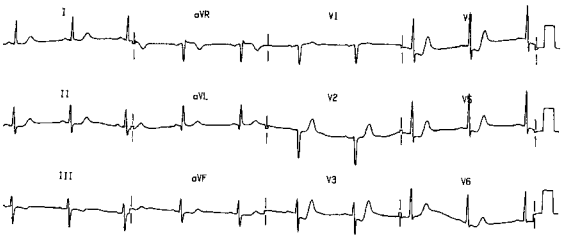
Electrocardiogram Interpretation

For Family Physicians

-- Beginner Workshop



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WARNING:
Parts of this manual are still ***Under Construction***.
I would greatly appreciate any comments about the manual.

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WORKSHOP TEACHING PLAN

Intended Participants:

Family Physicians who wish to review the basics of electrocardiogram interpretation.

Content:

- Review of the basics of ECG interpretation, including how an ECG recording is made, the components of an ECG recording, and normal values.
- Interpretation of some basic pathological conditions, for example abnormal axes, chamber enlargement and uncomplicated acute myocardial infarctions.

Learning Objectives:

Participants will learn to:

1. Understand how an ECG recording is made.
2. Recognize and define the components of the electrical heartbeat.
3. Explain how an ECG recording relates to the electrical physiology and anatomy of the heart.
4. Develop a preliminary understanding for how the ECG can be used to detect cardiac abnormalities.
5. Learn a systematic way to interpret an ECG recording.
6. Set personal goals for learning more about ECG interpretation.
7. Recognize when to refer an ECG recording for expert interpretation.

Format:

The first part of this workshop will be didactic. A detailed handout will be provided and discussed. Participants are encouraged to interrupt and ask questions throughout the workshop. There will be time toward the end to workshop to practice as a group on sample ECG recordings. Patient management will be de-emphasized during the workshop to allow maximum time for discussing basic electrocardiogram interpretation.

**Note: This Beginner Workshop is part of a series of ECG interpretation workshops for Family Physicians that covers beginner, intermediate and advanced levels (see Page 26).*

Introduction

Electrocardiogram (ECG) interpretation is an essential skill for family practice. Family physicians in urban and rural settings use electrocardiograms routinely while assessing their patients in private clinics, emergency departments and hospital inpatient wards. Family Physicians regularly have to make clinical decisions based on ECG interpretations from colleagues and specialists.

For example, during one eight month period in our rural community of about 5,000 residents in a territory covering 6,000 km^2, the community's four family physicians ordered more than1,281 ECGs. This works out to about 5-6 ECG recordings each day in their private clinics and the hospital <Thompson 1992>. This sample did not include ECG's done by one of the physicians in his private clinic.

Accuracy in ECG interpretation clearly is related to training and experience <>. Family physicians must acquire sound ECG interpretation skills before leaving residency training. Once in practice, they must continue to improve their interpretation ability through practice and continuing medical education. Very little has been published on methods for teaching ECG interpretation to Family Physicians.

This manual is an introduction to basic ECG interpretation. It is intended as a primer for beginners, or as a review of the basics for physicians already in practice. Intermediate workshops are being developed that teach interpretation of the large variety of disorders that can be found in 12- and 15-lead ECG recordings. Advanced workshops are being developed for family physicians who wish to learn about the rare and very difficult ECG's that we occasionally encounter in practice.

ECG Interpretation in Rural Family Practice

The rural context of practicing cardiology is characterized by extremely varied pathology, isolation from colleagues and specialists, and little time for continuing medical education <Thompson, Hindle and Rourke 1994>.

Most rural family physicians probably encounter an ECG at least once each day while on rounds in their rural hospital, in their clinics later in the day, or that night while on call in the local emergency department. They either have to do the initial interpretation themselves on the spot and then wait for the official interpretation from an expert reader several days later, or they have to figure out the significance of the expert reader's report when it finally does arrive. Either way, the buck usually stops with the rural family physician.

Assistance from computer interpretation can improve interpretation accuracy for family physicians with intermediate interpretation skills, but the role of computer interpretation remains controversial <Thompson 1992>. Fax machines allow family physicians to consult urban cardiologists in difficult or uncertain cases, but such consultation takes time to set up, and is disruptive for both physicians. For all these reasons, rural family physicians must have good ECG interpretation skills.

There is little literature on the use of ECG's in urban or rural family practice. I reported a study of 1,281 consecutive recordings over an eight month period for a small rural community in Alberta, Canada <Thompson 1992>. As Table 1 shows, ischemic or infarction patterns were by far the most common abnormality, present on 49% of all recordings. Given the obvious importance of being able to recognize ischemia and infarction early for patients in a rural setting, this means that rural family physicians must be able to work through the complexity of ECG interpretation for coronary artery disease accurately and promptly. Rural family physicians must be able to diagnose acute myocardial infarction and start thrombolysis immediately, often without direct access to an expert electrocardiographer.

Table 1. Incidence of ECG findings in 1,281 recordings in a rural community <Thompson 1992>.

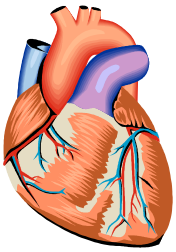
Normal Recordings:	
Normal	31.3%
Diagnoses Often Not Critically Significant:	
Sinus bradycardia	15.0%
1^{o} degree heart block	12.1%
Left axis deviation	11.1%
Left ventricular hypertrophy	5.1%
L atrial enlargement/abnormality	4.6%
PAC, PJC	4.5%
LBBB (complete/incomplete)	4.5%
LAFB	4.2%
Low voltages	3.7%
Right axis deviation	3.2%
RBBB (complete/incomplete)	2.7%
Short PR interval	2.1%
Nonspecific IV conduction delay	1.8%
Long QT interval	1.7%
LPFB	1.0%
Poor R wave progression	1.3%
?Sinus arrhythmia	1.3%
R atrial enlargement/abnormality	0.6%
Increased voltage N for age	0.4%
Right ventricular hypertrophy	0.1%
Diagnoses Often Critically Significant:	
Nonspecific ST, T change	20.8%
Infarct, not acute	18.5%
Arrhythmia other than SB, SA	10.6%
Significant ST, T change	8.7%
PVC	3.7%
Nonspecific J point elevation	3.2%
Consider drug/metabolic effect	2.0%
Pacemaker	1.5%
Acute infarct pattern	1.3%
Limb leads reversed	0.3%
WPW type A	0.3%
2^{o}, 3^{o} heart block	0.1%
Pericarditis	0.03%

Electrocardiogram Basics

Electrical Anatomy of the Heart

ECG recordings capture the anatomy of the heart in two, two-dimensional planes. The goal is to create a three-dimensional image of the heart in the reader's mind from these rather limited slices. During the six decades since 12-lead ECG recording has become routine in medicine, physicians have learned to squeeze the simple 12-lead like an orange, extracting an amazing amount of information from a fairly simple laboratory procedure.

Figure 1. Frontal view of the heart.

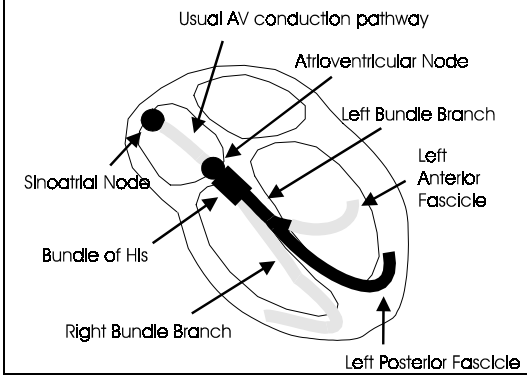


The heart is, of course, three-dimensional (Figure 1). Anatomically it sits roughly in the center of thorax with the center of mass displaced slightly to the patient's left on the frontal view, and is displaced slightly anteriorly on the lateral view. The electrical center of the heart is displaced to the lower left on the anterior view owing to the relative size of the left ventricle.

The position of the heart shifts in the thorax when the patient changes position, which is why ECGs normally are recorded in a supine position. Compromises have to be made when patients cannot tolerate lying flat.

Conduction System. Electrical activity in the heart normally originates from the ***sinoatrial (SA) node***, high in the right ventricle near the vena cavae (Figure 2). The signal then travels down several (typically three) histologically distinct ***atrioventricular tracts***, triggering contraction (depolarization) of the atria as it goes. The signal is held up in the ***atrioventricular (AV) node*** for a few refractory milliseconds, allowing the ventricles to expand with blood from the atria. The AV node is histologically distinct tissue lying in the base of the right atrium.

Figure 2. Schematic frontal view of the conduction system.



