Step-by-Step Method for Accurate Electrocardiogram Interpretation

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Electrocardiogram Technique

INTRODUCTION
Conventional Sequence Regarding Interpretation
The time-honored advice to students and staff is as follows: In every ECG, the following features should be examined systematically:

From: Contemporary Cardiology: Rapid ECG Interpretation, 3e by: M. Gabriel Khan © Humana Press Inc., Totowa, NJ
- Rate
- Rhythm
- P wave morphology
- PR interval
- QRS interval, QRS complex morphology
- ST segment
- T wave
- Electrical axis
- U wave, and QT duration

Some authors, advise the following sequence:
Assess: rate, rhythm, axis, hypertrophy, infarction

but this is not the conventional teaching of cardiology tutors.

New Sequence for Interpretation

This text departs somewhat from the conventional sequence and gives a new approach consistent with the changes in cardiology practice that have evolved over the past decade. The early diagnosis of acute MI depends on astute observation for abnormal changes in the ST segment. Determination of creatinine kinase MB (CK-MB) and troponins is not relevant in the early phase of acute MI, because these cardiac enzymes are not elevated and are nondiagnostic within the crucial first hour of onset of MI. The door-to-needle or balloon time must be minimized if maximal life-saving is to be achieved. Diagnosis depends on symptoms and ST segment changes. Thus, this text rushes the interpreter to the assessment of ST segment morphology and suggests an 11-step method or sequence for the rapid yet accurate interpretation of ECGs.

BRIEF HIGHLIGHTS OF AN 11-STEP METHOD

Figure 2-1 defines the ECG waveform; Fig. 2-2A–F shows features of the normal ECG; and Table 2-1 gives normal ECG intervals and parameters.

An 11-step method is advised to ensure accurate, yet rapid, interpretation of the ECG. Algorithms, illustrations, and many sample ECGs make the 11 steps easy to understand and apply. The 11 steps are briefly outlined in this chapter, and each step receives in-depth coverage in later chapters, which also give advanced diagnostic features for postgraduates.
Chapter 2 / Step-by-Step Method for Accurate ECG Interpretation

Fig. 2-1. Sodium influx, potassium efflux, the action potential, and the ECG. (From Khan, M. Gabriel: *On Call Cardiology*, 3rd ed., Philadelphia, 2006, WB Saunders, Elsevier Science.)

Fig. 2-2. A, Chest leads V₁ through V₆.

(continued)
Fig. 2-2.  Continued  

**B.** Limb leads I through aVF. Sinus rhythm, rate 65 beats/min; PR interval, 0.14 second; QRS duration, 0.08 second; QT interval, 0.36 second; axis, +30 degrees.  

**C.** Chest leads of a normal ECG with a QRS complex in V2 that is positive, indicating early transition. Compare with (A), in which transition is normal, occurring in lead V5; tall R waves in V1 and V2 are not caused by posterior infarction (see Table 2-3). Heart rate, 75 beats/min (see Table 2-2). Note normal small Q wave in V4 through V6.
Fig. 2-2. D, Limb leads of a normal ECG showing a deep but normal Q wave in lead III (see Table 2-1 for normal parameters). E, Leads V₄ through V₆ show small, normal Q waves less than 4 mm deep; leads V₁ through V₃ show normal R wave progression.

(continued)
**Fig. 2-2. Continued**  F, Normal ECG, sinus rhythm 75 beats/min; PR interval, 0.16; QRS duration, 0.08; normal QRS axis +60 degrees; QT interval, 0.35. The small notch on the R wave of leads II, III, and aVF is a normal finding in some individuals and does not indicate intraventricular conduction delay (*see* Chapter 4).
## Table 2-1

### Important Normal ECG Intervals and Parameters*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>PR interval</td>
<td>0.12 to 0.2 second (up to 0.22 second in adults).</td>
</tr>
<tr>
<td>P waves</td>
<td>&lt;3 small squares (0.12 second) in duration, and amplitude &lt;3 mm. Upright in lead I, inverted in aVR (if opposite, suspect reversed arm leads(^1) or dextrocardia) (see Step 6, Figs. 2-21 and 2-36).</td>
</tr>
<tr>
<td>QRS duration</td>
<td>0.05 to 0.1 second; ≥0.1 second, consider incomplete LBBB, incomplete RBBB, or WPW syndrome (see Steps 2 and 3, Figs. 2-4, 2-9, and 2-10).</td>
</tr>
<tr>
<td>Q waves</td>
<td>Normally present in aVR; occasionally in V(_1) or in aVL (vertical heart) (see Chapter 6). Often present in lead III: should be ≤0.04 second duration. Other leads except lead I: &lt;0.04 second duration and ≤3 mm deep; lead I ≤1.5 mm in patients older than age 30. Q waves may be up to 5 mm deep in several leads in individuals age &lt;30.</td>
</tr>
<tr>
<td>R waves</td>
<td>V(_1): 0 to 15 mm, age 12 to 20 (see Table 2-3). 0 to 8 mm, age 20 to 30. 0 to 6 mm, age &gt;30.(^3) V(_2): 0.2 to 12 mm, age &lt;30(^5) (see Step 5, Fig. 2-16). V(_3): 1 to 20 mm, age &gt;30.(^5)</td>
</tr>
<tr>
<td>ST segment</td>
<td>Isoelectric or &lt;1 mm elevation in limb leads and &lt;1 mm in precordial leads except for normal variant (see Step 4, Fig. 2-12).</td>
</tr>
<tr>
<td>T wave</td>
<td>Inverted in aVR; upright in I, II, and V(_3) through V(_6). Variable in III, aVF, aVL, V(_1), and V(_2) (see Step 8, Fig. 2-27).</td>
</tr>
<tr>
<td>Axis</td>
<td>O degrees to +110 degrees age &lt;40. –30 degrees to +90 degrees age &gt;40 (see Step 9, Fig. 2-30).</td>
</tr>
<tr>
<td>QT interval</td>
<td>See Table 2-5.</td>
</tr>
</tbody>
</table>

