the most common cause of left anterior fascicular block (LAFB).

Conduction delay or altered pathways of conduction in ventricles result in increased QRS interval (the total duration of ventricular depolarization) and ventricular activation time (interval from the beginning of QRS to the peak of the R wave). Injury and fibrotic changes in the involved myocardial cells depress the ST segment.

Ischemia occurs first, probably due to the increased oxygen demand in the hypertrophied cells, followed by injury and fibrotic changes as the disease progresses. The altered duration and pathways of repolarization produce T wave inversion in the leads with R waves of the greatest amplitude.

Axis shift, which may accompany enlargement, alters direction and amplitude of QRS complex.

**Left Ventricular Dilation** — see smaller QRS in limb leads and taller QRS in precordial leads (leads closer to heart demonstrate the increased size).

**Criteria for Right Ventricular Hypertrophy (RVH):**

- Right axis deviation >90°.
- An R/S ratio > 1 in lead V1 (in the absence of posterior MI or RBBB), OR
- An R wave > 7 mm tall in V1 (not R' of RBBB), OR
- An rsR' complex in V1, with a QRS duration of < 0.12 sec (incomplete RBBB), OR

RVH may show repolarization abnormalities in leads V1,3 and III.

- An S wave > 7 mm deep in leads V5 or V6; OR
- “Incomplete RBBB” pattern in V1 (S/R < 0.12 sec.)
- RBBB with either RAD (first 0.06 sec of the QRS; may be ±90° or more). Consider RVH in RBBB if the R/S ratio in lead I is less than in II.

RVH produces a false positive sign for left posterior fascicular block (LPFB). LPFB give right axis deviation; RAD is most commonly due to RVH. Before making the diagnosis of RVH, you must exclude the LPFB.

Main causes of RVH are chronic lung diseases, pulmonary hypertension, tricuspid regurgitation, and congenital lesions such as tetralogy of Fallot, pulmonic stenosis, ASD, and VSD.

Other causes of tall R waves in V1 are: W-P-W Syndrome (type A), RBBB, true posterior MI, wrong ECG lead placement — these must not be misdiagnosed as RVH.

**Right Ventricular Dilation** — see poor R wave progression as dilated RV pushes LV posteriorly resulting in loss of anterior forces.

**Ventricular Strain**

Strain is often associated with ventricular hypertrophy as the ventricle is straining against some kind of resistance (e.g., increased vascular or valvular resistance) and becomes hypertrophied in an attempt to compensate.

Characterized by moderate depression of the ST segment; generally curves upward or humps gradually in the middle of the segment.
• Is there low QRS voltage?

  - Defined as < 5 mm peak-to-peak in all limb leads or <10 mm in precordial leads.
  - Chronic causes — pulmonary disease, hypothyroidism, obesity, cardiomyopathy.
  - Acute causes — pleural and/or pericardial effusions

• Are prominent U waves evident?

  - Usually suggests digitalis or hypokalemia. Also seen in bradycardias or with antiarrhythmic drugs.

• Are there signs of myocardial injury, ischemia, or infarction?

  Right chest (or anterior) leads — V_1, V_2; also aV_R.
  Septal leads — V_1 to V_3 — located over interventricular septum.
  Left chest (or lateral) leads — V_5, V_6; also I and aV_L.

  V_1 and V_2 mirror changes occurring from the posterior side of the heart.

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**EKG: SECTION THREE**

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**D. AV Blocks**

Blocks can be transient, intermittent, or permanent and complete or incomplete. Blocks are caused by physiologic, pharmacologic, and pathologic processes. The site of a block is localized with a His bundle electrogram. The H represents impulse conduction over the His bundle and occurs between the A (atrial) and V (ventricle) deflections. The conduction time from the atria to the bundle is the A-H interval. The conduction time from the His bundle to the ventricle is the H-V interval. The bundle of His travels in posterior part of interventricular septum.

![Diagram of the heart showing AV blocks.]

**Traditional Classification of AV Blocks:**

• 1st degree AV block

• 2nd degree AV block — Mobitz I (Wenckebach phenomenon) and Mobitz II

• High degree AV block — momentary absence of conduction for several seconds

• 3rd degree AV block — complete heart block
**Newer, more useful classification of AV Blocks:**

- Diagnosis cannot be made on basis of surface ECG; requires invasive cardiac electrophysiology study (EPS) — His bundle ECG.

- **Type I AV block** — block located in the AV node; supra-Hisian (above His bundle); better prognosis.

  A-H prolongation during acute MI is often caused by ischemia, not necrosis. Thus, tend not to be permanent.

  A-H interval prolonged.

  A-H (atrial-His) interval makes up most of the PR interval on the surface ECG.

  Hallmark of blocks in the supra-Hisian region = PR prolongation.

  If A-H prolongation progresses to the point that the impulse fails to get through at all (3° block) a stable, dependable junctional escape rhythm generally tends to takeover and sustain life. If it is too slow, it can be pharmacologically by atropine but atropine only lasts about 20 min/dose.

  If complete heart block develops it does so gradually and predictably and thus can be anticipated.

  Transition usually takes place over several days in the setting of acute MI.

- **Type II AV block** — block located in the bundle branches or fascicles; infra-Hisian (below the AV node); worse prognosis.

  H-V (His-ventricular) interval represents time from His bundle depolarization until the beginning of ventricular depolarization; the impulse travels very fast once it gets out of the AV node so this interval is normally short. The H-V interval could double without a noticeable 1st degree AV block on the surface ECG.

  Hallmark of blocks in the supra-Hisian region = bundle branch and fascicular blocks.

  H-V prolongation during acute MI is usually caused by necrosis or other irreversible causes; requires permanent pacemaker.

  If H-V prolongation progresses to the point that 3° block develops, there may be no escape rhythm at all (ventricular standstill), or, at best, a slow and unpredictable idioventricular escape rhythm. Because the block is below pacer cells in the AV junction, junctional impulses will be blocked from conducting to the ventricles.

  Idioventricular rhythms do not respond to atropine and thus Isuprel must be used which is extremely potent and difficult to titrate.

  Isuprel may Δ idioventricular rhythm to ventricular tach. Lidocaine cannot be given for V. tach. in this setting as it might completely suppress the rhythm ⊖ complete asystole.

  Only thing you can do is increase the Isuprel drip.

  Onset of complete heart block is often sudden and unanticipated.

1. **First Degree Block** — discussed under 12-lead ECG interpretation.

   - Prolonged PR interval only (> 0.20 sec).

   - All sinus impulses conduct to the ventricles.

   - Causes — inferior MI, other cardiac disease, digitalis, β-blockers, degenerative changes with aging.

   - Relatively benign; rarely advances to complete block.

   - Treatment — none if chronic. Watch if new or progressive.

2. **Second Degree Block**
There are two types of 2° block; the differential dx. depends on the PR interval and the QRS complexes.

**Mobitz I (Wenckebach):**

- Mobitz I block is more common than Mobitz II.
- Mobitz I block is often a/w R CAD and is usually transient.
- It is a/w a prolonged A-H interval and irregular rhythm.
- QRS duration is normal and the P is upright and uniform.
- P-P interval is constant, but not all Ps are 1:1 with the QRSs.
- The R-R gets shorter as PR interval gets longer until an impulse is not conducted.
- Refers to a gradual prolongation of the PR interval, with occasional failure to conduct a P wave through the AV node to the ventricle.

**Characteristics (some or all of these may be present):**

- Start with normal or prolonged PR interval (underlying 1° block is most common); then with each successive beat the PR interval gradually lengthens until a beat is dropped. Usually due to prolonged A-H time.
- Following the dropped beat, the PR returns to what it was and the sequence is repeated.
- The greatest increase in the PR interval develops between 1st and 2nd beats in the cycle.
- R-R interval tend to shorten until the dropped beat. P-P interval should be normal and non-changing. P waves occur closer and closer to the preceding T wave.
- The longest cycle (containing the dropped beat) is less than two of the shorter cycles because is contains the shortest PR interval. (Less than double the R-R.)
- Tends to produce small groups of beats, esp. pairs because 3:2 Wenckebachs (3 P waves with 2 QRSs) are common.

Can occur as 2:1 Wenckebach. **Most 2:1 blocks are Wenckebach.**

- By itself, it is a benign condition.
Etiology — Most common cause is an inferior wall MI (usually there is transient ischemia of the AV node so the block may be reversible). Mobitz I blocks may be seen with disease in the AV node, digitalis toxicity (particularly when it occurs in combination with atrial tachycardia), and parasympathetic (vagal) tone. Commonly seen in athletic young patients, particularly during sleep.

Distinguished from 2nd AV Nodal Block, Mobitz type II by the fact that the PR interval of the P wave that follows the non-conducted P wave is at least 10 msec shorter than the PR interval of the P wave that precedes the non-conducted P wave.

Typically, the QRS complex is unchanged from the patient's normal QRS morphology.

By contrast, the PR interval does not change in Mobitz type II block.

Management — Therapy may not be necessary. If it is, atropine is the drug of choice, although a temporary pacemaker may be inserted. If the patient was receiving digitalis, it should be D.C.d.

Mobitz II

This block originates below the bundle of His in the BB system.

Usually an intermittent trifascicular block. Usually one bundle branch or fascicle is already permanently damaged, then damage occurs to the other. Sometimes one bundle vacillates between conducting and not conducting so that every other impulse is conducted resulting in a 2:1 block.

70% of the wide QRS blocks are Mobitz II, the others are Mobitz I.

Rare and often misdiagnosed.

There are 2 types of Mobitz II blocks — constant or periodic.

The constant type is regular; the periodic type is irregular.

Mobitz II rates are slow (1/2-1/3 of the normal rate).

The patient will have s/s, if the cardiac output decreases.

Characteristics:

- P wave is upright, uniform, and of SA origin.
- There is more than one P/QRS (2:1, 3:1, 4:1).
- PR interval is constant (but it can be prolonged).

Refers to occasional failure to conduct a P wave through the AV node to the ventricle without a change in the PR interval after the nonconducted P wave compared with before the nonconducted P wave.

A dangerous condition because it can progress to complete heart block and death without warning.

Placement of an external pacemaker may be lifesaving if a temporary pacemaker cannot be placed immediately. This condition, while dangerous, is very unusual.

QRS complex is usually wide, due to extensive disease of the His-Purkinje system, although a narrow QRS complex does not exclude the diagnosis.

Clinician should measure the change in PR interval carefully, as described for 2nd AV nodal block, Mobitz Type I.

Etiology of Mobitz II — includes anterior MIs, extensive destruction of the septum, shock and ventricular ectopic beats. This one is not caused by digitalis toxicity. It may progress to third degree block or ventricular standstill. This rhythm is usually irreversible.

Management — includes the administration of atropine, epinephrine, or Isuprel or the insertion of a temporary pacemaker followed by a permanent pacer. This rhythm is often not responsive to drug therapy. Usually not reversible.
3. **2° Atrioventricular Nodal Block (2:1 AV Block)**

- Diagnosed when the entire rhythm strip shows only conduction of every other P wave to the ventricle.

- Because the record does not show two consecutive P waves that conduct to the ventricle, it is not possible to measure prolongation of the PR interval, so that it is not possible to distinguish between 2° AV nodal block, Mobitz type I and the dangerous 2° AV nodal block, Mobitz type II. By convention, recordings obtained at other recent times are used to make this distinction.

4. **High Degree AV Block** — block which results in many dropped QRS complexes (vent. standstill).

- Type I (supra-Hisian) — benign phenomenon.

  May be seen with vasovagal reactions.

  In vagally mediated blocks, the sinus rate slows down and/or the PR interval prior to the dropped QRSs. The vagal stimulation affects both the SA node and AV conduction.

- Type II (infra-Hisian) — seen in association with Mobitz II can be extremely dangerous.

  If pt. with inferior MI and some degree of AV block (?1° or Wenckebach) vomits, the vagal stimulation may produce several seconds of complete block.

  Reverses after the vagal influence is gone.

  Treatment — same as for Mobitz II.

5. **Complete Heart Block (Third Degree)**

- There is no SA impulse conduction to the ventricles with this rhythm.

- The ventricular beat is idiojunctional if the AV junctional area is controlling the ventricular rhythm or idioventricular if the ventricle is controlling it.

- May be transient and cease after 2-4 days.
• There is a decrease in cardiac output, and angina, CHF, or syncope may develop.

• P waves are uniform.

• There is more than one P for each QRS, but the P is not related to the QRS.

• P-P and R-R intervals are regular.

• PR interval does not really exist since there is no relationship between the P and QRS.

• P waves sometimes fall on the QRS.

• QRS configuration is normal if the escape focus pacer is junctional and it is wide if the pacer is ventricular (focus is below BB bifurcation).

• Characterized by failure of conduction from the atria through the AV node to the ventricles.

• Atrial rhythm is independent of the ventricular rhythm, unless an accessory pathway that conducts antegrade is present.

• It is most easily distinguished from high-grade AV nodal block when the atrial and ventricular rhythms are regular but have different rates. Because of weak coupling between the chambers by the autonomic nervous system, these rates can be very close to each other and in fact can oscillate around each other.

• Complete heart block is one of three forms of AV dissociation. The other two forms are:
  _ Sinus arrest or sinus bradycardia with junctional rhythm or Idioventricular rhythm.
  _ Ventricular tachycardia.

Of these three forms, only complete heart block results from antegrade conduction block from the atria to the ventricles.

• Etiology — Complete block is seen after an inferior MI; sometimes after an anterior MI with RBBB and L axis deviation; rarely congenital; also seen with degeneration with age, myocarditis, cardiac surgery, and digitalis toxicity.

• Management — Isuprel may be used to treat the patient while preparing for pacemaker insertion.

**Interventions for Blocks Associated With Inferior Infarction**

• Determine if the patient is symptomatic from a slowed ventricular rate (check BP, urine, etc.).
• Monitor for development of Mobitz I or third degree block.

• Have atropine at bedside.

**Interventions for Blocks Associated With Anterior Infarction**

• Determine if patient is symptomatic from slowed ventricular rate (check BP, urine, etc.).

• Monitor for development of Mobitz II or third degree block.

• Have Isuprel and pacemaker at bedtime.

**Interventions for Blocks Associated With Drug Toxicity**

• Check digitalis and potassium levels.

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**PART IV: DIAGNOSIS OF DYSRHYTHMIAS**

Always state the impulse origin first and then add additional descriptions (e.g., sinus rhythm with right bundle branch block, first degree AV block, and frequent left ventricular PVCs). If there are two pacemakers driving the heart, mention the “higher” pacemaker first (e.g., sinus tachycardia with complete AV block and an idioventricular escape rhythm).

Remember, **TREAT THE PATIENT, NOT THE RHYTHM!**

**Questions for Every Rhythm:**

**What is the rate of the QRS?**

Rate > 100 is called *tachycardia*.

Rate < 60 is called *bradycardia*.

**Is the QRS regular or irregular?**

Irregular — atrial fibrillation, extra beats superimposed on sinus, sinus arrhythmia, others.

**Is the QRS narrow or wide?**

A wide QRS (> 3 small boxes) indicates a conduction delay or impulse arises in the ventricle.

