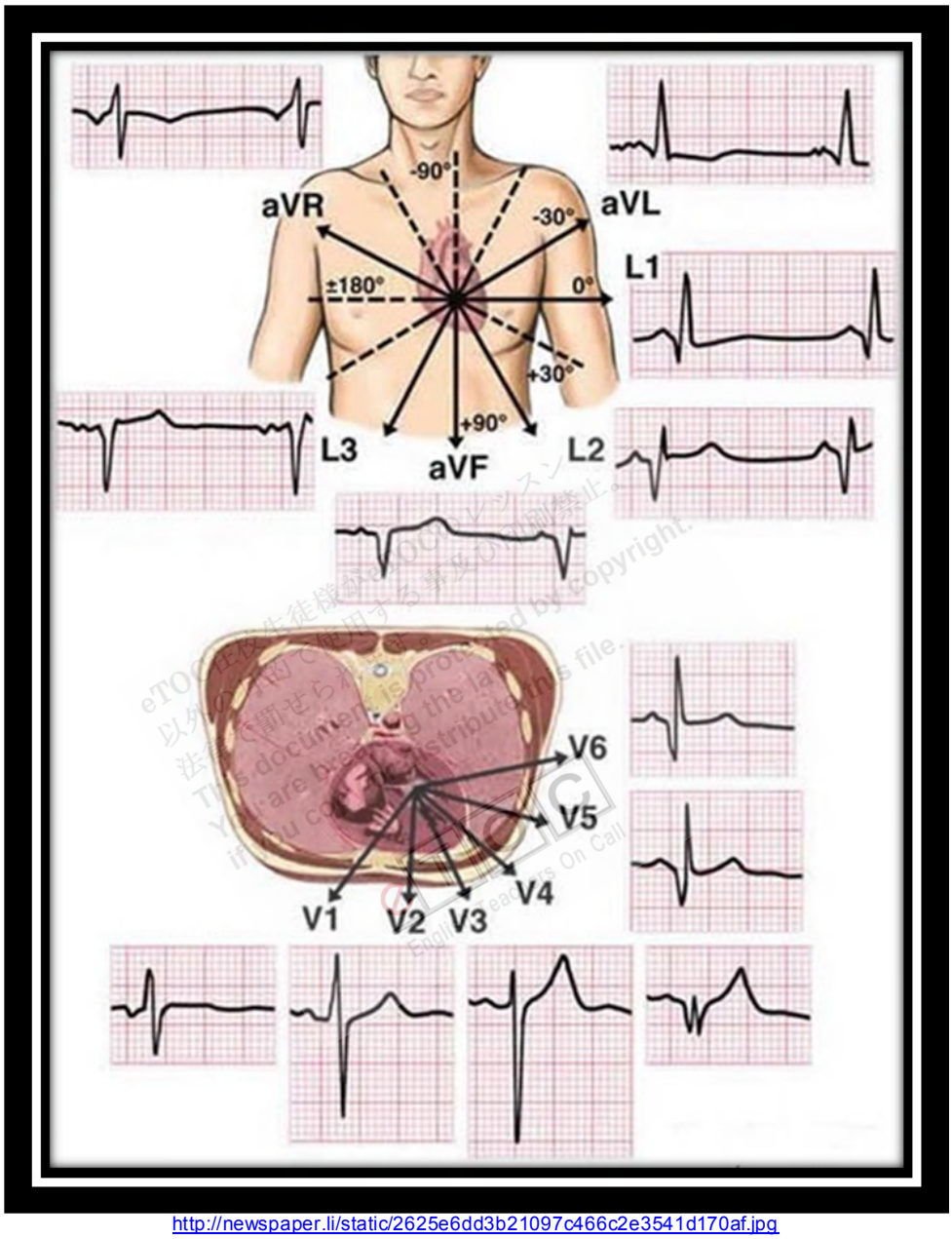
# Electrocardiography (ECG)