*ECG paper speed 25 mm/s.

\(^1\)Precordial leads remain normal.

\(^3\)Age >30 is relevant to the diagnosis of myocardial infarction (see Fig. 2-20 and compare with Fig. 2-18, poor R wave progression).
Step 1: see Fig. 2-3.

Assess:

- Rhythm, then the rate.
- Note that rhythm is assessed before rate, because it is clinically more important, and a normal rate of 60 to 100 beats/min is easily spotted.

Step 2: see Fig. 2-4.

Assess:

- PR and QRS intervals for blocks.
- Widening of the QRS duration suggests right bundle branch block (RBBB) or left bundle branch block (LBBB) (see Table 2-1 and Fig. 2-4).

Step 3: see Fig. 2-9.

If the QRS duration is increased in the absence of LBBB or RBBB, assess:

- For nonspecific intraventricular conduction delay (IVCD), a cause of which is Wolff-Parkinson-White (WPW) syndrome (see Fig. 2-9).
- Although WPW syndrome is uncommon, it is an important diagnosis that may be missed by computer analysis and by physicians. Because WPW syndrome is a cause of widening of the QRS complex, it is logical to consider this diagnosis in the same frame as bundle branch blocks; this approach avoids the embarrassment of missing the diagnosis. No other text considers WPW syndrome in the assessment of the 10 essential ECG features, and conventional teaching does not give the approach outlined in Step 3.
- Most importantly, it is imperative to exclude mimics of MI early in the assessment sequence. WPW syndrome may mimic MI. RBBB may reveal Q waves in leads III and aVF that may be erroneously interpreted as MI. The diagnosis of LBBB must be documented quickly, because the presence of LBBB obviates many diagnoses, particularly ischemia and hypertrophy, and the diagnosis of MI is difficult.
- Because ECG changes of bundle branch block may be observed in V1 and V2, the reader is requested to first focus on V1 and V2. Importantly, V1 usually reveals the morphology of P waves and is an excellent lead for the assessment of sinus rhythm and arrhythmia. Thus, sinus rhythm or rhythm disturbances can be rapidly documented; in addition, the PR interval can be assessed, and left atrial enlargement may be revealed.
• The thorough assessment of V₁ and V₂ provides considerable information.

• In addition, the assessment of V₁, V₂/V₃ may assist with the diagnosis of Brugada syndrome and right ventricular dysplasia, which may display particular forms of RBBB and have been shown to be causes of sudden death in young adults. We should not fear to divulge rare syndromes at an early stage to students, because these topics may serve to motivate them to higher levels of excellence. The steps that discuss these rare but important topics are directed to senior trainees and internists. It is logical to discuss basics mixed with advanced material because this may appeal to students and medical residents.

**Step 4: see Fig. 2-12.**

Assess:

• The all-important ST segment.

• The early diagnosis of acute MI depends on observation for ST segment changes. New terms have emerged: ST elevation MI (STEMI) and non–ST elevation MI (previously termed non–Q wave MI). The ST segment holds the key to the diagnosis. This text describes ST segment abnormalities in detail in this chapter and provides further discussion in Chapter 5.

**Step 5: see Fig. 2-16.**

Assess:

• For pathologic Q waves, which, with the prior assessment of the ST segment should determine the presence or absence of new or old MI.

• Search the V leads for the loss of R waves or poor R wave progression, which may indicate MI, lead placement errors, or other cause (*see* later discussion, figures in this chapter, and Chapter 6).

**Step 6: see Figs. 2-21 and 2-22.**

Assess:

• P waves for atrial hypertrophy.

**Step 7: see Fig. 2-24.**

Assess:

• For left ventricular hypertrophy (LVH) and right ventricular hypertrophy (RVH).
**Step 8**: see Fig. 2-27.
Assess:
- T waves for inversion, which can have many causes (see later discussion in this chapter and Chapter 8).

**Step 9**: see Fig. 2-30.
Assess:
- The axis and for fascicular blocks.
- The axis provides no specific diagnosis and is of ancillary assistance only. In the 21st century, I believe conventional teaching should change a little. We should not lose sight of the fact that medical students and interns are bright individuals who desire to move quickly to clinical problem solving. Thus, boring topics, particularly difficult ones to grasp such as axis determination, which provides little diagnostic yield, should be assessed after most others. Thus, determination of the axis is relegated to Step 9.