**Are there P waves?**

Look at more than one lead if in doubt.

**Does a P wave precede ever QRS?**

**Is the PR interval constant?** (> 1 large box = prolonged PR [1° AV block].)

**Definitions:**

*Cycle* — one complete systole and diastole sequence; may be measured from P-P or R-R or T-T intervals.

*Premature* — occurs early or ends a cycle shorter than the dominant cycle (active).

*Escape* — occurs late or ends a cycle longer than the dominant cycle (passive).
Ectopic — arises from outside the normal pacemaker of the heart; can arise from the atria, AV junction, or ventricles; can be premature beats, escape beats, or a continuous rhythm.

Block — an abnormal delay or failure of conduction; must be differentiated from normal physiologic delays (e.g., atrial flutter with 2:1 conduction or sinus node suppression following atrial ectopy.)

A. Sinus rhythms

1. Normal Sinus Rhythm — discussed under section on 12-lead ECG interpretation.

2. Sinus Bradycardia — discussed under section on 12-lead ECG interpretation.

3. Sinus Tachycardia — discussed under section on 12-lead ECG interpretation.

4. "Relative" Sinus Bradycardia — heart rate slows down sufficiently to make the patient symptomatic, esp. if normal rate was previously rapid (from 110 to 70 bpm).

5. Sinus Arrhythmia:

   • Irregular rhythm representing a cardiac adjustment to respiratory or neurologic Δs.

   Rate varies — during inspiration due to the baroreceptor response to increasing negative pressure; during expiration, there is a increase rate.

   • Rate is like a NSR, 60-100 bpm. Usually a benign finding.

   • P-P regularly irregular.

   • PR interval normal.

   • QRS for each P.

   • Treatment — none. Considered normal variant.

   • Sinus arrhythmias are normal in kids. Non-respiratory (pathologic) sinus arrhythmias are seen in elderly patients who have SA node disease or digitalis toxicity.

6. Sinus Arrest

   • Results from failure of the sinus node to activate the atria.

   • Usually benign when duration short (less than one to two seconds).

   • Can be life-threatening (because of the potential for longer periods of sinus arrest with asystole) when duration long (~ 3 seconds).

   • Causes — medications, including as ß-blockers, some Ca++ blockers such as diltiazem, Aldomet, and digitalis or quinidine toxicity, excess vagal stimulation, hyperkalemia, stress, or vasoconstriction.

   • Can also be part of the sick sinus ("tachy-brady") syndrome, one of the leading indications for implantation of permanent pacemakers in this country.
• With sinus arrest or pause, the impulse is never formed.

7. Sino-Atrial Block — unclear whether the sinus node pacer cells generate impulses which fail to be conducted out of the sinus node (SA exit block) or whether the pacer cells fail to generate impulses (SA arrest). Can be manifested by any of the following:

• Extremely slow sinus rate (e.g., 25/min) which cannot be justified simply as sinus bradycardia (may be 2:1, 3:1 etc. exit block).

• Occasional dropped beats (i.e., the entire P-QRS-T sequence is missing); may be sinus Wenckebach.

• No P waves at all (complete SA block or standstill).

One of 3 things can happen when you have complete SA block:

_ Cardiac standstill and death (rare).

_ Lower pacemaker comes to the rescue and takes over (junctional escape or ventricular escape rhythm).

_ Chronic atrial fibrillation, typically with a slow ventricular rate.

• Causes of SA block:

Digitalis, β-blockers, quinidine, etc.

Hyperkalemia

SA node ischemia/infarction (RCA or Cx artery disease).

Vagal stimulation.

Other cardiac disease.

Degenerative Δs with aging.

• Treatment of SA block:

Hold drug until toxicity ruled out.

Atropine and/or temporary pacing if patient symptomatic.

Permanent pacing if block persists and is not due to some reversible situation.

• SA node and intra-atrial blocks are rare. Blocks can be transient, intermittent, or permanent and complete or incomplete.

8. Sick Sinus Syndrome — Brady-Tachy Syndrome
• ECG manifestations:

Marked sinus bradycardia or arrest often resistant to atropine or Isuprel.

Longer (~ 12 sec) asystolic pause than can be explained by simple SA node suppressions following:

A single PAC.

DC cardioversion.

Spontaneous conversion of atrial flutter or fibrillation.

Rapid atrial pacing.

Periods of atrial fibrillation (or less commonly atrial flutter) either with a slow or rapid ventricular response alternating with periods of asystole, junctional escape rhythm, or ventricular escape rhythm or extremely slow junctional rate (< 40/min).

• Causes — same as for SA block.

• Symptoms:

Palpitations (either during tachy or brady phase).

Syncope or near syncope (during asystolic or brady cardiac periods).

CHF (inadequate CO because of slow heat rate, rapid heart rate, and loss of atrial kick).

Increased angina (inadequate CO) in patients with coronary artery disease (CAD).

• Treatment:

Permanent pacing. (Rapid rate cannot be treated with usual methods due to risk of bradycardia.)

After pacer inserted, drugs to convert atrial fibrillation or to slow ventricular rate in chronic atrial fib.

• Sick sinus syndrome may coexist with atrial flutter or fibrillation in elderly.

9. Hidden Atrial Activation

• Activation of the atrium by the sinus node can be inferred from surrounding sinus P waves. For example, if the P wave following a PVC (VPC) occurs at the time that would have been expected had the premature complex not occurred, then in can be inferred that the atrium was not activated retrogradely by the premature complex and that a hidden, or obscured, P wave did occur.
• Such an inference can be confirmed during invasive electrophysiologic study.

B. Ectopic Atrial Rhythms

1. Premature Atrial Contractions (PACs)

• Earlier than expected P waves — morphology of premature P wave usually differs from the sinus P.

• Atrial P wave often falls near the T wave and distorts it — P is flat, notched, or lost in the T in at least one lead.

• PR interval is usually normal.

• Rhythm is regular except for the premature contraction.

![Premature Atrial Complex (PAC)](image)

• PAC will have 1 of 3 fates: it will be non-conducted, conducted normally, or conducted abnormally.

Non-conducted PACs arrive at the AV junction before it has repolarized. Because the junction is refractory, the QRS is missing and there is no contraction for that beat.

As Dr. Marriott observes, "the commonest causes of pauses are non-conducted atrial premature complexes."

![Nonconducted PACs](image)

Conduction can be normal with a PAC if the PAC arrives at AV junction after it has repolarized; can then be transmitted to ventricles, initiating a normal, narrow QRS.

Aberrant conduction — traverses the bundle branches of the His-Purkinje system while one or both is in its relatively refractory period; resulting QRS complexes are wider than normal and have the morphology of BBB pattern.

• Atrial premature complexes are a normal finding in adults of all ages; frequency can increase during stress, ingestion of caffeine, after a heavy meal, and sympathomimetic drugs such as some OTC cold remedies.

• PACs are also seen in rheumatic dz., ischemia, hyperthyroidism, digitalis toxicity, MI, CHF, CAD, and hypokalemia.
2. Aberrant Conduction

- Occurs when the AV node is partially refractory and usually results in a RBBB pattern; impulse arrives at the LBB after it has repolarized, but at the RBB before it has repolarized; QRS is distorted (aberrantly conducted) because the impulse will be conducted normally to the LV, but relayed by aberrant pathways to the RV.

- $V_1$ and $V_6$ are triphasic and predominantly positive with aberrant PACs.

- Aberrant PACs look a lot like PVCs.

- Differential diagnosis should focus on whether a premature P wave precedes the wide QRS and whether a RBBB pattern occurs in $V_1$ or $V_6$.

Best criteria to support the dx. of aberrant supraventricular contraction is the presence of a premature P wave. Unfortunately the P wave of PAC may be buried so you may not see it; if a P wave precedes a PVC, it is usually sinus and not premature.

Aberrantly Conducted PAC

1. Normal supraventricular conduction.
2. Normal conduction in the left bundle branch.
3. Blocked conduction at the right bundle branch which is still refractory.
4. Normal conduction in the left ventricle.
5. Abnormal, delayed conduction in the right ventricle by aberrant pathways.

- **Cycle is reset** with PACs.

Look at the duration between the beat that precedes the one with the wide QRS and the normal beat which follows it.

A less than compensatory pause is often seen with PACs because of retrograde discharge of the SA node and resetting of cycle. The distance is less than you would expect from 2 normal beats.

(You have a compensatory pause with PVCs.)

3. Atrial Couplet

- Pair of atrial premature complexes in a row.

- Less common in normal subjects than are PACs, it can still be benign.

- Appearance of atrial couplets should also raise the index of suspicion for susceptibility to atrial fibrillation, atrial flutter, and supraventricular tachycardia (SVT).
4. Atrial Multiform Couplet

- Pair of PACs, with differing P wave morphologies, in a row.
- Unusual in normal subjects, but is itself benign.
- Appearance of multiform atrial couplets, esp. in patients with pulmonary dz., should raise the index of suspicion for susceptibility to multifocal atrial tachycardia, atrial fibrillation, and atrial flutter.

5. Wandering Atrial Pacemaker

- Supraventricular rhythm resulting from multiple ectopic foci in the atria.
- Characterized by 3 or more P wave morphologies.
- Rate < 100 bpm.
- Sinus rhythm gives way to an ectopic or junctional rhythm. May see fusion beats at the transitions.

Two opposing electrical currents (sinus and atrial ectopic) within the same chamber at the same time \( \Rightarrow \) P wave often narrower and of lesser amplitude than the normal sinus P wave.

- Benign, but reflects electrical abnormalities in one or both atria that increase the likelihood of multifocal atrial tachycardia or other atrial arrhythmias.

6. Multifocal Atrial Tachycardia (MFAT)

- Supraventricular rhythm resulting from multiple ectopic foci in the atria.
- Characterized by 3 or more P wave morphologies.
- Rate \( \geq \) 100 bpm.
- Seen most frequently in patients with severe pulmonary disease.
- Rapid ventricular rate can be symptomatic (hypotension, angina, CHF).
- Treatment — improvement of the concomitant pulmonary disease and consideration of administration of verapamil. *Digoxin is not effective in treatment of this rhythm.*

7. Atrial Escape Complex

- P wave that occurs later than would be expected from the sinus rate.
• Like all escape complexes, it can occur only when the normal cardiac pacemaker does not function, as in sinus arrest.

8. Atrial Tachycardia

• Abnormal supraventricular rhythm.

• Results from either an atrial automatic focus or a reentrant circuit that lies entirely within the atrium.

• Rate > 100 bpm.

• P wave morphology is usually different from that of the sinus P wave.

• Can be intermittent or incessant (present more than 50% of the time).

When incessant, it can cause symptomatic dilated cardiomyopathy that is reversible with control of the tachycardia.

• Can result from digitalis toxicity, particularly when it occurs in combination with 2\(^{nd}\) AV nodal block, Mobitz Type I (Wenckebach).

• Emergency treatment of this rhythm (when patient is symptomatic) — synchronized cardioversion with appropriate anesthesia.

• Short-term pharmacologic control — drugs that decrease AV nodal conduction (\(\beta\)-blockers, Ca\(^{++}\) blockers).

9. Atrial Flutter

• Atrial rate = 250-350 bpm; ventricular response usually ~ 75, 150, or 300 bpm.

• Atrial beats are regular.

• Ventricular beats are also usually regular but you may see a variable block, because the junctional area blocks some beats.

• Causes decreased filling of the ventricle if ventricular rate is too fast.

• P wave is called an F wave and has a sawtooth, picket fence shape in leads II, III, aV\(_5\), and V\(_1\).

• Even if flutter waves are not found, this rhythm should be suspected when the ventricular rate ranges from 140-160 bpm and there is no clear evidence of atrial activity.

• PR interval is unknown.
QRS is normal (narrow) but not 1:1 with the F (P) waves.

At least 2 different types (FYI) — one with regular morphology and a rate of 240-350 bpm and a rapid type with a rate greater than 350 bpm. The rapid type is difficult to treat and often Δs to V fib.

Carotid massage may slow the ventricular rate so flutter waves become apparent but massage does not terminate the dysrhythmia.

Treated if the ventricular rate is rapid or patient symptomatic — β- or Ca++ channel-blockers or digoxin; then either synchronized cardioversion or pacing.

Rapid atrial pacing may be used to interrupt flutter. For example, if the flutter rate is 280 bpm, the heart could be paced at 320 bpm. Pacing may produce A fib, which is easier to treat than flutter.

Supraventricular rhythm resulting from a reentrant circuit that lies within the R atrium or a single ectopic focus.

Usually begins with a premature beat and then is supported by reentry beats.

Can occur alone, but is usually associated with hypertensive cardiomyopathy, COPD, cor pulmonale, CAD, rheumatic heart dz., congestive cardiomyopathy, or hyperthyroidism.

• New onset is seen in about 5% of cases of acute MI. Clinician may also want to check for CHF, since worsening CHF can present with these rhythms.

10. Atrial Fibrillation

• P wave is called an f or fibrillatory wave and consists of a wavy line that is seen best in the inferior leads (I, II, aVF).

• PR interval — none.

• QRS is normal or aberrant.

• Atrial and ventricular rate and rhythm are irregular.

• R-R interval and baseline are variable.

• Atrial rate is > 350 bpm; the ventricular rate is 40-170 bpm. Ventricular response may be slow (< 60) or rapid (> 100).

• Characterized by an irregularly irregular ventricular rate that is usually rapid in young patients, but may be normal or even bradycardic in elderly patients or patients taking medications that cause AV nodal blockade.

• Most common supraventricular rhythm — found in 4/100 people age 55-64; 9/100 of those over 65.

• Seen more frequently in males than females.

• Ineffective quiver that occurs with A fib results in the loss of the atrial kick; also results in decreased CO, increased O2 demand, and an enlarged left atrium (? cause or effect).

• Usually due to re-entrant excitation with multiple reentry circuits within either atria, or both.

• May be chronic or paroxysmal.

• Can occur alone, but is usually a/w hypertensive cardiomyopathy, COPD, or congestive cardiomyopathy.

• New onset of either atrial flutter or A. fib. is seen in about 5% of cases of acute MI. Clinician may also want to check for CHF, since worsening CHF can present with these rhythms.

• Pulse deficit occurs — apical pulse is > than radial because some contractions are so feeble.

• Well tolerated by most patients. There are four different types (types I-IV) of A. fib.
Peripheral arterial emboli due to thrombi in the atria are seen in 30% of the patients; a/w an increased incidence of CVA after age 59, because the risk of emboli is 4-5x normal.

Low dose anticoagulation with warfarin significantly reduces the stroke rate without significantly increasing the hemorrhage rate.

Goal = 1.3 to 1.6 x normal PT (or an INR of 2.0-3.0).

ASA does not decrease risk of emboli in people over 70 y.o. as well as it does in middle-aged people.

HTN predisposes to emboli risk in A. fib. patients.

- Seen with atrial muscle dz., atrial distention, SA node dz., CHF with hypoxemia, stress, pericarditis, drug toxicity, hyperthyroidism, mitral stenosis, HTN, cerebral or cardiac ischemia, PE, Rh heart disease, acute infection, MI, cardiomyopathy, COPD, and digitalis toxicity.

