Canterbury District Health Board
Te Poari Hauora ō Waitaha

# Cardiology

# Self Learning Package

Module 2: Understanding a 12 lead ECG, Basic Electrophysiology and 12 lead ECG Placement

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## INTRODUCTION

Welcome to the ECG self learning package: Module 2. The electrocardiogram (ECG) is a diagnostic tool that measures and records the electrical activity of the heart in exquisite detail. This process checks for problems with the electrical activity of the heart. An ECG translates the heart's electrical activity into line tracings on paper and interpretation of these details allows diagnosis of a wide range of heart conditions. These conditions can vary from minor to life threatening. This package covers basic electrophysiology and 12 lead electrode placements.

The goal of this module is to review:

- Principles of electrophysiology
- The normal ECG complex
- Basic ECG interval measurements
- Measuring heart rate

This module will form the foundation of your ECG knowledge and enable you to understand the components of the normal ECG. You cannot learn the abnormal without first understanding the normal.

Learning outcomes form this module are:

- To state the measurements of ECG graph paper
- To identify the waveform components of the normal PQRST complex
- To state the normal PR interval measurement
- To state the normal QRS width measurement
- To describe three methods of measuring heart rate using ECG paper
- How to record a 12 lead ECG
- How to place a patient on telemetry.

## HOW TO USE THE ECG SELF-LEARNING PACKAGE

Follow these steps to complete the self-learning module:

1) Complete the pre reading at the start:

- ABC of clinical electrocardiography: Introduction. I-lead, rate, rhythm and cardiac axis.
- ABC of clinical electrocardiography: Introduction. II-Basic terminology.
- Understanding the 12 Lead ECG (Part 1).
- Understanding the 12 Lead ECG (Part 2).
- Precordial Electrodes Placement in Women.
- Taking a Quality ECG.

2) Complete the multi-choice question and evaluation, then return to the Cardiology CNE/CNS

Following the completion of this module you will receive **6 hours** professional development time, which will be credited to your individual training database.

# *ABC of clinical electrocardiography*
# Introduction. I—Leads, rate, rhythm, and cardiac axis

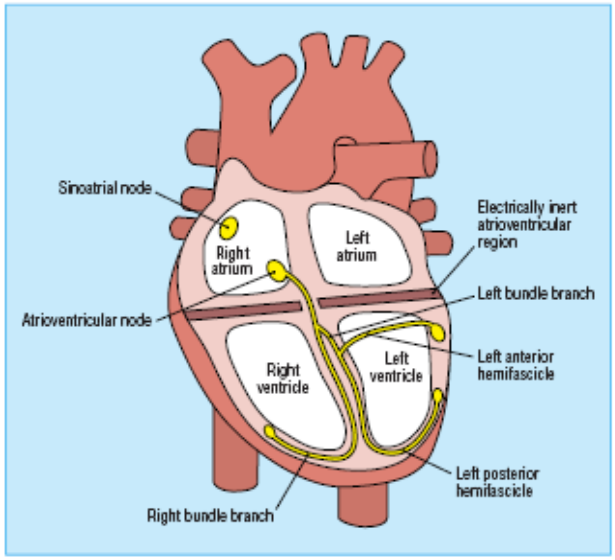
Steve Meek, Francis Morris

Electrocardiography is a fundamental part of cardiovascular assessment. It is an essential tool for investigating cardiac arrhythmias and is also useful in diagnosing cardiac disorders such as myocardial infarction. Familiarity with the wide range of patterns seen in the electrocardiograms of normal subjects and an understanding of the effects of non-cardiac disorders on the trace are prerequisites to accurate interpretation.

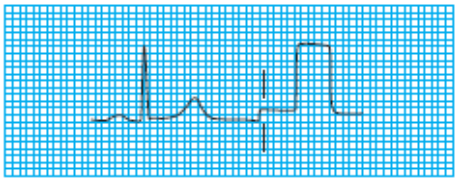
The contraction and relaxation of cardiac muscle results from the depolarisation and repolarisation of myocardial cells. These electrical changes are recorded via electrodes placed on the limbs and chest wall and are transcribed on to graph paper to produce an electrocardiogram (commonly known as an ECG).

The sinoatrial node acts as a natural pacemaker and initiates atrial depolarisation. The impulse is propagated to the ventricles by the atrioventricular node and spreads in a coordinated fashion throughout the ventricles via the specialised conducting tissue of the His-Purkinje system. Thus, after delay in the atrioventricular mode, atrial contraction is followed by rapid and coordinated contraction of the ventricles.

The electrocardiogram is recorded on to standard paper travelling at a rate of 25 mm/s. The paper is divided into large squares, each measuring 5 mm wide and equivalent to 0.2 s. Each large square is five small squares in width, and each small square is 1 mm wide and equivalent to 0.04 s.




The His-Purkinje conduction system



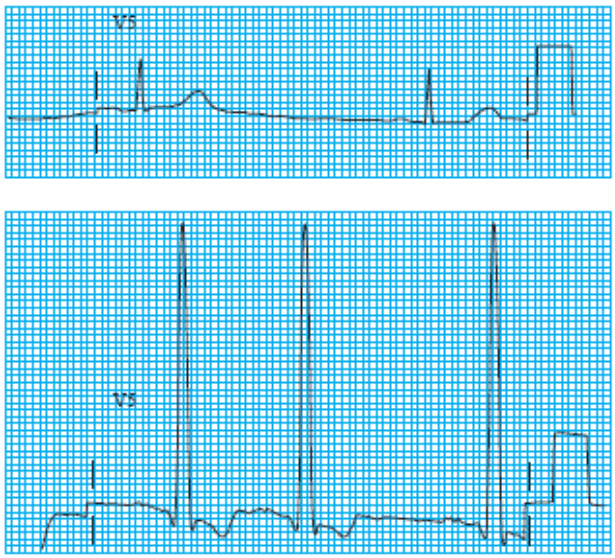

Standard calibration signal

**Throughout this article the duration of waveforms will be expressed as 0.04 s = 1 mm = 1 small square**

The electrical activity detected by the electrocardiogram machine is measured in millivolts. Machines are calibrated so that a signal with an amplitude of 1 mV moves the recording stylus vertically 1 cm. Throughout this text, the amplitude of waveforms will be expressed as: 0.1 mV = 1 mm = 1 small square.

The amplitude of the waveform recorded in any lead may be influenced by the myocardial mass, the net vector of depolarisation, the thickness and properties of the intervening tissues, and the distance between the electrode and the myocardium. Patients with ventricular hypertrophy have a relatively large myocardial mass and are therefore likely to have high amplitude waveforms. In the presence of pericardial fluid, pulmonary emphysema, or obesity, there is increased resistance to current flow, and thus waveform amplitude is reduced.

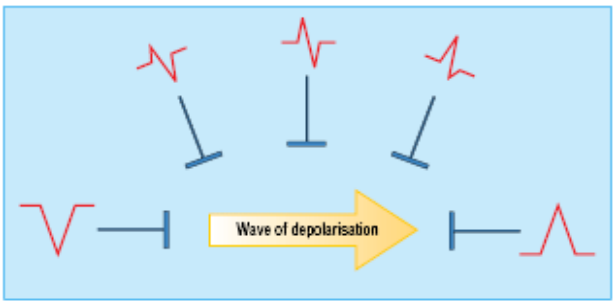
The direction of the deflection on the electrocardiogram depends on whether the electrical impulse is travelling towards or away from a detecting electrode. By convention, an electrical impulse travelling directly towards the electrode produces an upright ("positive") deflection relative to the isoelectric baseline,



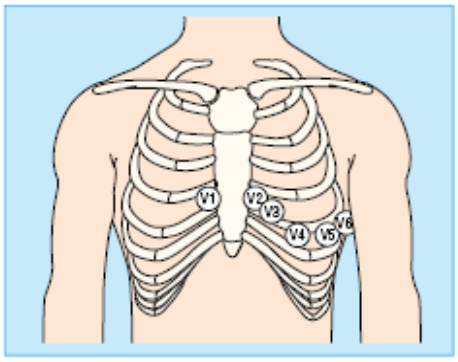

Role of body habitus and disease on the amplitude of the QRS complex. Top: Low amplitude complexes in an obese woman with hypothyroidism. Bottom: High amplitude complexes in a hypertensive man

whereas an impulse moving directly away from an electrode produces a downward ("negative") deflection relative to the baseline. When the wave of depolarisation is at right angles to the lead, an equiphasic deflection is produced.

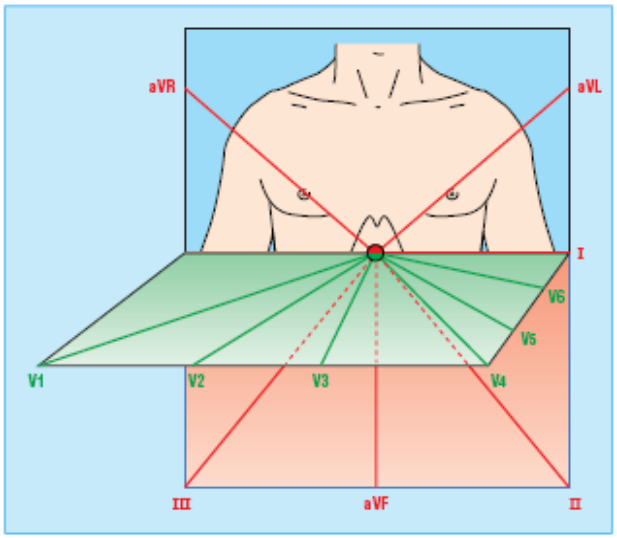
The six chest leads (V1 to V6) "view" the heart in the horizontal plane. The information from the limb electrodes is combined to produce the six limb leads (I, II, III, aVR, aVL, and aVF), which view the heart in the vertical plane. The information from these 12 leads is combined to form a standard electrocardiogram.




Wave of depolarisation. Shape of QRS complex in any lead depends on orientation of that lead to vector of depolarisation




Position of the six chest electrodes for standard 12 lead electrocardiography. V1: right sternal edge, 4th intercostal space; V2: left sternal edge, 4th intercostal space; V3: between V2 and V4; V4: mid-clavicular line, 5th space; V5: anterior axillary line, horizontally in line with V4; V6: mid-axillary line, horizontally in line with V4




Vertical and horizontal perspective of the leads. The limb leads "view" the heart in the vertical plane and the chest leads in the horizontal plane

The arrangement of the leads produces the following anatomical relationships: leads II, III, and aVF view the inferior surface of the heart; leads V1 to V4 view the anterior surface; leads I, aVL, V5, and V6 view the lateral surface; and leads V1 and aVR look through the right atrium directly into the cavity of the left ventricle.

**Anatomical relations of leads in a standard 12 lead electrocardiogram**

II, III, and aVF: inferior surface of the heart
V1 to V4: anterior surface
I, aVL, V5, and V6: lateral surface
V1 and aVR: right atrium and cavity of left ventricle

## Rate

The term tachycardia is used to describe a heart rate greater than 100 beats/min. A bradycardia is defined as a rate less than 60 beats/min (or < 50 beats/min during sleep).

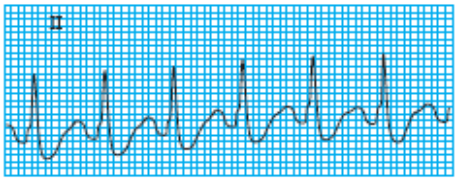
One large square of recording paper is equivalent to 0.2 seconds; there are five large squares per second and 300 per minute. Thus when the rhythm is regular and the paper speed is running at the standard rate of 25 mm/s, the heart rate can be calculated by counting the number of large squares between two consecutive R waves, and dividing this number into 300. Alternatively, the number of small squares between two consecutive R waves may be divided into 1500.

Some countries use a paper speed of 50 mm/s as standard; the heart rate is calculated by dividing the number of large squares between R waves into 600, or the number of small squares into 3000.

"Rate rulers" are sometimes used to calculate heart rate; these are used to measure two or three consecutive R-R intervals, of which the average is expressed as the rate equivalent.

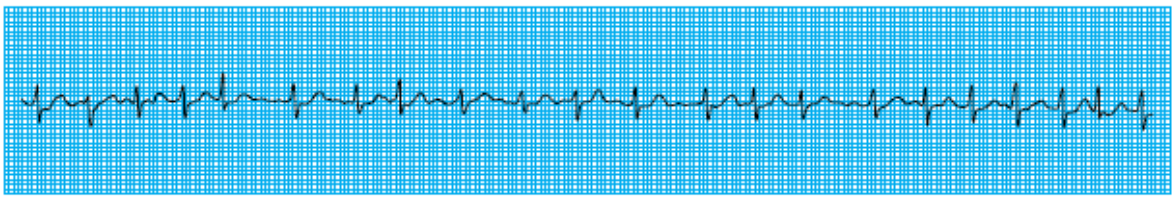
When using a rate ruler, take care to use the correct scale according to paper speed (25 or 50 mm/s); count the correct numbers of beats (for example, two or three); and restrict the technique to regular rhythms.

**Waveforms mentioned in this article (for example, QRS complex, R wave, P wave) are explained in the next article**




Regular rhythm: the R-R interval is two large squares. The rate is 150 beats/min (300/2=150)

When an irregular rhythm is present, the heart rate may be calculated from the rhythm strip (see next section). It takes one second to record 2.5 cm of trace. The heart rate per minute can be calculated by counting the number of intervals between QRS complexes in 10 seconds (namely, 25 cm of recording paper) and multiplying by six.



A standard rhythm strip is 25 cm long (that is, 10 seconds). The rate in this strip (showing an irregular rhythm with 21 intervals) is therefore 126 beats/min (6×21). Scale is slightly reduced here

## Rhythm

To assess the cardiac rhythm accurately, a prolonged recording from one lead is used to provide a rhythm strip. Lead II, which usually gives a good view of the P wave, is most commonly used to record the rhythm strip.

The term "sinus rhythm" is used when the rhythm originates in the sinus node and conducts to the ventricles.

Young, athletic people may display various other rhythms, particularly during sleep. Sinus arrhythmia is the variation in the heart rate that occurs during inspiration and expiration. There is "beat to beat" variation in the R-R interval, the rate increasing with inspiration. It is a vagally mediated response to the increased volume of blood returning to the heart during inspiration.