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The standard ECG provides 12 different **vector** views of the heart's electrical activity as reflected by electrical potential differences between positive and negative **electrodes** placed on the limbs and chest wall. Six of these views are vertical (using frontal leads I, II, and III and limb leads aVR, aVL, and aVF), and 6 are horizontal (using precordial leads $V_1$, $V_2$, $V_3$, $V_4$, $V_5$, and $V_6$). The 12-lead ECG is crucial for establishing many cardiac diagnoses, especially **arrhythmias** and **myocardial ischemia**. It can also

identify **atrial enlargement**, **ventricular hypertrophy**, and conditions that **predispose** to **syncope** or sudden death (eg, **Wolff-Parkinson-White syndrome**, **long QT syndrome**, **Brugada syndrome**). A useful ECG tutorial is available from the University of Utah.

**Table 4**

**Interpretation of Abnormal ECGs**

| Abnormal Component | Description | Possible Causes |
|---|---|---|
| P waves | Abnormal | Left or right atrial hypertrophy, atrial escape (ectopic) beats |
| P waves | Absent | Atrial fibrillation, sinus node arrest or exit block, hyperkalemia (severe) |
| P-P interval | Varying | Sinus arrhythmia |
| PR interval | Long | First-degree atrioventricular block |
| PR interval | Varying | Mobitz type I atrioventricular block, multifocal atrial tachycardia |
| QRS complex | Wide | Right or left bundle branch block, ventricular flutter or fibrillation, hyperkalemia |
| QT interval | Long | MI, myocarditis, hypocalcemia, hypokalemia, hypomagnesemia, hypothyroidism, subarachnoid or intracerebral hemorrhage, stroke, congenital long QT syndrome, antiarrhythmics (eg, sotalol,amiodarone, quinidine), tricyclic antidepressants, phenothiazines, other drugs |
| QT interval | Short | Hypercalcemia, hypermagnesemia, Graves disease,digoxin |

| | | |
|---|---|---|
| ST segment | Depression | Myocardial ischemia; acute posterior MI; digoxin; ventricular hypertrophy; pulmonary embolism; left bundle branch block; right bundle branch block in leads $V_1$ −$V_3$ and possibly in II, III, and aVF; hyperventilation; hypokalemia |
| ST segment | Elevation | Myocardial ischemia, acute MI, left bundle branch block, acute pericarditis, left ventricular hypertrophy, hyperkalemia, pulmonary embolism, digoxin, normal variation (eg, athlete's heart), hypothermia |
| T wave | Tall | Hyperkalemia, acute MI, left bundle branch block, stroke, ventricular hypertrophy |
| T wave | Small, flattened, or inverted | Myocardial ischemia, myocarditis, age, race, hyperventilation, anxiety, drinking hot or cold beverages, left ventricular hypertrophy, certain drugs (eg, digoxin), pericarditis, pulmonary embolism, conduction disturbances (eg, right bundle branch block), electrolyte disturbances (eg, hypokalemia) |
| U wave | Prominent | Hypokalemia, hypomagnesemia, ischemia |

**Table 5**

### Criteria for ECG Diagnosis of Left Ventricular Hypertrophy

| Criterion | Finding | Points |
|---|---|---|
| Romhilt-Estes (5 points = definite LVH; 4 | R or S wave ≥ 20 mm in any limb lead | 3 |

| | | |
|---|---|---|
| points =probable LVH) | *or*<br>S wave in $V_1$ or $V_2$ ≥ 30 mm<br>*or*<br>R wave in $V_5$ or $V_6$ ≥ 30 mm | |
| | ST-T changes typical of LVH | |
| | Digitalis | 1 |
| | No digitalis | 3 |
| | Left atrial changes: P terminal wave in $V_1$, amplitude≥ 1 mm, and duration ≥ 0.04 sec | 3 |
| | Left axis deviation ≥ −30° | 2 |
| | QRS duration ≥ 90 msec | 1 |
| | Interval between QRS and R-wave peak in $V_5$ or $V_6$ ≥0.05 sec | 1 |
| Sokolow-Lyon | $V_1$ S wave + $V_5$ or $V_6$ R wave ≥ 35 mm<br>*or*<br>aVL R wave ≥ 11 mm | |
| Cornell | Men: $V_3$ S wave + aVL R wave > 28 mm | |
| | Women: $V_3$ S wave + aVL R wave > 20 mm | |