- Treatment

Cardioversion can be accomplished with antiarrhythmic drugs or electrically.

Antiarrhythmic drug therapy may be 6 doses of flecainide or quinidine and, if that does not work, synchronous cardioversion may be used.

Anticoagulation therapy is begun 3 weeks before and continued 4 weeks after synchronous cardioversion.

Digitalis decreases the AV block and allows increased ventricular filling, so it is used to treat rapid ventricular responses.

Quinidine, propranolol, verapamil, and Pronestyl (also used to control rate) are also used.

Catheter ablation of the His bundle to create a 3º AV block is used for the patient with a rapid ventricular rate that does not respond to medications. This requires a permanent pacemaker.

Clonidine also used to treat A. fib. with a rapid ventricular response.

Clonidine decreases sympathetic outflow from brain, and increases parasympathetic outflow, thus slowing conduction across the AV node, decreases the ventricular response rate, and sometimes converting A. fib. to NSR; can be used in place of β- and Ca²⁺ blockers (with CHF), in place of β-blockers (asthma), and in place of digitalis (hypertrophic obstructive cardiomyopathy).

• **Digitalis toxicity is suggested by a regular ventricular response** (accelerated junctional rhythm) in combination with atrial fibrillation.

  - In the presence of an accessory AV pathway, atrial fibrillation can manifest as a rapid, irregularly irregular wide complex tachycardia that can resemble ventricular tachycardia closely; should be suspected particularly in young patients with very rapid tachycardia that is well tolerated hemodynamically; close examination of the ECG will reveal irregularly irregular RR intervals.

  - Important to obtain a 12-lead ECG before cardioversion because the location of the pathway, and therefore the risks of a subsequent curative catheter-mediated radiofrequency ablation procedure, can be estimated fairly accurately from the 12-lead ECG.

  - Do NOT give digitalis or verapamil to try to slow ventricular response if an accessory pathway is suspected. These drugs can accelerate conduction over accessory pathways, resulting in even more rapid ventricular activation which can, in turn, induce ventricular fibrillation.

  - Four issues related to care of patients with atrial fibrillation:

    1. Control of the rate of the ventricular response,
2. Conversion of the atrial rhythm to sinus rhythm,

3. Maintenance of sinus rhythm following conversion, and

4. Prevention of embolic stroke from thrombi that form in the fibrillating left atrium.

11. Supraventricular Tachycardia

- A generic name for a variety of specific supraventricular rhythms, including Atrioventricular Reentrant Tachycardia, Atrioventricular Nodal Reentrant Tachycardia, and Atrial Tachycardia.

- Also used in reference to any narrow complex rhythm to distinguish it from wide-complex rhythms that could arise in the ventricle.

- Use of the term includes atrial fibrillation, atrial flutter, junctional tachycardia, accelerated junctional rhythm, and multifocal atrial tachycardia.

12. Paroxysmal Supraventricular Tachycardia (PSVT)

- Old name was paroxysmal atrial tachycardia (PAT); no longer called PAT unless P waves are clearly seen.

- PSVT rhythms start and stop suddenly in comparison to sinus tachycardia, which begins and ends gradually.

- PSVT is also differentiated from sinus tachycardia by altered configuration of the P waves.

- PSVT is supported by either an AV nodal reentry circuit (AV nodal reentry tachycardia — AVNRT) or an AV reentry circuit using the AV node and an accessory pathway (circus movement tachycardia — CMT). Rate 170-250 bpm.

13. Atrioventricular Nodal Reentrant Tachycardia (AVNRT)

- Rate — 150-250 bpm (rarely above 250).

- QRS that is usually normal in contour and duration.

- P waves may or may not be seen; follow closely after the QRS if they are seen; retrograde P is usually lost in the QRS.

- A reentrant supraventricular rhythm whose circuit is located in the region of the AV node.

  Two functionally separate pathways down to the AV node — a slow (posterior) pathway and a fast (anterior) pathway. Slow pathway has the shortest refractory period.

  When an early atrial beat arrives at the AV node, it may be conducted only down the slow pathway to the ventricles, turning around in the AV node to return to the atria via the fast pathway.

  Atria and ventricles are activated simultaneously.

  Following a PAC, the PR interval at the onset of the tachycardia is long as the impulse uses the slower pathway.

  Subsequent P waves are buried in the QRS (retrograde activation.) QRS complexes are narrow.
• Only about 60% of narrow-complex tachycardias have this mechanism but 20% of all narrow-complex tachycardias are AVNRT, which use a concealed accessory pathway for retrograde conduction.

AV Nodal Reentry Tachycardia

(2 beats sinus, early beat, long PR ⊗ impulse "jumps over to slow pathway"

• Clinical significance of this rhythm depends on the rate.

• Most often paroxysmal and benign. Stops abruptly with effective treatment.

Usual initial treatments are the Valsalva maneuver, then IV adenosine (Adenocard).

Adenocard stimulates K⁺ conductance producing hyperpolarization, leading to AV block and coronary dilation.

If these are unsuccessful, one can try medication that reduces conduction through the AV node.

Rhythm can now be cured by catheter-mediated radiofrequency ablation.

In some cases it may be recurrent, symptomatic, and refractory to medical therapy.

• Presence is usually unrelated to preexisting heart disease.

• AVNRT is usually initiated by a PAC, and maintained by a reentry mechanism; impulse moves in an anterograde direction over a slow conducting pathway and retrograde over a more rapid pathway.

• Atria contract against closed AV valves, causing a reflux up the jugular vein.

• Other s/s are dyspnea, anginal pain, perspiration, fatigue, anxiety, dizziness, and polyuria; in the presence of heart dz., you may see CHF or shock.

• AV Nodal Pathways: 2 separate pathways leading to a common AV node exist in many normal individuals. Normally, impulses travel through the conduction fibers in an even and synchronous manner and collide with each other in the potential circuits. Ischemia can depress conduction or convert fast cells to slow cells setting up an environment for AV node reentry tachycardia (AVNRT).

14. AV Reciprocating Tachycardia (AVRT or "Circus Movement" Tachycardia)

• Orthodromic Tachycardia — most common form, proceeds antegrade (from atrium to ventricle) over the AV node, and retrograde over an accessory pathway.

Pt. has two AV pathways — the normal AV node and His bundle + an accessory pathway.

Usually initiated by a PAC but may be initiated by a PVC.

Impulse enters ventricles via the AV node and His bundle and returns to the atria via a rapidly conducting accessory pathway placing the P' wave close to the preceding QRS.

Impulse circulates around and around in this sequence.
Initial PR interval not prolonged.

Also a common arrhythmia in WPW syndrome.

- **Antidromic Tachycardia** — proceeds in the reverse direction; a wide QRS tachycardia except when the accessory pathway is located in the right anteroseptal location very close to the His bundle.

- When multiple pathways are present, it is also possible for the circuit to use 2 pathways as a circuit.

- P waves may or may not be seen, but they usually do not follow closely after the QRS if they are seen.

- Clinical significance — depends on the rate.

- Stops abruptly with effective treatment.

Usual initial treatments — Valsalva maneuver, then IV adenosine.

If these are unsuccessful, one can try medication that reduces conduction through the AV node, except that verapamil and digitalis SHOULD NOT BE GIVEN.

Can now be cured by catheter-mediated radiofrequency ablation.
C. Accessory AV Pathways

- "Preexcitation syndrome" is the term used to describe pathways that bypass the AV node and cause short PR intervals. Those pathways that insert into the ventricle wall produce delta waves that are seen in sinus rhythms.

Delta waves result from a fusion complex (2 beats trying to control ventricles at the same time).

- There are three common bypass systems:

  _Wolff-Parkinson-White (WPW) Syndrome:

  The abnormal pathway is called the Kent bundle or accessory AV connection. This pathway bypasses the normal AV conduction system and connects the atria directly to the ventricles.

  There is a delta wave and the PR is short. WPW is the most common preexcitation system.

  Term used to describe the presence of one or more accessory AV pathways that conduct in the antegrade direction, with or without retrograde conduction.

  Patients with this syndrome are susceptible to AV reentrant tachycardia and atrial fibrillation.
Mahaim Fiber: The abnormal pathway for this system is a fasciculo-ventricular or a nodo-ventricular connection. The pathway originates below the AV node and bypasses part or all of the ventricular conduction system and inserts directly into the ventricle wall. The pathway connects the lower AV node or bundle of His directly to the ventricle. The PR is normal, but a delta wave is produced.

Lown-Ganong-Levine Syndrome: The abnormal pathway for this system is the James bundle (atrio-Hisian tract). This pathway inserts into the conduction system just below the AV node, thus connecting the atria to the lower part of AV junction. A short PR is seen but there is no delta wave with this pathway.

Reciprocal Complex — A QRS complex that is caused by activation of a reentrant circuit rather than by the sinus node. This can be harbinger of AV nodal tachycardia or AV tachycardia.

Retrograde Atrial Activation — A P wave that occurs because of activation of a portion of the heart below the sinus node, including elsewhere in the atrium, the AV node (via the fast or a slow AV nodal pathway) or the ventricle (via an accessory pathway). Retrograde P waves typically are inverted in the inferior and right precordial ECG leads (II, III, aVF, and V1), in which the normal sinus P wave is upright.

E. Junctional Ectopic Beats and Rhythms

1. Junctional Dysrhythmias

• It is really the tissue around the AV node that is normally conductive.

• Junctional dysrhythmias are significant because they may be too fast or too slow to provide adequate cardiac output.

• Depending on the timing of retrograde conduction, the P occurs before (high junctional), after (low junctional), or during (mid junctional) the QRS.

• Junctional dysrhythmias often signal digitalis toxicity.

<table>
<thead>
<tr>
<th>UNCOMPPLICATED NODAL RHYTHMS</th>
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<tr>
<td><strong>P Wave Location</strong></td>
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<tr>
<td>&quot;High&quot; nodal</td>
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<tr>
<td>&quot;Mid&quot; nodal</td>
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<tr>
<td>&quot;Low&quot; nodal</td>
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2. Junctional Escape Complexes

• Junctional escape rhythms originate low in the AV node, high in the common bundle. They may be recognized by following clues

• The first beat of a junctional escape occurs later in the cycle than the next expected beat; the escape beat occurs after a beat has been missed

• Rate of 40-60 (inherent rate of junctional cells)

• The rhythm is regular

• P — inverted in II, III, and aVF (atria depolarization is retrograde)

• P and QRS not 1:1

• There is no PR interval unless the P appears before the QRS, then it is less than normal.

• QRS has normal morphology for the patient that is not preceded by a P wave and occurs later than would be expected from the sinus rate.
• Like all escape complexes, it can occur only when the normal cardiac pacemaker does not function, as in sinus arrest.

3. Junctional Premature Complexes

• Treated like premature atrial beats.

• If the atria and ventricles contract together, blood is forced back to the vena cava causing distended neck veins.

• QRS complex that occurs earlier than would be expected from the sinus rate, and that usually has a normal morphology for the patient.

• It can fail to conduct retrograde through the AV node, in which case it results in a compensatory pause. (The next P wave occurs at the same time as would be expected had the JPC not occurred.)

• More usually, it does conduct through the AV node, so that the following P wave may occur either sooner or later than would be expected.

• Relatively uncommon.

• Can be seen with increased frequency during stress, with ingestion of caffeine, and with sympathomimetic drugs such as some over-the-counter (OTC) cold remedies.

• Can also be misdiagnosed when the P wave of a PAC is obscured by the preceding T wave.

4. Junctional Premature Couplets

• An unusual rhythm, and most likely represents two cycles of one of the supraventricular tachycardias.

5. Junctional Rhythms

• A slow rhythm, with rates ranging from 40 to 60 beats per minute.

• QRS complexes have the patient's normal morphology.

• Usually, no P waves are seen. When P waves are present, they follow closely after the QRS complexes.

• Results from the backup pacemaker capability of the AV node during sinus arrest.

6. Accelerated Junctional Rhythms

• A supraventricular rhythm resulting from a focus in or near the AV junction.

• Rate ranges from 60 to 100 bpm.

• An abnormal rhythm that can result from digitalis toxicity, particularly when it occurs in combination with atrial fibrillation.

• Can also result from physiologic stress and other causes of increased sympathetic nervous system tone.
7. Junctional Tachycardia

• A supraventricular rhythm resulting from a focus in or near the AV junction.

• Rate is around 100 bpm.

See also: accelerated junctional rhythm.

• Usually results from a primary arrhythmia rather than as a response to physiologic stimulation.

• ECG usually cannot distinguish this rhythm from the more common types of supraventricular tachycardia, for which different treatments may be appropriate.

• Junctional tachycardia can be of several types — AVNRT (150-250), enhanced automaticity tachycardia (100-150), or accelerated junctional rhythm (digitalis induced) (65-100).

8. Interventions for Junctional Rhythms:

• Assess the ventricular rate, BP, sensorium, urine output, and skin changes • Determine if the dysrhythmia is due to digitalis

• Assess other possible causes like ischemia, infarction, heart disease

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**EKG: SECTION FOUR**

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F. Ventricular dysrhythmias

Ventricular dysrhythmias are frequently unstable and unpredictable. These dysrhythmias are also potentially lethal, because the heart does not provide a synchronous or coordinated contraction so SV and coronary flow are compromised.

1. Premature Ventricular Complexes

• Most common arrhythmia seen in a healthy adult. PVCs (or VPBs) are seen in 50-63% of healthy individuals and in 72-93% of post MI patients.

PVCs are a normal finding in adults of all ages. The frequency can be during stress, with ingestion of caffeine, and with sympathomimetic drugs such as some over-the-counter cold remedies.

The frequency is also in patients with a tendency to develop ventricular tachycardia.
PVCs are also a/w drugs like lidocaine, procainamide, quinidine, and propranolol; and a/w disease states like MI, cardiomyopathy, CVA, CHF, hypokalemia, mitral valve prolapse, infectious disease, ischemia, and electrolyte imbalance.

The carboxy-Hb of 4-6% that is seen in cigarette smokers is a/w numbers of PVCs in CV patients during exercise. Atmospheric pollution produces similar dysrhythmias.

- Originates in the ventricle and interrupts normal regularity.

A wide QRS complex that occurs earlier than would be expected from the sinus rate, and that almost always has an abnormal morphology.

- Fails to conduct retrograde through the AV node in half of patients, in which case it results in a compensatory pause. That is, the next P wave occurs at the same time as would be expected had the PVC not occurred.

- When it does conduct through the AV node, the following P wave may occur either sooner or later than would be expected.

- PVCs may be early or late — a late PVC is only slightly premature and is a/w a non-related P wave (the timing is similar to fusion beats).

- PVCs may be single or multiformed (formerly called single or multifocal).

- The HR depends on the number of PVCs.

- Patients are often unaware that they are having PVCs, but they may have palpitations, skipped beats, dizziness, SOB, chest discomfort, angina, or hypotension.

- PVCs are not preceded or initiated by a P wave.

If a PVC is preceded by a P wave, it is not a premature P — it is a nonconducted P.

- A PVC is initiated in the ventricle; therefore, there is no PR interval.