**Step 10**: see Fig. 2-32.
Assess:
- Miscellaneous conditions, such as long QT, pericarditis, pacing, and pulmonary embolism (see later discussion and Chapter 10).

**Step 11**: see Fig. 2-37.
Assess:
- For arrhythmia.
- Step 11 is indeed Step 1 if an abnormal rhythm is revealed in Step 1: assessment of rhythm (see later discussion in this chapter and detailed coverage in Chapter 11).

**Switching the Sequence**

Most importantly, these steps can be switched. After the assessment of the important ST segment in Step 4 and for Q waves indicative of acute or old MI in Step 5, Step 7 can switch with Step 9. Thus, the conventional approach is restored, with assessment of the P wave followed by that of the T wave, axis, hypertrophy, and miscellaneous conditions. Therefore, in essence, this text covers the 11 ECG features systematically with minor changes to the conventional approach and
offers relevant and important diagnoses during the sequence, which allows the reader to interpret ECGs with greater accuracy.

Close attention to the 11 steps for ECG interpretation outlined in this chapter and reference to detailed explanations given in subsequent chapters should allow students, staff, and practicing clinicians to be competent interpreters of most ECGs. Accurate, yet rapid, interpretation of the ECG requires a methodic approach.

**THE NORMAL ELECTROCARDIOGRAM**

Figure 2-2A–F shows normal ECG tracings. Figure 2-1 and Table 2-1 list important ECG intervals and parameters. The ECG interpretation should end with one of the following statements:

- Normal ECG
- ECG within normal limits
- Borderline ECG
- Abnormal ECG

**STEP 1: ASSESS RHYTHM AND RATE (FIG. 2-3)**

Focus on leads V1, V2, and II (see Fig. 2-2). Leads V1 and II are best for visualization of P waves to determine the presence of sinus rhythm or an arrhythmia, and V1 and V2 are best to observe for bundle branch block. If P waves are not clearly visible in V1, assess them in lead II, which usually shows well-formed P waves. Identification of the P wave and then the RR intervals allows the interpreter to discover immediately whether the rhythm is sinus or other and to take the following steps:

- Confirm, if the rhythm is sinus, that the RR intervals are equidistant (see Fig. 2-2A), that the P wave is positive in lead II, and that the PP intervals are equidistant and equal to the RR interval.
- Do an arrhythmia assessment if the rhythm is abnormal (see Fig. 2-3, Step 11 [Fig. 2-37], and Chapter 11).
- Determine the heart rate (Table 2-2).

**STEP 2: ASSESS INTERVALS AND BLOCKS (FIG. 2-4)**

- Determine the PR interval; if it is abnormal (>0.2 second), consider first-degree atrioventricular (AV) block (Table 2-1).
- Assess the QRS duration for bundle branch block; if it is ≥0.12 second, bundle branch block is present; assess both V1 and V6. Understanding the genesis of the QRS complex is an essential step and clarifies the ECG manifestations of bundle branch blocks (see Figs. 2-5 to 2-8 and Chapter 4).
**STEP 1**

Look at P waves and RR intervals in leads II and V1.
Look at leads V1 and V2; best for bundle branch block.

Determine

Rhythm
- Sinus?
  - Yes
  - No

Abnormal rhythm

Rate
(see Table 2-2)

Do arrhythmia assessment
(see Step 11 and Chapter 11)

- VPBs or APBs*
- Narrow QRS tachycardia
  (Figure 2-37)
- Wide QRS tachycardia
  (Figure 2-38)
- Bradyarrhythmia
  (Chapter 11)

*Ventricular premature beats, atrial premature beats

**Fig. 2-3.** Step-by-step method for accurate ECG interpretation. Step 1: Assess rhythm and rate.

**Right Bundle Branch Block**

The ECG criteria for RBBB are as follows:

- QRS duration ≥0.12 second.
- M-shaped complex in V1 and V2.
- Slurred S wave in leads 1, V5, V6; and an S wave that is of greater amplitude (length) than the preceding R wave (see Figs. 2-4, 2-6, and 2-7 and Chapter 4, Fig 4-2).

**Left Bundle Branch Block**

The ECG criteria for LBBB are as follows:

- QRS duration ≥0.12 second.
- A small R or QS wave in V1 and V2.
Table 2-2

<table>
<thead>
<tr>
<th>Determination of Heart Rate</th>
</tr>
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<tbody>
<tr>
<td>Heart rate (bpm)</td>
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</table>

<table>
<thead>
<tr>
<th>Number of large squares (bold boxes) in one RR interval*</th>
<th></th>
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<tbody>
<tr>
<td>1</td>
<td>300</td>
</tr>
<tr>
<td>1.5</td>
<td>200</td>
</tr>
<tr>
<td>2</td>
<td>150</td>
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<td>3</td>
<td>100</td>
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<td>60</td>
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<tr>
<td>9</td>
<td>33</td>
</tr>
<tr>
<td>10</td>
<td>30</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Number of QRS complexes in 6 seconds†</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>5 × 10</td>
<td>50</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
</tr>
<tr>
<td>7</td>
<td>70</td>
</tr>
<tr>
<td>10</td>
<td>100</td>
</tr>
<tr>
<td>15</td>
<td>150</td>
</tr>
<tr>
<td>20</td>
<td>200</td>
</tr>
</tbody>
</table>

*Normal paper speed 25 mm/s. One large box or five small squares (0.2 second) = 300 bpm (see Fig. 2-2C); four large boxes = 75 bpm.
†If the ECG paper has markers at 3-second intervals, count the number of QRS complexes in two of these 3-second periods (6 seconds) and multiply by 10 (see Fig. 2-2C). This method is advisable if there is bradycardia or irregular rhythm. For 5-second interval, multiply the number of QRS complexes by 12.