Ventricular contraction (depolarization) happens when the signal finally leaves the atrioventricular (AV) node and travels into the Purkinje cells of the ventricular conduction system, first entering a common ***Bundle of His***. The Bundle of His splits into the ***left and right bundle branches***. The left ventricle has more muscle mass than the right because it has to pump against high peripheral vascular resistance in the systemic circulation, while the right only has to pump against lower resistance in pulmonary circulation. The large left ventricular mass has to be depolarized as quickly as the right, and contraction has to proceed from the bottom of the ventricles to the top. To accomplish this synchrony, the left bundle branches into a fan of ***anterior fascicles*** that stick up toward the reader in Figure 2 then travel high to the left front of the heart, and into a fan of ***posterior fascicles*** that travel down and back into the bottom and back of the heart. Once these fascicles have conducted the signal rapidly into the septum and apex of the heart, the myocardium begins to depolarize more slowly, from the inner surface

(***endocardium***) to the outer surface (***epicardium***), squeezing the blood up and out of the ventricles.

The Twelve Traditional Leads

The 12-lead ECG isn't as old as our latest generation of medical students might believe. The three ***standard*** or ***limb leads 1,2 and 3*** have been in use since the late 1800's, but the ***augmented limb leads aVR, aVL and aVF***, and the ***precordial or chest leads V_{1-6}*** only came into use in the 1930's <Wagner page 22>. Although there are now systems that use hundreds of leads, the 12-lead ECG remains an extremely useful tool for family physicians.

The six precordial electrodes create a six-lead image of the heart on the transverse plane, but these leads cover only about 100 ^{o} of that plane (Figure 3).

As Figure 4 shows, the three electrodes that are placed on the two arms and left leg ("limb leads") create a 360^{o} six-lead electrical image of the heart on the frontal plane on the ECG recording (1, 2, 3, aVR, aVL, aVF). The lead on the right leg is system ground. A resistor placed into the limb lead circuit creates the negative pole for the six chest leads.

Figure 3. Transverse view, showing the chest lead positive poles.

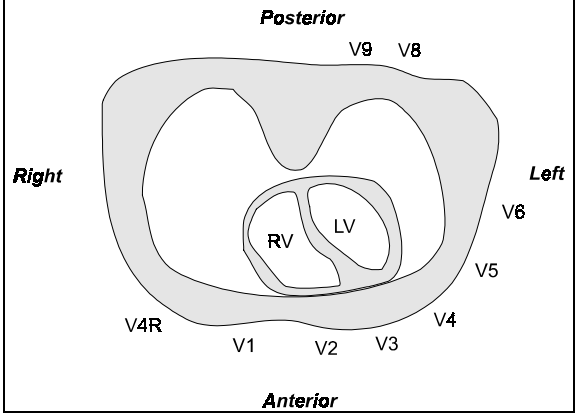
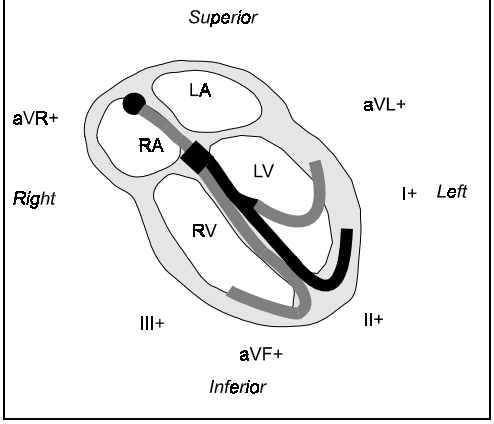


Figure 4. Frontal view, showing the limb lead positive poles.



Accurate lead placement is important. The definitions are anatomically precise and have not changed for decades, but in practice the leads often are placed in highly variable positions. This can lead to significant interpretation errors, especially while comparing serial recordings from the same patient. Figure 5 and Table 2 show how to locate these leads properly.

Figure 5. Anatomic locations of the six precordial leads.

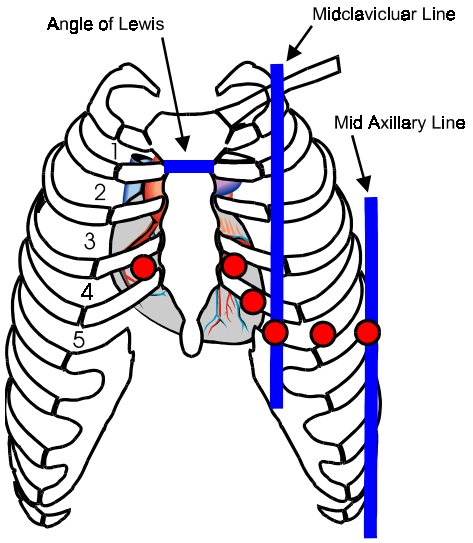


Table 2. Locating the precordial leads.

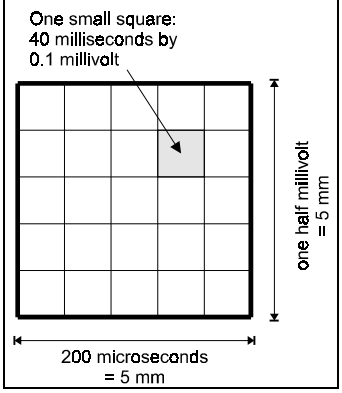
. The four limb leads are placed on the four limbs.
. Put V1 in the 4th intercostal space adjacent to the right sternum.
. Put V2 in the 4th intercostal space adjacent to the left sternum.
. Put V4 in the 5th intercostal space in the mid-clavicular line.
. Put V6 90° to the sternal axis at the same level as V4 and on the mid-axillary line.
. Put V3 halfway between V2 and V4.
. Put V5 halfway between V4 and V6, in the anterior axillary line.

Readers who want to know more about how the three limb leads produce a six-lead image, and how the precordial leads use the limb leads for their negative reference should read discussions about Einthoven's triangle and vector analysis in other textbooks <eg. Wagner pages 20-25>. They are worth the read if you are having trouble understanding how to visualize the electrical model of the heart produced by a 12-lead ECG.

Recently the fifteen-lead ECG came into vogue for the emergency department investigation of patients with chest pain by providing more direct views of the right ventricle and posterior of the heart (Figure 3). ECG leads can also be inserted into the esophagus or right atrium to sort out arrhythmias. ECGs produced with a hundred or more leads are used in electrophysiology and research studies, but that's outside the scope of this manual, fortunately. Fifteen leads is enough for this book!

Structure of a 12-Lead Recording

Figure 6. Grid dimensions.



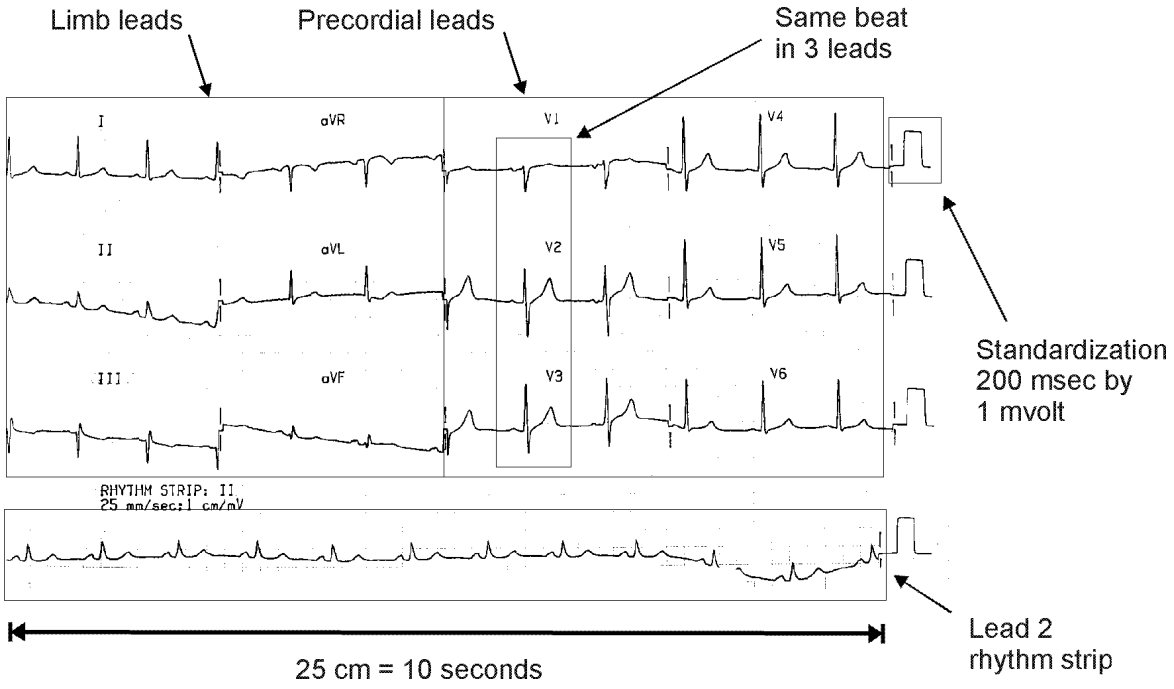
Time is measured for cardiac heartbeats in milliseconds on the x-axis. The standard 12-lead recording is made with the paper running through the machine at rate of 25 mm/sec. This produces tracings along the X-axis with a duration of 40 msec/mm ("one small square"), or 200 msec for each 5 mm thick line ("one large square") (Figure 6). There is enough horizontal room on the page for 25 cm of tracing, representing 10 seconds of real time.