- The QRS is always wide and bizarre, and has a longer duration than normal because depolarization originates in the ventricle and follows an abnormal pathway.

- The T wave is in the opposite direction from the R.

- There may be couplets, salvos, bigeminy, trigeminy, or a compensatory pause.

A couplet is 2 consecutive PVCs and a salvo is a short run of 3 or 4 PVCs.

A couplet can be a normal finding, but is more suggestive of electrical heart disease than are single PVCs.

Bigeminy (group of 2) refers to pairs of complexes and signifies a normal sinus beat followed by a PVC.

Trigeminy (group of 3) refers to a PVC with 2 consecutive sinus beats or vice versa.

- PVCs may be treated if they occur more than 6/min. The tx. consists of the administration of medications like lidocaine (bolus then drip), Pronestyl, bretylium, or Inderal.

The cause (electrolyte imbalance, etc.) also should be corrected.

Compensatory Pause: A PVC wipes out the expected sinus related QRS and there is a compensatory pause before next normal cycle.

The pause occurs because the AV node is refractory and does not allow retrograde conduction so the next sinus impulse comes when it is supposed to.

The duration is what you would expect from 2 normal beats.
The PVC hides the normal SA impulse — similar to the QRS hiding atrial repolarization on a normal ECG.

- **R on T Phenomenon:** If an R falls on a T wave, it may cause VT or V-fib.

- **Fusion Beats**

Fusion beats are usually a combination of a SA impulse and a PVC, or a SA impulse and an artificial pacemaker impulse.

Fusion or capture complexes confirm the coexistence of supraventricular and ventricular foci. The same chamber of the heart is depolarized by 2 simultaneous impulses leading to blending or fusion of the two.

The two impulses are both trying to control the heart at same time; the complex has characteristics of the supraventricular and the ventricular beat.

The first part of the complex looks normal because of the sinus influence, but the last half is abnormal.

The fusion complex is usually more narrow than the PVC.

You need to look at both the normal and the PVC beats and then look at the fusion beat to differentiate this dysrhythmia.

Pacemakers produce ventricle foci so a fusion beat is expected in patients with pacemakers.

Fusion beats are seen with ventricular ectopy so their presence can be used to help differentiate SVT with aberrancy from VT.

Fusion beats are treated as PVCs. If the overall rate is OK, it is usually not necessary to report fusion beats unless there are more than 5/min.

**Differentiating PVCs from Aberrant PACs — Precordial Concordance** (see page 6) confirms ventricular ectopy. Normally the QRSs in precordial leads change from downward in V1 to upward in V6. When the depolarizing current is directed toward base of the heart from a ventricular focus, this progression is altered and all of the precordial leads are upright or downward.

PVCs tend to be mono- or biphasic in V1 and V6.

V6 is usually predominantly negative in the presence of a L ventricular PVC.

V1 is predominantly negative in the presence of a R ventricular PVC.

**2. Idioventricular Escape Rhythms**

- Originates in the His-Purkinje system.

- The first beat of an escape rhythm occurs later in the cycle than expected (after a beat is missed).

- Usually regular with a rate of 20-40 (inherent ventricular rate).

The slow rate ∅ — cardiac output and also permits the emergence of lethal dysrhythmias.

- Usually there is no P or PR interval (if present, they bear no relation to the QRS).

- The QRS is wide and bizarre, because it originated in the ventricle and was not conducted normally.

- Diagnosed when only ventricular escape complexes are present, and they occur at 20 to 40 beats per minute.

- This rhythm is barely consistent with life.

- If you see this, you should start CPR immediately, and should move the patient to an ICU as soon as possible.
• **DO NOT** give lidocaine or any other antiarrhythmic medication for this rhythm. You could cause asystole and death by inhibiting the only spontaneous rhythm the patient's heart is able to generate.

**Management:** The ventricular rate needs to be accelerated. The rhythm is enhanced with drugs, pacing, or calcium.

### 3. Ventricular Escape Complexes

• A wide QRS occurring later than would be expected from the sinus rate.

• Like all escape complexes, it can occur only when the normal cardiac pacemaker does not function, as in sinus arrest.

### 4. Accelerated Ventricular Rhythm

• Diagnosed when only ventricular escape complexes are present, and they occur at 60 to 100 beats per minute.

• Usually seen in the setting of acute MI.

• If the patient is in sinus rhythm, the rate of this rhythm tends to be about the same as the rate of the sinus rhythm. In this case, the two rhythms will speed up and slow down so that they alternately capture the ventricle, with characteristic periods of fusion QRS complexes during the Δs in rate.

• This rhythm is usually benign. Because it occurs in the setting of acute MI, patients who exhibit it are already in an ICU where any malignant sequelae can be treated readily.

### 5. Ventricular Tachycardia (VT)

• VT is usually initiated by PVCs, but carried on by re-entry mechanisms.

• A single focus VT looks like a series of PVCs.

• It is usually regular or slightly irregular with a rate of 150-250 bpm.

• The beats are not preceded by P waves (the P wave is lost in the QRS); however, a dissociated P may be occasionally seen.

• There is no PR interval.

• The QRS is wide and bizarre and its duration is increased.

• If it is seen, the T wave is in the opposite direction of the QRS.

• The atria and ventricles beat independently so the cardiac output is decreased.

Often there is no palpable pulse.

• VT often changes to V-fib (and carries a high risk of sudden death).

• **Etiology**

VT is normal in rare individuals.

It is seen 2° to electrolyte (hyper- or hypokalemia, hyper or hypocalcemia) and metabolic disorders (hypoxemia, acidosis), drug effects (digitalis toxicity), after an MI, or in association with cardiomyopathy and mitral valve prolapse.

It may be caused by the R on T phenomena.

• Diagnosed when three or more PVCs occur in a row at a rate of 100-120 beats per minute or faster.
• The major clinical distinctions are between hemodynamically unstable versus stable VT and between sustained versus unsustained VT.

Hemodynamically unstable ventricular tachycardia is a life threatening emergency for which the ACLS protocol should be initiated immediately. Synchronized cardioversion is usually the tx. of choice. Awake patients should be sedated heavily before cardioversion if at all possible.

• **Sustained ventricular tachycardia** is defined as having a duration of 30 seconds or more, or being hemodynamically unstable.

The immediate treatment is specified by the ACLS protocol.

For long-term treatment, it is important to realize that these patients have a 20% to 40% sudden death mortality, when untreated, over the 12 months following initial presentation.

Empiric treatment with antiarrhythmic drugs does not reduce this mortality.

Effective treatment with drugs and/or an implantable cardioverter defibrillator reduces the sudden death mortality over the next 12 months to 0-2%. Therefore, consultation with a cardiac electrophysiologist is recommended during the initial hospital stay to ensure adequate evaluation and tx. before discharge from the hospital.

• Management — If the patient is stable lidocaine is the treatment of choice — bolus then drip.

Other treatments include procainamide and quinidine, which prolong the refractory period and break the cycle; potassium chloride; or β-blockers.

Counter shock is used if the patient is unstable.

If the patient is receiving digitalis, the digitalis should be D.C.’d.

Vagal stimulation will not stop VT; the ectopic focus must be depressed.

Calcium-channel blockers are also ineffective.

6. **Polymorphic Ventricular Tachycardia or Torsades de Pointes** (twisting of points)

• Special form of VT characterized by changing QRS morphology, sometimes accompanied by slight changes in the rate. • A particularly malignant form of VT that is thought to be intermediate between ordinary monomorphic VT and ventricular fibrillation.

• **For etiology, think of proarrhythmia, as from type IA antiarrhythmic medications, hypokalemia, hypomagnesemia, profound bradycardia, and idiopathic prolonged QT syndrome.**

7. **Ventricular Fibrillation**

• A lethal rhythm characterized by absence of both organized electrical and organized mechanical activity. There are rapid, irregular ineffective ventricle contractions.

• Equivalent to cardiac death. There is no cardiac output with V-fib. Initiate CPR immediately.

• There is no P, QRS, or T wave.

• V-fib is multifocal.

• **Coarse V-fib** is thought to be recent onset V-fib and thus easier to revert with unsynchronized countershock than **fine V-fib**.

Epinephrine may convert fine fib into coarse fib (one reason we use it in CPR).

VENTRICULAR FIBRILLATION (V-FIB)

• Signs and symptoms of V-fib include loss of consciousness within seconds, seizures, no pulse, and dilated pupils.

• Etiology — R on T, VT, MI, digitalis or quinidine toxicity, electrolyte disturbances, CAD, dying heart.
• Management of V-fib — includes the use of drugs like lidocaine and procainamide, or unsynchronized countershock (stops the heart by depolarizing all of the cells at once so SA can take over).

V-fib rarely spontaneously terminates.

8. Parasystole

• Results from intermittent capture of the ventricle by a ventricular focus that has an entrance block.

(It is not depolarized when the remainder of the ventricle is activated.)

• Characterized by PVCs with variable coupling intervals (intervals from the preceding normal QRS complex to the premature complex) and with constant intervals between the premature complexes. Detection of the latter constancy usually requires finding the least common denominator of the intervals between premature complexes, because of the intermittency of ventricular capture by the focus.

• Rare.

• Usually considered benign, although any premature ventricular activation can induce malignant ventricular rhythms in the ischemic myocardium.

9. Asystole Rhythm (Ventricular Standstill)

• A terminal, lethal phenomenon.

• P waves may or may not be present — atrial electrical activity stops at same time or shortly after the cessation of ventricular activity.

• Initiate CPR immediately. This is an agonal rhythm that is not consistent with life.

• Diagnosed when only ventricular escape complexes are present, and they occur very slowly.

10. Pulseless Electrical Activity (PEA) — formerly called electromechanical dissociation or EMD.

• Complexes appear on the monitor but there is no pulse or BP, because the electrical conduction is not coupled to the mechanical contraction.

• When giving CPR to these patients, you check effectiveness with the femoral or carotid pulse.

• PEA is seen with tamponade, severe hypovolemia, tension pneumothorax, massive pulmonary emboli, massive MIs, ruptured papillary muscles, CHF, etc.

PART V: Differential Diagnosis of Wide QRS tachycardias

PART VI: ST Segment Monitoring

PART VII: ELECTRICAL INTERVENTION

Pacemaker Electrocardiography and Trouble-Shooting

Pacemakers

All pacemakers deliver current to the heart. There are unipolar and bipolar pacemakers. An unipolar system has a single negative electrode in the chamber of the heart being paced. A bipolar system has both poles in the chamber of the heart being paced. The ECG pacing spike is larger in the unipolar system. Patients with pacemakers should carry a pacemaker I.D.

Pacemaker codes:

• 1st letter: chamber paced

Ventricle, atrium, dual chamber, or single chamber
• 2nd letter: chamber sensed

Ventricle, atrium, dual chamber, or single chamber

• 3rd letter: mode of response

Inhibits pacing, triggered, dual (both I and T), or none (0)

• 4th letter: programmable functions

Programmable, multiprogrammable, communicating, or rate modulating

• 5th letter: tachyarrhythmia function (antitachycardia pacing)

Meaning of selected individual letters

• V = ventricle

• A = atrium

• D = double (with mode of response this means atrial triggered and ventricular inhibited)

• 0 = none

• T = triggered

• I = inhibited

• R = reverse

• P = programmable (rate and/or output)

• M = multi-programmable

The more common models include fixed rate (asynchronous — VOO, AOO, DOO), ventricular demand (VVI, VVT), atrial demand (AAI, AAT), atrial synchronized (VAT), and AV sequential (DVI). If the patient expects to do any activity, he needs a rate adjusting pacemaker. This type of pacemaker stores the previous R-R and generates the next beat based on it.

Problems with pacemakers include loss of capture (the electrical stimuli fails to produce a QRS complex, the pacing electrode is displaced, or the milliamperage is too low); non-sensing (the pacemaker fails to recognize spontaneous heartbeats — this results in competition), and loss of pacing artifact (the pacing stimulus does not produce a spike on the ECG — this is the result of battery failure, pulse generator breakage, or wire disconnections or breakage).

Pacemaker cells have been harvested and reproduced in test tubes and replaced, but there is no way to "reglue" them yet.

Pacemaker Terms

• Rate — the heart rate at which the pulse generator will pace.

• Standby rate — the lowest rate at which the pulse generator will pace.

• Electrical output or milliamperes (mA) — amount of current needed to trigger myocardial depolarization (0.1-20 mA).

• Sensitivity or millivolts (mV) — the voltage needed to respond to the heart's electrical activity (0.5-20 mV).

• Mode — represented by an abbreviation based on the pacemaker code; indicates the pacemaker's capabilities.
• **Atrioventricular (AV) interval** (also called the AV delay) in dual-chamber pacemakers — time between an atrial event (either sensed or paced) and a paced ventricular event. The pacemaker tries to sense an R wave during this interval. Expressed in milliseconds (msec). 250 msec is a commonly programmed AV interval. If the pacemaker senses an intrinsic R wave, ventricular pacing doesn't occur. If an R wave is not sensed, ventricular pacing occurs at the end of the AV interval. The pacemaker's AV delay may be longer or shorter than the patient's intrinsic PR interval.

• **Ventriculatrial (VA) interval** in dual-chamber pacemakers — time between a ventricular event (either sensed or paced) and a paced atrial event. The pacemaker tries to sense atrial activity during this time. Also measured in msec (e.g., 700 msec).

• **Pulse interval** in dual-chamber pacemakers — the total of the AV and VA intervals in msec.

Represents an entire cycle.

• **Programmed standby rate** — number of pulses per minute.

• **Hystereses** — a prolonged pulse interval intended to give the heart an opportunity to beat spontaneously. For example, if a pacemaker is set at 70 pulses/minute, the intrinsic rate will be allowed to fall to 60 pulses/minutes before the pacemaker delivers a stimulus. If hysteresis ends and natural depolarization doesn't occur, the pacemaker returns to its faster rate.

• **Special antitachycardia functions** — the response of the pacemaker to tachycardia such as by burst pacing.

**AUTOMATIC IMPLANTABLE CARDIOVERTER DEFIBRILLATORS**

A lead system and pulse generator are implanted. This system is capable of sensing either ventricular tachycardia or fibrillation and cardioverting or defibrillating the patient automatically. The AICD will not shock the patient unless the actual HR is greater than the set rate. The device can recharge in 10-30 sec (x 4).

The device helps treat ventricular tachycardia and ventricular fibrillation by monitoring heart activity and delivering countershocks when dysrhythmias arise. The AICD is approved for patients who have survived at least one episode of cardiac arrest not a/w an acute MI and patients with recurrent tachydysrhythmias who haven't had a cardiac arrest.

Within 10-35 seconds after the problem is sensed, the AICD typically delivers its first shock at 25 joules.

If the first shock fails to convert the rhythm, the AICD delivers a 2nd shock at 30 joules and, if necessary, a 3rd and 4th shock at 30 joules. After the 4th shock, a normal ECG must be sensed for at least 35 seconds before a new four-shock sequence can occur.

The AICD decreases mortality in patients with sudden cardiac death unassociated with MI from 40% to 2% within 2 years after the initial sudden death event.