**Cardinal features of sinus rhythm**

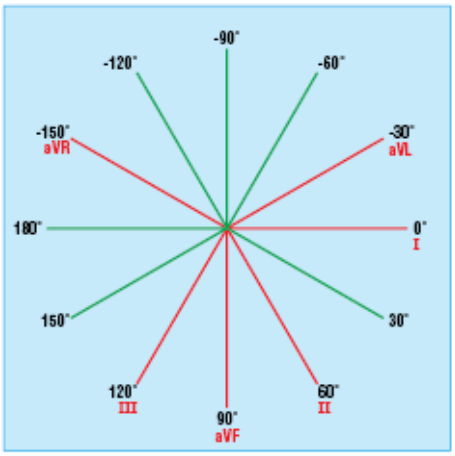
- The P wave is upright in leads I and II
- Each P wave is usually followed by a QRS complex
- The heart rate is 60-99 beats/min

**Normal findings in healthy individuals**

- Tall R waves
- Prominent U waves
- ST segment elevation (high-take off, benign early repolarisation)
- Exaggerated sinus arrhythmia
- Sinus bradycardia
- Wandering atrial pacemaker
- Wenckebach phenomenon
- Junctional rhythm
- 1st degree heart block

## Cardiac axis

The cardiac axis refers to the mean direction of the wave of ventricular depolarisation in the vertical plane, measured from a zero reference point. The zero reference point looks at the heart from the same viewpoint as lead I. An axis lying above this line is given a negative number, and an axis lying below the line is given a positive number. Theoretically, the cardiac axis may lie anywhere between 180 and −180°. The normal range for the cardiac axis is between −30° and 90°. An axis lying beyond −30° is termed left axis deviation, whereas an axis >90° is termed right axis deviation.




Hexaxial diagram (projection of six leads in vertical plane) showing each lead's view of the heart

**Conditions for which determination of the axis is helpful in diagnosis**

- Conduction defects—for example, left anterior hemiblock
- Ventricular enlargement—for example, right ventricular hypertrophy
- Broad complex tachycardia—for example, bizarre axis suggestive of ventricular origin
- Congenital heart disease—for example, atrial septal defects
- Pre-excited conduction—for example, Wolff-Parkinson-White syndrome
- Pulmonary embolus

Several methods can be used to calculate the cardiac axis, though occasionally it can prove extremely difficult to determine. The simplest method is by inspection of leads I, II, and III.

**Calculating the cardiac axis**

| | Normal axis | Right axis deviation | Left axis deviation |
|---|---|---|---|
| Lead I | Positive | Negative | Positive |
| Lead II | Positive | Positive or negative | Negative |
| Lead III | Positive or negative | Positive | Negative |

A more accurate estimate of the axis can be achieved if all six limb leads are examined. The hexaxial diagram shows each lead's view of the heart in the vertical plane. The direction of current flow is towards leads with a positive deflection, away from leads with a negative deflection, and at 90° to a lead with an equiphasic QRS complex. The axis is determined as follows:

- Choose the limb lead closest to being equiphasic. The axis lies about 90° to the right or left of this lead
- With reference to the hexaxial diagram, inspect the QRS complexes in the leads adjacent to the equiphasic lead. If the lead to the left side is positive, then the axis is 90° to the equiphasic lead towards the left. If the lead to the right side is positive, then the axis is 90° to the equiphasic lead towards the right.

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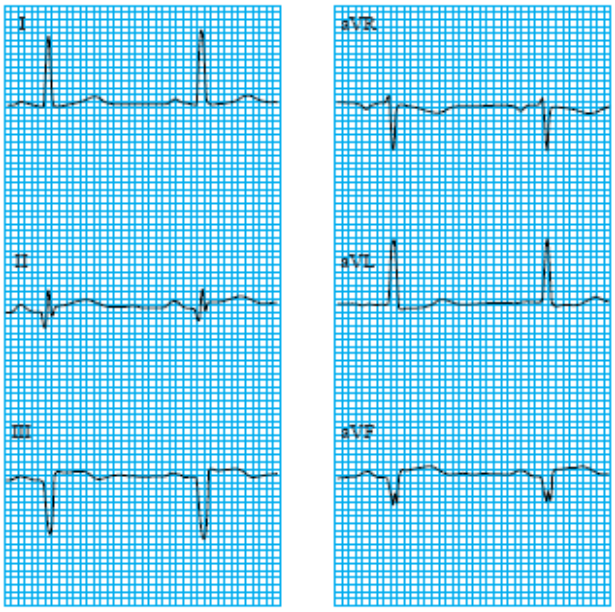
*BMJ* 2002;324:415–8




**Determination of cardiac axis using the hexaxial diagram (see previous page). Lead II (60°) is almost equiphasic and therefore the axis lies at 90° to this lead (that is 150° to the right or −30° to the left). Examination of the adjacent leads (leads I and III) shows that lead I is positive. The cardiac axis therefore lies at about −30°**

The ABC of clinical electrocardiography is edited by Francis Morris, consultant in emergency medicine at the Northern General Hospital, Sheffield; June Edhouse, consultant in emergency medicine, Stepping Hill Hospital, Stockport; William J Brady, associate professor, programme director, and vice chair, department of emergency medicine, University of Virginia, Charlottesville, VA, USA; and John Camm, professor of clinical cardiology, St George's Hospital Medical School, London. The series will be published as a book in the summer.

## The emperor's pointer

At first I thought it was just me. I was attending my first scientific meeting, a young doctor eager to acquire new knowledge. It was clear that the first speaker had firmly embraced the concept of the PowerPoint presentation, and he was treating his audience to beautifully coloured slides. Like so many of us, he tended to put just a little too much information on each slide. One of his slides showed a comparison between groups, and we were promised that the third group would differ dramatically from the other two. I saw two lines cross the slide, one close to the other, but no third line. I looked at the key at the bottom of the graph and again saw no more than two groups. I was still waiting for a sudden and victorious appearance of the third group on the graph when the speaker turned to the next slide as if nothing had happened.

At another point, he showed us a pictogram of a human cell that was engaging in complex metabolic activity, with multiple schematics of receptors, proton pumps, and mitochondrial pathways. A small detail of this cell's activity was apparently of great interest to the speaker, for he was enthusiastically aiming his laser pointer at it. I waited for the dot or arrow to appear on the slide, but nothing happened. "Turn it on," I thought, but instead he went on to the next slide, leaving me in the dark about what had been so interesting in that cell. A similar thing occurred a few slides later, but no one in the audience seemed to bother to tell the speaker to turn on his magical pointing device. It had to be me, then. Apparently, his laser pointer was invisible to me, as were some of his wonderfully coloured lines and bars and legends. I couldn't see the emperor's clothes.

And then it dawned on me: I was a man. And what are men, at least some men? Yes, they are colour blind. Presentation after presentation, I have failed to see the highlights in so many slides. And even this trusted journal joins in the conspiracy. For no particular reason, some issues appear without a date on the cover, such as the first issue in November 2001. Or is it that sometimes, some parts of this journal are invisible to me?

But I can't be alone. Suppose I'm at a large international meeting with an audience of 1500 people, of whom 1000 are male. About 70 of them will be colour blind and therefore not able to see the little red dot or arrow being pointed at that interesting red line. There may be even more, because it seems that a lot of men who once had dreams of becoming a pilot but were turned down because of colour blindness have become doctors.

What can we do about this? Using a big flashing yellow arrow might help; or, as in *Wheel of Fortune*, using a female assistant to point out the area of interest on a slide with a cane (she herself can be guided by the speaker using his pointer); or, perhaps, just putting less data on each slide.

Jurgen A H R Claassen *registrar in geriatric medicine, UMC St Radboud, Nijmegen, Netherlands*

We welcome articles up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake*, or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk. Permission is needed from the patient or a relative if an identifiable patient is referred to.

# *ABC of clinical electrocardiography*
# Introduction. II—Basic terminology

Steve Meek, Francis Morris

This article explains the genesis of and normal values for the individual components of the wave forms that are seen in an electrocardiogram. To recognise electrocardiographic abnormalities the range of normal wave patterns must be understood.

## P wave

The sinoatrial node lies high in the wall of the right atrium and initiates atrial depolarisation, producing the P wave on the electrocardiogram. Although the atria are anatomically two distinct chambers, electrically they act almost as one. They have relatively little muscle and generate a single, small P wave. P wave amplitude rarely exceeds two and a half small squares (0.25 mV). The duration of the P wave should not exceed three small squares (0.12 s).

The wave of depolarisation is directed inferiorly and towards the left, and thus the P wave tends to be upright in leads I and II and inverted in lead aVR. Sinus P waves are usually most prominently seen in leads II and V1. A negative P wave in lead I may be due to incorrect recording of the electrocardiogram (that is, with transposition of the left and right arm electrodes), dextrocardia, or abnormal atrial rhythms.

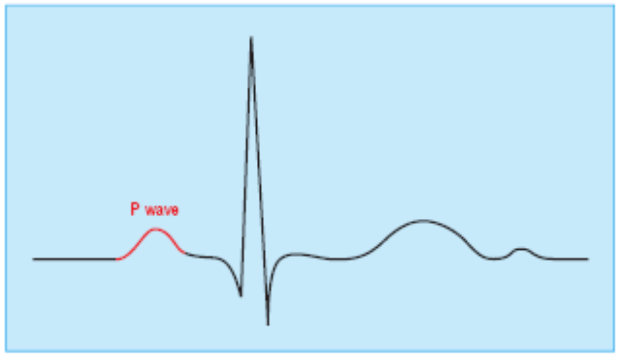
P waves are usually more obvious in lead II than in lead I

The P wave in V1 is often biphasic. Early right atrial forces are directed anteriorly, giving rise to an initial positive deflection; these are followed by left atrial forces travelling posteriorly, producing a later negative deflection. A large negative deflection (area of more than one small square) suggests left atrial enlargement.

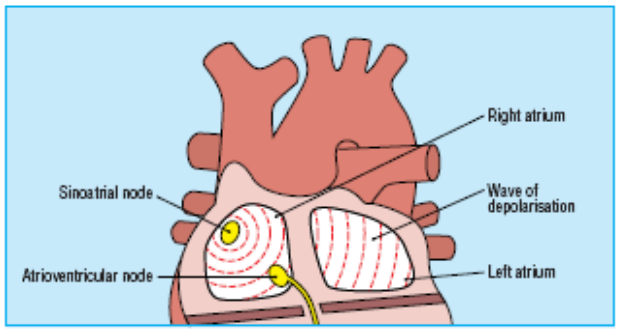
Normal P waves may have a slight notch, particularly in the precordial (chest) leads. Bifid P waves result from slight asynchrony between right and left atrial depolarisation. A pronounced notch with a peak-to-peak interval of > 1 mm (0.04 s) is usually pathological, and is seen in association with a left atrial abnormality—for example, in mitral stenosis.

## PR interval

After the P wave there is a brief return to the isoelectric line, resulting in the "PR segment." During this time the electrical impulse is conducted through the atrioventricular node, the bundle of His and bundle branches, and the Purkinje fibres.



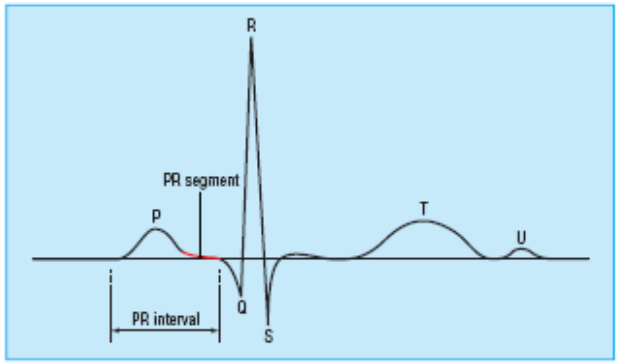

Complex showing P wave highlighted




Atrial depolarisation gives rise to the P wave

**Characteristics of the P wave**

- Positive in leads I and II
- Best seen in leads II and V1
- Commonly biphasic in lead V1
- < 3 small squares in duration
- < 2.5 small squares in amplitude




Normal duration of PR interval is 0.12-0.20 s (three to five small squares)

The PR interval is the time between the onset of atrial depolarisation and the onset of ventricular depolarisation, and it is measured from the beginning of the P wave to the first deflection of the QRS complex (see next section), whether this be a Q wave or an R wave. The normal duration of the PR interval is three to five small squares (0.12-0.20 s). Abnormalities of the conducting system may lead to transmission delays, prolonging the PR interval.

## QRS complex

The QRS complex represents the electrical forces generated by ventricular depolarisation. With normal intraventricular conduction, depolarisation occurs in an efficient, rapid fashion. The duration of the QRS complex is measured in the lead with the widest complex and should not exceed two and a half small squares (0.10 s). Delays in ventricular depolarisation—for example, bundle branch block—give rise to abnormally wide QRS complexes ($\geq$0.12 s).

The depolarisation wave travels through the interventricular septum via the bundle of His and bundle branches and reaches the ventricular myocardium via the Purkinje fibre network. The left side of the septum depolarises first, and the impulse then spreads towards the right. Lead V1 lies immediately to the right of the septum and thus registers an initial small positive deflection (R wave) as the depolarisation wave travels towards this lead.

When the wave of septal depolarisation travels away from the recording electrode, the first deflection inscribed is negative. Thus small "septal" Q waves are often present in the lateral leads, usually leads I, aVL, V5, and V6.

These non-pathological Q waves are less than two small squares deep and less than one small square wide, and should be $<$25% of the amplitude of the corresponding R wave.

The wave of depolarisation reaches the endocardium at the apex of the ventricles, and then travels to the epicardium, spreading outwards in all directions. Depolarisation of the right and left ventricles produces opposing electrical vectors, but the left ventricle has the larger muscle mass and its depolarisation dominates the electrocardiogram.

In the precordial leads, QRS morphology changes depending on whether the depolarisation forces are moving towards or away from a lead. The forces generated by the free wall of the left ventricle predominate, and therefore in lead V1 a small R wave is followed by a large negative deflection (S wave). The R wave in the precordial leads steadily increases in amplitude from lead V1 to V6, with a corresponding decrease in S wave depth, culminating in a predominantly positive complex in V6. Thus, the QRS complex gradually changes from being predominantly negative in lead V1 to being predominantly positive in lead V6. The lead with an equiphasic QRS complex is located over the transition zone; this lies between leads V3 and V4, but shifts towards the left with age.

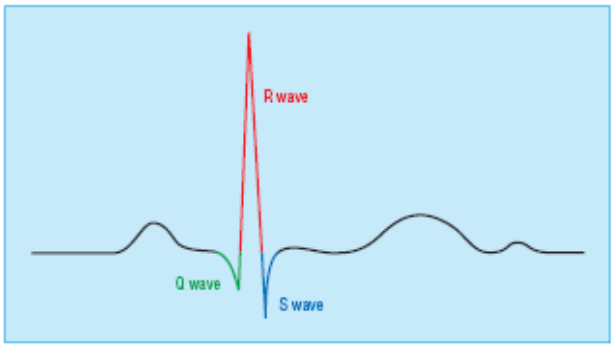
The height of the R wave is variable and increases progressively across the precordial leads; it is usually $<$ 27 mm in leads V5 and V6. The R wave in lead V6, however, is often smaller than the R wave in V5, since the V6 electrode is further from the left ventricle.

The S wave is deepest in the right precordial leads; it decreases in amplitude across the precordium, and is often absent in leads V5 and V6. The depth of the S wave should not exceed 30 mm in a normal individual, although S waves and R waves $>$30 mm are occasionally recorded in normal young male adults.