Strength of the electrical signal from the heart is measured in millivolts on the y-axis. The ECG normally records a ***gain*** of 1 mV/cm on the Y-axis (Figure 6). Some machines allow the operator to adjust the Y-axis gain, and might even make the adjustment automatically. Some operators might turn the gain down to half (2 mV/cm) to make the tracing look neater if the QRS complex has high voltages, which could lead the interpreter to miss otherwise

significant Y-axis abnormalities, such as critically important but subtle ST segment elevation or depression.

Figure 7 shows the layout of a standard North American 12-lead electrocardiogram. The six limb leads are grouped on the left, and the six precordial leads on the right. Note that the first three lines all repeat the same heartbeat vertically, allowing the reader to view the electrical heartbeat from three vantage points at once. Most North American machines also add an additional 10 second lead II rhythm strip at the bottom of the page, providing a slightly wider time frame for interpreting lead II morphology, as well as creating a 20 second window of observation for the rhythm. Lead II is used because conduction normally flows down the heart on the lead II axis, toward its positive pole

Figure 7. Layout of a 12 lead ECG recording.



Modern machines allow the operator to make a variety of recordings other than the standard 12-lead, such as 3-lead recordings over a longer time frame, or single lead rhythm recordings over several minutes. These latter recordings produce very small complexes on both axes, but this is a very useful option when patients might have intermittent dysrhythmia.

Positive and Negative Deflections

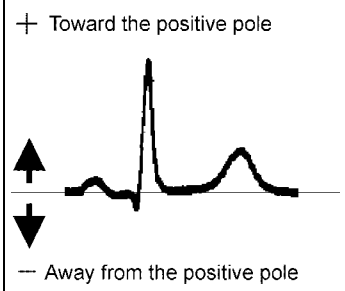
Few principles of electrocardiography are as important to understand as why the ECG pen scribes a line up or down at different points in the heartbeat cycle.

For common reference, each time we refer to a lead, for example when we say, “Lead 2 shows…”, we mean the view from the positive terminal of the lead (Figure 3 and Figure 4). Most of us understand electricity as having a positive pole and a negative pole. The electrical heartbeat can be simplified the same way. Each lead has a negative and a positive pole, as shown in Figure 15 for the six limb leads.

The ***baseline*** or ***isoelectric line*** refers to a time in electrical heartbeat when there is no electrical activity, or when negative and positive forces are balanced. The ECG pen scribes a mark on the baseline for a lead in two situations: when the heart is electrically silent, and when the positive and negative electrical forces in the heart are exactly equal from the vantage point of that lead.

When electricity flows along the conduction system of the heart, or when heart muscle depolarizes and repolarizes, we see those events reflected in every lead. A flow of any of these three events toward the positive end of the lead causes the ECG to scribe a line above the baseline (Figure 8). A flow away causes the ECG to scribe below the baseline.

Figure 8. Positive and negative deflections.



Since the heart is a three dimensional object, electricity rarely flows in the same direction exactly. The ECG shows how much ***net*** flow is occurring toward the lead. The pen will scribe higher above the baseline if any combination of the following occur at that moment:

- net flow is more directly toward the positive pole of the lead,
- net flow is through larger heart mass toward the positive pole of the lead,
- there is less distance between the positive electrode and the site of electrical activity in the heart.

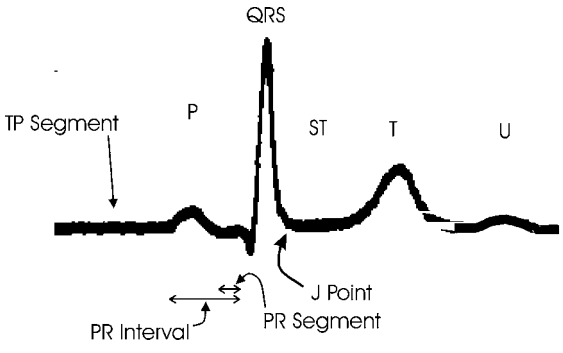
The Electrical Heartbeat

Table 3 and Figure 9 show the classic components of the electrical heartbeat for lead 2, and Table 5 gives criteria for normal measurements and shapes.

Table 3. Components of the electrical heartbeat.

Component	Physiologic Correlation
P wave	Atrial depolarization
PR interval	AV node refractory period
PR segment	Atrial repolarization (in the terminal portion)
QRS complex	Ventricular depolarization
J point	End of ventricular depolarization
ST segment	Blood flow from the ventricles
T wave	Ventricular repolarization
U wave	Uncertain
TP Segment	Rest prior to next heartbeat cycle, and the "isoelectric line"

Figure 9. Components of the electrical heartbeat.



The Normal Sequence. The heartbeat sequence (Figure 9) starts with electrical depolarization exiting the SA node and spreading over the atria, causing the atria to contract and producing the ***P wave***.

The ***PR segment***, the flat line between the end of the P wave and the beginning of the QRS complex is caused by a refractory pause that occurs in the AV node, allowing time for the

blood to leave the atria and enter the ventricles, and for the ventricular myocardium to stretch in preparation for pumping blood into the pulmonary and systemic arterial circulations.

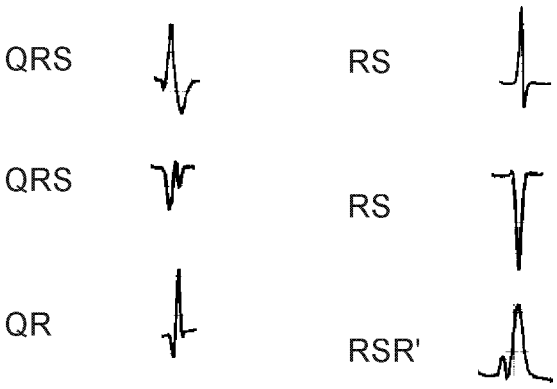
The duration of atrial contraction and relaxation can be measured only incompletely by the ***PR interval***, measured from the beginning of the P wave to the beginning of the QRS complex. This is because atrial repolarization, which produces the normally hidden ***Ta wave*** ("atrial T wave"), normally occurs during the period when relative more massive ventricular depolarization of the QRS complex occurs.

The QRS complex hides the ***Ta wave***, so it is normally not possible to measure the whole period of atrial contraction and relaxation. The Ta wave, when it can be seen, is a very small deflection opposite in polarity to the P wave. If you find an ECG in a patient with atrioventricular block in whom the QRS complex is missing after some P waves, look for the Ta wave at the point where a QRS complex would normally be. It's like finding a four leaf clover, if you're interested in such things.

The ***QRS complex*** is caused by ventricular depolarization. After the PR segment refractory pause, depolarization finally leaves the AV nodal tissue, entering the common Bundle of His, and then splitting into the left and right bundles of the ventricular conduction system. In the left ventricle the depolarizing signal then fans out into the anterior and posterior fascicles of the left bundle branch. The ventricular conduction system causes synchronized depolarization of the ventricular myocardium, ejecting blood from the ventricles.

The term "QRS" is a misnomer, because normal ventricular depolarization looks very different from different lead vantage points. Not every lead shows a sequence of Q wave, followed by R wave, followed by S waves during ventricular depolarization like the classic image shown first in Figure 10. In some leads there might be no Q wave at the start of ventricular depolarization because net electrical flow is toward the positive end of the lead at that moment.

Figure 10. QRS morphology examples.



The conventions used to describe variations in the QRS complex are shown in Figure 10. The first negative deflection is always called a Q wave, any positive deflection is an R wave, and any negative deflection after a Q or R wave is an S wave. The second occurrence of a wave is labeled with a prime symbol (').

The ***intrisicoid deflection*** is the point where the R wave begins its abrupt downstroke. It represents the moment when the epicardial muscle under the lead depolarizes <Chou and Knilans p 13> (Figure 11). It is useful to measure the time from the onset of the QRS complex to the intrisicoid deflection when diagnosing ventricular bundle branch blocks. The greater the delay in the intrisicoid deflection, the greater the delay in ventricular depolarization in the part of the heart represented by any given lead. I think this is the best way to master bundle branch block recognition, once you've climbed the learning curve.