Fourth generation AICD transvenous systems are now available. A pacing and defibrillating electrode are placed in the right ventricle. The device is programmed to deliver bradycardia, anti-tachycardia, low energy cardioversion, and high energy defibrillation. The newer devices ignore the gradually increasing HR in an exercising heart, but respond to sudden increases in HR by delivering a shock. These newer devices also monitor the response to the shock and step up the voltage if the heart does not respond.

**COUNTERSHOCK**

Countershock is the delivery of a high intensity charge to heart. This charge causes complete depolarization of the heart and interrupts the dysrhythmia, allowing the SA node to regain control. There are two types: synchronous cardioversion and defibrillation (asynchronous).

**Synchronous cardioversion** delivers a charge of 25-50 watt/sec (up to 400 if necessary) when the patient's QRS is sensed. The charge is delivered during the QRS to prevent the R on T phenomena. The charge is synchronized to fall someplace other than on a T wave. Synchronized cardioversion is used to manage fast dysrhythmias that have definite QRS complexes, for example, atrial fibr with fast ventricular response or V tach. The patient should be NPO 8 hours before if possible; digitalis should be withheld for 24 hours. The patient should be sedated. The procedure may create a paradoxical reaction; additionally, the patient may become irritable, confused, and hyperactive. Check the BP, rhythm, and respiration before, during, and after the procedure and observe for the s/o of emboli. This procedure should not be used in patients who have digitalis toxicity.

**Defibrillation or asynchronous cardioversion** uses a nonsynchronized mode to treat fast dysrhythmias without QRS complexes (V fib). This procedure is also useful for patients who have pulseless VT. Direct current is applied to the chest wall to interrupt or convert dysrhythmias to normal sinus rhythm. The charge should be delivered as soon as possible. The procedure is unsuccessful if the cardiac muscle is anoxic. The required energy levels vary with body size — less than 50 kg, use 3.5-6.0 watt sec-1 kg-1; greater than 50 kg, use full out. The average charge is 200-400 watt/sec. Saline pads with electrode paste prevent bridging. The paddles should be placed on the right upper sternum under the clavicle and left of the apex of the heart under the arm. After the shock there is always a period of electrical instability so if possible before shock, fix hypokalemia, hypoxia, digitalis toxicity, acid/base imbalance.

**Cough** — A cough increases arterial and cardiac pressure and can be as effective as CPR. It gains time to get the crash cart.
APPENDIX A: CONDUCTION SYSTEM

APPENDIX B: CORONARY CIRCULATION

Location of Arteries and Structures Nourished By Them

APPENDIX C: Comparison of Q-Wave and Non-Q-Wave Myocardial Infarctions

<table>
<thead>
<tr>
<th>Pathology</th>
<th>Q Wave</th>
<th>Non-QWave</th>
</tr>
</thead>
<tbody>
<tr>
<td>Larger area of infarction</td>
<td>Smaller area of infarction</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Enzyme elevation</th>
<th>Q Wave</th>
<th>Non-QWave</th>
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</thead>
<tbody>
<tr>
<td>CK</td>
<td>Greater elevation</td>
<td>Less elevation</td>
</tr>
<tr>
<td>SGOT</td>
<td>Greater elevation</td>
<td>Less elevation</td>
</tr>
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<table>
<thead>
<tr>
<th>ECG</th>
<th>Pathologic Q waves present</th>
<th>Absent pathologic Q waves</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST-segment elevation</td>
<td>ST-segment depression (in acute phase) (in acute phase)</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Imaging</th>
<th>Q Wave</th>
<th>Non-QWave</th>
</tr>
</thead>
<tbody>
<tr>
<td>Technetium-99M</td>
<td>Helpful in diagnosis</td>
<td>Not helpful in majority of cases pyrophosphate</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>Shows larger regional wall</td>
<td>Shows smaller area of wall motion abnormality</td>
</tr>
<tr>
<td>PET scan</td>
<td>Reveals homogenous defects</td>
<td>Nonhomogeneous defects</td>
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<table>
<thead>
<tr>
<th>Angiographic findings</th>
<th>Q Wave</th>
<th>Non-QWave</th>
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<tbody>
<tr>
<td>Total coronary occlusion commonly seen in infarct related artery soon after Q-Wave</td>
<td>Total occlusion is infrequently observed in early hrs in infarct-related artery; mod. increase next days</td>
<td></td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Reinfarction</th>
<th>Q Wave</th>
<th>Non-QWave</th>
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<tbody>
<tr>
<td>Can occur</td>
<td>Higher incidence</td>
<td></td>
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<table>
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<tr>
<th>Postinfarct angina</th>
<th>Q Wave</th>
<th>Non-QWave</th>
</tr>
</thead>
<tbody>
<tr>
<td>Can occur</td>
<td>Higher incidence</td>
<td></td>
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</tbody>
</table>

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<thead>
<tr>
<th>Calcium channel blockers</th>
<th>Q Wave</th>
<th>Non-QWave</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early and late beneficial effects not</td>
<td>Diltiazem specifically reduces incidence</td>
<td></td>
</tr>
<tr>
<td>equivocally proven</td>
<td>of early reinfarction and severe angina</td>
<td></td>
</tr>
</tbody>
</table>
Nifedipine reduces postinfarct angina and unstable angina

**Beta-adrenergic blockers** Improve long-term survival _________________ Not effective

**Mortality**

1. Early ___________ Higher in-hospital mortality rate _________________ Lower in-hospital mortality rate

2. Late ——— _________________ Similar or even higher than Q-wave infarction

**APPENDIX D: TESTS USED IN DIAGNOSIS OF ACUTE MI**

Myocardial cells that are irreversibly injured release a number of enzymes into the circulation where they can be measured.

Increased CK, LDH, AST (SGOT) with increased CK-MB and LDH-1 isoenzymes; temporal pattern of release is of diagnostic importance.

Isoenzymes may help rule out noncardiac causes that may elevate the routine enzymes.

**Creatine Phosphokinase (CK)**

- Onset of elevation starts 4-8 h after MI, peaks at 24 h, and returns to normal in 72-96 h except in the case of large infarctions when CK clearance is delayed. Coronary reperfusion causes an earlier peak (within 12 h).

- Sensitivity > 90%.

- Specificity — CK is elevated in other conditions.

Two- to threefold increase of total CK may follow IM injection.

Other causes of increased total CK — muscular dystrophy, myopathies, direct current (DC) cardioversion, cardiac catheterization, hypothermia, stroke, trauma, surgery, convulsions, prolonged immobilization, COPD associated with LV CHF, pulmonary embolism, shock, myxedema, extensive 3rd degree burns, strenuous exercise, diabetic ketoacidosis, acute alcohol intoxication (rhabdomyolysis), and small bowel infarction. Amount of CK increase may be correlated with severity of infarction.

**CK-MB Isoenzyme** — more specific for myocardial necrosis than CK alone. Not present in significant concentrations in extracardiac tissue; particularly useful when skeletal muscle and brain damage are suspected.

- Onset of elevation occurs by 4-6 h, peaks within 18-24 h, and returns to normal within 48-72 h.

- **Most specific and sensitive enzyme clinically available for diagnosis of MI.**

- CK-MB present in substantial amounts only in myocardium and rarely in skeletal muscle of patients with muscular dystrophy of Duchenne type. Its presence in normal skeletal muscle is debatable.

- Can also be elevated in myocardial contusion, myocarditis, cardiopulmonary resuscitation, hypothyroidism, after DC cardioversion, and cardiac surgery.

- Not elevated following IM injection, noncardiac surgery, exercise, uncomplicated cardiac catheterization, or pneumonia.

- Reperfusion (by angioplasty, thrombolysis, or spontaneously) of myocardium causes an early peaking of CK-MB (8-12 h).

**Aspartate Aminotransferase (AST; previously SGOT)**

- Activity increased in 8-12 h. Peaks at 18-36 h and returns to normal within 3-5 days.

- Very sensitive.

- Specificity is poor due to its elevation in other conditions — liver disorders, hepatic congestion, biliary tract diseases, shock, skeletal muscle trauma, oral contraceptives, pericarditis, pulmonary emboli, myocarditis, cardiac catheterization, cardiac surgery.
Lactic Dehydrogenase (LDH)

- Onset of elevation occurs in 24-48 h; peaks at 3-6 days, and remains elevated for as long as 7-14 days. Last of three standard enzymes to become elevated.
- Very sensitive.
- Specificity is poor due to elevation in hemolysis, leukemia, lymphoma, liver disorders, renal disorders, burns, heart failure and hepatic congestion, DC cardioversion, myxedema, skeletal muscle trauma, and pulmonary emboli.

**LDH Isoenzyme** — elevation may suggest cardiac disease or hemolysis.

- 5 LDH isoenzymes but LDH\(_1\) predomnates in heart.
- Rises before total LDH (within 8-24 h) and may rise when there is no rise in total LDH.
- Following acute MI, the ratio of LDH\(_3\) to LDH\(_2\) becomes > 1.
- Sensitivity > 95%; generally done only when the initial CK and CK-MB might have missed MI (after 48 h). LDH\(_1\) isoenzyme is of diagnostic value in patients with acute MI who present after significant delay after onset of symptomatology.
- Levels may be elevated in minimal hemolysis.

**Other Enzymes Used for Diagnosing MI**

- Myoglobin.
- New radioimmunoassay against troponin I may be a more specific marker for myocardial injury.
- CK isoenzyme-(3). CKMM subtype.
- Myosin light chains.
- Tropomyosin.

**Nonspecific Lab. Findings**

- Elevated WBC and neutrophil counts (appear in few hours and persist for 3-7 days).
- Elevated erythrocyte sedimentation rate.
- Alanine aminotransferase (ALT; previously SGPT) — time course of elevation is intermediate between CK and LDH; lacks tissue specificity.

**Chest X-Ray**

- May be normal in acute MI or cardiomegaly may be present.
- If pulmonary capillary wedge pressure is > 18-25 mm Hg, signs of CHF may occur — pulmonary vascular redistribution to the apices, interstitial pulmonary edema (blurring of pulmonary vasculature, perihilar haze, Kerley B lines, lattice pattern), and pleural effusion.

**Imaging Techniques: Infarct Avid Imaging** \(^{99m}\text{Tc Pyrophosphate}\)

- Usually positive between 1 day and 1 week of MI.
- Peak uptake occurs 2-3 days post-MI.
• Returns to normal between 1 and 2 weeks.

1. False positive
a. Previous MI, unstable angina, calcified aneurysm, pericarditis, myocardial contusion, calcified aortic or mitral valve, cardioversion, penetrating wounds, cardiomyopathy, malignancy, amyloidosis, calcified cartilage, rib fractures, breast tissue.

b. Sensitivity and specificity are high for transmural (Q-wave) infarcts (~ 90%), but are lower for non-Q-wave infarcts.

2. Uses
a. To diagnose MI when clinical history is not clear.

b. To diagnose MI when delay in hospitalization occurs and enzyme peaks are missed.

c. To detect MI in patients when their cardiac enzymes are altered after cardiac surgery.

d. To detect RV infarcts.

e. To diagnose MI when ECG is difficult to interpret (in presence of LBBB pacemaker rhythm).

f. To detect infarct extension.

g. To locate and size an MI.

h. Infarct avid imaging should be used in conjunction with clinical history, ECG, and tests for enzyme levels. Other promising new techniques include MRI and PET. Monoclonal antibodies may also prove useful in the future.

**Imaging Techniques to Assess LV Function and Complications in Acute MI**

1. Echocardiography and Doppler
a. Limited role in diagnosis of acute MI.

b. Useful in assessing regional wall motion abnormality and global LV dysfunction.

c. Useful for diagnosing complications of acute MI, e.g., mitral regurgitation due to ruptured papillary muscle or papillary muscle dysfunction, ventricular septal defect, infarct expansion, cardiac rupture, LV aneurysm, pseudoaneurysm, LV thrombus, pericardial effusion.

d. May play a useful role in prognostication of acute MI.

e. May have potential role in assessing reperfusion after thrombolytic therapy.

2. Radionuclide ventriculography: Uses.

a. To determine ejection fraction (EF).

b. To detect regional wall motion abnormality.

c. To help differentiate true LV aneurysm from false aneurysm.

d. Separation of RV and LV dysfunction.

e. Detection of pericardial effusion.

**APPENDIX E: ANTIARRHYTHMIC AGENTS**
ANTIARRHYMICS

Meds Quinidine (IA) Beta-blockers — Bretylium Nifedipine

Procainamide (IA) 'olol' drugs Amiodarone Verapamil

Lidocaine (IB) Propranolol Other calcium

Phenytoin (IB) Acebutolol channel

Mexiletine (IB) Esmolol blockers

Encainide (IC) Atenolol

Flecainide (IC)

Propafenone (IC)

Actions Blocks Na+, Block β₁ receptors, Slows repolarization, Blocks Ca²⁺ movement

Slows conduction, Blocks SNS effects, Blocks K⁺ channels

Marked inhibition through AV, and on heart at AV node

ECG Characteristics of Class IA Antiarrhythmics — Quinidine, procainamide, disopyramide, moricizine

• ? slight widening of QRS complex; increased widening of QRS = early sign of toxicity.

• Prolonged QT interval.

• Quinidine and procainamide may also produce U waves and flattened or inverted T waves.

ECG Characteristics of Class IB Antiarrhythmics — Lidocaine, mexiletine, tocainide

• ? shortened QT interval.

• Phenytoin may slightly shorten the PR interval.

• Mexiletine apparently doesn't change the ECG.

ECG Characteristics of Class IC Antiarrhythmics — Flecainide, encainide, propafenone, indecainide, lorcainide, aprindine

• ? lengthened PR interval.

• Widened QRS complex.

• Prolonged QT interval.

ECG Characteristics of Class II Antiarrhythmics — β-adrenergic blockers, propafenone
• slight lengthening PR interval.

• shorten QT interval slightly.

• Decrease heart rate.

**ECG Characteristics of Class III Antiarrhythmics** — Bretylium, amiodarone, N-acetylprocainamide (acecainide), sotalol

• ? widening of QRS complex.

• ? Prolongation of QT interval.

• Decrease heart rate.

**ECG Characteristics of Class IV Antiarrhythmics** — Ca**++-channel blockers

• ? lengthening of PR interval.

• Decrease heart rate.

**Beta Blockers Available In United States**

**Medication**

**Acebutolol** 3-4 400 mg/d

Sectral ®

**Atenolol** 6-9 50 mg/d

Tenormin ®

**Betaxolol** 14-22 10 mg/d

Kerlone ®

**Bisoprolol** 9-12 5 mg/d

Zebeta ®

**Carteolol** 5-6 2.5 mg/d

Cartrol ®

**Esmolol** 0.15 50 µg/kg/min

Brevibloc ®

**Labetalol** 6-8 200 mg bid

Normodyne ®, Trandate ®

**Metoprolol** 3-7 100 mg/d

Lopressor ®

**Nadolol** 20-24 40 mg/d
Beta-Adrenergic Blockers (class II drugs)

- Given to reduce the HR, contractility, and ventricular wall tension; decreases oxygen demand and prolongs diastolic filling, thus increasing coronary filling.

- Beta blockers are also antiarrhythmic.