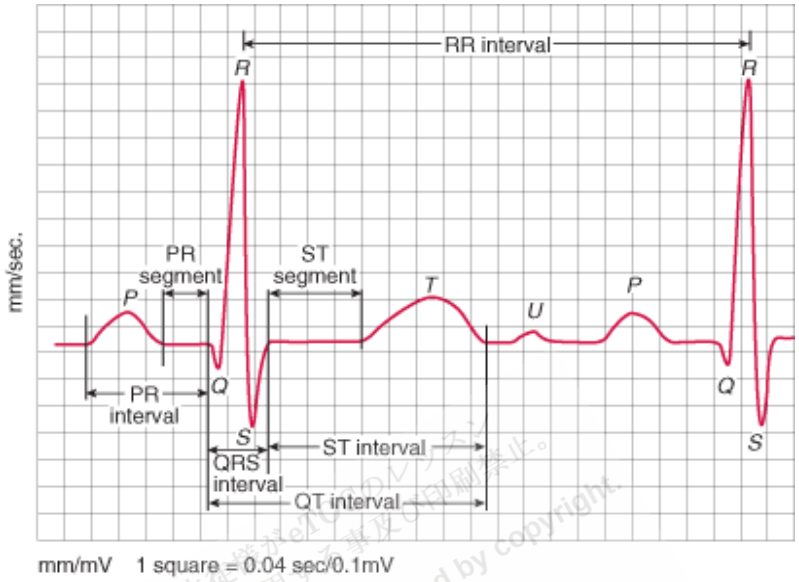
LVH = left ventricular hypertrophy.

**Standard ECG Components**

By convention, the ECG tracing is divided into the P wave, PR interval, QRS complex, QT interval, ST segment, T wave, and U wave.

**Fig. 2**

**ECG waves.**




**P wave** = activation (**depolarization**) of atria. **PR interval** = time interval between onset of atrial depolarization and onset of ventricular depolarization. **QRS complex** = depolarization of ventricles, consisting of the Q, R, and S waves. **QT interval** = time interval between onset of ventricular depolarization and end of ventricular repolarization. **R-R interval** = time interval between 2 QRS complexes. **T wave** = ventricular repolarization. ST segment plus **T wave (ST-T)** = ventricular repolarization. **U wave** = probably after-depolarization (relaxation) of ventricles.

***P wave:*** The P wave represents atrial depolarization. It is upright in most leads except aVR. It may be **biphasic** in leads II and $V_1$; the initial component represents right atrial activity, and the 2nd component represents left atrial activity.

An increase in **amplitude** of either or both components occurs with atrial enlargement. Right atrial enlargement produces a P wave > 2 mm in leads II, III, and aVF (P pulmonale); left atrial enlargement produces a P wave that is broad and double-peaked in lead II (P mitrale). Normally, the P axis is between 0° and 75°.

***PR interval:*** The PR interval is the time between onset of atrial depolarization and onset of ventricular depolarization. Normally, it is 0.10 to 0.20 sec; **prolongation** defines 1st-degree atrioventricular block.

***QRS complex:*** The QRS complex represents ventricular depolarization. The Q wave is the initial downward **deflection**; normal Q waves last < 0.05 sec in all leads except $V_{1-3}$,

in which any Q wave is considered abnormal, indicating past or current **infarction**. The R wave is the first upward deflection; criteria for normal height or size are not absolute, but taller R waves may be caused by ventricular hypertrophy. A 2nd upward deflection in a QRS complex is designated R′. The S wave is the 2nd downward deflection if there is a Q wave and the first downward deflection if not. The QRS complex may be R alone, QS (no R), QR (no S), RS (no Q), or RSR′, depending on the ECG lead, vector, and presence of heart disorders.