Another short refractory pause occurs during the ***ST segment*** as the heart allows the blood to flow out of the ventricles, then the ventricles repolarize during ***the T wave*** in preparation for the next heartbeat.

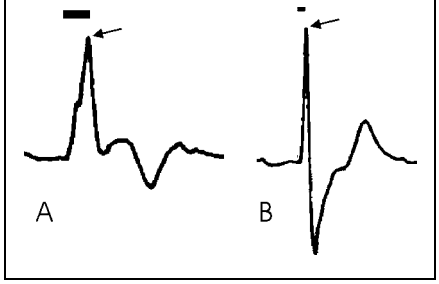


Figure 11. Intrisicoid deflection (arrow). In A the intrinsicoid deflection is delayed (thick bar), in B it is not.

The ***QT interval*** is the time between the start of ventricular contraction and the end of ventricular relaxation. The QT interval is a useful way to monitor some antriarrhythmic drug effects and some diseases.

The ***U wave*** is sometimes seen in normal hearts. The U wave is caused by normal electrical phenomena that occur in the heartbeat cycle after the main mass of ventricular myocardium repolarizes, although the exact mechanism is controversial, like so much about cardiac conduction.

The ***baseline*** or ***isoelectric line*** is a period of electrical inactivity between episodes of depolarization and repolarization that can be used as a reference for detecting abnormal y-axis deflections, such as ST segment elevation or depression. In hearts beating at a normal rate, the ***TP segment*** between the end of the U wave and the start of the P wave usually is clearly visible and provides a good baseline. At higher heart rates or in hearts with abnormal electrical heartbeats, it can be more difficult to find a good baseline. The first part of the PR segment usually is an electrically quiet period in the electrical heartbeat. The latter part of the PR segment occurs as atrial repolarization (Ta wave) is beginning, which might cause that part of the PR segment to be depressed because atrial repolarization occurs away from most leads, except in aVR where the PR segment might be slightly elevated.

The Electrical Heartbeat Image Varies From Lead to Lead. The electrical heartbeat generally appears in a variety of shapes that are quite different from the classic lead 2 appearance shown in Figure 9, depending on the orientation of the lead that recorded the sequence (Figure 3 and Figure 4). Lead aVR, for example, is the only limb lead which is positive in the upper right side of the heart (Figure 4). This means that all of its complexes are inverted relative to the five other limb leads. That's why it has been called the "orphan lead", because it is all alone on the right superior aspect of the heart. At least it was, until the 15-lead ECG introduced lead V_4R to clinical practice to give a better look at the right side of the heart (Figure 3), half way between the inferior aspect seen through lead 3, and the superior aspect seen through lead aVR. All the other leads also have characteristic normal looks, depending on their orientation to the heart's flow of electricity (Table 5).

Normal Criteria for the Electrocardiogram

Table 5 shows criteria for normal shapes and sizes of ECG findings. Any attempt to define ECG normality is difficult because of the variations that occur in healthy persons. In this book we try to give the family physician reader some suggestions for measurement cutoff points and patterns that can be used in a practical way to separate normal from abnormal, but the interpreter must always be slightly suspicious about ECG's that seem to be on the edges of normal. ECG diagnoses must always be thought of in terms of probabilities, even for straight-forward, normal-looking recordings.

The Cut-Off Point Concept for Defining Normal. Most rules that have been suggested for ECG interpretation tend to be cut-off points. There is so much variation in ECG normality that there tends to be some overlap between normal and abnormal.

As an example of the cut-off point concept, consider the PR interval. Wagner points out that the PR interval varies with age (Table 4) <Wagner page 39>. ECG interpretation is difficult because of variations like this. The widely used practical definition for the PR interval of 120-200 msec clearly is a compromise that we use to simplify interpretation on day-to-day basis. Interpreters always need to remember that these rules of thumb are practical cut-offs that in some cases can lead to inaccurate clinical decisions.

Table 4. Variation in the PR interval with age.

Children	0.10 - 0.12 sec
Adolescents	0.12 - 0.16 sec
Adults	0.14 - 0.22 sec

Table 5. Normal criteria. <Chou and Knilans 4th ed. 1996, Wagner 9th ed.>.

Component	Physiological Events	Normal Dimensions	Other Normal Features and Comments
P wave	Atrial contraction (depolarization)and ejection of blood from the atria.	≤ 110 msec. <0.25 mvolts in the limb leads.	• Upright in 1, 2, aVF, V_{4-6}. • Inverted in aVR. • Variable in 3, aVL, V_{1-3}. • Leads 1-3: single peak, or two peaks if they are < 40 msec apart. • Second half in V_1 positive, or < 0.1 mV negative.
Limb lead P wave axis	Net electrical vector of atrial contraction.	0^o to $+75^o$	
PR interval	Atrial contraction, flow of blood into the ventricles and pause before ventricular contraction (atrial depolarization and start of repolarization).	120 - 200 msec.	• Usually measure in lead 2, but ideally use the lead with the largest, widest P wave and the longest QRS duration. • Shorter at faster rates and younger persons. • Longer in larger hearts and older persons. • Normal in children: 100 - 120 msec. • Normal in adults: 140 - 220 msec.
PR segment	Blood flow into the ventricles (end of atrial depolarization and start of repolarization).	Usually isoelectric, but can be slightly displaced opposite to the P wave owing to atrial repolarization.	• Often isoelectric, enabling it to be used as the baseline reference from which other segments are either elevated or depressed.
Ta wave	Atrial relaxation (repolarization).	Opposite polarity to the P wave.	• Occurs during the PR segment and the QRS complex.

Component	Physiological Events	Normal Dimensions	Other Normal Features and Comments
QRS complex	Ventricular contraction (depolarization)	< 120 msec. > 0.5 mV in all the limb leads. > 1.0 mV in all of the precordial leads	• Measure in the lead with the longest QRS duration. • Often hard to find the endpoint of the QRS complex. • *Any* initial upward deflection means that an R wave, not a Q wave, is present. • Q in aVR. • Q often in one of leads 2, 3 or aVF. • Q in lead 3 sometimes 40-50 msec wide. • small narrow Q < 40 msec x 0.1 mV (one small square) in 1, aVL, aVF and V_{5-6}. • R wave increases in amplitude across the precordial leads, usually becoming larger in voltage than the S wave in V_2 - V_4 (transition zone) in adults, in V_1 in neonates. • R wave < 15 mm lead 1, < 10 mm aVL, <19 mm 2, 3, aVF. • Total QRS amplitude above and below the baseline > 5 mm in all limb leads and > 10 mm in all precordial leads. • See Figure 10 for variations in QRS shapes.
Intrinsicoid deflection	Ventricular epicardial depolarization.	Delay < 35 msec in right precordial leads, and <45 msec in left precordial leads.	• Measure from the start of the QRS complex to the start of the *abrupt* downstroke of the R wave. • See Figure 11.
Limb lead QRS axis	Net electrical vector of ventricular contraction.	-30^o to $+105^o$	• See text page 19 and Figure 15. • Adult under age 40: 0^o to $+105^o$. • Over age 40: -30^o to $+90^o$. • Overweight: more leftward, Thin: more rightward.
ST segment	Flow of blood out of the ventricles (end of ventricular depolarization and start of ventricular repolarization).	Usually within 1 mm of the isoelectric baseline.	• Horizontal, with a smooth entry to the T wave. • Limb leads: isoelectric in 75% of normal persons; in the remaining 25% slight elevation is more common. • Precordial leads: slight elevation in 90% of normal persons, more common in men, usually in V_2 and V_3, where it can reach 3 mm. • ST depression is rare in 1, 2, and aVF in normal persons, and is considered abnormal in the precordial leads. • Can be slightly upsloping or downsloping in normal persons, but the degree is important, as is change from previous ECG.
T wave	Ventricular relaxation (repolarization)	< 0.5 mvolts in the limb leads. < 1.0 mvolt in the chest leads.	• More gradual slope in the first half than the second half. • Mild biphasic appearance in anterior chest leads in children. • Upright in 1, 2 and V_{4-6} (except in very young children), inverted in aVR, variable in all other leads. • Upright, flat or slightly inverted in 3. • Upright in aVL, aVF if QRS > 0.5 mV. • Can be inverted in V_{1-2} in adult men, V_{1-3} in adult women (shallowly in V_3). • Always upright in V_5-V_6. • Diphasic T waves: negative-positive always abnormal and positive-negative can be abnormal.
Limb lead T wave axis	Net electrical vector of ventricular relaxation.	Same general direction as the QRS axis.	• Should be within 45^o of the QRS axis on the frontal plane (frontal leads). • Should transition to negative within three leads of the QRS transition on the transverse plan (precordial leads). • Variation from the QRS axis suggests pathology.