For regular rhythm: start with a complex that lies on a bold vertical grid line.
Rate = 300 bpm ÷ number of large boxes (0.2 second) in one RR interval.
Normal rate is between 60 bpm (five boxes) and 100 bpm (three boxes); therefore, no need to calculate exact rate.
Or: rate = 1,500 ÷ number of small (1 mm, 0.04 second) squares in one RR interval.

• A notched R wave in leads 1, V5, and V6 (see Figs. 2-4 and 2-8 and Chapter 4).

In the presence of LBBB, vector forces are deranged and the ECG cannot be used for the diagnosis of ischemia or ventricular hypertrophy. The diagnosis of acute MI in the presence of LBBB is difficult to make and can be erroneous (see discussion of LBBB and acute MI in Chapter 6).
Fig. 2-4. Step-by-step method for accurate ECG interpretation. Step 2: Assess intervals and blocks.
V(I) = vector I produces a small r wave in leads V₁ and V₂, Q in leads V₅ and V₆.
V(II) = vector II produces an S wave in lead V₁ and an R wave in lead V₅ or V₆.
V(III) = vector III produces the terminal S in leads V₅ and V₆ and the terminal r or r′ in V₁, V₂, and aVR.

V₁ = lead V₁ electrode.
V₅ = lead V₅ electrode.
R = right ventricle muscle mass.
L = left ventricle muscle mass.
S = septum.

**Fig. 2-5.** Vectors I, II, and III, labeled V(I), V(II), and V(III), underlie the genesis of the normal QRS complex. (From Khan, M. Gabriel: *On Call Cardiology*, 3rd ed., Philadelphia, 2006, WB Saunders, Elsevier Science.)

**Fig. 2-6.** Genesis of the QRS complex in right bundle branch block. (From Khan, M. Gabriel: *On Call Cardiology*, 3rd ed., Philadelphia, 2006, WB Saunders, Elsevier Science.)
Fig. 2-7. A, QRS duration in V₁ ≥ 0.12 second; RSR’ (M-shaped complex) in V₁; and wide, slurred S wave in leads 1, V₅, and V₆ indicate right bundle branch block.
Fig. 2-7. B. Limb leads; slurred, wide S wave in lead I, and the amplitude (length or duration) of the S wave is greater than the preceding R wave.
**STEP 3: ASSESS FOR NONSPECIFIC INTRAVENTRICULAR CONDUCTION DELAY AND WOLFF-PARKINSON-WHITE SYNDROME** (FIG. 2-9)

- If the QRS duration is prolonged ≥0.11 second and bundle branch block appears to be present but is atypical, consider WPW syndrome, particularly if there is a tall R wave in V₁ and V₂ (Table 2-3; see also Figs. 11-27 and 11-28).
- Assess for a short PR interval ≤0.12 second and for a delta wave (Fig. 2-10).
Fig. 2-8. Continued
**STEP 3**

QRS ≥0.11 But not typical RBBB or LBBB configuration

1. Atypical RBBB

2. Atypical LBBB

**CONSIDER**

Spot for Delta Wave + PR ≤0.12* (see Figure 2-10)

Present

3. WPW SYNDROME*

1, 2, and 3 excluded

Diagnosis

INTRAVENTRICULAR CONDUCTION DELAY (IVCD)

Consider Brugada syndrome and right ventricular dysplasia, rare forms of RBBB patterns; see Chapter 4

* = in ~20% the QRS is <0.11 seconds; in ~23% the PR interval is 0.12 second or slightly longer

**Fig. 2-9.** Step-by-step method for accurate ECG interpretation. Step 3: Assess for nonspecific intraventricular conduction delay and Wolff-Parkinson-White (WPW) syndrome (see Figs. 2-10 and 2-11). (See Chapter 4 for Brugada syndrome and right ventricular dysplasia, rare forms of right bundle branch block [RBBB] patterns, and Chapter 11 for Wolff-Parkinson-White syndrome.)