- Contraindicated in CHF, pulmonary edema, asthma, COPD, heart block, and hypotension.

- Chronic routine use for 2 years decreases total mortality, sudden death, and reinfarction rate.

- IV metoprolol (Lopressor ®) 5 mg given q 5-10 min to 15 mg followed by 50-100 mg PO BID if no contraindications (hypersensitivity, overt CHF, 2° or 3° AV block, cardiogenic shock) or complications; later atenolol 50-100 mg daily.

Ca++ Antagonists

- Potential value as anti-injury agents in the setting of reperfusion, as antiplatelet agents, and as coronary vasodilators; may thus minimize platelet activation in the vicinity of lysed thrombi by decreasing shearing forces.

- Diltiazem, at least, apparently adversely affects prognosis in patients with CHF or impaired LV function.

Unique Adverse Reactions of Calcium Channel Blockers

Medication Initial Dosage

Diltiazem 30 mg QID

Sustained-release 60-120 mg BID

Nicardipine 20 mg TID

Nifedipine 10 mg TID

Sustained-release 30 mg QD
Verapamil 80 mg TID

Sustained-release 240 mg QD

Digitalis

- Increases automaticity in cardiac muscle cells, but decreases AV conduction by increasing parasympathetic tone.

Lidocaine (Class I)

- Suppresses reperfusion arrhythmias. Lidocaine is the agent of choice for ventricular tachycardia and ventricular fibrillation.
- Lidocaine decreases automaticity in the ventricles.
- Slows the impulse movement through the AV junction and His bundle by blocking Na⁺ movement (it decreases the rate of phase zero depolarization).
- Lidocaine has no anticholinergic effects.
- Lidocaine's production of a negative inotropic action, neurologic side effects, and an increased incidence of asystole preclude its prophylactic use.

Magnesium

- Appears to have favorable effects on cardiac arrhythmias, coronary blood flow, platelet aggregation, and myocardial metabolism.
- Administration of MgSO₄ is controversial (no change in mortality compared to placebos in 1995 trials).
- MgSO₄ IV — 2-6 gm IV over 15 min followed by infusion of 3-20 mg/min for 5-48 h.

Quinidine (Class I)

- Suppresses automaticity, especially in ectopic cells.
- Has an indirect atropine-like effect so it increases conduction.

APPENDIX F: THROMBOLYTICS

Reperfusion — PTCA or IV thrombolytic therapy with plasminogen activators.

Recent studies — 160-325 mg aspirin plus 5000 units of IV heparin should be given with institution of thrombolytic therapy; then 325 mg aspirin daily and a continuous infusion of heparin for 2-5 days.

Every minute counts — ideally within 1-3 h after onset of symptoms; beneficial if given within 6 h; some benefit appears possible up to 12 h.

Clinical factors that favor proceeding with thrombolytic therapy include anterior wall injury, hemodynamically complicated infarction, and widespread ECG evidence of myocardial jeopardy.

tPA (tissue plasminogen activator) — more effective at restoring flow than streptokinase or APSAC.

Recommended total dose = 100 mg; begin with 5-10 mg bolus followed by 60 mg IV over first hour and 20 mg each in 2nd and 3rd hours. New data suggests that it may be beneficial beyond 6 hours.

Streptokinase — 1.5 million units IV over 1 h.

APSAC (anisoylated plasminogen streptokinase activator complex) — single dose of 30 mg over 2-5 min.

Both streptokinase and APSAC are antigenic; once one has been used repeat administration with either agent should be avoided.
Allergic reactions to streptokinase and APSAC — approximately 2%.

**Contraindications for Thrombolytics** — history of CVA, trauma or invasive or surgical procedure within 2 weeks (or prolonged CPR), marked hypertension (systolic > 180 mm Hg and/or diastolic > 100 mm Hg), active peptic ulcer disease, bleeding diathesis, suspected aortic dissection, hemorrhagic retinopathy, intracranial neoplasm; hepatic failure = relative contraindication.

**Complications** — hemorrhagic stroke most serious and occurs in approximately 0.4% of cases; rate increases with advancing age; vascular access sites.

If thrombolytic agents are given in combination with aspirin, the post MI mortality decreases by 23-42% (with no increase in cerebral hemorrhage). Thrombolyis can paradoxically initiate platelet aggregation and activate the clotting cascade. There is a slight risk of stroke in patients who are over 75 y. o. tPA was given by paramedics in the Washington "MITI" study.

**Aspirin** inhibits platelet function and causes vasoconstriction. The current recommendation is that men over 50 with risk factors take 325 mg every other day (the study has not been completed for women or men over 50 who do not have risk factors).

**Heparin** is used to prevent reocclusion (the IV form of heparin is given with tPA). The desired aPTT with heparin therapy is usually 60-85 seconds. Adjust heparin to keep PTT at 1.5-2 times normal.

**Hirudin**, which is derived from leeches, is currently being used as a substitute for heparin in some studies; currently investigational; powerful antithrombin somewhat similar to heparin.

**PTCA** may be performed when thrombolytic agents cannot be used or if the patient develops cardiogenic shock. The latest research (JAMA, July 1996) recommends a CABG over PTCA as therapy. PTCA as primary intervention — generally reserved for those with contraindications to pharmacologic thrombolysis — advanced age (> 70 years) cardiogenic shock (systolic BP <100 mm Hg); infarction resulting from occlusion of previous CABG.

**APPENDIX G: MISCELLANEOUS DRUGS USED AFTER MYOCARDIAL INFARCTION**

**ACE Inhibitors:**

- Facilitate the formation of prostaglandin vasodilators, nitrous oxide, and free-radical scavengers.; also appear to have some antiarrhythmic properties.

- Patients with significant LV dysfunction have increased circulating natriuretic peptide; treatment with ACE inhibitors improves their survival.

- ACE inhibitors improve mortality and prevent CHF and recurrent MI in patients with ejection fraction of ___. 40%.

- These drugs appear to modulate heart remodeling post MI result in less hypertrophy; prognosis is worse with cardiac dilation, because dilation increases stress. LV end systolic volume is a good predictor of survival post MI.

- ACE is present in all vascular beds and many tissues (kidney, heart, brain, adrenal, testes).

Tissue angiotensin has a local effect on the function of the heart and vessels.

- Tissue specific ACE inhibitors have fewer side effects.

- Captopril is least tissue specific of the ACE inhibitors currently on the marker; captopril target dose 50 mg TID or pharmacologically equivalent doses of other ACE inhibitors.

- Many patients must stop taking the inhibitors because a major side effect is a chronic cough — the cough is due to bradycardic enhancement.

**Analgesics** — judicious use of narcotics; inhibits further release of catecholamines and subsequent predisposition to arrhythmias and increased myocardial oxygen consumption (MVO₂).

**Sublingual NTG** — up to three 0.4 mg doses at 5 min. intervals if hypotension does not occur; idiosyncratic reactions can cause marked hypotension and bradycardia.

**Morphine** — opiates depress sympathetic nervous system as well as central nervous system resulting in mild arteriolar dilation and venodilation and thus decreased afterload and decreased preload; 2-4 mg IV every 5 min with relative impunity until pain is relieved or toxicity is manifested by hypotension, vomiting, or respiratory depression. 2-3 mg/kg may be needed.
Has vagotonic effect which may cause bradycardia or advanced degrees of heart block/Morphine-induced hypotension (decreases sympathetically mediated arteriolar and venous constriction resulting in venous pooling a subsequent decrease in cardiac output and BP) — minimize with supine position, elevation of legs, IV fluids and atropine (if HR is not increased) and patient does not have overt or incipient pulmonary edema. Fairly rare although nausea and vomiting are common side effects.

**Demerol** — a vagolytic agent; therefore the drug of choice if the patient is hypotensive or bradycardic.

**Nitrous Oxide** — effective; also decreases afterload; generally well tolerated for as long as 24–48 h when used intermittently.

**Intravenous Beta-Blockers** — decrease ischemia by lowering MVO$_2$.

**NSAIDs and Glucocorticoids (except aspirin)** — AVOID; can impair healing, produce larger infarct scar, and increase risk of myocardial rupture.

*Research has shown (NEJM, October/1994) that the combination of thrombolytic agents, ASA, and beta-blockers improve survival. No improvements were seen with prophylactic use of lidocaine or calcium blockers. (Internists and family practitioners were less aware of these treatments than cardiologists.)*

Measures should also be taken to decrease O$_2$ demand:

The patient should be on bed rest followed by limited physical activity

Beta-adrenergic blockers to decrease heart rate

Arterial vasodilators such as nifedipine, nitrates (IV NTG) or angiotensin-converting enzyme (ACE) inhibitors to decrease afterload

Diuretics to decrease preload;

Oxygen to avoid hypoxemia.

**Gastrointestinal Agents** — Measures should also be undertaken to prevent nausea and vomiting and stool softeners are needed to decrease straining; straining can precipitate bradycardia and be followed by increased venous return to heart.

**Hypolipidemic Agents** are important for secondary prevention of death and morbidity post MI.

**Nitrates**

- May decrease excessive coronary reactivity and hence ischemia; used to manage acute ischemic syndromes.

- Some nitrates cause venous and arterial dilation, thus decreasing both preload and afterload.

- Alleviate coronary artery spasm and increase collateral flow.

- May attenuate platelet adhesion and aggregation, although this is not proven.

- Usually given by continuous IV for 24-36 h, then intermittently by an oral route (with a 10 h free period). It is important to avoid rebound tachycardia and hypotension when titrating IV nitrates.

**NITRATES**

**Antianginal Medication**

**Erythritol tetranitrate** Sublingual/ 5 mg

**Cardilate ®** Transmucosal 5 mg

Chewable 10 mg
APPENDIX H: ISCHEMIA/Infarction

**EVALUATION OF MI:** The diagnosis is based on ECG changes, enzymes, physical examination and possibly radio-nucleotide imaging.

**Four Hemodynamic Categories of MI**

- No pulmonary congestion or hypo-perfusion (low mortality, less than 5%).
- Pulmonary capillary wedge > 18 mm Hg and cardiac index > 2.2 (pulmonary congestion with normal cardiac output and mortality of less than 10%).
- Pulmonary capillary wedge < 18 mm Hg and CI < 2.2 (no pulmonary congestion but hypo-perfusion and mortality less than 25%).
- Pulmonary capillary wedge > 18 mm Hg and CI < 2.2 (pulmonary congestion with hypo-perfusion and mortality less than 50%).

**Hemodynamic Parameters of 4 Categories of MI**

- Normal CO and wedge • High to normal CO and wedge (need diuresis, inotropics, afterload reduction)
- Low CO and wedge (hypovolemic — need fluids) • Low CO and high wedge (need inotropics, afterload reduction, intra-aortic pump)

**MANAGEMENT POST MI:** The patient's rhythm and enzyme changes should be monitored. A change in compliance post-MI can produce acute pulmonary edema when you attempt to improve preload by volume loading, so the wedge pressure must be monitored when attempting a volume challenge.

**MYOCARDIAL INFARCTION COMPLICATIONS**

- **Aneurysm Formation**

Aneurysms form in 15% of MI patients. Can occur weeks to month after infarct.

May lead to CHF, systemic embolization of mural thrombi, and dysrhythmias.

May present as worsening dyspnea due to heart failure, symptoms of arrhythmias, or symptoms of arterial emboli, or may be asymptomatic.

Signs include abnormal precordial bulge, double apical impulse, dyskinetic impulse, S₃, S₄; signs of CHF may be associated with MR or VSD.

ECG — shows persistent ST elevation.
Chest x-ray — may be within normal limits or show cardiomegaly; abnormal bulge or angulation in area of aneurysm; left atrial enlargement; calcium in ventricular wall.

- **Arterial Embolism From Site of Infarct** (usually within first 3 weeks)

  May be directly related to both the site and size of the infarct.

  Can result in symptoms of stroke, or in renal or mesenteric infarction, or in cold, painful extremity.

- **Cardiac Tamponade**

  Heart is surrounded by fluid held in the pericardial sac resulting in constriction of the heart with a resulting decrease in cardiac filling and a decrease in heart sounds.

  Other signs and symptoms include a paradoxical pulse, decreased ECG voltage, neck vein distention, increased heart size, an enlarged liver and spleen, rales, wheezes, extreme anxiety, and decreased urinary output (the patient may go into shock).

  There is usually a sudden decrease in BP and cardiac output and an increase in CVP, PAP, and HR.

  Also seen with cardiac surgery, acute pericarditis, ventricle rupture, or aortic dissection.

  Evidence of tamponade may be seen on x-ray, scan, echo, or pericardiocentesis.

  Management of tamponade includes the administration of steroids and diuretics and a pericardiocentesis or pericardiectomy.

- **Cardiogenic Shock** — “power failure”.

  Cumulative loss of ~ 40% of LV muscle mass usually results in cardiogenic shock.

  BP inadequate to perfuse kidneys and other organs adequately.

  Symptoms — restlessness, decreased mentation, shortness of breath.

  Signs included obtundation, confusion; tachycardia; cool, moist skin;...

  Characterized by systolic BP of < 80-90 mm Hg, cardiac index < 1.8 L/min/m²; and PWP > 18 mm Hg;

  urinary output < 20-30 cc/h; associated signs of CHF; metabolic acidosis.

  *Diagnosis not based on hypotension alone.*

  Mortality directly related to decreased stroke volume; mortality rate > 70%.

  Risk factors include advanced age, decreased LV ejection fraction on admission, large infarct, history of diabetes mellitus, and previous MI.

  Relatively small infarct superimposed on a previously compromised heart may precipitate hemodynamic disaster.

  Treatment — attempt to maintain coronary perfusion pressure by increasing BP with vasopressors, intra-aortic balloon pump, and manipulation of blood volume to a level that ensures optimal LV filling pressure (PWP approximately 20 mm Hg)

  Cardiogenic shock is seen in 15% of MI patients — vicious cycle of progressively irreversible hemodynamic changes resulting in decreased peripheral and coronary perfusion (circulatory failure) and increased pulmonary congestion. Hypotension, acidosis, hypoxemia and myocardial depressant factor also depress myocardial function.

  Mortality = 80-90% mortality.

  *If the wedge is increased, the patient needs venous dilation; if it is decreased, the patient needs fluid.*
Both vasopressor and vasodilator therapy are used in shock.

Inotropic agents increase stroke volume and decrease heart rate, wedge pressure, and systemic vascular resistance. Therefore the BP should increase.

• **CVA Secondary to Thromboemboli**: Necrosis of the endothelium roughens the surface, predisposing to thrombus formation.

• **Congestive Heart Failure** — some degree of CHF in approximately 40-50% of patients with acute MI; acute LV failure signifies that > 25% of LV is dysfunctional or ischemic.

Clinical signs = rales, S₃ and S₄ gallops; CXR changes.

Characterized by increased PWP and PAP but these may result from decreased diastolic ventricular compliance (diastolic failure) or decreased stroke volume with secondary cardiac dilatation (systolic failure).

Anterior wall infarcts are associated with more severe CHF than are inferior infarcts.

"Stunned myocardium" — acute ischemic episode may cause myocardial injury that is reversible if the myocardium is reperfused; temporarily unable to contract.