Component	Physiological Events	Normal Dimensions	Other Normal Features and Comments
QT interval	Complete ventricular cycle, from contraction to relaxation (depolarization through repolarization).	Varies with the heart rate, see Table _.	• Varies with heart rate, age and gender. • Usually less than half the RR interval at normal sinus rates (65-90 bpm). • Often hard to measure the end of the T wave, so it can be a difficult parameter to measure accurately. • Chou's method: use 40 msec as the upper limit of normal at 70 bpm and add or subtract 20 msec for each 10 bpm change; subtract 70 msec for the lower limit of normal (good for rates of 45-115 bpm) <p 16>.
TP segment	Generally silent phase between the end of one heartbeat and the start of another.	Usually isoelectric	• Can be used as a baseline reference like the PR segment. • Unreliable as baseline at rates where the following P wave starts to merge: use the PR segment instead.
U wave	Cause uncertain (possibly ventricular afterpotentials or ventricular Purkinje repolarization).	Usually 5-25% the amplitude of the T wave.	• Usually same polarity as the T wave.

Not all Abnormal Findings are Pathological. As research into electrocardiography progresses, it is increasingly clear that ECG measurements and patterns in normal persons overlap with pathological ECG findings (Table 6). Well known examples include long PR intervals and the ST elevation of early repolarization in healthy young adults. Rarely, however, these findings genuinely reflect pathology. Careful clinical correlation is always required for such findings.

Table 6. Variant findings that can occur in healthy persons.

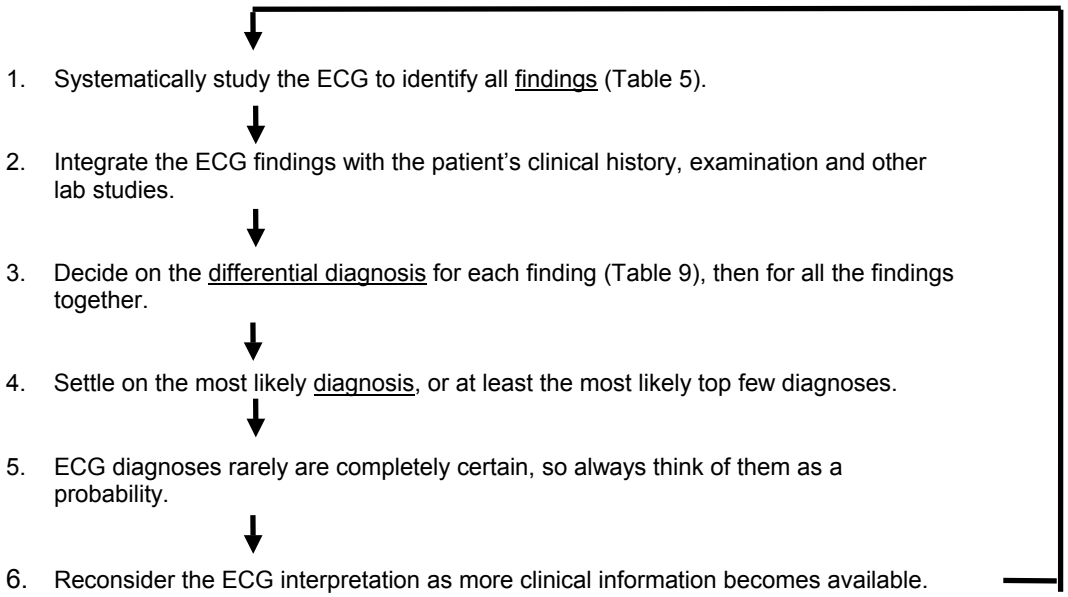
Finding	Comments
Changing P wave morphology	Electrophysiologic studies show that the sinus pacemaker can shift within the SA node, and that the pacemaker impulse can originate in the right atrium outside the sinus node <Chou p 3>.
Long PR interval	Can be normal in young persons and healthy athletes.
High QRS voltages	Can be normal in younger persons, black persons, and healthy athletes.
ST elevation anterior chest leads.	Can represent early repolarization in healthy young adults.
T wave inversion anterior precordial leads.	Can be normal in women.
RSR' pattern in V_1	Occurs in a small number of healthy persons (2.4% - Chou page 17> .
Sinus bradycardia	Can be normal in athletes.*
Heart blocks	Can be normal in athletes.*
Ventricular hypertrophy	Can be normal in athletes.*
ST segment and T wave changes	Can be normal in athletes.*

*As Chou and Knilans point out <page 20>, it remains unclear why some athletes suffer sudden death.

A Systematic Approach to Electrocardiogram Interpretation.

Figure 12 shows how to proceed from reading the ECG to using it for making a diagnosis. First identify all the ***findings*** you can. Then integrate this information with the clinical data. Settle on a list of possible ***diagnoses***, then pick the one or two that are most likely. Be clear in your own mind about how likely it is that the patient has the diagnosis, because all ECG diagnoses are only probabilities, ranging from very low to very high. Finally be prepared to re-evaluate your decision as more ECGs are done and more clinical data become available.

Figure 12. Systematic approach to ECG interpretation.



Finding the Findings

ECG interpretation as a model of human diagnostic thinking has been the subject of considerable research. It is a complex task. There is evidence that both expert humans and good computer programs achieve the same accuracy and "pickup" rate on average, but for different reasons. Humans often know things that the computer doesn't, like the results of other laboratory studies done on the patient, or the history. Computers, on the other hand, can be more infallible and systematic than humans for some features of ECG interpretation. We also know that more experienced and better trained readers are far more accurate than lesser skilled readers, and can see more in an ECG.

The first step to making an ECG diagnosis is to identify normal and abnormal ***findings***. There are many ways to approach an ECG, but I tend to use the one shown in Figure 13.

Identity and History. I start by ensuring that I know the age, gender and history of the patient. I often know the patient in our rural community, even if they are in another physician's practice. I always read their ECG with their file of old ECGs so that I can spot temporal changes that might give clues to the diagnosis. I designed an ECG requisition so that the ordering physician could tell me why they've ordered the ECG, the patient's relevant history, and list relevant drugs that could affect the ECG. We encourage our nurses and laboratory technicians always to write a description of the patient's chest pain on the recording, for example, "*chest pain gone, just relieved by nitroglycerine*", or "*with chest pain severity 5/10*". All of this information promotes more meaningful and accurate ECG interpretations.

Figure 13. Systematic approach to finding findings.

Name, age, gender, history.
Standardization.
Rate.
Rhythm.
- P waves associated with QRS?
- RR interval regular?
- QRS narrow?

Axis.
- P wave
- QRS complex
- T wave

P Waves.
- Height
- Width
- Shape

PR Interval.
- Duration

QRS Complex.
- Height
- Width
- Pattern
- Q waves

ST Segments.
- Deviation from baseline
- Shape

T Waves.
- Height
- Shape

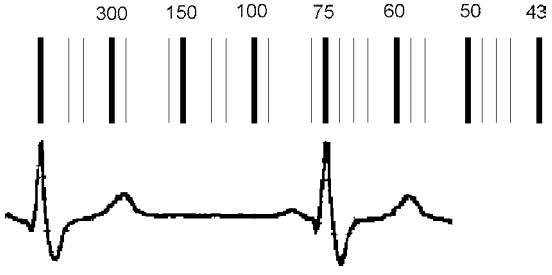
QT Interval.
- Duration

Other findings (see text).
Consider referral.

Standardization. Next note the standardization used to make the recording, both on the Y-axis (voltage, normally one millivolt per cm) and on the X-axis (rate of recording, normally 25 mm/sec, or 200 msec/5 mm). Some machines allow the operator to change the voltage standardization, but that would change all the criteria for interpretation that you would use for spotting Y-axis abnormalities, like ST elevation or the height of the R wave. When using a computer-assisted interpretation, it is important to note the filters that were turned on. I found that our Hewlett-Packard program tends to miss small Y-axis deviations (such as small P waves) when we turned on a 40 Hz filter to avoid background noise produced by our hospital's electrical supply, for example <Thompson 1992>.

Rate. Calculate the rate by knowing that the Y-axis standardization is 25 mm/sec. One small square (1 mm) is 40 msec in duration. Therefore 2.5 cm represents one second. Count the number of complexes across the 25 cm page (10 seconds), multiply by 6 and you get the average rate per one minute. This is a useful way to estimate rate for irregular features, such as the number of PVC's per minute, or the QRS rate in atrial fibrillation.