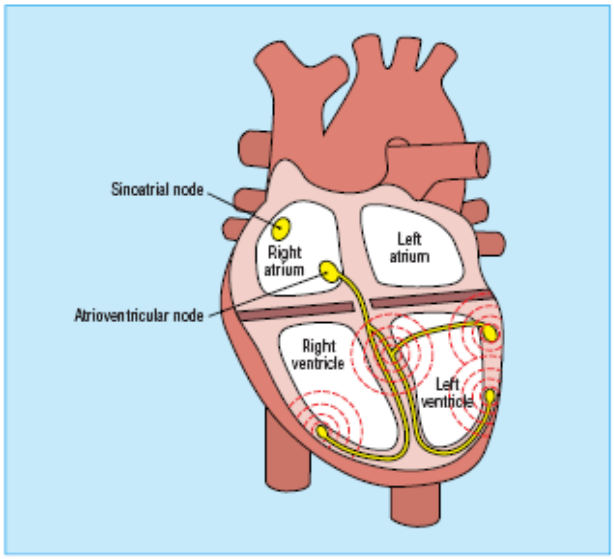
**Nomenclature in QRS complexes**

*Q wave:* Any initial negative deflection
*R wave:* Any positive deflection
*S wave:* Any negative deflection after an R wave

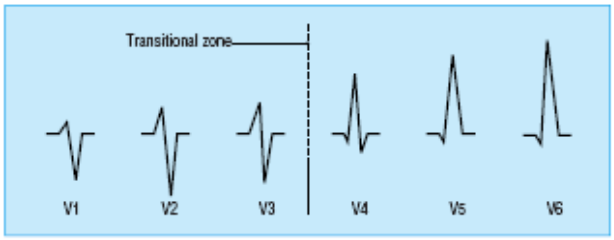
**Non-pathological Q waves are often present in leads I, III, aVL, V5, and V6**




Composition of QRS complex




Wave of depolarisation spreading throughout ventricles gives rise to QRS complex




Typical change in morphology of QRS complex from leads V1 to V6

## ST segment

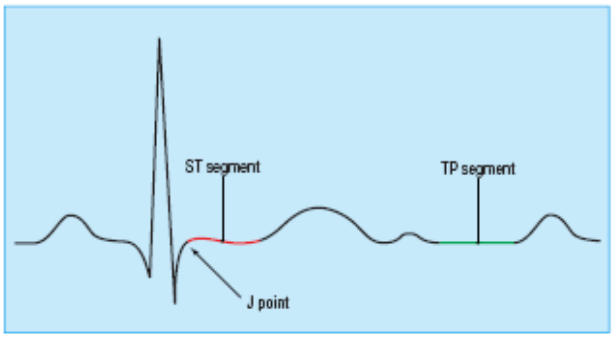
The QRS complex terminates at the J point or ST junction. The ST segment lies between the J point and the beginning of the T wave, and represents the period between the end of ventricular depolarisation and the beginning of repolarisation.

The ST segment should be level with the subsequent "TP segment" and is normally fairly flat, though it may slope upwards slightly before merging with the T wave.

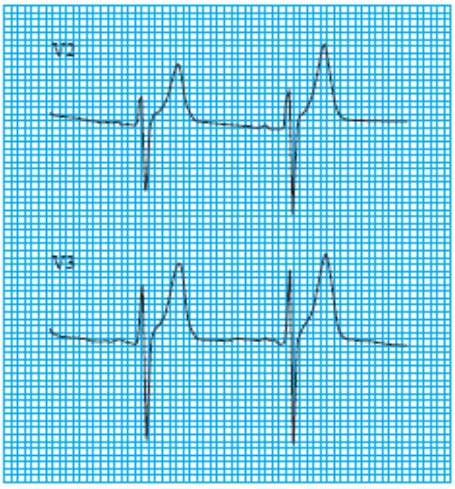
In leads V1 to V3 the rapidly ascending S wave merges directly with the T wave, making the J point indistinct and the ST segment difficult to identify. This produces elevation of the ST segment, and this is known as "high take-off."

Non-pathological elevation of the ST segment is also associated with benign early repolarisation (see article on acute myocardial infarction later in the series), which is particularly common in young men, athletes, and black people.

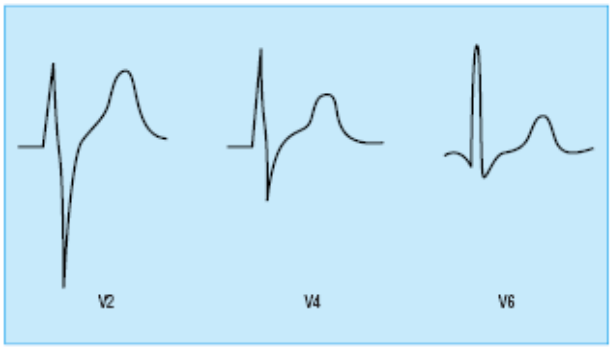
Interpretation of subtle abnormalities of the ST segment is one of the more difficult areas of clinical electrocardiography; nevertheless, any elevation or depression of the ST segment must be explained rather than dismissed.




The ST segment should be in the same horizontal plane as the TP segment; the J point is the point of inflection between the S wave and ST segment




Complexes in leads V2 and V3 showing high take-off




Change in ST segment morphology across the precordial leads

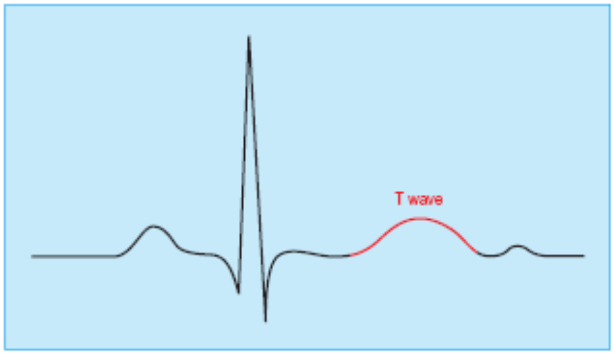
**The T wave should generally be at least 1/8 but less than 2/3 of the amplitude of the corresponding R wave; T wave amplitude rarely exceeds 10 mm**

## T wave

Ventricular repolarisation produces the T wave. The normal T wave is asymmetrical, the first half having a more gradual slope than the second half.

T wave orientation usually corresponds with that of the QRS complex, and thus is inverted in lead aVR, and may be inverted in lead III. T wave inversion in lead V1 is also common. It is occasionally accompanied by T wave inversion in lead V2, though isolated T wave inversion in lead V2 is abnormal. T wave inversion in two or more of the right precordial leads is known as a persistent juvenile pattern; it is more common in black people. The presence of symmetrical, inverted T waves is highly suggestive of myocardial ischaemia, though asymmetrical inverted T waves are frequently a non-specific finding.

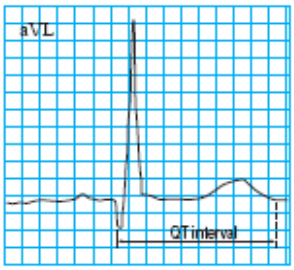
No widely accepted criteria exist regarding T wave amplitude. As a general rule, T wave amplitude corresponds with the amplitude of the preceding R wave, though the tallest T waves are seen in leads V3 and V4. Tall T waves may be seen in acute myocardial ischaemia and are a feature of hyperkalaemia.




Complex showing T wave highlighted

## QT interval

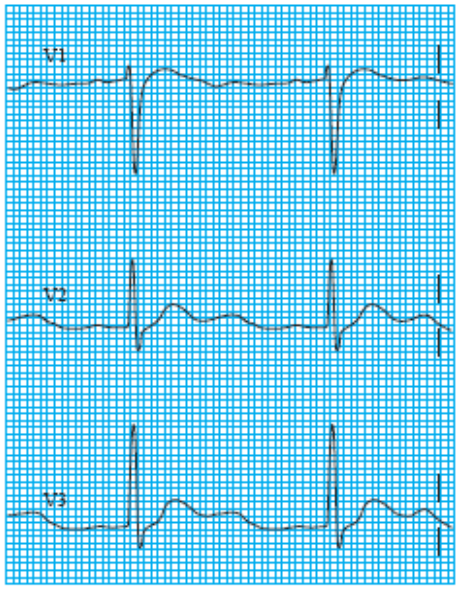
The QT interval is measured from the beginning of the QRS complex to the end of the T wave and represents the total time taken for depolarisation and repolarisation of the ventricles.




The QT interval is measured in lead aVL as this lead does not have prominent U waves (diagram is scaled up)

The QT interval lengthens as the heart rate slows, and thus when measuring the QT interval the rate must be taken into account. As a general guide the QT interval should be 0.35-0.45 s, and should not be more than half of the interval between adjacent R waves (R-R interval). The QT interval increases slightly with age and tends to be longer in women than in men. Bazett's correction is used to calculate the QT interval corrected for heart rate (QTc): $QTc = QT/\sqrt{R\text{-}R}$ (seconds).

Prominent U waves can easily be mistaken for T waves, leading to overestimation of the QT interval. This mistake can be avoided by identifying a lead where U waves are not prominent—for example, lead aVL.




Obvious U waves in leads V1 to V3 in patient with hypokalaemia

## U wave

The U wave is a small deflection that follows the T wave. It is generally upright except in the aVR lead and is often most prominent in leads V2 to V4. U waves result from repolarisation of the mid-myocardial cells—that is, those between the endocardium and the epicardium—and the His-Purkinje system.

Many electrocardiograms have no discernible U waves. Prominent U waves may be found in athletes and are associated with hypokalaemia and hypercalcaemia.

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The ABC of clinical electrocardiography is edited by Francis Morris, consultant in emergency medicine at the Northern General Hospital, Sheffield; June Edhouse, consultant in emergency medicine, Stepping Hill Hospital, Stockport; William J Brady, associate professor, programme director, and vice chair, department of emergency medicine, University of Virginia, Charlottesville, VA, USA; and John Camm, professor of clinical cardiology, St George's Hospital Medical School, London. The series will be published as a book in the summer.

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### "A little white tablet, doctor"

Few doctors will not recognise this reply from patients asked to recall their medication. After a similarly vague history of presenting complaint, a persistent clinician might try to narrow down what the tablet is prescribed for, at least, though often (and alarmingly perhaps) this is often a fruitless exercise. There can be occasions, however, when the colour is the key after all.

When I was a senior house officer working in accident and emergency I was asked to take a telephone call from a patient wanting advice. He explained that he had gone to the high street pharmacy to collect a repeat prescription for his "rat poison." On returning home, he was surprised to see that he seemed to have been given a different brand from his normal one, and he was now unsure what dose to take. I asked him what he normally took:

"One blue and one brown tablet, doctor."

I calculated this as 4 mg of warfarin and explained that he should continue on this dose until his next check up, reassuring him that different pharmacies probably used slightly different packaging. He wasn't so sure. "But they're all the same colour, doctor," he replied.

Assuming he had probably been given a supply of 1 mg brown tablets, I asked him to describe them. "Little white tablets, doctor."

Now concerned and puzzled, I advised him to bring them to the department as he lived locally. Sure enough, in a new bottle marked "Warfarin—take as directed by your doctor" were about 50 small white tablets with a "5" embossed on them. We identified them as bendrofluazide and immediately alerted the pharmacy about the dispensing error.

Thankfully, to my knowledge, the other potentially more catastrophic half to this story never emerged—that is, the patient who agonised over which colour of his new brand of "water pills" to take first, the brown, the blue, or the pink.

Martin Turner *research fellow in neurology, King's College London.*

We welcome articles up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake,* or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk. Permission is needed from the patient or a relative if an identifiable patient is referred to. We also welcome contributions for "Endpieces," consisting of quotations of up to 80 words (but most are considerably shorter) from any source, ancient or modern, which have appealed to the reader.

# Understanding the 12-lead ECG part I

**Find how the ECG translates the heart's electrical activity into a waveform and what it tells you about your patient's condition.**

BY GUY GOLDICH, RN, CCRN, MSN

RESPONDING TO THE CALL bell, you find George Smythe, 67, sitting up in bed and complaining of chest discomfort. Mr. Smythe had a laparoscopic cholecystectomy earlier today. You take his vital signs and perform a chest pain assessment, which includes the onset, location, quality, intensity, duration, and any radiation of the discomfort. You ask about associated signs and symptoms and factors that aggravate or relieve the pain. Following your facility's protocol, you administer supplemental oxygen at 2 to 4 liters/minute via nasal cannula and page the physician on call, who orders stat serum cardiac biomarkers, a 12-lead electrocardiogram (ECG), and sublingual nitroglycerin.

Do you know what to look for to determine if Mr. Smythe's 12-lead ECG is abnormal? Could you recognize signs that he's having a myocardial infarction (MI)? If you can independently interpret a 12-lead ECG, you can anticipate and prepare for the emergency care your patient may need.

In this article, I'll cover the basics of 12-lead ECG interpretation, focusing on a normal ECG. Next month, I'll discuss ECG abnormalities.

## What's happening in the heart

The heart's internal conduction circuit initiates each heartbeat and coordinates all parts of the heart to contract at the proper time. A normal heartbeat is initiated in the sinoatrial (SA) node, a specialized group of cells in the right atrium. The SA node depolarizes at a rate of 60 to 100 times/minute, causing the atria to contract and propel blood into the ventricles.