Treatment — preload reduction (furosemide and NTG) to decrease pulmonary congestion and decrease MVO₂; digoxin is of little value.

Furosemide's initial effect is venodilation; this effect is achieved within 5 min of IV administration; avoid hypovolemia from excessive diuresis.

Pulmonary congestion, decreased contractility and abnormal wall motion often occur.

• **Dressler's Postinfarction Syndrome** — delayed form of acute pericarditis; can occur 1 week to several months after acute myocardial infarction.

Poorly understood; possibly antigen-antibody response (autoimmune disorder) to necrotic myocardium.

Pericardial pain, fever, friction rub, pleural effusion, pleuritis, tachycardia, and arthralgias may accompany this syndrome.

Treatment — aspirin; occasionally glucocorticoids if NSAIDs fail but steroids could alter healing.

• **Dysrhythmias** — most common complication; affect > 90% of acute MI patients; ischemic myocardium electrically unstable.

Both sinus tachycardia and atrial fibrillation are associated with increased mortality and are seen most often with anterior infarctions.

Causes — ischemia, hypoxemia, autonomic nervous system imbalances, lactic acidosis, electrolyte abnormalities, alteration of impulse conduction pathways or conduction defects, drug toxicity, or hemodynamic abnormalities.

Treatment — correct any electrolyte imbalances, provide adequate oxygenation, reduce sympathetic nervous system stimulation, and diminish MVO₂.

Hypokalemia = risk factor; maintain K⁺ at approximately 4.5 mmol/L.

**Ventricular Dysrhythmias**

Risk for ventricular fibrillation is highest during the first 4 hours after infarction.

β-blockers — effective in abolishing PVCs in MI patients and in preventing ventricular fibrillation.

Use routinely if no contraindications.

Treat (lidocaine, bretylium, amiodarone, electrocardioversion, defibrillation) sustained or symptomatic ventricular arrhythmias; carefully watch frequent PVCs (> 5/min), R-on-T, multifomed PVCs, ventricular bigeminy and PVCs occurring in couplets.

Prophylactic administration of lidocaine controversial; cardio depressant and proarrhythmic; may predispose pt. to bradycardia and asystole, CNS depression, and seizures.
Torsades de pointes — polymorphic ventricular tachycardia; change in amplitude and cycle length cause appearance of oscillation around baseline; associated with preceding QT prolongation (often > 0.60 sec).

Etiology includes hypoxemia, hypokalemia, hypomagnesemia, intracranial events such as SAH, 3rd AV block, toxicity due to quinidine, dig., phenothiazines, or tricyclics.

Treatment — overdrive pacing, magnesium sulfate.

Accelerated idioventricular rhythm (slow VT) — rate 60-100 bpm:

Common with inferoposterior infarct where it is usually associated with sinus bradycardia.

Often occurs transiently during thrombolytic therapy at time of reperfusion.

Usually benign and doesn’t precede classic ventricular tachycardia.

### Supraventricular Dysrhythmias

Bradycardia from parasympathetic nervous system overactivity may be treated with atropine or transvenous pacing if patient is symptomatic; elevating legs also helpful.

Especially common with inferior infarcts.

AV blocks and intraventricular conduction disturbances:

Mortality associated with AV block and anterior infarcts markedly greater than that associated with inferior infarcts.

Anterior infarcts — usually associated with trifascicular blocks of extensive necrosis.

Inferior infarcts — usually associated with AV nodal ischemia.

Sinus tachycardia — usually secondary to anxiety, pain, excessive sympathetic stimulation resulting in adrenal tone, pericardial inflammation, hypoxemia from pulmonary congestion, venous congestion, CHF or other remediable factors.

Atrial fibrillation/flutter — may be indicative of failure or atrial infarction.

• **Myocardial Infarct Expansion**

Disproportionate thinning and stretching of the infarct within first 5-10 days post-MI.

Associated with LV rupture, papillary rupture, VSD.

Symptoms of increased CHF and chest pain without ECG or enzyme evidence of further infarction.

• **Myocardial Infarct Extension**

Increased ischemic pain.

Reappearance of increased CK-MB 48 h after initial symptoms.

ECG evidence of further infarction.

• **Organic Brain Syndrome** may occur post MI, if blood flow to brain is decreased; decreased cerebral perfusion; thromboemboli may cause TIA or CVA.

• **Papillary Muscle (or Chordae Tendineae) Dysfunction or Rupture** (1st-2nd week)

Permits systolic regurgitant flow from LV to LA.
Contributing factors — thinning of wall, poor collateral flow, shearing effect of contraction against stiffened necrotic area, marked necrosis at terminal end of blood supply, and aging of myocardium with laceration of myocardial microstructure. 80% of time, it occurs in inferior or posterior MI.

Posterior papillary muscle more commonly affected as its only blood if from post. descending branch of RCA or dominant LCA.

Anterior papillary muscle normally has a dual blood supply from tributaries of diagonal branch of LAD and marginal branch of LCx.

Dysfunction may be transient; symptoms may be mild or catastrophic.

Symptoms — often sudden onset of _ SOB; symptoms of _ output ( _ mentation, lethargy).

Complete or partial rupture \( \not\) acute mitral insufficiency \( \not\) sudden _ LAP and _ PAP, _ CO, and acute pulmonary edema. Systolic apical murmur; usually occurs after \( S_1 \) and radiates to axilla.

\( S_1 \) and \( S_2 \) widely split.

May see large \( r \) waves in PWP tracing.

LA \( v \) wave amplitude may reach levels of 50-70 mm Hg.

Suspect when shock, acute pulmonary edema, apical thrill, and high-pitched holosystolic murmur at apex develop in AMI pt.

Differential diagnosis — septal rupture; use bedside Doppler.

Complete papillary muscle rupture = absolute surgical emergency; rapid dx. and surgical intervention is imperative.

Medical management of ruptured papillary muscle to support pt. preoperatively — decrease preload (results in improved alignment of papillary muscles and decreased size of regurgitant valve orifice) and _ afterload ( _ resistance _ backflow ) with IABP, dopamine, nitroprusside or NTG infusion; ? diuretics and/or digitalis.

MR may also be result of alteration in size of shape of ventricle due to impaired contractility or aneurysm formation

• **Pericarditis**

Sharp, positional pain; worse when recumbent; pain _ with inspiration; pain _ when pt. sits up and leans forward.

Pericardial friction rub 1-3 days after acute MI. Rub may be evanescent. Tachycardia.

Treatment — ASA. NSAIDs may delay infarct healing.

Pericarditis may be due to a transmural infarction that produces a rough epicardial layer and irritation of pericardial surface resulting in inflammation. It is usually seen 2-4 days post MI.

• **Postinfarction Angina** (within 1st 10 days)

Suggests viable myocardium still subject to ischemia.

Angina pectoris; \( S_4 \); hypertension; hypotension in severe cases; pale, clammy skin; ECG evidence of ischemia.

• **Pulmonary Embolus** (anytime)

Esp. common with CHF and prolonged bedrest.

Symptoms — dyspnea, pleuritic chest pain (with pulmonary infarction), diaphoresis, anxiety, hemoptysis; calf pain with thrombophlebitis of extremity.

Signs — tachypnea, tachycardia, fever, pleural rub (with pulmonary infarction), hypotension if embolus massive; loud \( P_2 \); ± distended neck veins; hypoxia.
**Rupture of Heart Structures:** The papillary muscles, chordae tendineae or ventricular wall may rupture and produce pulmonary edema and shock. These structures usually rupture after day 3.

Ventricular septal rupture is seen with anterior or inferior MIs and causes shunting of blood, decreased CO, increased right heart work, and pulmonary congestion. The septum usually ruptures 10-11 days after the MI. The symptoms include a palpable thrill, a loud systolic murmur, CHF, increased PAP, and decreased BP and cardiac output.

More common in women and previously hypertensive patients.

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**Myocardial Rupture** — ruptured LV causes massive hemorrhage into the pericardial cavity (hemopericardium).

Causes death by impairment of cardiac filling (cardiac tamponade).

Sudden disappearance of pulse, BP, and consciousness while ECG shows sinus rhythm.

Higher incidence with 1st infarct, hx. of hypertension, no hx. of angina, and relatively large Q-wave infarcts.

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**Rupture of the Ventricular Wall** is rare and when it does occur it causes tamponade and death within a few minutes. It is seen more in women than men, especially if they have HTN. It is most likely to occur 5-21 days after the MI.

**Septal Rupture** — rupture of a septal infarct; rarely; acquired VSD. (within 1st 2 weeks)

Present with severe CHF (dyspnea, mental arrest, lethargy) a/w sudden appearance of holosystolic murmur at apex or left mid- or lower sternal border; often acc. by a parasternal thrill. 

pulseless electrical activity.

Differential diagnosis — papillary muscle rupture; use bedside Doppler.

Preoperative treatment — — afterload (~ resistance → backflow ) with IABP, nitroprusside or NTG infusion.

**RV Infarction** — 1/3 of patients with inferoposterior infarcts demonstrate at least a minor degree of RV necrosis; some also have extensive RV infarct.

**Presentation** — JVD, Kussmaul’s sign (paradoxical pulse — BP — exceeds 10 mm Hg on inspiration while venous pressure remains steady or ), and hepatomegaly with or without ST elevation in right precordial leads, esp. V2,R

Volume expansion often improves CO and hypotension.

**Sudden Death** — risk factors = age > 65 years, previous angina pectoris, hypotension or cardiogenic shock, acute systolic hypertension at time of admission, DM, dysrhythmias or conduction defects, previous MI.

**Thromboembolism** — often clinically silent; found in 45% of patients at autopsy.

Thrombi may form on the endocardial surface of myocardial infarcts or ventricular aneurysms + DVT.

**Ventricular Aneurysm** — = dyskinesis; local expansile paradoxical wall motion during systole.

Infarct heals as thin fibrous scar which fails to move with rest of the ventricular myocardium.

Most common at the apex; occur months to years after AMI.

Don’t predispose to nor a/w cardiac rupture.

May be complicated by ejection fraction, SV, CHF, arterial embolism, and ventricular tachyarrhythmias.

Physical finding of greatest value = double, diffuse, or displaced apical impulse.

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**APPENDIX I: DIAGNOSTIC AND THERAPEUTIC PROCEDURES**
IN CORONARY ARTERY DISEASE

• Cardiac Catheterization:

Involves the insertion of a radiopaque catheter into an artery (left side) or vein (right).

Used with stress test/pacing to assess the functional impact of CAD.

Used to evaluate pressures, cardiac output, O₂ levels, coronary circulation, ventricular function, heart dz., and hemodynamic function.

Catheterization is also used to perform angiography. Dye is used to visualize ventricle motion and coronary blood flow.

Histamines and steroids are given prior to the procedure to the possibility of a reaction to the radiopaque iodine dye.

NTG and calcium blockers are used during the procedure to spasm.

Dye is an osmotic diuretic so the patient must be well hydrated.

Smokers have an risk of complications as nicotine produces aggravation of coronary spasm after vessel manipulation.

• Coronary Artery Bypass Graft (CABG):

Performed for revascularization.

Veins used for the procedure take on arterial characteristics (get thicker walls) within one month.

30% of the grafts thrombose in first year. Platelet derived growth (PDGF) factor has been implicated in the thrombosis process — clinical studies are currently being conducted with Ab to PDGF.

• Chest X-ray:

Used to assess heart size and position, chamber size, pulmonary blood flow, and line placement.

• Directional Atherectomy (DVI):

A rotating blade cuts away lesions, leaving scalloped edges and a vacuum sucks the lesion out after it is cut.

This tool actually creates a potent stimulus for hyperplasia and rescarring.

• ECG Stress Test:

ECG of a heart at rest can be normal even in the presence of CAD.

Stress test is used to document ischemia and necrosis.

Patient walks on a moving treadmill during the test. A continuous ECG is recorded during the procedure.

Speed and grade of the treadmill q 3 min. until the patient's HR reaches a target HR (220-age) x (0.85) or the patient fatigues — unless symptoms develop. (Patients taking β-blockers are unable to their HR.)

S & S that necessitate stopping the test include pallor, ataxia, hypotension, ST changes, tachycardia, block, bradycardia, and chest pain.

Patient is monitored for an additional 5-10 minutes after test.

False + exercise test may occur if any of the following are present: hyperventilation, abnormal electrolytes, vasomotor instability, and certain drugs — Valium; antihistamines; digitalis [As the ST]; and lithium [As ST, T, and HR].
Contraindications — severe LV dysfunction, an acute MI within 10 days, unstable angina, and uncompensated aortic stenosis or cardiomyopathy.

All post MI patients need a stress test or catheter test to uncover residual ischemia.

ST depression during the test (post MI) is predictive of mortality.

- **Echocardiography:**

  Non-invasive method of recording cardiac structures and motion using the echo (reflection) of high frequency ultrasound.

  Transesophageal echo recording is the invasive form of this test.

  Tells about the thickness of the septum and walls and about the diameter of the LV cavity, the motion of heart, the heart size and shape, and valve structure.

  Fibrotic, calcified, and scarred structures reflect more sound waves; therefore, appear more dense.

  Smokers are poor candidates for echo due to interference by carbon monoxide.

  Two modes of echocardiography — M and two dimensional.

  M mode uses a single ultrasound beam and information is displayed on a strip chart along with an ECG for reference.

  Two dimensional mode is more advanced and rotates throughout the chest wall; information is recorded on a printout and videotape.

  Used to dx mitral/aortic valve dz., pericardial effusion, LV size and function, tumors, idiopathic hypertrophic subaortic stenosis, shunts, and hypokinesis.

  Echo combined with dobutamine gives a better picture of the heart damage. (Drug is infused during the echo and wall motion is monitored.)

- **Electrophysiology:**

  Clinical indications include refractory or incapacitating supraventricular tachydysrhythmias, sustained ventricular tachycardia unrelated to an acute MI, sudden cardiac death unrelated to a myocardial infarction, unexplained syncope in patients with CV disease, recurrent wide QRS complex tachycardia, and nonsustained ventricular tachycardia in patients with cardiovascular dz.

  Procedure involves the percutaneous introduction of 1-6 catheters. Rapid pacing is then done to stress the heart and conduction system and study the origin of the dysrhythmia and the electrophysiological properties of accessory bypass tracts.

  Drug therapy may be used during the test to evaluate the effectiveness of various cardiac drugs on the dysrhythmia.

  Procedure takes 4-6 hours. During this time, a His-bundle electrogram is recorded to locate the site of conduction abnormalities.

  Recordings are made from the RA, the RV, and the His bundle.

  The electro- or histogram divides the PR interval into 3 components — the PA time (intra-atrial conduction, 37 msec), the A-H time (AV nodal conduction, 77 msec), and H-V time (conduction through the His-Purkinje system, 40 msec).

  PA and H-V times are mostly unaffected by the ANS.

  A-H time varies with vagal and sympathetic stimulation and with HR.

  Sinus node recovery time, refractory periods, and action potential duration can be evaluated with this technique.

  Techniques allow for the precise localization and catheter ablation of an accessory bypass tract. With cryoablation, the cells are frozen; with thermal ablation, the catheter tip heats the area and destroys the cells, with radiofrequency ablation, current is used to destroy the cells.