Figure 14. Rate estimation.



A more convenient way to estimate rate, once you memorize the steps, is shown in Figure 14. If the X-axis standardization is 25 mm/sec, then the following rule works. Find a complex on or very near a thick 5 mm line on the recording. If the next similar complex lies on the next 5 mm line, then the complexes are occurring at a rate of 300 per minute. If the next complex lies on the line 10 mm away, then the rate is 150 per minute. In Figure 14 the ventricular rate is 75 bpm.

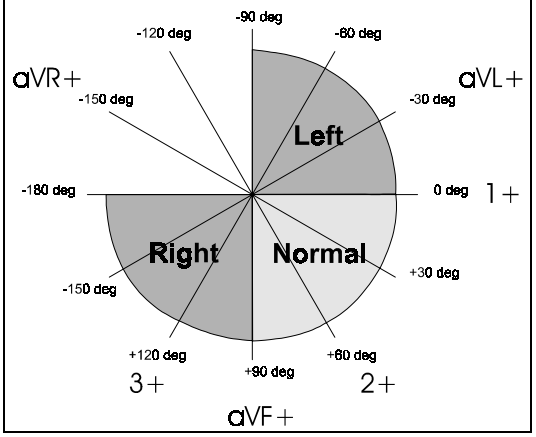
Rhythm. Always start with simple observations. Identify P waves and QRS complexes. It is highly unlikely that you will never see clear evidence of ventricular conduction in a live and awake patient, but P waves can be absent or not connected to the QRS complexes. Is the RR interval regular or irregular? Note whether the QRS complex is wide (> 3 mm or 120 msec) or narrow.

From four basic observations -- *rate, P wave and QRS complex synchrony, RR interval regularity, and width of the QRS complex* – you can begin to sort out the rhythm. Remember that an ECG diagnosis is always a guess about the true state of affairs in the heart. This is especially true with rhythm diagnosis from a 12-lead ECG, so don't feel too frustrated if you can't be certain about the rhythm. Always refer to an expert electrocardiographer if you are not sure, and do not be surprised if the expert isn't sure either.

Look for extra complexes such as premature atrial, junctional and ventricular contractions. Note their proximity to the P, QRS and T complexes.

Axis. "Axis" generally refers to the electrical axis of either atrial contraction (the P wave), ventricular contraction (the QRS complex) or ventricular relaxation (the T wave). The electrical current of a heartbeat normally flows from the SA node into the ventricles, roughly toward the positive pole of lead 2. Although some of the electricity flows away from lead 2's positive pole, the *mean* electrical vectors for the P wave, QRS complex and T wave are roughly toward the positive pole of lead 2, producing a limb lead axis for each that normally lies roughly between 0^o and 90^o.

Figure 15. Limb lead axis.



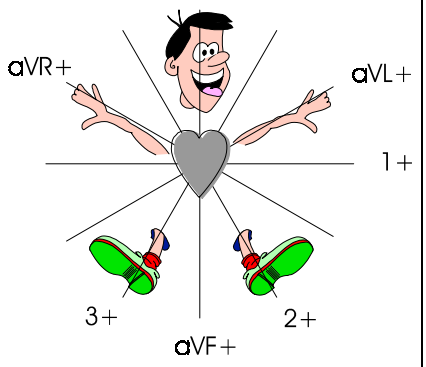
The normal axis range ranges -30^o to 105^o over the whole population, but varies with age. Normal variation in electrical axis is wide, so wide that any attempt to define a cutoff is arbitrary. Figure 15 shows the general definitions for "left" and "right" axis. Most normal persons lie between $+30^o$ and $+ 75^o$ <Chou page 6>. Very small numbers of normal hearts have a limb lead QRS axis lying even beyond -30^o to $+110^o$ <ref?>.

Table 7. Wagner's axis definitions.	
Left axis deviation (LAD) any age group.	-30^o to -90^o.
Right axis deviation (RAD) in adults.	$+90^o$ to $+180^o$.
Extreme axis deviation (EAD) any age group.	-90^o to -180^o.

Authors differ in their definitions of axis deviation. Chou suggests using the rule of calling the axis normal if it lies between 0^o and $+105^o$ in persons under the age of 40, and -30^o and $+90^o$ over 40 <Chou and Knilans page 6>. Wagner's suggestions for adult axis deviations are shown in Table 7 <Wagner page 44>.

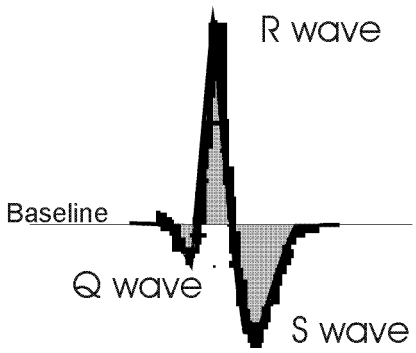
There are lots of tricks for estimating the limb lead axis, but the first problem is to remember where the leads lie on the frontal plane. One trick for visual learners is to picture the limb leads around the "limb lead kid" shown in Figure 16. His right arm corresponds with lead aV Right, his left arm with lead aV Left, and his feet with leads 2 and 3.

Figure 16. The limb lead kid.



First decide whether the net QRS is positive or negative in lead 1. To do this, estimate visually whether the ***net areas*** between the X-axis and the QRS curve appear to be larger above or below the X-axis baseline, as demonstrated in Figure 17 (this is calculus, remember?). In Figure 17 the net area is negative. Don't be fooled by the taller R wave into thinking that this QRS is positive. If the net area is negative, then the current is flowing away from the positive electrode of lead 1.

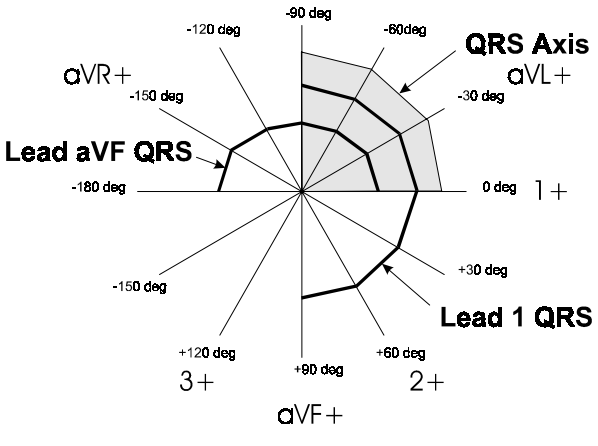
Figure 17. Using area to determine whether the QRS is positive or negative.



Take the Left Bundle Branch Block ECG on page 33 as an example. In lead 1 the net QRS area is clearly positive, meaning that the net flow of electricity is toward the positive pole of lead 1 (Figure 18).

Next look at the lead lying 90° from lead 1: lead aVF. Leads 1 and aVF are called **orthogonal pairs** because they lie 90° to each other. Because the QRS in Lead 1 is positive, the QRS axis must lie on the right side of aVF, as demonstrated in Figure 18. Because the net QRS area in aVF is negative, then the net flow of electricity has to be away from aVF's positive pole. Combining the information from leads 1 and aVF, the QRS axis must lie somewhere between 0° and -90°.

Figure 18. Sample axis calculation.



Then look at another pair of leads that lie 90° to each other, say leads 2 and aVL. Do the same for the remaining pair, leads 3 and aVR . You'll be able to narrow the QRS axis to one of the twelve 30° slices of the limb lead 360° pie this way. You can get more precise, to within about 5-10°, if you determine whether the net QRS is a lot or a little positive or negative in the various leads.

Sometimes the axis cannot be calculated readily. The easy answer is to call it "indeterminate". The harder solution is to calculate two axes, one for the initial half of the QRS and the other for the terminal half <Marriott 8th page 38>. I must admit that I rarely do this.

What's the point of straining your brain by calculating the axis? Clinically, not an awful lot most of the time. It can be very useful to know when the axis suddenly shifts from normal to the left or right. If this happens during an acute MI, then you have to be suspicious that one of the left bundle fascicles is being damaged by infarction, raising the possibility that your patient will enter high-grade heart block. You'd like to know about that if you practice in a rural hospital without ICU capability for immediately managing high-grade heart block.

The QRS axis is part of the definition for a number of heart disorders. Unusual discordance between the T wave axis and QRS axis (>50^{o}) is said by Wagner to be a sign of myocardial abnormality <Wagner page 38>. The electrical axis must be calculated on every 12-lead ECG, even though hurts until you get used to doing it.

P Waves. Study the P waves for the condition of the atria. Note the width, height and shape of the P waves. Calculate the P wave axis and compare it to the QRS axis for clues regarding underlying pathology.