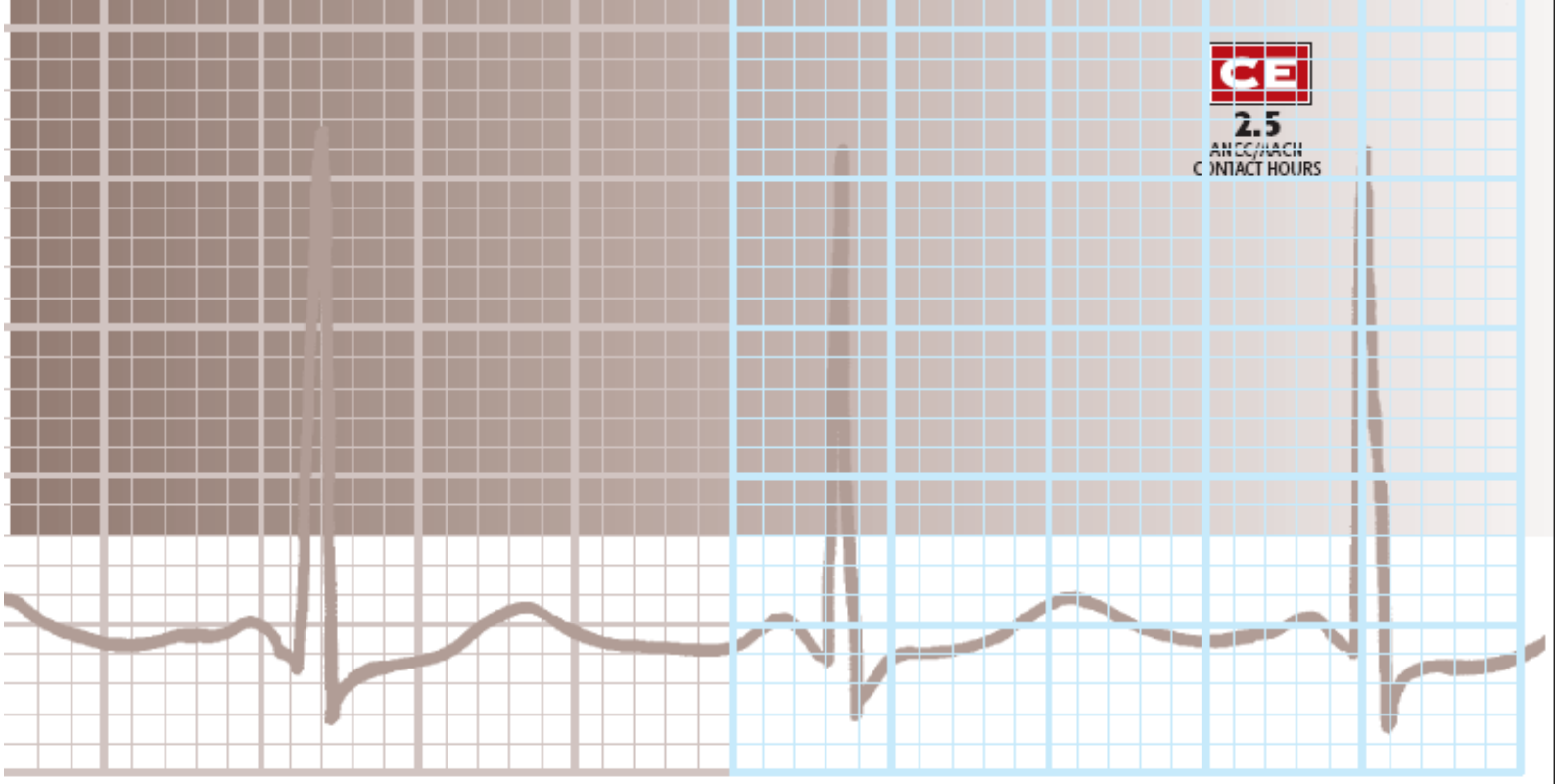
Atrial depolarization produces the first element on the ECG waveform: the *P wave*. The P wave is the first part of the cardiac cycle and appears as a small, semicircular bump (see *Tracing a normal ECG waveform*).

The wave of depolarization continues through the atria until it encounters the next important structure, the *atrioventricular (AV) node*. The AV node receives the atrial impulse and (after a brief pause to let the ventricles fill) transmits it to the ventricles via the *bundle of His*. A collection of cardiac conduction fibers, the bundle of His splits into the right and left *bundle branches*.

The bundle branches are high-speed conducting fibers that run down the intraventricular septum and transmit the cardiac impulse to the *Purkinje fibers*. These fibers form a complex network that mingles with ventricular myocardial cells. The function of the Purkinje fibers is to rapidly stimulate ventricular muscle fibers, resulting in the next major event in the cardiac cycle: *ventricular depolarization*.

Ventricular depolarization generates the *QRS complex*, the electrical equivalent of ventricular systole. (Remember that electrical activity precedes mechanical activity, and the ECG shows only electrical activity.) If you palpate a carotid or radial pulse while looking at a cardiac monitor, you should feel a pulse with each QRS complex on the monitor.

The QRS complex normally has a duration of 0.06 to 0.1 second. A duration greater than 0.12 second




usually indicates prolonged ventricular conduction caused by a bundle-branch block. The QRS complex is variable in appearance and may have a different shape (or morphology) in different patients or even look different in various ECG leads in the same patient. The QRS complex may have one, two, or three wave components, depending on the lead and your patient's condition.

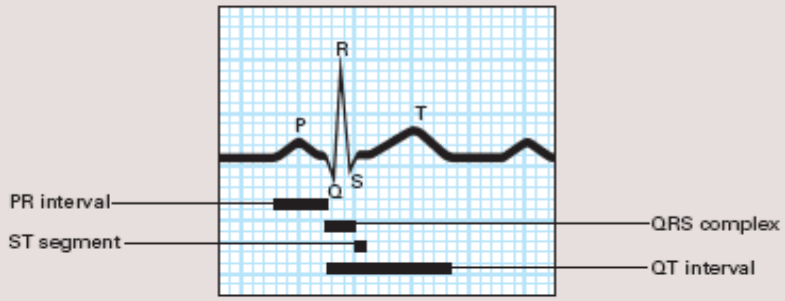
The last major wave component of the ECG is the *T wave*, which is larger than the P wave and rounded or slightly peaked. Immediately following the QRS complex, it represents ventricular repolarization or a metabolic rest period between heartbeats. During repolarization, electrolytes such as potassium, sodium, and calcium cross the cell membrane (back to their original location) to prepare the cardiac cell for the next depolarization.

Besides the three waveforms, the normal ECG cardiac cycle tracing has two important *segments*, or flat (isoelectric) parts of the tracing between the waveforms: the *PR interval* and the *ST segment*.

The PR interval is the period from the beginning of the P wave to the beginning of the QRS complex. It consists of the P wave plus the short isoelectric segment that terminates at the start of the QRS complex. The normal PR interval lasts 0.12 to 0.2 second; this represents the time from SA node depolarization to ventricular depolarization. If the PR interval is *less* than 0.12 second, then the cardiac impulse didn't follow the normal conduction pathway. If the PR interval is *longer* than 0.2 second, then a disease process may be affecting the cardiac conduction pathway, keeping it from functioning properly.

The ST segment consists of the isoelectric line between the end of the QRS complex and the beginning of the T wave. The ST segment reveals information about the heart's oxygenation status. For example, myocardial ischemia (a temporary, reversible decrease in oxygenation) often results in an ST segment below the baseline of the ECG tracing. When myocardial cells are injured (reversible physical damage from lack of oxygen), the ST segment often is elevated above the baseline. So ST-segment elevations are a key indicator of MI. I'll discuss this in detail in the next part of this series. For tips on how to use the ECG to calculate heart rates and more, see *Paper training*.

**Tracing a normal ECG waveform**




## Catching the wave

If you examine a 12-lead ECG, you'll notice that some QRS com-

plexes have upward deflections and others have downward deflections. Here's why.

Each ECG lead has a positive (or sensing) electrode and a negative electrode, which acts as an anchor. The positive electrode looks toward its negative electrode and senses whether electrical energy is being directed toward or away from the positive electrode.

When electrical energy is directed *toward* the positive monitoring electrode, the QRS complex has an upward deflection. When the electrical energy is directed *away* from the positive monitoring electrode, the QRS complex has a downward deflection. The more directly aligned the direction of the electrical energy with the positive electrode, the more upright the complex. If the electrical energy approaches the positive monitoring electrode at a glancing angle, the complex will still be upright, but less upright than if the energy were directly aligned with the positive electrode.

Energy arriving at a perpendicular angle to the positive electrode results in either a waveform with little deflection (isoelectric) or equal amounts of positive and negative deflection.

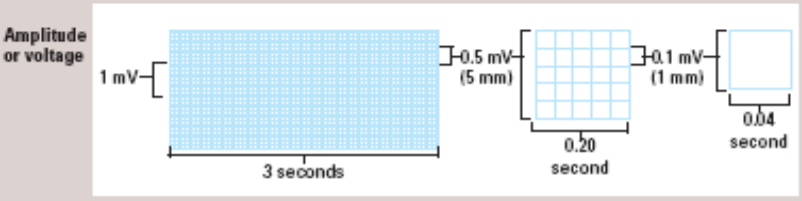
As the energy is directed away from the positive electrode, the QRS complex becomes progressively more negative. When energy flow is directed totally away from the positive electrode, the QRS complex is deflected directly downward.

## Paper training

You can use the markings on ECG paper to calculate events within the cardiac cycle. The ECG paper is a grid of large and small blocks. On the horizontal axis, a large block is equal to 0.2 second and a small block is equal to 0.04 second. The vertical axis represents voltage or electrical energy, with each vertical millimeter (small block) being 0.1 millivolt of electrical energy. However, in practice, deflections are typically described as being in millimeters, not millivolts.

By counting the number of small squares and multiplying by 0.04, you can calculate the duration of any event in the ECG tracing. A QRS complex that's 2.5 small squares wide is 0.1 second. You also can use the ECG paper to calculate heart rates, using one of two methods. In the 6-second method, you start by looking for the markings (usually short vertical lines) at the top of the rhythm strip or ECG paper. These markings divide the ECG paper into 3-second intervals. Count the number of QRS complexes contained in two intervals (6 seconds) and multiply by 10. This method works for both regular and irregular heart rhythms.

In the division method, count the number of small squares between any two heartbeats. Make sure you use the same part in both QRS complexes—usually the peak of the complex works the best. Divide 1,500 by the number of small squares and you'll have the heart rate in beats per minute. This method is accurate only with regular heart rates because irregular heart rhythms have a varying number of small squares between any two QRS complexes.




## Going with the flow: A look at vectors

All cardiac cells are electrochemical, meaning they generate electrical energy during depolarization. This electrical energy, called a *vector*, has strength (measured in millivolts) and direction (measured in degrees from an arbitrary zero point called the electrical axis). Each cardiac cell generates its own microvector. The mathematical average of these microvectors is the *mean QRS vector* or *mean vector*, which follows the conduction pathway of the heart—downward and to the left. The mean vector flows slightly to the left of the ventricular septum because the left ventricle has more and larger cardiac cells.

Generally, each person has a unique mean vector direction, which remains constant unless his cardiac status changes. For example, left ventricular hypertrophy secondary to heart failure pulls the mean vector even more sharply to the left side.[1] A person who has a mean vector in an abnormal direction is said to have an *axis deviation*. (For details, see *Axis deviation: As easy as pie (charts)*.)

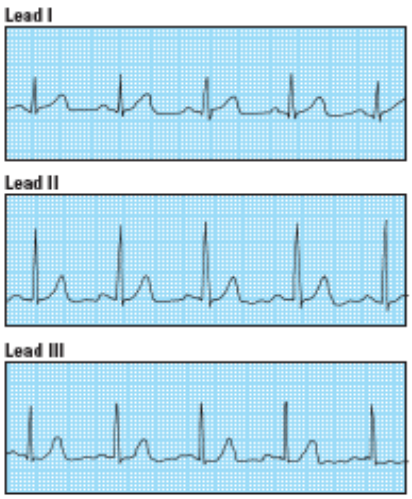
## Putting it all together

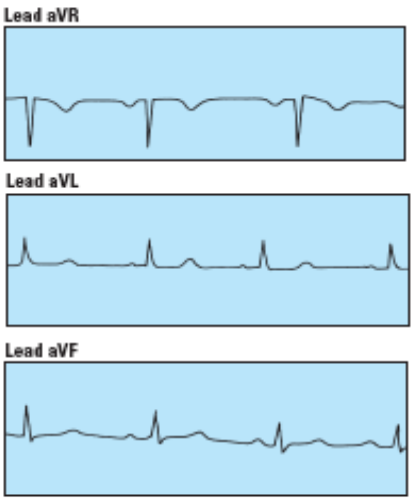
The mean vector is a representation of the overall electrical properties of the heart. A 12-lead ECG is the electrical record of the mean vector from 12 different monitoring sites (leads) on the surface of the body. As when you look at any object, you need to see all the angles to get a complete picture.

## Looking at limb leads

The first six leads of the 12-lead ECG come from four electrodes placed on the patient's arms and legs; the right lower leg electrode is the ground electrode. The limb leads record the mean vector in the up-down and left-right direction along the body's frontal plane. Because they use separate positive and negative electrodes, they're called bipolar or standard leads.

- **Lead I** has the positive electrode on the left arm and looks toward the negative electrode on the right arm for electrical energy. Because




the mean vector travels from upper right to lower left, energy flows toward the positive electrode of lead I, resulting in an upward deflection of the QRS. And because the mean vector doesn't flow directly toward lead I, but approaches it at a somewhat broad angle, the upward deflection of the QRS complex is moderate.

- In **lead II**, the positive electrode is on the left foot and the negative electrode is on the right arm. Because the mean vector flows directly at the positive lead II electrode, this lead usually has the most upright QRS complexes and the most prominent P waves of the entire 12-lead ECG. That's why lead II is a favorite monitoring lead in many intensive care and telemetry units.
- **Lead III** puts the positive electrode on the left foot and the negative one on the left arm. The mean vector flow approaches lead III downward from the right, again producing an upward QRS deflection. Because the angle is narrower than the angle between the mean vector and lead I, the lead III QRS complex is more upright than the lead I QRS complex.

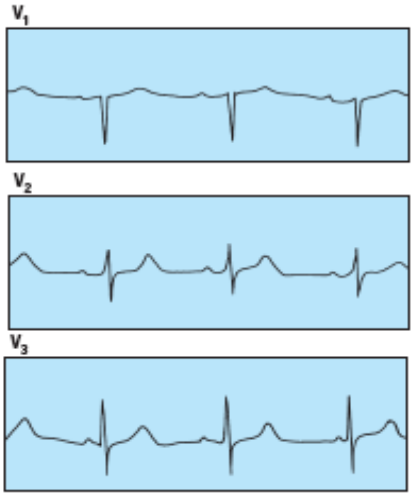
The second set of limb leads are called the augmented or unipolar leads and use a single positive monitoring electrode. The negative electrode is an electrically calculated location at the center of the heart.[2]

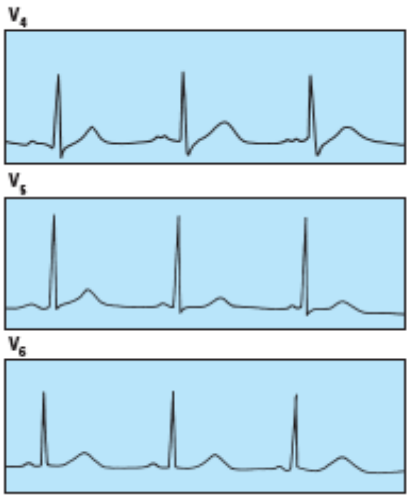
- **Lead aVR** is the only limb lead on the right side of the body. Its positive monitoring electrode is located on the right arm and looks downward and to the left. The mean vector also flows downward and to the left, directly *away* from lead aVR, resulting in a negative deflection for all waveforms. In a normal ECG, lead aVR is the only limb lead with a downwardly deflected QRS.
- **Lead aVL** positions a positive electrode on the left arm and looks to the right and *downward* toward to the center of the heart (in contrast to lead I, which looks strictly to the right). The mean vector approaches aVL at a very broad angle, producing the least upright QRS complex among the limb leads.
- **Lead aVF** has its positive monitoring lead on the left leg and looks straight up to the center of the chest. The mean vector approaches aVF at a fairly direct angle, although not as directly as lead II, so lead aVF has very upright QRS complexes with prominent P waves. Because leads II, III, and aVF all look upward at the oncoming mean vector, their waveforms share many qualities, such as highly positive QRS complexes and prominent P waves. Because these leads look upward at the bottom or inferior ventricular wall of the heart, they're known as the inferior leads.