WPW syndrome may mimic an inferior MI (see Chapters 6 and 11 for discussion of WPW syndrome). If WPW syndrome, RBBB, or LBBB is not present, interpret as nonspecific intraventricular conduction delay (IVCD) and assess for the presence of electronic pacing (see Figs. 2-7, 2-8, 2-11, and 10-16).
Table 2-3

Causes of Tall R Waves in $V_1$ and $V_2$

1. Thin chest wall or normal variant, age <20, early transition (see Fig. 2-2C)
2. Right bundle branch block (see Fig. 2-7)
   \textit{Note:} Slurred S wave in leads I, $V_5$, and $V_6$
3. Right ventricular hypertrophy (see Fig. 7-8)
   \textit{No} slurred S wave in leads I, $V_5$, and $V_6$
4. Wolff-Parkinson-White syndrome (see Fig. 2-10)
5. True posterior infarction (see Fig. 6-19)
   \textit{Note:} Associated inferior MI, no slurred S in $V_5$ and $V_6$, and T upright in $V_1$ and $V_2$
6. Hypertrophic cardiomyopathy
7. Duchenne muscular dystrophy
8. Low placement of leads $V_1$ and $V_2$
9. Dextroposition (see Fig. 10-9)

\textbf{Fig. 2-10.} Tall R waves in leads $V_1$ and $V_5$; QRS duration $\geq 0.11$ second; and delta wave in $V_3$ through $V_5$ indicate Wolff-Parkinson-White syndrome.
Fig. 2-11. Sinus rhythm 72/min; ventricular premature beats; QRS duration 0.14 s, 140 ms: Intraventricular conduction delay (IVCD). Abnormal ECG.
STEP 4: ASSESS FOR ST SEGMENT ELEVATION OR DEPRESSION (FIG. 2-12)

- Focus on the ST segment for elevation or depression (see Fig. 2-12). ST elevation ≥1 mm (0.1 mV) in two or more contiguous ECG leads in a patient with chest pain indicates ST elevation MI (STEMI). The diagnosis is strengthened if there is reciprocal depression (Fig. 2-13).
- Figure 2-13A shows marked ST elevation in leads II, III, and aVF, with marked reciprocal depression in leads I and aVL, diagnostic of acute inferior MI.
- Figure 2-13B shows marked ST segment elevation in V₁ through V₅, caused by extensive acute anterior MI.

![Diagram of Step 4: ECG Interpretation]

- **ST segment elevation**
  - Yes
    - ≥1 mm elevation in two or more limb leads II, III, and aVF
      - Acute inferior MI
  - No
    - ≥1 mm elevation in two or more contiguous precordial leads V₁ to V₆
      - Acute anterior MI

- **ST depression**
  - ≥1 mm in two or more leads
  - Yes
    - Troponin or CK-MB positive?
      - Yes
        - Acute anterior MI
      - No
        - Acute inferior MI

Yes
- ST elevation MI (see Figure 2-13)
- Non-ST elevation MI (non-Q wave infarction) (see Figure 2-14, A)
- Ischemia (see Figure 2-14, B and C and Chapter 5)

*Reciprocal depression increases probabilities of acute myocardial infarction (MI).

**Fig. 2-12.** Step-by-step method for accurate ECG interpretation. Step 4: Assess for ST segment elevation or depression.
Fig. 2-13. A, Marked ST segment elevation in leads II, III, and aVF with reciprocal depression in leads I and aVL indicate acute inferior infarction. B, Marked ST segment elevation in leads V₁ through V₅ indicates acute anterior infarction. C, Electrocardiogram of a 79-year-old woman with an apparent acute subendocardial myocardial infarction attributed to subtotal occlusion of the left main coronary artery, associated with global hypokinesis and an estimated left ventricular ejection fraction of 10%. ST segment is depressed in leads I, II, III, aVL, aVF, and V₂ through V₆. Apparent “reciprocal” ST segment elevation is seen in leads aVR and V₁. (From Surawicz B, Knilans TK: Chou’s Electrocardiography in Clinical Practice, 5th ed., Philadelphia, 2001, WB Saunders, Elsevier Science.)

- Figure 2-13C shows the ECG of a patient with a subtotal occlusion of the left main coronary artery. Note the ST elevation in aVR is greater than the ST elevation in V₁, a recently identified marker of left main coronary disease. (See Chapter 5, particularly Figs. 5-11 and 5-12, for an in-depth discussion of Step 4: ST segment elevation.)
- Figure 2-14A shows features of non–ST elevation MI (non–Q wave MI).
- Figures 2-14B and 2-14C illustrate ECG features diagnostic of myocardial ischemia.
Fig. 2-13. Continued
A. Marked ST segment depression and elevated creatine kinase (CK) and CK-MB indicate non–Q wave myocardial infarction. 

B. ECG patterns of myocardial ischemia. 

*Upsloping ST depression is nonspecific; commonly seen with tachycardia. (From Khan, M. Gabriel: On Call Cardiology, 3rd ed., Philadelphia, 2006, WB Saunders, Elsevier Science.)

C. Leads V₄ through V₆ show ST segment depression; V₄ through V₆ are in keeping with myocardial ischemia from a patient known to have unstable angina.

Fig. 2-14. A, Marked ST segment depression and elevated creatine kinase (CK) and CK-MB indicate non–Q wave myocardial infarction. B, ECG patterns of myocardial ischemia. *Upsloping ST depression is nonspecific; commonly seen with tachycardia. (From Khan, M. Gabriel: On Call Cardiology, 3rd ed., Philadelphia, 2006, WB Saunders, Elsevier Science.) C, Leads V₄ through V₆ show ST segment depression; V₄ through V₆ are in keeping with myocardial ischemia from a patient known to have unstable angina.
Elevation of the ST segment may occur as a normal variant (Fig. 2-15). See Chapters 5 and 6 for further discussion of ST segment abnormalities and MI.