QRS Complex. Study the QRS complex for a clues about the condition of the ventricles. Note the width, height, shape and pattern of the complex.

Q waves can be difficult to assess. They can appear normally (Table 5), but their absence can be abnormal in some circumstances (Table 9). Always assess Q waves relative to other clinical information about the patient. One of the most difficult problems is to decide whether to call Q waves abnormal in the inferior leads. Small Q waves often appear normally in leads 1, aVL, aVF and V_{5-6} because initial depolarization in the ventricles is from left to right in the septum, directed away from those leads. If Q waves are not clearly diagnostic, then call them "nondiagnostic".

Lead Reversal. Whenever the P wave or QRS morphology doesn't quite make sense or seems to have changed dramatically, consider improper placement of the leads on the patient. Limb lead reversal is more common than reversing the precordial leads. A common clue to limb lead reversal is that the QRS and T wave polarities reverse an become positive in lead aVR, where they normally are negative because aVR is the only lead out in the upper right side of the heart (see the normal ECG example on page 31). At the same time the polarities also reverse in aVL, where they normally are positive.

ST Segments. Study the ST segments separately from the T waves first, then together with the T waves. Look for deviation from the baseline, and shape of the ST segment. Shapes include flat, upsloping, downsloping, curved and straight. Note where the ST segment is relative to baseline 2 mm (80 msec) after the J point. Note carefully how the ST segment enters the T wave: is it smooth, or does it have an ominous abrupt entry?

T Waves. Note height and shape of the T waves. Shapes include smooth symmetry, asymmetry sharp peaking, inversion, and biphasic. Check again for the way the ST segment enters the T wave.

QT Interval. Calculate the QT interval to spot unusually short or long duration.

Other findings. Finally, carefully study the ECG for clues to more subtle and rare findings. Some examples include:

- delta waves signifying accessory pathways in Wolf-Parkinson-White syndrome.

- the Osborne wave in hypothermia.
- pathological U waves in some ischemic and metabolic disturbances.

Consider Referral. When in doubt, always refer the ECG to an expert electrocardiographer for an opinion.

Putting Findings Together to Make a Diagnosis

The goal of ECG interpretation is to identify clues about the following aspects of the heart:

- ***anatomy*** (e.g., chamber enlargement, orientation of the heart in the chest).
- ***electrical conduction problems*** (e.g., dysrhythmia, heart blocks).
- ***tissue pathology*** (e.g., ischemia, infarction, cardiomyopathy, pericardial inflammation, presence of accessory pathways, metabolic disturbances, effect of drugs).

Table 9 shows some differential diagnoses for some abnormal findings. Unfortunately there is no substitute for lots of ECG-reading experience in order to make this table work. I've tried to make the table a bit easier to use by sorting the diagnoses into three categories of abnormal: common, uncommon-but-high-urgency, and uncommon-but-low-urgency.

Sensitivity, Specificity and Predictive Values.
Sensitivity is the likelihood that the finding will be present on the ECG when the disease is present in the patient. ***Specificity*** is the likelihood that the finding will be absent on the ECG when the disease is absent in the patient. ***Predictive values*** are more useful: they tell us the likelihood that a patient with (or without) the ECG finding actually has (or does not have) the disease associated with that finding.

Table 8. Sensitivity, Specificity and Predictive Values.

	Disease Present	Disease Absent	
Finding Present	A	B	$N_{positive}$
Finding Absent	C	D	$N_{negative}$
	$N_{disease}$	$N_{nodisease}$	

Sensitivity	True positive tests, number of ECG's with the finding where the associated disease was present.	$\frac{A}{Ndisease}$
Specificity	True negative tests, number of ECG's without the finding where the disease was absent.	$\frac{D}{Nnodisease}$
Positive Predictive Value	Probability that a patient with the finding actually has the associated disease.	$\frac{A}{Npositive}$
Negative Predictive Value	Probability that a patient without the finding actually does not have the associated disease.	$\frac{D}{Nnegative}$

Unfortunately the ECG is neither very sensitive nor specific for many types of heart disease. In autopsy studies of left ventricular hypertrophy, for example, the ECG has a sensitivity of only about 50%, while specificity ranges as low as 21% <Wagner page 51>. In another example, the initial ECG is diagnostic for acute myocardial infarction in only 50% of cases < Gibler & Aghababian, p 142 in Aufderheide and Brady>. This lack of sensitivity, specificity and predictive value means that clinicians have to apply findings from the ECG to each case with care, integrating clinical information from other sources.

It never ceases to amaze me how much information an expert electrocardiographer can get from a simple 12-lead ECG. Nevertheless, I am also impressed that at the end of most virtuoso performances, the expert always qualifies the interpretation as a likelihood. Many 12-lead diagnoses are little more than guesses that should be further evaluated with more precise and accurate methods, or simply by close clinical observation of the patient. I think it makes sense to always report interpretations as, "*The ECG suggests* …". For practical purposes this qualifying statement is always left out, but it should always be assumed.

Cardiac Pathology Varies With Time. The ECG shows a very small window of time: only ten seconds, enough for only twelve heartbeats at 72 beats per minute. Many types of cardiac pathology appear and disappear over very short periods of time. Intermittent arrhythmia and ischemia can come and go within a few minutes, for example.

ECG Diagnoses can be Secondary. In many cases ECG findings can be caused by diagnoses that are secondary to yet another diagnosis. For example bundle branch block can be secondary to ischemia caused by inflammatory diseases like the coronary artery disease in Kawasaki's Disease. The lesson here is to always think through the reasons for the finding in any given patient.

Table 9. Differential diagnoses for some abnormal findings. 

	Differential Diagnoses		
Finding	**Common**	**Uncommon but high urgency.**	**Uncommon but low urgency.**
Sinus bradycardia	Healthy athlete. Sleep. Drug effects.	Acute myocardial infarction. Drug effects. Intoxication. Raised intracranial pressure. Hyperkalemia. Myocarditis. Quinidine toxicity. Digitalis toxicity.	Vasovagal syncope. Vomiting. Micturition. Hypothyroidism. Idiopathic degeneration of the sinus node. Cardiomyopathy.
Sinus tachycardia	Anxiety. Febrile illness. Simple exertion.	Acute ischemia or infarction. Acute intoxication. Thyrotoxicosis. Congestive heart failure. Anemia. Drug effects. Pericarditis. Myocarditis.	Congenital heart disease.
Atrioventricular block other than first degree.	Coronary artery disease. Age-related fibrosis.	Ischemia or infarction. Pericarditis. Myocarditis. Intracranial hemorrage. Myocardial contusion. Digitalis toxicity. Quinidine toxicity.	Cardiomyopathy.
Biphasic P wave with a negative terminal force in V_1 (> -40 msec)	Left atrial enlargement.		
Wide notched P wave in the inferior limb leads (>120 msec)	Left atrial enlargement.		
Tall, normal duration P waves in the inferior leads (> 2.5 mvolts).	Right atrial enlargement.	Cor pulmonale. Pulmonary embolism.	Left atrial enlargement.

	Differential Diagnoses		
Finding	**Common**	**Uncommon but high urgency.**	**Uncommon but low urgency.**
Rightward frontal P wave axis.	COPD.		
Prolonged PR interval (> 200 msec)	Normal variant. First degree AV block.	Acute or evolving injury to the conduction system.	Cardiomyopathy. Dilantin effect.
Elevated PR segment in aVR and depressed PR segment in lead 2	Normal variant.	Pericarditis.	
Prominent R wave in V_1		Acute posterior MI. Wolf-Parkinson-White Syndrome.	
RSR' in V_1	Normal variant.		Right ventricular hypertrophy.
Abnormal Q waves.	Old or subacute subendocardial or transmural myocardial infarction. Conduction defects.	Pulmonary embolism. Intracranial hemorrhage.	Normal variants. Ventricular hypertrophy. Cardiomyopathy. Mitral valve prolapse - systolic click syndrome.
Absence of Q waves.	Bundle branch block.		
Wide QRS complexes	Ventricular origin. Bundle branch block.	Pacemaker effect. Acute conduction defect. Wolff-Parkinson-White Syndrome. Quinidine toxicity. Procainamide toxicity.	Ventricular enlargement.
Large amplitude QRS complexes.	Normal variant. Ventricular hypertrophy. Bundle branch block.		Cardiomyopathy.
Low amplitude QRS complexes.	Obesity. COPD.	Pericarditis. Pericardial effusion. Hypothyroidism. Pneumothorax.	Cardiomyopathy.
Poor R wave progression.	Normal variant. Nonspecific abnormality. Ventricular hypertrophy.	Old anterior myocardial infarction.	
Delta wave.	Wolff-Parkinson-White Syndrome.		
Osborne wave.		Hypothermia.	
Wide S wave in left leads (lead 1, V_5, V_6).	Right bundle branch block.		
Delayed intrisicoid deflection.	Bundle branch block.		Ventricular enlargement.
Frontal left QRS axis deviation.	Normal if mild. Left fascicular hemiblock. Ventricular hypertrophy.	Acute, evolving injury to the conduction system.	
Frontal right QRS axis deviation.	Normal variant. COPD.	Metabolic disturbance. Cor pulmonale.	Right ventricular hypertrophy.
ST segment elevation.	Normal variant. Myocardial infarction. Bundle branch block.	Myocardial ischemia. Pericarditis. Aortic dissection. Pulmonary embolism. Pneumothorax. Intracranial hemorrhage. Myocardial contusion. Hyperkalemia. Pacemaker.	Mitral valve prolapse - systolic click syndrome. Ventricular aneurysm. Cardiomyopathy.