### Six chest leads weigh in

The six chest or precordial leads lie across the anterior chest and measure the mean vector in the horizontal plane.

- **Lead $V_1$** is located at the right sternal border, fourth intercostal space, and lies above the right ventricle and septum.
- **Lead $V_2$** is at the left side of the sternum, fourth intercostal space.
- **Lead $V_3$** is midway between leads $V_2$ and $V_4$.
- **Lead $V_4$** is at the midclavicular line in the fifth intercostal space.
- **Lead $V_5$** is at the anterior axillary line in the fifth intercostal space.
- **Lead $V_6$** is at the midaxillary line, fifth intercostal space, and is positioned above the lateral wall of



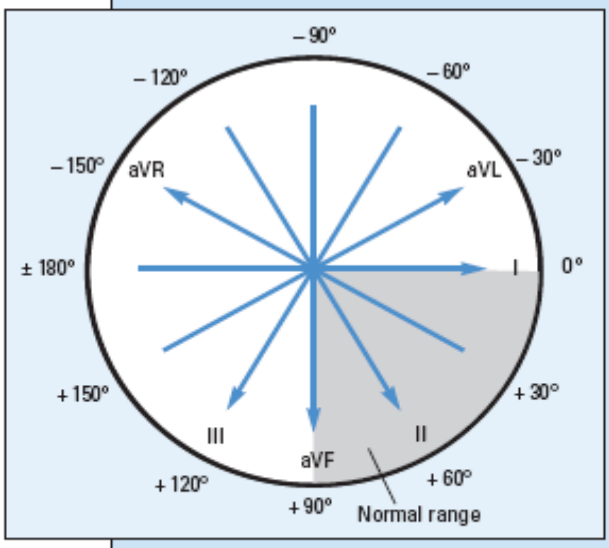



# Axis deviation: As easy as pie (charts)

Combining your assessment skills with an understanding of axis deviation can give you a more detailed picture of your patient's condition. The hexaxial reference system and the quadrant method can help you visualize problems with cardiac conduction.

## Hexaxial reference system

The normal QRS complex (or vector) represents the average electrical signal that the heart generates during depolarization. Within the heart, the mean vector generally flows from upper right to lower left. The exact direction of that flow (called the electrical axis) can be used as an assessment tool in the 12-lead ECG because an abnormal axis can give you clues about what's going wrong in the heart's electrical system.




To measure the electrical axis, imagine all six limb leads displayed simultaneously around a central point in a circle, which represents the heart (see the illustration at left). In this hexaxial system, the leads divide the circle into equal 30-degree segments.

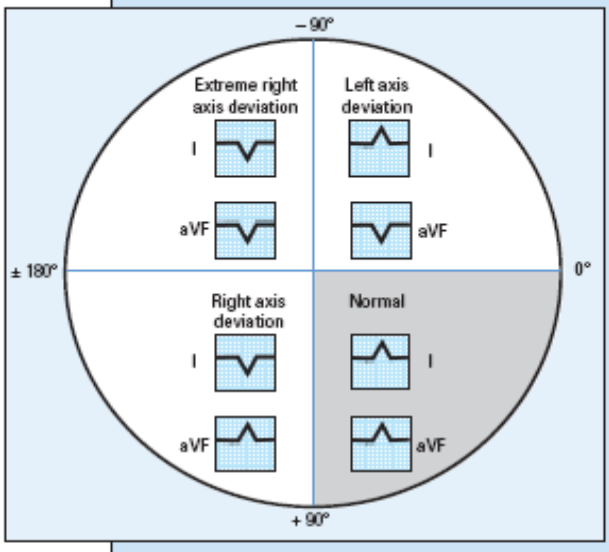
Each lead can be assigned a number of degrees, and the mean vector's direction can be given in degrees. If the mean vector is aligned directly with lead I, its axis is 0 degrees. A mean vector directed halfway between leads II and aVF has an axis of 75 degrees. (Although you can calculate your patient's electrical axis, all modern 12-lead ECG machines provide this information automatically.)

The normal electrical axis of the heart falls between -30 and +90 degrees. Although this is a wide range, it's a numeric equivalent of the concept that the electrical conduction of the normal heart is right to left and top to bottom.

A *left axis deviation* occurs when the electrical axis of the heart is between -30 and -90 degrees. A *right axis deviation* occurs when the electrical axis is in the +90-to-+180-degree range. A mean vector having an electrical axis within the range of -90 to -180 degrees is called an *indeterminate axis* or *extreme right axis deviation*.

## Quadrant method

To approximate axis deviation using the quadrant method, divide the cirde (which represents the patient's heart) into four quadrants (see the illustration below). You need only two ECG leads to make this assessment. Examine leads I and aVF. If lead I is upright, then the vector is flowing right to left. If lead aVF is upright, the vector is directed top to bottom. If they're *both* upright, the electrical axis must fall into the lower left or normal quadrant. This quadrant roughly matches the criteria for *normal electrical axis,* indicating a normal direction of electrical conduction.



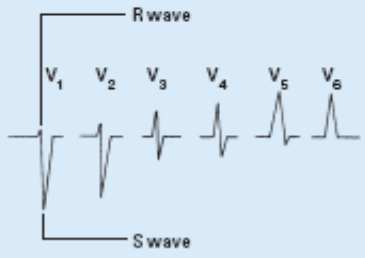

*Left axis deviation* occurs when lead I is upright and lead aVF is down or negative. The electrical axis is located in the upper right quadrant. The mean vector is abnormally directed to the left side of the heart. A left axis deviation can be caused by many different pathologic conditions. Some left bundle-branch blocks will produce a left axis deviation because the cardiac vector flows abnormally from the right side of the heart to the left. Because the mean vector is not conducted by infarcted tissue and flows away from it, an inferior-wall myocardial infarction will produce a left axis deviation (due to a negative QRS in lead aVF). Many patients with pacemakers have a left axis deviation because the pacemaker leads are on the right side of the heart.

Finally, some structural body changes will produce a left axis deviation. In advanced pregnancy, the enlarged uterus may occupy so much space in the abdomen that the elevated diaphragm pushes the heart to a more horizontal or leftward-lying position, producing a left axis deviation. Similarly, short and squat or morbidly obese patients may have a left axis deviation because of the heart's position in the chest.

You can recognize a *right axis deviation* when lead I is negative and lead aVF is upright. The mean vector is abnormally directed to the right side of the heart. Causes of right axis deviation include chronic obstructive pulmonary disease and right ventricular hypertrophy. In both instances, enlargement of the right cardiac chambers pulls the mean vector to the right side. A right bundle-branch block causes the mean vector to flow from left to right, resulting in right axis deviation. Children and tall, thin adults may have a normal right axis deviation if the heart hangs down in a more vertical position.

If both leads I and aVF are negative, then the axis deviation is termed *indeterminate axis* or *extreme right axis deviation*. The mean vector is directed upward and to the right. If you find an indeterminate axis deviation on your patient's ECG, check the leads; incorrect ECG lead placement is a common cause of this finding. Other causes are some types of pacemakers, abnormal cardiac rhythms such as ventricular tachycardia, congenital heart disease, or dextrocardia (heart positioned on the right side of the chest).

**R-wave ups and downs**




the left ventricle.

The mean vector in the horizontal plane is influenced by the overwhelming power of the left ventricle and can be thought of as flowing toward the left side. Because the mean vector flows away from lead $V_1$, this lead has a downward QRS deflection; the QRS is almost totally upright in leads $V_5$ and $V_6$ because the mean vector flows directly at these leads. The QRS complex becomes progressively more upright across the chest wall from $V_1$ to $V_6$, a change known as *R-wave progression* (see *R-wave ups and downs*).[3] This is another characteristic of a normal ECG.

## Putting it all together

Prepared with our new knowledge of 12-lead ECGs, let's examine Mr. Smythe's 12-lead ECG. His heart rate is normal, and you see clear P waves, QRS complexes, and T waves. The PR interval is 0.14 second, which falls within the normal range. The QRS complex should be less than 0.12 second; Mr. Smythe's QRS complexes are 0.08 second wide. The T waves are upright and normal looking. Finally, the ST segment is level with the baseline.

Mr. Smythe's limb leads are all upright with the normal exception of aVR. Lead II is the most upright and aVL is the least upright. The chest leads demonstrate downward lead $V_1$ and upright leads $V_5$ and $V_6$ with normal R-wave progression across the chest wall.

You conclude that Mr. Smythe has a normal 12-lead ECG, indicating no electrical abnormalities in heart function. However, he's not out of the woods yet. Some types of ischemic chest pain aren't apparent on routine ECG, so the physician may consider following up with a stress test.[4]

Mr. Smythe's normal ECG, negative cardiac enzymes, and benign patient history led the medical team to rule out a cardiac source for his discomfort. He was discharged home the next day with a prescription for pantoprazole and told to follow up with his health care provider if his chest discomfort recurs.

In this article, you've learned to recognize the features of the normal ECG. Next month, I'll examine some abnormalities of the 12-lead ECG and discuss how to assess for MIs and arrhythmias.

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The author has disclosed that he has no significant relationship with or financial interest in any commercial companies that pertain to this educational activity.

# Understanding the 12-lead ECG part II

**Learn to recognize bundle-branch blocks, myocardial infarction, and common dysrhythmias.**

BY GUY GOLDICH, RN, CCRN, MSN

LAST MONTH, I described the components of the 12-lead electrocardiogram (ECG) and how to recognize a normal ECG. In this article, I'll explain some advanced techniques that you can use to interpret common ECG abnormalities: bundle-branch blocks, myocardial infarction (MI), and common dysrhythmias.

## Bundle-branch blocks: Obstruction in the conduction

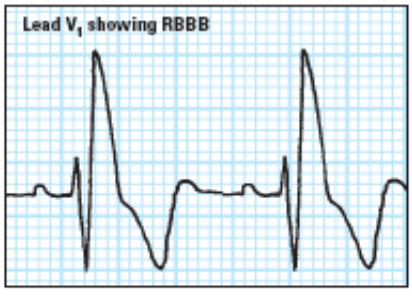
Probably the most common ECG abnormality you'll encounter is a bundle-branch block, which appears on the ECG as a wider-than-normal QRS complex (more than 0.12 second in duration). As you know, the cardiac impulse, originating in the sinoatrial (SA) node, normally travels through the bundle of His into the right and left bundle branches in the septum. The two bundle branches terminate in the Purkinje fibers. When the impulse reaches them, ventricular depolarization begins.

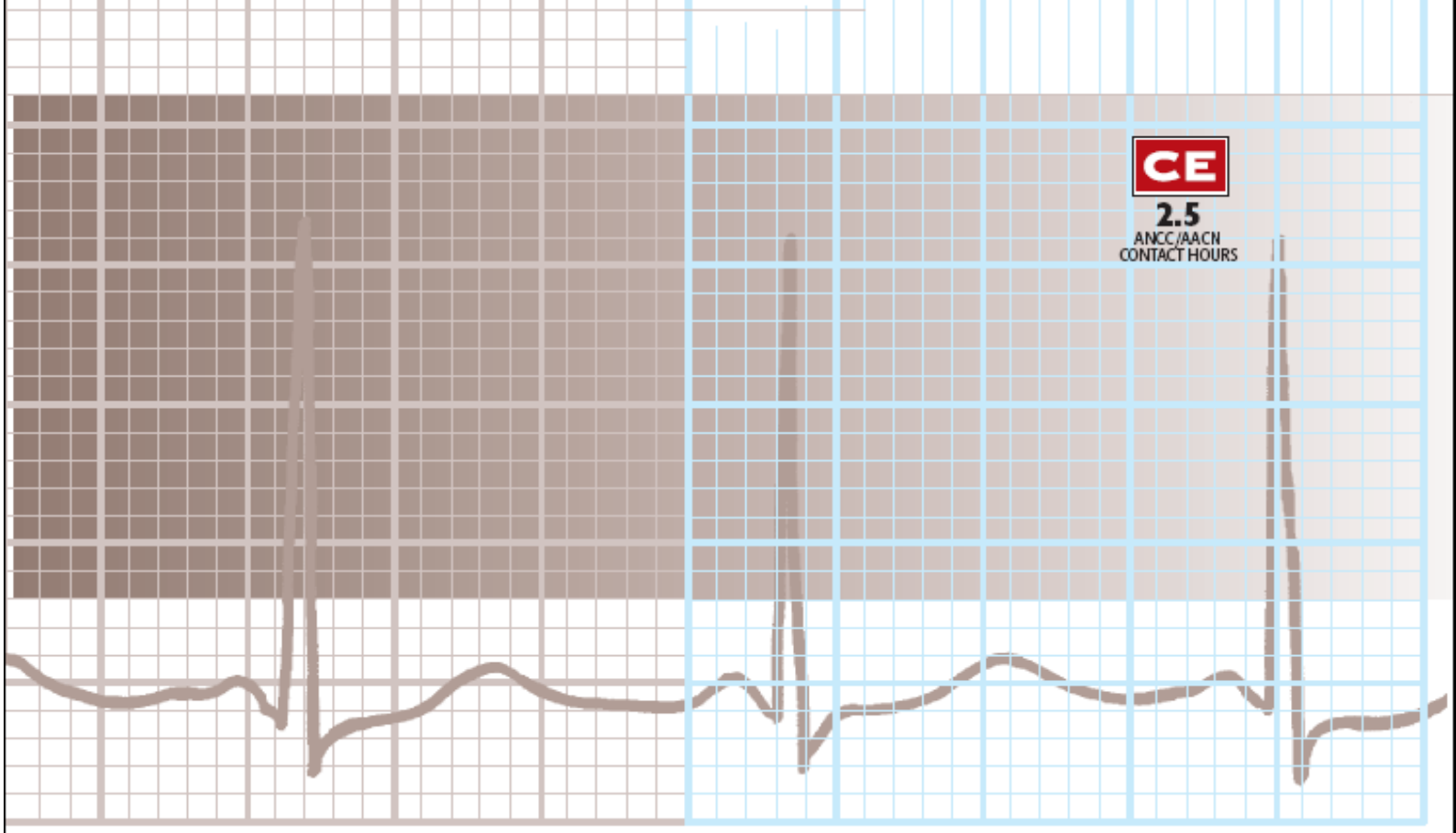
Normally the impulse is delivered to myocardial cells on both sides of the heart simultaneously, so depolarization begins at the same time on both sides of the heart. The result is a very fast, synchronous contraction of the ventricles. On ECG, the normal QRS complex duration from two intact bundle branches is 0.12 second or less (three or fewer small squares of the ECG paper).

A bundle-branch block occurs when one of the two bundle branches can't conduct the cardiac impulse to the myocardial cells. The most common cause of chronic bundle-branch block is ischemic heart disease. When an artery supplying the bundle branch narrows, the flow of oxygenated blood is reduced and the bundle branch can't conduct impulses normally.