*Note:* This text advises scrutiny of the ST segment before assessment of T waves, electrical axis, QT interval, and hypertrophy because the diagnosis of acute MI or ischemia is vital and depends on careful assessment of the ST segment.
Exclude other causes of ST elevation:

- Normal variant: 1- to 2-mm ST segment elevation, mainly in leads V_2 through V_4, nonconvex, and with fishhook appearance. Common in African Americans: even 4-mm ST segment elevation (see Fig. 2-15 and sections on acute myocardial infarction in Chapters 5 and 6).
- Coronary artery spasm: ST returns to normal with nitroglycerin or with pain relief.
- LBBB: QRS >0.12 second and typical configuration (see Fig. 2-8B, and Chapter 4).
- Left ventricular aneurysm and known old infarct with old Q waves (see Chapter 6).

**STEP 5: ASSESS FOR PATHOLOGIC Q WAVES (THAT IS, LOSS OF R WAVES) (FIG. 2-16)**

- Assess for the loss of R waves—pathologic Q waves—in leads I, II, III, aVL, and aVF (see Figs. 2-17A and 2-17B and Chapter 6 for detailed discussion).
Fig. 2-16. Step-by-step method for accurate ECG interpretation. Step 5: Assess for pathologic Q waves (i.e., loss of R waves).
Fig. 2-17. A, Loss of R wave in leads III and aVF (i.e., pathologic Q waves associated with marked ST segment elevation in leads III and aVF) and minimal elevation in lead II and reciprocal depression in leads I and aVL indicate typical acute Q wave inferior infarction. B, Wide, deep pathologic Q waves in leads II, III, and aVF and isoelectric ST segment indicate old inferior myocardial infarction. C, Variation in QRS configuration caused by rotation. (From Khan, M. Gabriel: *On Call Cardiology*, 2nd ed., Philadelphia, 2001, WB Saunders, Elsevier Science.)
Fig. 2-17. Continued
Assess for R wave progression in V2 through V4. Figure 2-17C illustrates the variation in the normal QRS configuration that occurs with rotation. The R wave amplitude should measure from 1 mm to at least 20 mm in V3 and V4 (see Table 2-1). Loss of R waves in V1 through V4 with ST segment elevation indicates acute anterior MI (Fig. 2-18A).

- Loss of R wave in V1 through V3 with the ST segment isoelectric and the T wave inverted may be interpreted as anteroseptal MI age indeterminate (i.e., infarction in the recent or distant past) (Fig. 2-18B). Features of old anterior MI are shown in Fig. 2-18C and lateral infarction is shown in Fig. 2-19.

Fig. 2-17. Continued
Fig. 2-18. A, Loss of R waves in V_2 through V_5 (i.e., pathologic Q waves associated with abnormal ST segment elevation) indicates acute anterior infarction. B, Loss of R wave in V_1 through V_3 (i.e., pathologic Q waves associated with an isoelectric ST segment) and T wave inversion indicate anteroseptal infarction, age indeterminate, infarction occurring approximately 1 to 12 months before the recording of this tracing; comparison with previous ECGs and clinical history required to determine the age of infarction. C, Loss of R waves in V_2 through V_5 (i.e., pathologic Q waves in V_2 through V_4 not associated with acute ST segment changes) indicates old anterior infarction. D, Loss of R waves in V_4 and V_5 indicate anterior myocardial infarction, age indeterminate.

(continued)
Fig. 2-18. Continued
Fig. 2-18. Continued
Fig. 2-19. Pathologic Q waves in V₄ through V₆ and ST segment in keeping with an old anterolateral infarct; clinical correlation necessary to confirm the presence of an old infarction.

Poor R wave progression in V₂ through V₄ may be caused by the following:

- Improper lead placement.
- Late transition (Fig. 2-20).
- Anteroseptal or anteropical MI.
- LVH (see Chapter 7).
- Severe chronic obstructive pulmonary disease, particularly emphysema—emphysema may cause QS complexes in leads V₁ through V₄,
Fig. 2-20. Poor R wave progression in leads V₂ through V₅. Note: The negative QRS complex in V₅ is caused by late transition and not by other causes of poor R wave progression such as anterior infarction. ECG within normal limits.

which may mimic MI; a repeat ECG with recording electrodes placed one intercostal space below the routine locations should cause R waves to be observed in leads V₂ through V₄ (see Chapter 6)
• Hypertrophic cardiomyopathy.
• LBBB (Fig. 2-8B).

In women, albeit rarely, the R wave in V₂ or V₃ may be less than 1 mm tall; this may cause an erroneous diagnosis of anteroseptal infarction.

In summary: An abnormal, pathologic Q wave is defined in adults as one that has a duration of >40 ms, but the definition does not apply to leads aVR and V₁, which may normally lack the initial R wave. In
addition, in leads III, aVF, and aVL, the initial R wave may be absent; the resultant QS or QR pattern represents a normal variant. A QS pattern is disturbing to students and clinicians. The student is warned: Sound knowledge of normal variants and features of normal ECG deflections that look abnormal but are indeed normal must be mastered by the competent interpreter of ECGs. A QS is often observed in lead aVL in thin subjects with a vertical heart. A QS in lead III is common in individuals with a horizontal heart position, some of whom are obese. See Chapter 6 for an in-depth discussion of Step 5: Q wave abnormalities.