- **Heart Transplant:**
Indications for transplant include L. ventricular failure, congenital disorders, and cardiomyopathy. An immunosuppressive protocol (cyclosporine, steroids, azathioprine, OKT3) is used. Bacterial and viral infections are seen in over 60% of the patients. Cyclosporine is nephrotoxic so most patients need a kidney transplant after the heart transplant. CAD in the transplanted organ does not cause the usual symptoms.

<10% of these patients have chest pain and< 50% have ECG changes. The CAD is often misdiagnosed and the patient is treated for rejection instead. In 1989, the one yr. survival was 80% (5 yr., 60%). (Liver: 1/2 patients die on waiting list; kidney: one y. survival is 85-95%; 10 yr. is 75-85%).

• Holter Monitor:

A Holter monitor is a small portable monitor that is worn for 12-48 hours. One or two lead ECG recordings are usually obtained via 3-5 electrodes. Monitors are available now that can do 4 lead ECGs. The additional leads provide clearer information regarding atrial activity and screen out artifact abnormalities that appear only on one channel. The recordings can identify supraventricular dysrhythmias with aberrancy and document ST segment abnormalities. Usually modifications of leads V1 and V5 are used. The patient records activities and symptoms in a diary which are later correlated with the strip.

• Intervascular Stents:

Stents are coils that are left in vessels to support the wall and push back dissections. Complications included thrombosis and clotting, with restenosis in 41% of the cases, spasm, collapse, and dislodgement. They now use a balloon to open the stent even more. Gianturco-Roubin is one of the more common stents. Newer stents are biodegradable.

• Lasers:

There are several types of lasers available. Lasers are used in CV surgery to unplug vessels. The laser vaporizes plaque and produces a smoother wall than PCTA. Light is converted to thermal energy and protein is coagulated while water is vaporized. A relativity smooth wall results and PLTs do not adhere so there is a minimum inflammatory response. Lasers are also used to reseal tears due to angioplasty but may create tears themselves. Lasers produce either thermal or mechanical perforations in 20% of the CAD treatments. Mechanical perforations occur because the fiber optics are relatively inflexible. Laser therapy is useful for total occlusions that PCTA cannot fix or to enlarge a stenosis so that PCTA can be performed. Laser therapy is not an improvement over PCTA therapy and it is more time consuming. 45% of the vessels restenose in 6 months.

• Magnetic Resonance Imaging (MRI):

With MRI, the nuclei of certain isotopes (for example, hydrogen atoms in water and fat) are lined up in an external magnetic field. The nucleus of each atom behaves like a tiny bar magnet when patient is surrounded by a large magnet. A short radio frequency is used to apply a second external magnetic field that changes the orientation of the nuclear magnets; the nuclei then tilt. As they realign themselves they emit a radio frequency that has a spatial form and this energy is used to form the MRI image. Normally the nuclei of hydrogen atoms in the body are randomly oriented. By varying the timing, orientation and strength of the magnetic fields, it is possible to obtain a wide range of image contrasts. Other types of MRI are used to obtain images of such things as blood, capillary permeability, and turbulence. Fast MRI allows imaging of the heart in a fraction of a second. Echo-planar MRI acquires images in 30 ms, while turbo-FLASH obtains images in 300 ms — both techniques produce peripheral nerve stimulation as a side effects.; therefore, there may be a potential to trigger a dysrhythmia. Contraindications for MRI studies include pacemakers, ferromagnetic cerebral aneurysm clips, cochlear implants or metal fragments in the eye. Sternal wires, clips used in bypass surgery and prosthetic valves are now considered safe.

• Percutaneous Transluminal Coronary Angioplasty (PTCA):

Another variant of dye contrast cardiac catheterization and is an alternative to bypass surgery. PTCA improves coronary flow by enlarging the lumen of the artery. The lesion is compressed by a pressurized balloon. The stenosis produced by the balloon leads to an inflammatory/injury response with resulting fibrosis. Lesions in older patients may be calcified and non-compressible. Usually cardiac patients have thrombosis and stenosis, so they often need both PTCA and fibrinolytic drug therapy. The PTCA should not be done for at least 24 hours after administration of the drug. Post PTCA care involves monitoring the ECG and providing care similar to that following catheterization. The arterial sheaths are removed 3-5 hours after the procedure; they are not removed right away because of the danger of bleeding from the heparin given during the procedure. If heparin therapy is continued after the procedure, the sheaths are left in overnight. Patients must stay on their backs while the sheaths are in place and they should not lift their heads up off the pillow. They can bend the leg without the sheath to get relief from back strain. Pressure is applied on the groin site for 15-30 minutes after sheath removal. Pressure is applied over the area if the patient coughs or sneezes. Bleeding is a serious risk so we apply a pressure bandage and sandbag after sheath removal. The circulation should be checked (pulse, color, temperature, sensation) looking for emboli, thrombi s/s, especially the first 4 hours after the procedure. The patient is placed on bedrest for 6-8 hours after sheath removal. The patient is NPO the first hour, then given fluids 2nd hour; solids are reintroduced after 2 hours. Fluids are forced to help eliminate the dye. Coagulation, cardiac enzymes, and electrolytes are evaluated. Complications include angina due to occlusion of catheter, coronary artery dissection (intimal tear), spasm, MI, bradycardia, ventricular tachycardia and fibrillation, CVAs, emboli, infection, allergic reaction, systemic hypotension, bronchospasm, death, HA, hot flashes due to vasodilation, and N/V as side effects.; therefore, there is a potential to trigger a dysrhythmia. Contraindications for MRI studies include pacemakers, ferromagnetic cerebral aneurysm clips, cochlear implants or metal fragments in the eye. Sternal wires, clips used in bypass surgery and prosthetic valves are now considered safe.
infection in healthy individuals — it has been suggested that injury associated with PTCA activates latent HCMV. HCMV inactivates p53 in smooth muscle cells, predisposing them to increased growth.

• Pharmacologic Stress Test:

This method is used if the patient cannot exercise, has an aortic aneurysm or ventricular pacemaker, or is taking calcium or beta blockers and is contraindicated in COPD patients. In the past, we gave dipyridamole ($t_{1/2}$ is 2 minute), which inhibits adenosine uptake by the RBCs and thus produces coronary vasodilation. We are using adenosine ($t_{1/2}$ is 2 sec) in some hospitals now. Patients are more likely to have chest pain, SOB, flushing, and AV block with adenosine, but the side effects are short lived. The side effects with dipyridamole last longer than adenosine, but require no therapy. The 201tl is injected after the dipyridamide or adenosine.

• Phonocardiography:

Phonocardiography uses sound recording to evaluate the timing of the cardiac cycle and the characteristics of murmurs. Audible vibrations produced by the heart, great vessels and valves are recorded by 3 microphones and converted to electrical signals then recorded on paper. The microphones are placed over the base and apex of the heart. There are no risks to this procedure. An ECG and the carotid pulse are recorded also for reference points.

• Pull-Back Atherectomy:

This tool leaves a round lumen and is used to cut calcified lesions.

• Radioisotopes:

Low dose radiation is used to measure ventricle function or myocardial perfusion, looking at living versus dead cells. This tool tells about ischemia versus death.

• Roto-Borer:

The roto-borer is a high speed rotational coronary angioplasty that is used to grind plaque on calcified lesions. Scar tissue recloses the hole made by the tool.

• Technetium Pyrophosphate (Teboroxime) (99Tc):

A technetium scan is used to evaluate gated cardiac function. The ECG provides a "gate" (physiological marker) for end-diastole and end-systole. The R-R interval is divided into segments by the computer and a gamma counter records radioactivity from each segment. Technetium is used to identify a MI that is 12 hours to 6 days old. Technetium employs hot spot imaging — the damaged cells pick up the isotope because of their high calcium concentration and the infarct becomes radioactive. Bone also picks up the isotope. False positive readings occur with breast tumors, old age, aneurysms, and cardiomyopathies. The scan is performed 2-3 hours after injection of the isotope. The operator takes several 5 minute scans from different angles. The patient must lie quiet. This test can be used to tag RBCs; to evaluate wall motion, ventricle performance, and ejection fraction; and to see pre- and post-fibrolytic agent reperfusion changes.

• Thallium 201 (201tl) Scan

A thallium scan is a cold spot scan. The isotope is picked up by living heart cells. The infarct on the scintigram is a dark spot and is called cold spot. The patient must be NPO 2-4 hours before the test so digestion does not divert the radioactivity. This scan is used to assess perfusion after exercise. Areas with decreased perfusion are not able to take up isotope during exercise, but will 4 hours later so we do 2 scans. The isotope is injected via a heplock 1 min. before the patient stops exercising and a scan is performed immediately after the exercise and again 4 hours later. Areas that reperfuse in 4 hours suggest reversible ischemia (CAD). This tool will show whether the heart can increase its ejection fraction with exercise.

APPENDIX J: DEFINITIONS

Block — an abnormal delay or failure of conduction; must be differentiated from normal physiologic delays (e.g., atrial flutter with 2:1 conduction or sinus node suppression following atrial ectopy.)

Cycle — one complete systole and diastole sequence; may be measured from P-P or R-R or T-T intervals.

Ectopic — arises from outside the normal pacemaker of the heart; can arise from the atria, AV junction, or ventricles; can be premature beats, escape beats, or a continuous rhythm.

Escape — occurs late or ends a cycle longer than the dominant cycle (passive).
Paradoxical Pulse: a diagnostic sign. There is an abnormal drop in systolic BP when the patient inspires. With inspiration the vessels of the lung increase in size secondary to the increased negative pressure and the blood pools causing a decrease in SV and pulse strength. This sign is seen with positive pressure ventilation, right ventricular infarct, hypovolemia, and in 1/3 of tamponade patients.

Premature — occurs early or ends a cycle shorter than the dominant cycle (active).

Pulse Pressure: affected by stroke volume and compliance. The greater the CO, the greater the pressure increase during systole and the fall during diastole; therefore, the greater the pulse pressure. The greater the compliance, the smaller the pressure rise (arteriosclerosis decreases compliance, as does some HTN.)

Pulsus Alternans: a diagnostic sign. There are alternating strong and weak beats due to a change in L ventricular contractile force. This sign is seen on the ECG as alternating tall and short QRSs. Pulsus alternans are seen with tamponade, L ventricular failure, digitalis toxicity, paroxysmal SVT, AV block, falling BP, and aortic insufficiency. It is common among patients at increased risk for ventricular dysrhythmias.

Ventricular Contractile Asynergy:

• Hypokinesis: generalized reduction in myocardial contraction

• Asyneresis: localized or discrete area of reduced wall motion

• Akinesis: total absence of wall motion in a discrete area

• Dyskinesis: paradoxical systolic expansion of a portion of the L ventricular wall

• Asynchrony: a disturbance in the temporal sequence of L ventricular wall contraction

APPENDIX K: MISCELLANEOUS

Sex after a MI

The patient's sexual habits prior to the cardiac event should be assessed and used as a guide for teaching. Eighty percent of the post MI patients return to daily activities including sex in 4-6 weeks. Sexual activity can be resumed when the patient can climb two flights of stairs without chest pain, SOB, or extreme fatigue. The heart is usually stressed only 4-6 min. of a 10-16 min. sexual experience. An orgasm will generally increase the HR above 150 bpm and the BP to 160/90 mm Hg for 15-20 seconds similar to climbing two flights of stairs. Angina and palpitations may occur post-orgasm. The patient should be instructed to call the MD if the s/s last longer than 15 min. Masturbation, manual and oral stimulation are OK; anal intercourse may produce dysrhythmias. Patients should avoid sex if they are cold or too hot, immediately after a shower, or after a heavy meal or moderate or heavy consumption of alcohol. They should wait 2 hours after food/drink.

THE AGING CV SYSTEM

The peak maturity of all systems is at age 19-23. We do not notice our losses, because we have massive reserves (there is a net loss of cells but we have 5x the number that we need). Humans notice a loss of immediate recall in the mid to late 40s.

Exercise in a 20 y. o. produces a 20 fold increase in cardiac output, but it only produces a 3-4 fold increase in a 70 y. o. The maximal HR at age 65 → 40% from age 40 due to parasympathetic dominance; the SV reaches its limits at time of peak HR. Oxygen demand and ineffective metabolism prevent additional HR beyond that level.

Orthostatic hypotension may develop because the baroreceptors are not as effective and elderly patients may faint with severe stress. The blood vessels lose their elasticity with age and there is expansion of the vessels when cardiac output increases. The vessel Δs also $\not\in$ in auto-regulatory control of flow to the brain, coronaries, and kidney.

With aging there is a decrease in the ability to regulate temperature and in the recognition of referred pain. The decrease in pain recognition may lead to silent MIs, blunting angina, and perception of L arm pain.

The elderly experience decreased digitalis tolerance and decreased tolerance to other drug, i.e. nitrates.

INFECTIVE ENDOCARDITIS

The endocardium lines the heart chambers, covers the valves, and is continuous with the endothelial lining of the blood vessels adjacent to heart. It provides a smooth surface and prevents friction between the blood and the heart.
Endocarditis is an inflammation of the endocardium, especially the valves. **Non-bacterial endocarditis** is associated with blood stasis, MI, trauma, aging, collagen diseases, and artificial valves. **Bacterial endocarditis** is associated with bacteria, viruses, fungus, rickettsiae, and parasites — strep. and staph, are the most common organisms. The organisms enter the blood stream via dental cleaning, bladder catheterization, URI, skin infections, etc.

**Risk factors** include mitral valve prolapse, prosthetic valves, ventricular septal defect, previous attack of bacterial endocarditis, male gender, IV drug abuse, long term central vessel catheterization, and recent cardiac surgery.

**Pathophysiology:** A valve is usually damaged, leading to an inflammatory reaction. The damage exposes the basement membrane allowing the release of chemicals that are chemotaxic for PLTs. PLT activation and thrombus formation then create nonbacterial thrombotic endocarditis or infective carditis if micro-organisms are present in the blood.

Not all micro-organisms are capable of colonizing — the organisms must be able to survive interactions with complement, Ab, PLTs, etc. and they must also adhere to the damaged surface where they then proliferate and propagate the vegetation. Within 3-6 hours after the initiation of the infection, bacterial colonies form within aggregates of fibrin and PLTs and increase in size by 24 hours. The bacteria may activate the clotting cascade and promote the production of fibrin. As they multiply deep in the fibrin network, the bacteria become less susceptible to body defense mechanisms.

**Clinical manifestations:** acute, subacute, and chronic. The patient may have murmurs (75%), fever, petechiae on knees, elbows, ankles, eyes, and lips (25-50%), splenomegaly (50%), CHF (50-80%), immune complex deposition in various organs, anorexia, weight loss, night sweats, positive serum C&S, and back pain (25%). Osler nodes are pea sized tender reddish lesions on the fingers and toes. Right sided lesions are seen in drug addicts and in patients with congenital defects. ECGs, C&S, and scans of organs that emboli may have lodged in are used for evaluation. Management includes antimicrobial therapy for 4-6 weeks (IV then PO), prophylactic antibiotics, and rest.

**REFERENCES**