A common cause of acute bundle-branch block is acute MI. If the MI involves the ventricular septum, one of the bundle branches may become infarcted, leading to a loss of conduction. Although uncommon, physical injury of a bundle branch during an invasive procedure such as cardiac catheterization or heart surgery also may produce a bundle-branch block.[1]

• In a **right bundle-branch block (RBBB)**, impulse conduction to the right ventricle is blocked. The cardiac impulse is conducted only to the left side of the heart where left ventricular depolarization



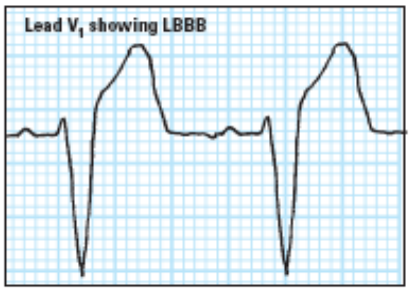



begins. The right side of the heart depolarizes only in response to the cell-to-cell wave of depolarization that travels from the left side of the heart. This cell-to-cell depolarization is much slower than the normal synchronous depolarization; that's why the QRS complex is significantly wider than normal.

Examine lead $V_1$ to identify an RBBB. In lead $V_1$, the normal QRS complex consists of a small R wave, then a large S wave. As you recall, lead $V_1$ looks at the right side of the heart. A small vector originating in the septum toward $V_1$ creates a small upward R wave, then the predominant mean QRS vector creates the large S wave as the mean QRS vector flows away from lead $V_1$.

In RBBB, the path of the mean QRS vector is changed due to left-to-right slow conduction; lead $V_1$ now records a delayed R wave approaching it, resulting in a positive R wave. So the key identifier of RBBB in lead $V_1$ is a QRS complex wider than 0.12 second with a delayed (longer than 0.07 second) positive main R wave. Some RBBBs may display a triphasic waveform ("rabbit ears") consisting of a small r wave, downward S wave, and a second, larger R wave.[2]

- In a **left bundle-branch block (LBBB)**, electrical impulses don't reach the left side of the heart normally, so once again, synchronous depolarization of the ventricles doesn't occur. Depolarization begins in the right side of the heart and travels in a right-to-left direction via slow cell-to-cell depolarization. Lead $V_1$ records the mean QRS vector directed away from its positive lead, resulting in a wide downward complex. Because the mean vector takes a relatively longer time to cross to the left side of the heart, the QRS complex is wider than 0.12 second. The key to recognizing an LBBB is a wide, downward S wave or rS wave in leads $V_1$ and $V_2$.

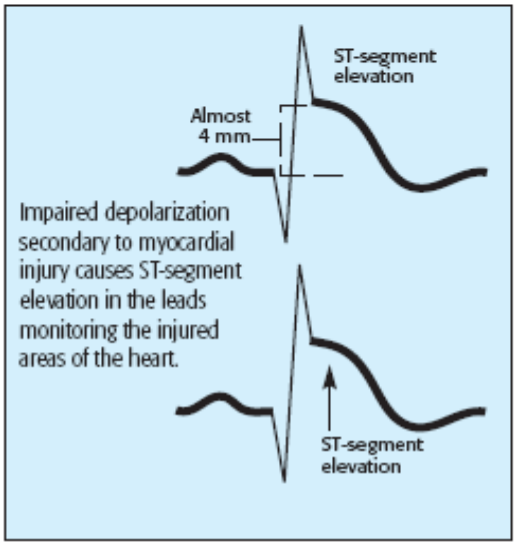



## Recognizing an MI

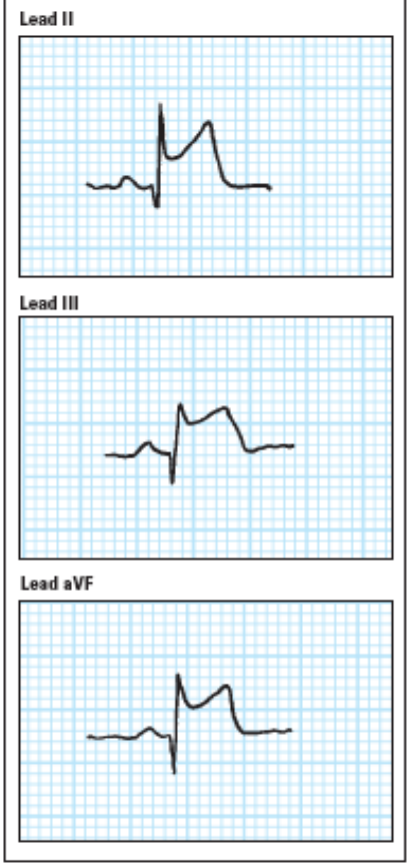
One of the most critical functions of the 12-lead ECG is to determine whether a patient is experiencing an acute MI. A series of predictable ECG changes that occur during an MI help you identify it quickly and initiate appropriate treatment.

Among one of the earliest changes in the ECG tracing is an elevation of the ST segment, indi-

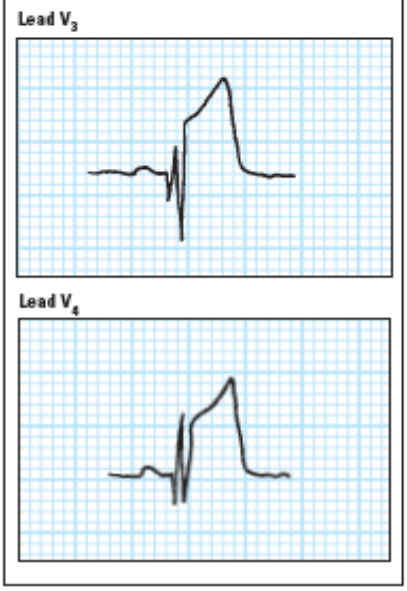
## Understanding ST-segment elevation




## Inferior-wall STEMI




## Anterior-wall STEMI

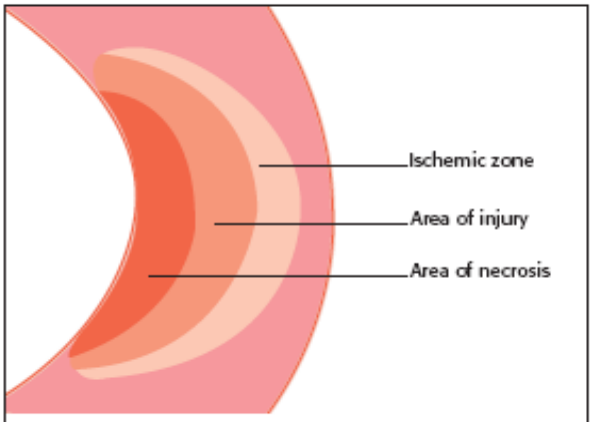



cating reversible myocardial injury (see *Understanding ST-segment elevation*). In a normal ECG, the ST segment is level with the tracing's baseline. When myocardial cells sustain injury from MI, depolarization is impaired, resulting in ST-segment elevation in the leads monitoring the affected areas of the heart. An ST-segment-elevation MI (STEMI), the most serious type of MI, is associated with more complications and a higher risk of death.[3]

The leads with ST-segment elevations identify the area of myocardial injury, so you can determine the region of the heart affected by knowing which area is monitored by which ECG lead. Let's look at some examples.

- Because leads II, III, and aVF all monitor the inferior (or bottom) wall of the heart from slightly different directions, they're usually described as the inferior leads. This area of the heart is perfused by the right coronary artery. A patient with a STEMI involving the inferior wall of the heart will have elevated ST segments in leads II, III, and aVF (see *Inferior-wall STEMI*).
- Another common infarct lead pattern occurs when an MI involves the intraventricular septum, which is perfused by the left anterior descending (LAD) coronary artery. In a *septal MI*, the leads monitoring the septum's electrical activity will display elevated ST segments. Precordial (or chest) leads $V_1$ and $V_2$, which are located on the anterior chest wall directly over the septum, most accurately monitor the septum's electrical activity. (These leads also are known as the septal leads.) The patient experiencing a septal MI will have ST-segment elevations in leads $V_1$ and $V_2$.
- Directly to the left of the septal area of the heart is the large frontal or *anterior* wall of the heart, which is also perfused by the LAD coronary artery. As the most muscular and powerful pumping wall of the heart, the anterior wall is responsible for a large proportion of cardiac output. Anatomically, leads $V_3$ and $V_4$ are located directly above the anterior wall of the heart and monitor its electrical activity. An anterior-wall STEMI will cause the ST segments in these leads to be elevated (see *Anterior-wall STEMI*).
- The lateral wall of the heart, perfused by the left circumflex artery,

## Tissue damage after MI




After an MI, the heart muscle has three zones of damage. Necrotic tissue dies from lack of blood flow. Injured cells may recover and ischemic cells can be saved if the area is reperfused promptly.

is located to the left of the anterior wall and follows the curve of the left lateral chest wall. Relatively muscular, it also contributes significantly to the heart's pumping ability. The ECG lead pattern that monitors the lateral wall's electrical activity is more complex because the lateral wall is monitored by a combination of precordial (chest) leads and frontal (limb) leads.

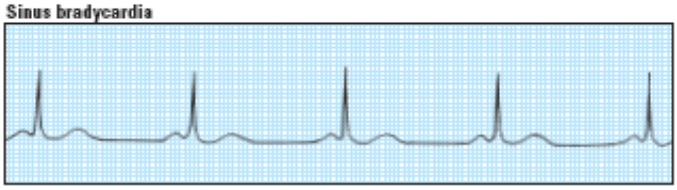
Chest leads $V_5$ and $V_6$ are located on the left lateral chest wall and monitor electrical activity by looking down at the lateral heart wall. Leads I and aVL also monitor the lateral wall's electrical activity. A patient with a lateral-wall STEMI will have ST-segment elevations in leads I, aVL, $V_5$, and $V_6$.

Although patients can have MIs affecting a single heart wall, such as a discrete septal MI or anterior-wall MI, the area of infarction may involve more than one area of the heart. In such a case, ST-segment elevations appear in the leads monitoring all of the involved areas. For example, if the infarction extends into both the septum and the anterior wall, the ST-segment elevations would appear in leads $V_1$, $V_2$, $V_3$, and $V_4$. The areas involved in the MI are reflected by the descriptive name; in this case, an *anterioseptal* MI. For information on how MI affects heart muscle, see *Tissue damage after MI.*

### Identifying common dysrhythmias

Now let's examine some common ECG rhythm abnormalities you may encounter in your practice, keeping in mind that you always treat the patient, not the rhythm. When you find an abnormal rhythm or a rhythm change, assess your patient and document level of consciousness, vital signs, chest pain, shortness of breath, and other signs and symptoms associated with the dysrhythmia. By using your assessment skills, nursing judgment, and knowledge of ECGs, you can determine the level of urgency of the situation.

Sinus bradycardia



- **Sinus bradycardia** is a sinus rhythm slower than the lower normal sinus rate of 60 beats/minute. The P waves, QRS complexes, and T waves are all normal. Sinus bradycardia is commonly caused by ischemic heart disease that causes the SA node to malfunction. Sinus bradycardia can also be caused by acute MI and some types of medications, such as beta-blockers. Well-conditioned athletes may have normal resting heart rates slower than 60 beats/minute. Assess your patient for hemodynamic stability if he has a new or profound sinus bradycardia. Contact the health care provider if your patient is symptomatic. Signs and symptoms that may accompany sinus bradycardia include hypotension, lethargy, fatigue, chest pain, and difficulty breathing. Be prepared to transfer your patient to the intensive care unit (ICU) for a temporary pacemaker.
- **Sinus tachycardia** is a sinus rhythm that's faster than the

Sinus tachycardia

upper normal sinus rate of 100 beats/minute. Sinus tachycardia can produce heart rates of 100 to 150 beats/minute. At faster rates, the heart's myocardial oxygen demand increases, and a patient with preexisting heart disease may experience chest pain or other cardiac symptoms. Sinus tachycardia usually is related to a physiologic cause, such as fever, infection, pain, physical exertion, anxiety, hypoxia, or shock. If you can identify and treat the cause, the heart rate will usually decrease. To manage sinus tachycardia with an unknown cause, the health care provider may order a beta-blocker such as metoprolol or atenolol.[4]

• **Atrial fibrillation (AF)**, one of the most common dysrhythmias you'll see in practice, has two predominant characteristics: an irregularly irregular heart rhythm and no meaningful P waves. Normally, after passive ventricular filling, the atria contract regularly and eject their load of blood into the ventricles (atrial kick). In AF, atrial kick is lost. Instead of contracting normally, the atria quiver due to random and chaotic depolarization of atrial cells. The random atrial depolarization is also responsible for the irregular ventricular rate, which can vary from 40 to 180 beats/minute. Atrial fibrillation has many causes, including atrial enlargement from chronic obstructive pulmonary disease or other lung disease, thyroid disease, ischemic heart disease, acute MI, stress or fatigue, and excessive use of caffeine, alcohol, or cigarettes.

Atrial fibrillation

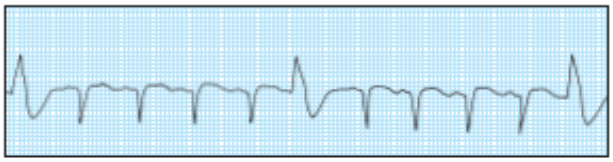
You may first encounter AF during a routine vital signs check. If your patient has a new irregular heart rate, or has an abnormally fast or slow heart rate, obtain an order for a 12-lead ECG. Look for an irregularly irregular rhythm and f waves, the two hallmarks of AF. Perform a thorough physical assessment because patients can rapidly become hemodynamically unstable or develop worsening heart failure. If your patient has unstable or symptomatic AF, administer supplemental oxygen and establish or maintain intravenous (I.V.) access before transferring him to the ICU or telemetry unit for treatment with I.V. diltiazem or a beta-blocker.

If the patient is stable, the health care provider may order various oral medications to control or convert AF, such as digoxin, diltiazem, amiodarone, or metoprolol.

All patients with AF lasting more than 48 hours are at high risk for thrombus formation because of irregular blood flow in the atria. If released into the circulation, these thrombi can cause arterial obstruction resulting in life-threatening complications such as stroke. As ordered, administer I.V. heparin and start the patient on oral warfarin to prevent thrombus formation.[5]

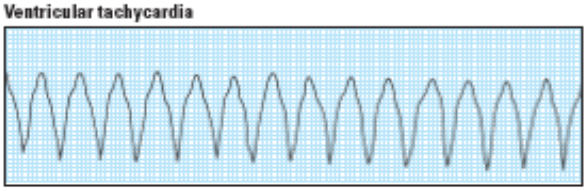
• **Premature ventricular contractions (PVCs)** are characterized by a wide, abnormal QRS complex because conduction is through the ventricular tissue and not the His-Purkinje system. Look for a QRS greater than 0.12 second that appears large, abnormal, and premature (occurring *before* the next sinus beat). Caused by irritable ventricular tissue that depolarizes early and unpredictably, PVCs can be triggered by heart failure, electrolyte imbalances, stimulants such as caffeine, hypoxia, acute MI, mitral valve prolapse, thyroid disease, and injury or infarct of the myocardial tissue. Rare or isolated PVCs seldom require aggressive treatment. However, if you notice that the frequency of PVCs is increasing, or if you see new groups or "runs" of PVCs, contact the health care provider for further evaluation.