**STEP 6: ASSESS P WAVES** (FIG. 2-21)

- Assess the P waves for abnormalities including atrial hypertrophy (Figs. 2-22 and 2-23; see also Fig. 3-3).

**Assess P waves in leads II and V₁ for hypertrophy.**

- **Peaked ≥3 mm amplitude**
  - Yes: Probable right atrial enlargement (see Figure 2-22 and Chapter 7)
  - No: Normal

- **Broad >2.5 mm (≥0.11 second) or bifid in lead II or diphasic in V₁ (see Figures 2-22 and 2-23)**
  - No: Check for RVH and causes of RVH, right ventricular strain, and other features of pulmonary embolism (see Chapter 7)
  - Yes: Left atrial enlargement.*
    - Check for mitral stenosis, mitral regurgitation, LV failure, dilated cardiomyopathy, LVH and causes of LVH (see Figure 2-23 and Chapter 7)

* = Left atrial abnormality: enlargement, hypertrophy, or increased atrial volume or pressure.

**Fig. 2-21.** Step-by-step method for accurate ECG interpretation. Step 6: assess P waves.
**Fig. 2-22.** A, Left atrial enlargement: P wave duration greater than three small squares (0.12 second) in lead 2; in lead V₁, the negative component of the P wave occupies at least one small box: $1 \text{mm} \times 0.04 \text{ second} = \text{P terminal force} \geq -0.04 \text{ mm s.}$ B, Right atrial enlargement: lead 2 shows P amplitude $>3 \text{ mm}$; in V₁, the first half of the P wave is positive and $>1 \text{ mm}$ wide (see Figs. 2-23, 7-2, and 7-3). (From Khan, M. Gabriel: *On Call Cardiology*, 3rd ed., Philadelphia, 2006, WB Saunders, Elsevier Science.)

**Fig. 2-23.** Lead V₁ shows right and left atrial hypertrophy. Lead II shows peaked P waves caused by right atrial enlargement.
STEP 7: ASSESS FOR LEFT AND RIGHT VENTRICULAR HYPERTROPHY (FIG. 2-24)

- Assess for LVH (see Figs. 2-24 and 2-25 and Chapter 7) and RVH (see Fig. 2-26 and Chapter 7 for further details).
- Criteria for LVH and RVH are not applicable if bundle branch block is present. Thus, it is essential to exclude LBBB and RBBB early in the interpretive sequences as delineated previously in Steps 2 and 3.

STEP 8: ASSESS T WAVES (FIG. 2-27)

- Assess the pattern of T wave changes (see Fig. 2-27). T wave changes are usually nonspecific (Fig. 2-28). T wave inversion associated with ST segment depression or elevation indicates myocardial ischemia (Fig. 2-29). See Chapter 8 for further information on T wave abnormalities.

**STEP 7**

a. **Assess for left ventricular hypertrophy (LVH),**

1. S wave in $V_1 + R$ wave in $V_5$ or $V_6 > 35$ mm = LVH $\approx 90\%$ specificity; sensitivity $< 40\%$
2. R wave in aVL + S wave in men $\geq 24$ mm and in women $\geq 18$ mm = LVH $\approx 90\%$ specificity; sensitivity $< 40\%$
3. Specificity of (1) or (2) increased to $\approx 98\%$ in presence of
   a. Left atrial enlargement or
   b. ST segment depression and T wave inversion (strain pattern) in $V_5$ or $V_6$ (see Figures 2-23 and 2-25)

b. **Assess for right ventricular hypertrophy (RVH).**

1. R wave in $V_1$ $\geq 7$ mm$^\dagger$
2. S wave in $V_5$ or $V_6$ $\geq 7$ mm
3. R/S ratio in $V_1$ $\geq 1$
4. R/S ratio in $V_5$ or $V_6$ $\leq 1$
5. Right axis deviation $\geq +110^\circ$
   Any two of above = RVH likely (see Figure 2-26)
6. Specificity increased if ST depression and T wave inversion in $V_1$ to $V_3$ or right atrial hypertrophy (see Figures 2-26, 7-7, and 7-8

$^\dagger$Age $> 30$; $\geq 40$ mm, age 20 to 30.

**Fig. 2-24.** Step-by-step method for accurate ECG interpretation. Step 7: Assess for left ventricular hypertrophy (LVH) and right ventricular hypertrophy (RVH) (not applicable if QRS duration $\geq 0.12$ second or in presence of LBBB or RBBB).
Fig. 2-25. Note that the standardization at half voltage in V₁ through V₆ is markedly increased; ST-T strain pattern in V₅ and V₆ and left atrial enlargement are typical features of left ventricular hypertrophy.
Fig. 2-26. A, Leads V₁ through V₆: A tall R wave in V₁, R/S ratio in V₁ >1, and R/S ratio in V₅ or V₆ <1 are features of right ventricular hypertrophy. B, Limb leads: right-axis deviation +140 degrees, peaked P wave in lead II, and right atrial enlargement are all in keeping with right ventricular hypertrophy.
Fig. 2-27. Step-by-step method for accurate ECG interpretation. A, Step 8: Assess T wave changes. B, Step 8: Alternative approach for the assessment of T wave changes.
Fig. 2-28. T wave inversion in V2 through V5 not associated with ST segment depression or elevation; nonspecific ST-T wave changes; cannot exclude ischemia, but the tracing is not diagnostic. Abnormal ECG.
**Fig. 2-35.** ECG showing electronic pacing and ventricular capture; rate is 60 beats/min. No further analysis is possible because of pacemaker rhythm.