Normally, the QRS interval is 0.07 to 0.10 sec. An interval of 0.10 to 0.11 sec is considered incomplete bundle branch block or a nonspecific **intraventricular conduction delay**, depending on QRS **morphology**; ≥ 0.12 sec is considered complete bundle branch block or an intraventricular conduction delay. Normally, the QRS axis is 90° to −30°. An axis of −30° to −90° is considered left axis **deviation** and occurs in left anterior fascicular block (−60°) and inferior MI. An axis of 90° to 180° is considered right axis deviation; it occurs in any condition that increases pulmonary pressures and causes right ventricular hypertrophy (cor pulmonale, acute pulmonary embolism, pulmonary hypertension), and it sometimes occurs in right bundle branch block or left posterior fascicular block.

***QT interval:*** The QT interval is the time between onset of ventricular depolarization and end of ventricular repolarization. The QT interval must be corrected for heart rate using the formula:

$$QT_c = \frac{QT}{\sqrt{RR}}$$

where $QT_c$ is the corrected QT interval; R-R interval is the time between 2 QRS complexes. All intervals are recorded in seconds. $QT_c$ prolongation is strongly implicated in development of **torsades de pointe** ventricular tachycardia. $QT_c$ is often difficult to calculate because the end of the T wave is often unclear or followed by a U wave with which it merges. Numerous drugs are implicated in prolonging the QT interval.

***ST segment:*** The ST segment represents completed ventricular myocardial depolarization. Normally, it is horizontal along the baseline of the PR (or TP) intervals or slightly off baseline.

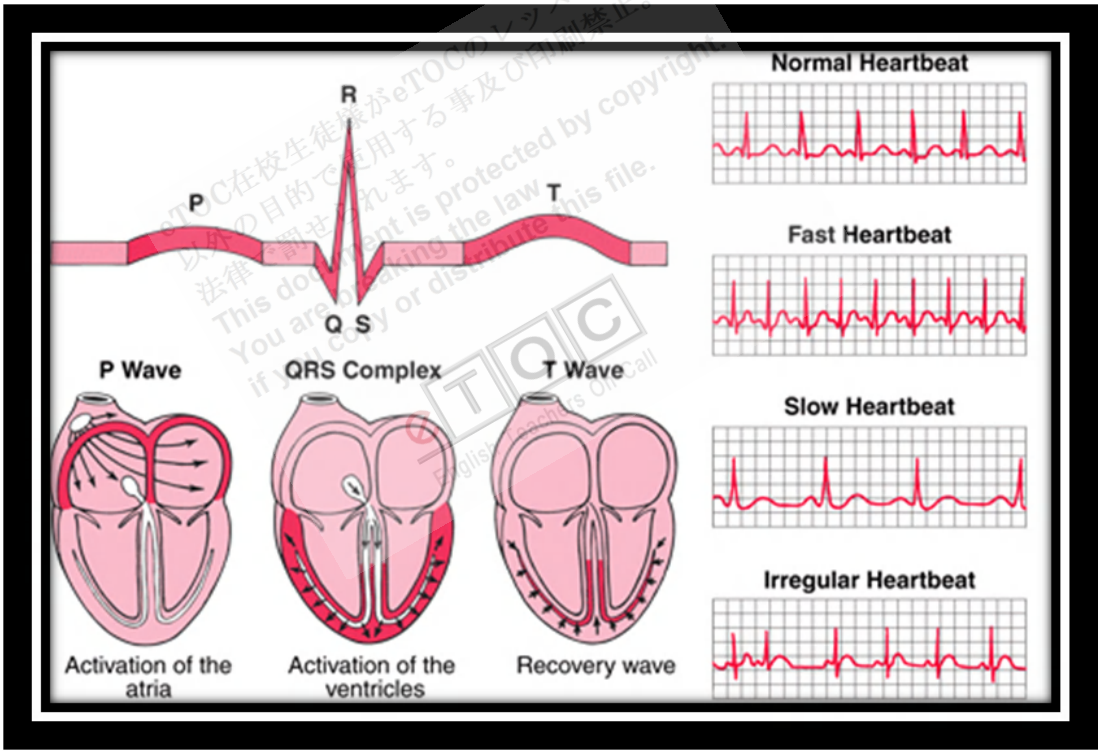
**ST segment elevation** can be caused by