Isolated PVCs



• **Ventricular tachycardia (VT)** is a very rapid (100 to 250 beats/minute) series of wide-complex ventricular depolarizations. In this dysrhythmia, abnormal ven-

Ventricular tachycardia



tricular tissue rapidly depolarizes, taking rhythm control away from the sinus node. Along with the rapid rate, VT is characterized by wide, bizarre QRS complexes usually followed by large T waves in the opposite direction of the major QRS deflection.

If your patient is unconscious, apneic, and pulseless, call a code and start cardiopulmonary resuscitation. If your patient has a pulse and is awake, treat this situation as a medical emergency. Call for the physician stat (and the rapid response team, if your facility has one), bring a crash cart with a monitor/defibrillator to the bedside, and prepare to transfer the patient to the ICU.

## Summing up

Like any new skill, interpreting 12-lead ECGs takes practice and commitment. Make a habit of reviewing your patients' ECGs routinely. Seek guidance from colleagues experienced in ECG interpretation, such as senior staff nurses, clinical nurse specialists, and clinical nurse-educators. Many physicians will also be happy to review an ECG with you if they know you're interested in learning to spot problems early.

With practice and experience, ECG interpretation will become a valuable nursing tool, helping you to recognize problems promptly and provide even better patient care.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# Precordial electrode placement in women

P.W. Macfarlane, R. Colaco, K. Stevens, P. Reay, C. Beckett, T. Aitchison

***Background.*** **Precordial ECG electrode positioning was standardised in the early 1940s. However, it has been customary for the $V_3$ to $V_6$ electrodes to be placed under the left breast in women rather than in the correct anatomical positions relating to the 4th and 5th interspaces. For this reason, a comparison between the two approaches to chest electrode positioning in women was undertaken.**
***Methods.*** **In total 84 women were recruited and ECGs recorded with electrodes in the correct anatomical position and also in the more commonly used positions under the breast. As a separate study, 299 healthy women were recruited to study normal limits of leads $V_3$ to $V_6$ recorded with electrodes in the correct anatomical positions and compare them with published normal limits with electrodes in the more commonly used locations.**
***Results.*** **It was shown that there was less variability with electrodes in the correct anatomical positions and that there were significant differences between the new limits of normality compared with the old established limits.**
***Conclusion.*** **Expansion of the database and further analysis of the data is required to make a definitive recommendation with respect to precordial electrode placement in women. (*Neth Heart J* 2003;11:118-22.)**

Key words: EGG, precordial electrode, women, normal limits, ECG analysis

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The electrocardiogram still remains one of the most commonly used, if not the most commonly used, noninvasive investigative technique in medicine. It is well known that there can be significant day-to-day variation in ECG appearances, which have been quantified,[1] while the effect of using marked positions on the chest to minimise variation has also been assessed.[2] Studies have been undertaken on the effect of electrodes being placed one interspace too high or too low[3] but ultimately, there is no failsafe approach that can guarantee accurate electrode placement given the many variables involved, not the least of which is the training of the medical or technical staff involved in ECG recording. Indeed, medical staff have very little formal training and may often be called upon to record ECGs in the acute situation, out of hours, when no trained technician is available.

On the other hand, little attention seems to have been paid to the effect of electrode placement on ECG interpretation in women. In 1938, a committee of the American Heart Association and cardiologists representing the Cardiac Society of Great Britain and Ireland recommended initially standardising on one chest electrode position.[4] However, it was a subsequent paper published by the American Heart Association[5] which defined the positions now commonly used for recording the precordial leads $V_1$ to $V_6$. A supplementary report by this committee was also published in the following year.[6]

As is well known, the reference point for the positioning of precordial leads is the 4th intercostal space. $V_1$ and $V_2$ are positioned at this level and $V_4$ is defined as being placed at the intersection of the 5th intercostal space and the mid-clavicular line. $V_3$ is placed intermediate to $V_2$ and $V_4$ while $V_5$ and $V_6$ are positioned at the same level as $V_4$. The positioning of $V_4$ according to the above recommendations has proved problematic in women and technicians worldwide have commonly positioned this electrode underneath the left breast in women in the mid-clavicular line rather than strictly at the defined anatomical reference of the fifth intercostal space.

In an earlier publication aimed at technicians in the United Kingdom, it was suggested that precordial

**Table 1.** Mean R-wave amplitudes (mV) in V3 to V6 according to location.

| | Lead | | | |
|---|---|---|---|---|
| | $V_3$ | $V_4$ | $V_5$ | $V_6$ |
| On breast | 0.686mV | 0.950mV | 1.251mV | 1.208mV |
| Below breast | 0.720 | 0.978 | 1.132 | 1.074 |
| Mean difference | 0.034 | 0.028 | -0.119 | -0.134 |
| 95% CI (below, on) | 0.007, 0.060 | -0.009, 0.065 | -0.152, -0.087 | -0.160, -0.108 |
| P value | 0.01 | 0.14 | <0.01 | <0.01 |

CI=confidence interval.

electrodes should be placed underneath the breast.[7] On the other hand, Rautaharju et al.[8] have advocated that electrodes should be placed on top of the breast in keeping with the strict anatomical recommendations.

In addition, one of the authors (PWM) had the subjective impression that poor R-wave progression was more prevalent in females than males, leading to more diagnoses of possible anterior myocardial infarction. One possible reason for this might be the inaccurate positioning of $V_3$ to $V_5$ in women.

The question which had to be asked, therefore, was which is the more reliable position for electrode placement, i.e. one that could lead to more consistent and accurate recording? Alternatively, the question might be asked as to whether it really made any difference to ECG measurements if electrodes were placed on, rather than below, the breast.

We set out to examine the problem and consider the implications. This preliminary communication summarises the work so far undertaken.

## Methods

ECGs were recorded in a group of female volunteers recruited from the cardiorespiratory, medical and surgical units at Glasgow Royal Infirmary with the chest electrodes $V_1$ to $V_6$ placed strictly in accordance with the recommendations described above (approach 1) and in addition, with the $V_4$ electrode placed at the highest point below the breast in the appropriate longitudinal reference line. In this case, $V_3$ to $V_6$ were then placed according to the standard definitions, i.e. $V_3$ was midway between $V_2$ and $V_4$ while $V_5$ and $V_6$ were at the same horizontal level as $V_4$, whatever level that might be (approach 2). Two consecutive recordings were made with approach 1 and a further two consecutive recordings were made with approach 2 to assess repeatability of each approach. Electrodes were removed and the whole process was repeated 30 minutes later so that a total of eight recordings were made on each female volunteer.

To obtain an estimate of breast volume, bra cup size (A to E) was also noted.

As a separate component to the project, 12-lead ECGs were also recorded in a group of healthy female volunteers in whom the precordial electrodes were placed strictly according to the anatomical reference points (approach 1). This study was aimed at comparing measurements with normal limits determined several years ago using electrodes placed under the breast in women.[7]

As part of the study, electrocardiograms from all male and female patients in Glasgow Royal Infirmary were reviewed over a period of two weeks to identify the incidence of reports of poor R-wave progression, reversed R-wave progression and low R waves.

## Statistical methods

The comparison between normal ranges using anatomical reference marks and previously published normal ranges was made on a simple basis of comparing differences between means.

On the other hand, a more complex model was used for determining whether there was any difference in ECG wave amplitudes because of the variation in electrode positioning on or below the breast in females. This model included checks for between-female patient variability, within-patient long-term variability, i.e. over 30 minutes and within-patient short-term variability, i.e. over one minute.

## Results

### *Variation in precordial electrode placement*

Altogether, 84 women (mean age 60±10 years) were recruited into the repeated measurements study for which ethical permission had previously been obtained. There was no consistent trend found in R-wave measurements with electrodes placed on or below the breast as shown in table 1. Measurements in $V_5$ and $V_6$ with electrodes on the breast were significantly higher than below. On the other hand, the reverse was true for $V_3$ while for $V_4$ there was no significant difference between the two positions.[8]

### *Repeatability*

The eight recordings from each of the 84 females were studied to answer the question on stability of electrode placement and hence measurement. Table 2 shows the total variance between replicates, i.e. measures repeated one minute and 30 minutes apart, from which it can

**Table 2.** Variance ($\mu V^2$) between replicates.

| Lead | On breast | Below breast | Ratio On/Below | P value | Approx 95% CI |
|---|---|---|---|---|---|
| $V_3$ | 26111($\mu V^2$) | 30551 | 0.85 | 0.3 | (0.45, 1.59) |
| $V_4$ | 52298 | 61106 | 0.86 | 0.3 | (0.46, 1.61) |
| $V_5$ | 11237 | 29972 | 0.37 | 0.001 | (0.20, 0.69) |
| $V_6$ | 17447 | 19456 | 0.90 | 0.4 | (0.48, 1.69) |

CI=confidence interval.

be seen that there is less variability of measurements from electrodes on the breast, essentially for all leads but significantly so only for $V_5$.

There were 41 women with bra cup size A or B, 28 with size C and 15 with D or E. It was found by comparing these three groups that as breast (cup) size increased, the repeatability of measurements within each volunteer decreased for $V_3$ and $V_4$. The finding of reduced variability of R-wave measurements on as compared with under the breast was replicated across all breast sizes.

### *Normal limits*

A total of 299 women were recruited to the study on normal limits. Their age distribution is shown in table 3 from where it can be seen that it was easier to recruit younger females to the study. Figure 1 shows the difference between the existing and revised upper and lower limits of normal R waves for leads $V_3$ to $V_6$ recorded from females aged 30 to 39 years. Table 4 shows differences in mean R, S and T+ values using the two approaches to electrode positioning in 18- to 29-year-old females.

### *Poor R-wave progression*

Altogether, 1315 patients (755 men and 560 women) effectively were recruited for this part of the study. The prevalence of all forms of poor R-wave progression, including reversed R-wave progression and low R waves in $V_2$ to $V_4$, was 19% in women and 11% in men. In the group of 84 women, the incidence of poor R-wave progression was not high enough to determine whether this ECG finding could be related to breast size.

**Table 3.** Study population subgroups according to age (years). Mean±SD age values (years) for each age range are also shown.

| Category | Age range (years) | Number of volunteers | Mean age ± SD (years) |
|---|---|---|---|
| 1 | 18-29 | 121 | 22.2±3.0 |
| 2 | 30-39 | 49 | 34.5±5.9 |
| 3 | 40-49 | 69 | 44.9±2.7 |
| 4 | ≥50 | 60 | 56.1±6.5 |

## Discussion

This ongoing study has drawn together various threads of a project designed to determine whether precordial electrodes in women should be placed on or beneath the breast. Several points can be made from a philosophical point of view. It has long been the practice in women for electrodes to be placed below the recommended positions perhaps as much for convenience and cosmetic reasons as anything else. The suction electrodes which were commonly used throughout the period from around 1930 to 1990 tended to leave weals on the skin, particularly if left in place for too long. Furthermore, personal communication with Proudfit,[9] who worked in the laboratory of Wilson and his colleagues who developed the precordial lead recording system,[10] indicated that chest electrodes were indeed placed underneath the breast, sometimes supported by towelling if necessary.

Personal observation and communication suggest that normal limits of precordial leads, certainly as developed in this laboratory,[11] were derived from electrodes placed underneath the breast. However, modern technology based on adhesive electrodes makes it much more acceptable and straightforward to place electrodes on top of the breast. As indicated in this study, this results in a slightly more reproducible recording than would be the case with the alternative positioning.

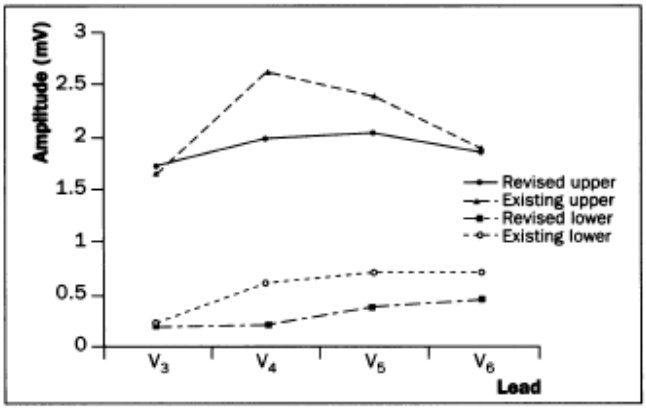


*Figure 1. Upper and lower 96% limits of normal ranges ($V_3$ to $V_6$) of R-wave amplitude in females aged 30 to 39 years. Existing[11] and revised limits are shown. See text for further details.*

**Table 4.** Mean amplitudes (mV) of R, S and T+ waves in leads $V_3$ to $V_6$ in 18- to 29-year-old females from the existing normal values (EXS) and the newly revised data (REV). P values (two-sample t test) for the differences are also shown. Note that, by convention, S waves have a negative value. Measurements were available from up to 318 females to derive the existing normal values. Not every individual had an R and S wave in every lead.

| | Lead | | | |
|---|---|---|---|---|
| | $V_3$ | $V_4$ | $V_5$ | $V_6$ |
| R amp (REV) | 0.752 | 1.0489 | 1.17 | 1.092 |
| R amp (EXS) | 0.81 | 1.302 | 1.342 | 1.219 |
| P value | <0.1 | <0.0005 | <0.0005 | <0.0025 |
| S amp (REV) | -0.731 | -0.532 | -0.281 | -0.164 |
| S amp (EXS) | -0.794 | -0.382 | -0.244 | -0.15 |
| P value | <0.05 | <0.0005 | <0.1 | >0.2 |
| T+ amp (REV) | 0.555 | 0.561 | 0.472 | 0.373 |
| T+ amp (EXS) | 0.533 | 0.426 | 0.399 | 0.337 |
| P value | <0.4 | <0.0005 | <0.0005 | <0.01 |

The effect of age and gender on ECG amplitude has been well documented.[11,12] ECG criteria for diagnosing left ventricular hypertrophy (LVH), for example, must be age and gender based but rarely are, with some exceptions, e.g. as built into the Glasgow Programme.[13] Such criteria differences may well be a function of body mass index but the differences between male and female ECG voltages tend to narrow with increasing age. Electrode positioning in women is not likely to have a significant influence on this particular diagnosis and the lack of sensitivity of ECG criteria is simply a reflection of the inadequacy of the ECG in diagnosing LVH with high sensitivity and specificity.

Some authors advocate the use of body surface mapping with multiple chest electrodes, e.g. 64, which minimises the effect of misplacement of one electrode. The gain in sensitivity, however, is marginal which can be explained in some cases by concentric LVH leading to a cancellation effect among electrical forces and hence a normal voltage, so that no matter how many electrodes are used, the ECG will not detect LVH.

