Basics of EKG Interpretation: A Programmed Study

Acknowledgement is given to Leslie K. Muma, MS, RN, NP for assistance in preparation of this learning module.

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://www.usfca.edu/fac-staff/ritter/ekg.htm

Recommended Texts:


Recommended Web Site:

- http://www.ce5.com/ekg.htm
**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, II, and aV_{F}.* The six limb leads are used to determine the **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.

**PRECORDIAL PLACEMENT OF UNIPOLAR ELECTRODES FOR 12 LEAD ECGs**

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.

V₉ = same horizontal plane as V₄-V₆; over spine.

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:
V₃R = halfway between V₁ and V₄R.

V₄R = 5 RICS at MCL.

V₅R = same horizontal plane as V₄R at AAL (anterior axillary line).

V₆R = same horizontal plane as V₄R at MAL (mid-axillary line).

**PLACEMENT OF BIPOLAR LEADS (I, II, III, MCL₁, MCL₆) 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:* LL electrode should always be placed below the umbilicus in order to avoid problems in patients with ventricular hypertrophy.

*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.

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 V6 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.

Full Amplitude Half Amplitude Double Amplitude

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 V4-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 V1-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.

**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 V3 is abnormal unless there is LVH. *LVH causes loss of R height from V1-V3 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, aV₁, V₅, and V₆ (left lateral leads).

Small in aV₁ and V₅ — 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, aV₁, 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-TU 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 QT table is not available, the Q-T interval can be corrected for heart rate using Basset's formula:

\[
QTc = \frac{QT \text{ interval (secs)}}{\sqrt{RR \text{ interval (secs)}}}
\]

- 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 proarhythmia (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, aV₁, or aV₅.
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.
$V_1$ 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 $V_3 =$ 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](image)

Leads With Leads With ?

ST Segment Reciprocal

Category Elevation Changes Coronary Artery

Inferior II, III, aVF I, aV_L RCA or LAD of LCA

Anterior $V_1, V_2, V_3, V_4$ II, III, aV_F LAD

Extensive I, aV_L, V_1, V_6 II, III, aV_F L main or LAD + Cx

Anterior

Apical $V_5, V_6$ (? $V_3, V_4$) None Terminal LAD or RCA

Anterolateral I, aV_L, V_4, V_5, V_6 II, III, aV_F Cx

Anteroseptal $V_2$ and $V_3$ or in $V_1$ and $V_4$ None 1st division of LAD only

Anterobasal I, V_6 Cx

Lateral I, aV_L, V_5, V_6 V_1, V_2 OM or Cx of LCA

Posterolateral $V_4, V_5, V_6; aVF$ Cx of LCA (Apical)

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

http://www.usfca.edu/fac-staff/ritter/ekg.htm
Posterior None V₁, V₂ Cx; ? RCA; variable

True Posterior V₇, V₈, V₉ V₁, V₂ Variable — LCx or RPL (posteroseptal)

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

Posterobasal V₆; aVF Cx

Right Ventricular V₄ 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

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<th>Specificity</th>
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<td>1 mm ST elevation Proximal RCA</td>
<td>93%</td>
<td>88%</td>
</tr>
<tr>
<td>No ST elevation with Circumflex</td>
<td>85%</td>
<td>97%</td>
</tr>
<tr>
<td>Upsloping ST segment Distal RCA</td>
<td>74%</td>
<td>92%</td>
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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 V₃. 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, aV₅, 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 aVF.

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 V₁-2 or V₂-3 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 aVL.

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. Antero-septal infarcts predispose the patient to tachy- and brady- dysrhythmias and septal rupture.

Anterior (antero-septal) 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

Limb leads normal

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

**ST elevation in aVL** — 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 (V3-V5 or V1-V6) overlying infarct.

Loss of R waves plus deep Q waves in V3-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 LCxA.

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

ST and T waves initially may be elevated in V6 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 V₄R** — right ventricle is involved.

ST segment elevation in V₁ 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 S₃ 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, V₅-V₆** — apex is involved.

**ST depression in V₁-V₃ (esp. V₂-V₃) or ST elevation in V₇-V₉** — 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 bradydysrhythmias; 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 V₁-V₃**

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 (V₁-2). Then hold a mirror over the ECG and look for Q waves and an elevated ST.

- See only reciprocal changes in V₁-V₂ (? V₃) unless posterior leads (V₇-V₉) are obtained.

With a posterior MI, there are large R waves and ST depressions in leads V₁-2.

Reciprocal changes may also be seen in leads I and aV₅L.

- 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 (posteroseptal) 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 V₅ and V₆.

ST depression in V₃ and V₄; persist for at least a few days.

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

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, V₃, and V₄; 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 V₄₋₆ 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.

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**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 V2-4. Deep S wave in lead I and V6. 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 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.

Hypomagnesemia and Hypermagnesemia

- Marked decreased Mg++ 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 (rSR').

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 aVR 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/or 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, aVF) — 10 points

Anterior Q Waves (leads V2-V4) — 30 points

Lateral Q Waves (leads I, aVL, V5) — 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

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http://www.usfca.edu/fac-staff/ritter/ekg.htm 9/27/04
• 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, aVF)

• QRS duration < 120 msec

Wolff-Parkinson-White • Short PR with delta wave

• QRS duration > 120 msec

Normal variant

• Normal ECG