- Early repolarization

- Left ventricular hypertrophy
- Myocardial ischemia and infarction
- Left ventricular aneurysm
- Pericarditis
- Hyperkalemia
- Hypothermia
- Pulmonary embolism

**ST segment depression** can be caused by

- Hypokalemia
- **Digoxin**
- Subendocardial ischemia
- Reciprocal changes in acute MI

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***T wave:*** The T wave reflects ventricular repolarization. It usually takes the same direction as the QRS complex (**concordance**); opposite polarity (**discordance**) may indicate past or current infarction. The T wave is usually smooth and rounded but may be of low amplitude in hypokalemia and hypomagnesemia and may be tall and peaked in hyperkalemia, hypocalcemia, and left ventricular hypertrophy.

***U wave:*** The U wave appears commonly in patients who have hypokalemia, hypomagnesemia, or ischemia. It is often present in healthy people.

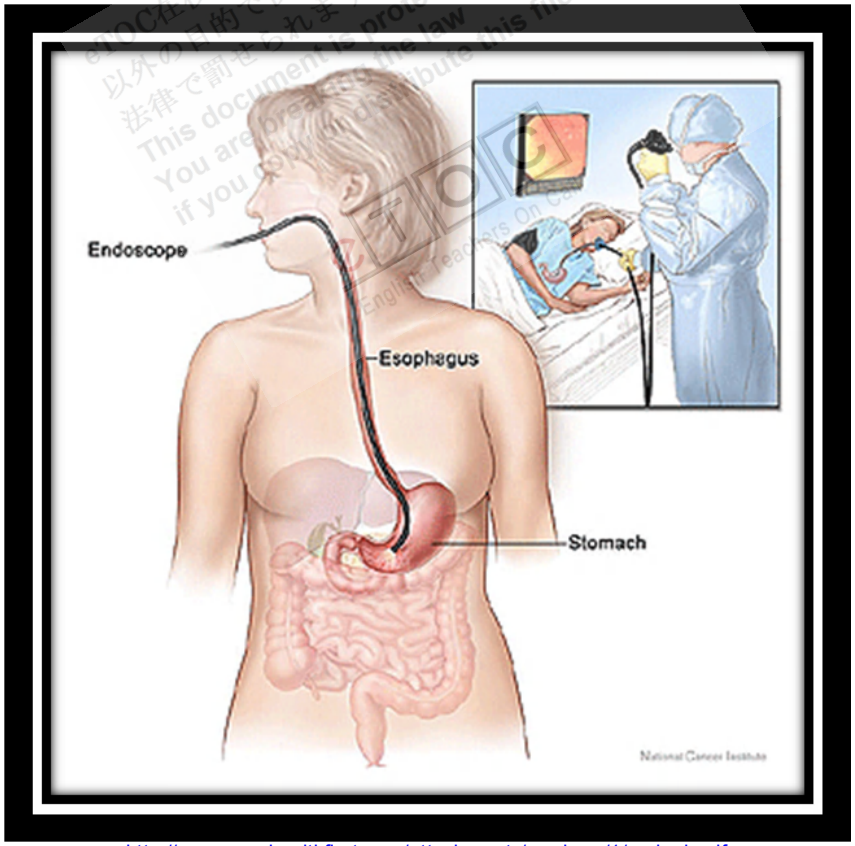
**Specialized ECG Tests**

A standard 12-lead ECG represents only a single brief period of cardiac activity; enhanced techniques can provide additional information.

***Additional precordial leads:*** Additional precordial leads are used to help diagnose right ventricular and posterior wall MI.

Right-sided leads are placed across the right side of the chest to mirror standard left-sided leads. They are labeled $V_1R$ to $V_6R$; sometimes only $V_4R$ is used, because it is the most sensitive for right ventricular MI.