The application of a large number of electrodes permits the use of so-called inverse models to calculate the electrical activity on the epicardial surface[14] but there has, as yet, been no major study showing improved diagnosis of a variety of cardiac abnormalities with this approach.

It was interesting to find that poor R-wave progression in chest leads was more prevalent in females than in males but, paradoxically, the average R-wave amplitude in $V_4$ with electrodes placed on the breast was lower than that with electrodes placed beneath the breast. This could be due to attenuation of voltages due to breast tissue. This simply indicates that criteria for poor R-wave progression have to be carefully developed particularly in women in whom false positive reports of anterior myocardial infarction are not uncommon.[15]

The female volunteers in the study had a variety of cardiac and respiratory problems. A wide spectrum of pathology was therefore embraced, but as each individual acted as her own control, the inhomogeneity of the population studied was immaterial in terms of repeatability of measurements and effect of electrode placement variation.

The other component of the study clearly indicates that revised normal limits of the ECG in females would have to be used if chest leads were placed at the correct anatomical level. However, a larger number of volunteers would be required to give added strength to this conclusion.

## Conclusion

Expansion of the database and further analysis of the data is required in order to make a definitive recommendation with respect to precordial electrode placement in women. Such a recommendation would be based not only on objective experimental data but also on more subjective considerations involving ease of electrode placement. ■

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The article should contain an abstract. The abstract should have the following headings: Objectives/Background, Methods, Results, Conclusions and must not contain more then 200 words. Do not cite references in abstracts. Classification of the article: introduction, patients and methods, results, discussion, conclusion, acknowledgements, references, glossary, appendix.
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# Taking a Quality 12 lead ECG

**Tony Curran (Cardiology Nurse Educator)**

An EKG is an important part of the initial evaluation of a patient who is suspected to have a heart related problem. Small sticky electrodes are applied to the patient's chest, arms and legs. However, with some systems, the electrodes may be applied to the chest, shoulders and the sides of the lower chest, or hips. Wires are used to connect the patient to an EKG machine. The electrical activity created by the patient's heart is processed by the EKG machine and then printed on a special graph paper. It takes a few minutes to apply the EKG electrodes, and one minute to make the actual recording.

It is important to place 12 lead ECG electrodes in a correct position because, when looking for evidence of ischemia or MI is essential to compare 2 or more successive ECG recordings and when assessing the ECG recording changes detected must be related to the electrode-heart position. Misplaced electrodes or unconventional positioning of a patient results in misinterpretation or omission of pathological signs

## 7 Step to obtaining a quality 12 lead ECG:

1) Ensure patient is comfortable.

2) Remove any chest hair (males).

3) Clean skin if necessary – use a damp cloth to degrease and clean the skin, this provide hydration which enhance sign pick up by the carrier gel of the ECG electrodes. (Important note: the use of alcohol wipe is counter intuitive as this will dry out the skin and increase skin impedance.

4) Apply appropriate adhesive electrodes to limbs/chest.

5) Attach ECG cables to tabs.

6) Ensure the patient is relaxed and still.

7) Record ECG making sure speed is 25 mm/sec and calibration is correct.

## Problems and troubleshooting:

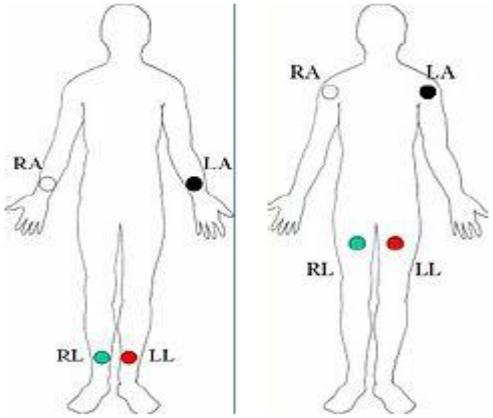
- AC interference – switch off non essential equipment at power source.
- Muscle tremor – assist patient to relax, move limb electrodes to upper limbs if possible.
- Baseline wander – cause by sweat or poor electrodes contact. Dry skin, wipe with damp cloth or shave hair.
- Broken leads/cables – replace leads/cables.
- Flat line in one chest electrode – check proper adhesion to skin, replace electrodes. Ensure electrodes are still moist when applying, when applying, choose spots on the body that are flat and fleshy, not muscular. Check connection between cable and ECG module.

## Skin Preparation:

Skin preparation is often required to help produce an ECG that is artefact free and accurate. This process is designed to minimise the skin to electrode impedance.

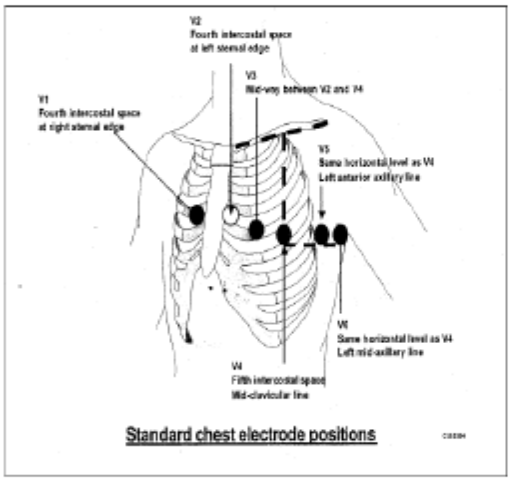
- The removal of chest hair may be required to ensure adequate contact with the skin. Verbal consent should be obtained from the patient and documented. Clean razor to be used and disposed of in the sharps bin.
- Exfoliation may be required and should be undertaken with very light abrasion using a paper towel, gauges swab or proprietary abrasive tape designed specifically for this purpose. Abrasive tape also performs a dual function, it not only removes the outmost layer of dead skin, it creates a small erythema causing blood vessels to rise to the surface, this assist in a more define d ECG tracing.
- On occasions the skin will require cleaning. Best method is using a damp cloth with mild soap – no alcohol wipes.

## Electrodes Placement: limb Leads:




## Electrode placement: Precordial (chest) leads:

| Electrodes | Position |
|---|---|
| V1 | Fourth intercostals space at the right sternal edge. |
| V2 | Fourth intercostals space at the left sternal edge. |
| V3 | Midway between V2 and V4. |
| V4 | Fifth Intercostals space in the mide – clavicular line. |
| V5 | Left anterior axillary line at same horizontal level as V4. |
| V6 | Left mid axillary line at same horizontal level as V4 and V5. |



Standard chest electrode positions

## Locating chest electrode positions:

Care should be taken when counting the intercostals spaces down from the clavicle. The small space between the clavicle and the first rib is not mistaken for the first intercostals space. In order to avoid this common error the sternal angle should be used as the main reference pioint. This anatomical landmark denotes the position of the sternal angle at the manubriosternal joint.

To locate the sternal angle, a finger should be run down the sternaum from the sternal notch until a bony ridge is met. From this ridge, slide the finger down and to the side, locating the second intercostals space. Count down to the third and fourth spaces. Locate the edge of the sternum and place V1 there and repeat the procedure in the left side to correctly position V2.

V4 is placed in the fifth intercostals space in line with the mid point of the clavicle. Once V4 is correctly place, location of V3 can be identified. This position is between V2 and V4. V5 and V6 positions are identified by following the horizontal line from V4 (not the rib line) and place V5 in line with the anterior axilla and V6 in line mid axilla.

When recording an ECG from a female patient, the lateral chest electrodes (V4, V5 & V6) are palced beneath the left breast. It is suggested to never place electrodes above the bony structure or on a breast tissue in females. But "Breast tissue appears to have a practically negligible effect on ECG amplitudes, and in women, the placement of chest electrodes on the breast rather than under the breast is recommended in order to facilitate the precision of electrode placement at the correct horizontal level and at the correct lateral positions (refer to reading).

However if you cannot avoid moving onto a breast, write it down on a recording. The same applies for obtaining ECG recording from a patient in a sitting position (e.g. due to cardiac failure or breathlessness).

## Recording:

In order to record a good quality ECG, the patient must be relaxed and comfortable. Make sure the patient's fists are not clenched, their arms are not stiff or they are moving their fingers. These simple movements will be recorded on the ECG as well as cardiac activity and this type of interference will make the ECG more difficult to interpret. Remember that some patient will not be able to relax completely due to pain/discomfort, so you will need to assess the situation and where possible make them comfortable.

On occasions it may be necessary to adapt the recommended ECG recording techniques. For example, wheelchair bound patients may need to remain in their chair during the recording process. If this is the case highlight this variation by documenting on the ECG and patient notes **'ECG recorded while patient in wheelchair'.** Always note down any change in patient's position, electrode placement or specific medical condition (pacemaker, dextrocardia, MI in the past, angina...).

Patient details (name, d.o.b. and hospital number) should be either entered into the ECG machine date base or a ID label arrached to the ECG tracing. Press the appropriate button on the ECG machine to initiate a recording (usually labelled 'start' or 'auto'). A 12 lead ECG and rhythm strip should be recorded at 23 mm/sec. To assist in reducing somatic muscle interference on the ECG, switch on the filter button. It is important to remember even thou the filter is used to reduce interference, it can distort the ECG. So it is important to try and relax the patient to reduce any somatic muscle interference.

Any changes on the ECG, advise the medical team urgently. If the patient has any cardiac symptoms at the time of recording, such as chest pain or palpitations, document and refer to management protocols. If the ECG is technically correct and of good quality, ensure that it is correctly labelled (patient ID) then remove all of the electrodes from the patient and dispose accordantly.

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## Module 2: Understanding a 12 Lead ECG, Basic Electrophysiology and 12 lead ECG placement.

**Questions:**

**1. The normal heartbeat is initiated in the**
a. SA node. c. bundle of His.
b. AV node. d. Purkinje fibers.

**2. The structure that briefly delays an impulse (to let the ventricles fill) is the**
a. SA node. c. bundle of His.
b. AV node. d. Purkinje fibers.

**3. The QRS represents**
a. atrial repolarization.
b. atrial depolarization.
c. ventricular depolarization.
d. ventricular repolarization.

**4. Which QRS duration indicates a bundlebranch block?**
a. 0.04 second c. 0.10 second
b. 0.08 second d. 0.14 second

**5. The T wave represents**
a. atrial depolarization.
b. atrial repolarization.
c. ventricular depolarization.
d. ventricular repolarization.

**6. Which reveals information about the heart's oxygenation status?**
a. PR interval c. ST segment
b. QT interval d. QRS complex

**7. Electrical energy generated during depolarization is called**
a. an axis. c. axis deviation.
b. a vector. d. the cardiac cycle.

**8. Which of the following represents normal axis?**
a. QRS complex positive in leads I and aVF
b. QRS complex negative in leads I and aVF
c. QRS complex positive in lead I and negative in lead aVF
d. QRS complex negative in lead I and positive in lead aVF

**9. Which represents left axis deviation?**
a. QRS complex positive in leads I and aVF
b. QRS complex negative in leads I and aVF
c. QRS complex positive in lead I and negative in lead aVF
d. QRS complex negative in lead I and positive in lead aVF

**10. Left axis deviation can be caused by**
a. pulmonary embolism.
b. right ventricular hypertrophy.
c. chronic obstructive pulmonary disease.
d. an inferior-wall MI.

**11. Which of the following leads view the horizontal plane of the heart?**
a. V1 through V6
b. I, II, III
c. aVR, aVL, aVF
d. I, II, III, aVR, aVL, aVF

**12. Which chest lead is placed at the right sternal border, fourth intercostal space?**
a. lead V1 c. lead V3
b. lead V2 d. lead V4

**13. R-wave progression refers to the QRS complex becoming progressively more**
a. positive from lead I to lead III.
b. positive from lead aVR to lead aVF.
c. positive from lead V1 to V6.
d. negative from lead V1 to V6.

**14. Myocardial injury generally is represented by an ST segment that is**

a. above the baseline.
b. level with the baseline.
c. below the baseline.
d. isoelectric.

**15. On the horizontal axis, a small box on ECG paper is equal to**
a. 0.04 second. c. 0.1 millivolt.
b. 0.1 second. d. 1 mm.

**16. A bundle-branch block occurs when one of two bundle branches can't conduct cardiac impulses to the**
a. atria. c. myocardial cells.
b. bundle of His. d. atrioventricular node.

**17. The *most common* cause of a chronic bundle-branch block is**
a. ischemic heart disease.
b. an acute MI.
c. an acute coronary syndrome.
d. trauma to one of the bundle branches.

**18. In addition to a QRS complex wider han 0.12 second, which ECG abnormality is a key indicator of an RBBB?**
a. a negative R wave in lead V1
b. a positive R wave in lead V3
c. a negative R wave in lead V3
d. a positive main R wave in lead V1

**19. Which is one of the earliest changes indicative of reversible myocardial injury?**
a. QT prolongation
b. Q-wave deepening
c. ST-segment elevation
d. PR interval shortening

**20. Which leads view the inferior wall of the heart?**
a. V1 through V6 c. I and aVL
b. I, II, and III d. II, III, and aVF

**21. Elevated ST segments in V1 and V2 indicate which type of MI?**
a. anterior wall c. lateral wall
b. septal wall d. inferior wall

**22. Which heart wall is perfused by the circumflex branch of the left coronary artery?**
a. lateral c. anterior
b. inferior d. septal

**23. A patient with ST-segment elevation in leads I, aVL, V5, and V6 may have**
a. an anterior-wall MI.
b. an inferior-wall MI.
c. a lateral-wall MI.
d. ischemic heart disease.

**24. An anterioseptal MI would have ST elevation in leads**
a. V1 and V2 only. c. V5 and V6 only.
b. V3 and V4 only. d. V1 through V4.

**25. Which *isn't* a possible cause of sinus bradycardia?**
a. atropine
b. beta-blockers
c. acute MI
d. a well-conditioned heart

**26. Sinus tachycardia usually is related to**
a. medications.
b. an MI.
c. a physiologic cause.
d. a posterior-wall MI.

**27. One hallmark of AF is**
a. a regularly irregular rhythm.
b. a prolonged QRS complex.
c. an irregularly irregular rhythm.
d. a prolonged PR interval.

**28. Atrial contraction just before ventricular contraction is called**
a. atrial kick. c. synchrony.
b. repolarization. d. f waves.

**29. Which medication may be used to treat AF?**
a. digoxin c. captopril
b. atropine d. diltiazem

**30. Premature ventricular contractions are characterized by wide, abnormal**
a. QRS complexes. c. P waves.
b. U waves. d. T waves.

**31. If your patient is in pulseless VT, which action would you take first?**
a. Call the rapid response team.
b. Obtain a stat 12-lead ECG.
c. Call a code – (arrest code/green button).
d. Ensure adequate vascular access.

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**Topic: Module 2: Understanding a 12 lead ECG, Basic Electrophysiology and 12 Lead ECG Placement.**

We want to ensure that the training/education you have received is effective and relevant. We would be grateful if you would complete this evaluation. Please circle the most appropriate rating.

**(The response range from 1 for limited use, to 5 for very useful)**

**Circle your choice**

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   **1 2 3 4 5**

   **Comments:** _____

   _____

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