**Fig. 2-36.** Mirror-image dextrocardia with situs inversus. The patient is a 15-year-old girl. There is no evidence of organic heart disease. A, Tracing recorded with conventional electrode placement. B, Tracing obtained with the left and right arm electrodes reversed. The precordial lead electrodes also were located in the respective mirror-image positions on the chest. The tracing is within normal limits. (From Chou TC: *Electrocardiography in Clinical Practice*, 4th ed., Philadelphia, 1996, WB Saunders, Elsevier Science.)
STEP 11: ASSESS ARRHYTHMIAS (FIG. 2-37)

Tachyarrhythmias should be analyzed as the following:

- **Narrow complex tachycardia**: Figure 2-37A gives the differential diagnosis of narrow QRS complex tachycardia.
- **Wide complex tachycardia**: Figure 2-37B gives the differential diagnosis of wide QRS complex tachycardia. See Chapter 11 for relevant ECGs.

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**Fig. 2-37.** Step-by-step method for accurate ECG interpretation. Step 11: Assess arrhythmias: differential diagnosis of narrow QRS tachycardia (A) and wide QRS tachycardia (B).
and detailed discussion of arrhythmia diagnosis including that of bradyarrhythmias.

**ELECTROCARDIOGRAM TECHNIQUE**

- Ensure the standardization is 1 mV displayed as a 10-mm deflection (10 small squares in amplitude).
- Always record the ECG at a standard paper speed of 25 mm/s.
- Remember that artifacts such as baseline drift are often caused by loose or improperly installed sensors.
- Most ECG machines have two modes of operation: automatic or manual. Familiarize yourself with the procedure in the ECG department of your hospital so that you can do the ECG, if called, when there is no technician or nurse available to do the procedure.
- Attach the electrodes (bulb suction cup or flat sensors) on a smooth, fleshy part of the lower arm or forearm and on the fleshy parts of the lower leg.
- Attach the chest lead sensors as indicated in Fig. 2-38 (bulb sensor suction cups or flat sensors).

Ensure that electrodes are properly placed. Incorrect lead placement can lead to serious errors with interpretation (Fig. 2-39).
V1 = 4th interspace at the right margin of the sternum
V2 = 4th interspace at the left margin of the sternum
V3 = Midway between positions for V2 and V4
V4 = 5th interspace at junction of left midclavicular line (apex)
V5 = At horizontal level of position V4 at left anterior axillary line
V6 = Same horizontal line as for position V4 but in the midaxillary line

Fig. 2-38. Chest leads placement. V1, 4th interspace at the right margin of the sternum; V2, 4th interspace at the left margin of the sternum; V3, midway between positions for V2 and V4; V4, 5th interspace at junction of left midclavicular line (apex); V5, at horizontal level of position V4 at left anterior axillary line; V6, same horizontal line as for position V4 but in the midaxillary line.

Fig. 2-39. A, Atrial fibrillation and pseudo–inferior infarction resulting from electrode misplacement. With Q waves and ST elevation in leads 2, 3, and aVF and with reciprocal depression of the ST segment in aVL and chest leads, this tracing suggests acute inferior infarction. However, lead 1, with virtually no deflections, is the tip-off: The two arm electrodes are on the two legs (and the leg electrodes are on the arms). B, Limb leads with the electrodes attached correctly. (From Marriott JLH: Practical Electrocardiography, 8th ed., Baltimore, 1988, Williams & Wilkins.)
Fig. 2-40. Arm electrodes interchanged. Otherwise ECG within normal limits.
Untrained technicians often place leads V₅ and V₆ too anteriorly; this may not give a true recording of the left ventricular muscle mass. The leads must be placed in the anterior and midaxillary line (see Fig. 1-15). Incorrect placement of V₂ and V₃ may render a false interpretation of old anteroseptal MI. Thus, much care is needed in placing the chest leads. Feel for the bony points (see Fig. 1-14) to position V₂, V₃, and V₄. Small changes in electrode position can cause significant changes in the record obtained with these leads.

The most common error is the reversal of the left and right arm leads. The ECG records the following: the P wave is negative in lead I and upright in aVR; lead I is a mirror image of I, and therefore the entire complex that is usually positive becomes negative; there is reversal of lead aVR and aVL (aVR is aVL: aVL shows a negative P wave and a relatively negative complex because it is aVR); and there is reversal of leads II and III (lead II is III and lead III is II). Figure 2-40 shows the effect of the reversal of the arm leads. The P, QRS, and T waves are inverted in leads I and aVL; the precordial (V) leads remain normal, however, and thus rule out dextrocardia, in which the limb leads are similar but there is loss of R waves or poor R wave progression from V₂ through V₆ (see Fig. 2-36).