Additional left-sided leads can be placed in the 5th intercostal space, with $V_7$ at the posterior axillary line, $V_8$ at the midscapular line, and $V_9$ at the left border of the spine. These leads are rarely used but may help diagnose a true posterior MI.

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***Esophageal lead:*** An esophageal lead is much closer to the atria than surface leads; it is an option when the presence of P waves on a standard recording is uncertain and when detecting atrial electrical activity is important, as when atrial or ventricular origin of wide-complex tachycardia must be differentiated or when atrioventricular **dissociation** is suspected. An esophageal lead may also be used to monitor **intraoperative myocardial ischemia** or to detect atrial activity during **cardioplegia**. The lead is placed by having the patient swallow an electrode, which is then connected to a standard ECG machine, often in the lead II port.

***Signal averaging:*** Signal averaging of QRS waveforms creates a digital composite of several hundred cardiac cycles to detect high-frequency, low-amplitude potentials and microcurrents at the terminal part of the QRS complex. These findings represent areas of slow conduction through abnormal myocardium, indicating increased risk of **reentrant** ventricular tachycardia. Signal-averaged ECG is still largely a research technique but is occasionally used to assess risk of sudden cardiac death (eg, in post-MI patients without evidence of conduction delay, patients with myocardial ischemia and unexplained syncope, and those with nonischemic cardiomyopathy) and to assess efficacy of surgery to correct the arrhythmia. This technique may also be useful for assessing the **proarrhythmic effects** of antiarrhythmic drugs and for detecting rejection of heart transplants. Signal averaging of P waves is being studied as a way to identify patients at risk of atrial fibrillation.

***Continuous ST-segment monitoring:*** This type of monitoring is used for early detection of ischemia and serious arrhythmias. Monitoring can be automated (dedicated electronic monitoring units are available) or done clinically using serial ECGs. Applications include emergency department monitoring of patients with crescendo angina, evaluation after percutaneous intervention, intraoperative monitoring, and postoperative care.

***QT dispersion:*** QT dispersion (the difference between the longest and shortest QT intervals on a 12-lead ECG) has been proposed as a measure of **myocardial repolarization heterogeneity**. Increased dispersion suggests electrically **heterogeneous myocardium** caused by ischemia or fibrosis, with increased risk of reentrant arrhythmias and sudden death. QT dispersion predicts mortality risk but is not widely measured because measurement error is common, values in patients with and without disease overlap substantially, there is no reference standard, and other validated risk predictors are available.

***Heart rate variability:*** This measurement reflects the balance between sympathetic and parasympathetic (vagal) input to the heart. Decreased variability suggests decreased vagal input and increased sympathetic input, which predict increased risk of arrhythmias and mortality. The most common measure of variability is the mean of the standard deviations of all normal R-R intervals in a 24-h ECG recording. Heart rate variability is used primarily in research, but evidence suggests that it provides useful information about left ventricular dysfunction after MI, heart failure, and hypertrophic cardiomyopathy. Most Holter monitors have software that measures and analyzes heart rate variability.

***Holter monitor:*** Holter monitoring is continuous monitoring and recording of the ECG, BP, or both for 24 or 48 h. It is useful for evaluating **intermittent arrhythmias** and, secondarily, for detecting hypertension. The Holter monitor is portable, enabling patients to participate in normal daily activities; it may also be used for sedentary hospitalized patients if automated monitoring is unavailable. Patients are asked to record symptoms and activities so that they may be correlated with events on the monitor. The Holter monitor does not automatically analyze the ECG data; a physician does so at a later date.

***Event recorder:*** Event recorders are worn for up to 30 days and can detect infrequent rhythm disturbances that 24-h Holter monitoring may miss. The recorder may operate continuously and also be activated by the patient when symptoms occur. A **memory loop** enables information to be stored for seconds or minutes before and after activation. The patient can transmit ECG data by telephone or satellite to be read by a physician. If patients have serious events (eg, syncope) at intervals of > 30 days, an event recorder may be placed subcutaneously (implantable loop recorder); it can be activated by a small magnet. Battery life for subcutaneous recorders is 24 mo.

**Reference: http://www.merckmanuals.com**