	Differential Diagnoses		
Finding	**Common**	**Uncommon but high urgency.**	**Uncommon but low urgency.**
ST segment depression.	Normal variant. Nonspecific abnormality. Rate-related. Myocardial ischemic disease. Ventricular conduction defect. Ventricular hypertrophy.	Pericarditis. Aortic dissection. Pulmonary embolism. Intracranial hemorrhage. Wolff-Parkinson-White Syndrome. Hypokalemia. Pacemaker.	Digitalis effect. Mitral valve prolapse - systolic click syndrome. Cardiomyopathy. Chronic hypomagnesemia.
Tall T waves.	Normal variant. Conduction defects. Ventricular hypertrophy.	Acute or subacute ischemia or infarction. Hyperkalemia. Intracranial hemorrhage. Acute hypomagnesemia.	
T wave inversion.	Normal variant. Nonspecific abnormality. Ventricular conduction defect. Ventricular hypertrophy. Non-paced beats in patients with pacemakers.	Myocardial ischemia. Subacute myocardial infarction. Pericarditis. Myocarditis. Myocardial contusion. Following Stokes-Adams seizures in complete heart block. Intracranial hemorrhage. Spontaneous pneumothorax. Pulmonary embolism. Wolff-Parkinson-White Syndrome. Pacemaker.	Post tachycardia. Mitral valve prolapse - systolic click syndrome. Cardiomyopathy.
Diphasic T waves.	Normal variant. Ischemia or infarction, especially if biphasic negative-positive.		Cardiomyopathy
Prolonged QTc interval.	Myocardial ischemia (most common cause). Drug effects.	Metabolic abnormality.	Normal variant. Quinidine effect. Procainamide effect. Sotalol effect.
Shortened QTc interval.		Hypercalcemia.	
Negative U wave.		Highly specific for organic heart disease.	
U wave inversion.		Ischemic heart disease.	Left ventricular hypertrophy.
Tall U wave.		Electrolyte imbalance. Hyperthyroidism. Intracranial hemorrhage. Digitalis. Hypokalemia. Hypercalcemia.	Quinidine. Procainamide. Mitral valve prolapse.

Table 9 cannot be used on its own to generate differential diagnoses. In most cases the abnormal finding alone is not a sufficient criterion to establish a diagnosis – the correct combination of findings is usually needed. Table 9 does not include a number of conditions, for example congenital heart disease

Other ECG Interpretation Workshops.

This beginner workshop is an introduction to the basic concepts. In intermediate and advanced ECG interpretation workshops we will explore abnormal findings and differential diagnoses in much greater depth.

The ***intermediate workshops*** are intended for Family Medicine residents and Family Physicians in practice. Each cover a single group of disorders:

- Acute coronary ischemic syndromes (completed),
- Acute myocardial infarction (completed),
- Nonspecific ST changes (completed),
- Bundle branch blocks,
- Tachycardias,
- Heart blocks and bradycardias,
- Rare-not-to-be-missed ECG diagnoses,
- Drug effects and metabolic disturbances.

The ***advanced workshops*** are intended for expert-level Family Physicians already in practice. These are usually physicians with advanced privileges in ECG interpretation, or physicians with a special interest in ECG interpretation. The workshops are unstructured. A cardiologist will be asked to attend to help the group work through difficult ECG's. Workshop attendees will be asked to bring their own difficult ECG's, and to help each other learn advanced tricks about ECG interpretation.

A ***Maintenance of Competence Workshop*** is on the drawing board. This will be an examination-style experience. Attendees will be given a series of ECG's to interpret for 90 minutes, then the ECG's will be discussed in the next hour, after a short break. The answer sheets will be collected before the discussion session. A scored result will be scored anonymously and mailed privately to the participants later, with their score plotted on a histogram of scores for the entire group. This workshop will be of interest to the following participants:

- Family Physicians planning to challenge the College of Physician and Surgeons' ECG Interpretation examination.
- Teachers of Family Medicine residents.
- Family Physicians in practice who wish to assess their own ability.
- Expert interpreters who wish to check their own ability.
- Researchers who wish to determine the range and variation of ECG interpretation competence among Family Physicians.

Learning Objectives in Electrocardiogram Interpretation for Family Physicians

Family Physicians must be able to recognize and manage patients with significant electrocardiogram abnormalities. This is especially true for Family Physicians working in rural hospitals, urban emergency departments, urgent care centres and inpatient wards. Clinic office patients are less acute, but all Family Physicians who work in non-urgent ambulatory clinic settings must know how to use the electrocardiogram appropriately.

Table 10. Learning objectives in ECG interpretation for Family Physicians.

1. Knows when to perform an electrocardiogram.
2. Is able to interpret the electrocardiogram such that he or she can identify the findings listed in Table 10.
3. Is able to integrate their electrocardiogram interpretation with clinical information to make the diagnoses listed in Table 10.
4. Knows when and how to refer an ECG to an expert electrocardiographer.
5. Appropriately manages the patient based on the interpretation of the ECG.

Table 11. Minimum list of ECG findings and diagnoses that a family physician should be able to recognize.

General	• Components of the ECG tracing. • Normal versus abnormal ECG. • Lead reversal.	**AV Block**	• 1^{o} AV Block. • 2^{o} AV Block, types 1 and 2. • 3^{o} AV Block.
Rate	• Estimate rate to within 10 bpm.	**Axis (P, QRS and T)**	• Estimate direction to within $10^{o.}$ • Type of abnormality (left or right shift).
Rhythm	• Normal sinus rhythm. • Sinus arrhythmia. • Sinus bradycardia. • Sinus tachycardia. • Atrial focus other than sinus. • Premature atrial, junctional and ventricular beats. • Atrial fibrillation. • Atrial flutter. • Junctional rhythm. • Idiopathic ventricular rhythm. • Ventricular tachycardia. • Ventricular fibrillation. • Pacemaker.	**Conduction Abnormalities** **Chamber Enlargement**	• Complete and incomplete left and right bundle branch blocks. • Left anterior and posterior fascicle blocks. • Nonspecific interventricular conduction delay. • Short PR interval. • Wolf-Parkinson-White Syndrome. • Left and right atrial enlargement. • Left and right ventricular enlargement.
Ischemic Syndromes	• Nonspecific ST segment and T wave changes. • Ischemia (anterior, lateral, high lateral, inferior, posterior). • Old myocardial infarction (anterior, lateral, inferior). • Acute myocardial infarction (anterior, lateral, high lateral, inferior, and posterior). • Q wave infarction. • Non Q wave infarction. • Infarction of uncertain age.	**Miscellaneous Disorders**	• Repolarization. • Pericarditis. • Changes suggesting drug or metabolic effect: Bradycardia secondary to beta blockers and digoxin. Hyper- and hypokalemia. Hyper- and hypocalcemia. QT prolongation secondary to medications.

Maintaining ECG Interpretation Competence in Practice

A physicians' ECG interpretation ability improves by reading ECG's continuously. It is clear from the literature that ECG interpretation ability varies widely among Family Physicians, and that less experienced physicians are less capable of interpreting ECG's accurately than more experienced ECG readers.

One solution in a small rural Ontario medical staff is that each Family Physician reads the ECG's for the group each week in rotation. Every three months the group audits everybody's interpretations to keep up skills.

In Alberta the College of Physicians and Surgeons certifies Family Physicians to bill for ECG interpretations through a written examination. This helps to identify more expert Family Physician interpreters in each rural community who can do all the ECG interpretations for the group, as well as provide feedback to everyone about their own interpretations.

In some rural communities and urban Family Physician clinics all ECG's are routinely sent to an expert interpreter (Family Physician or internist) for reading. This practice ensures that findings and diagnoses are less likely to be missed, and gives feedback to the Family Physician about their personal interpretation skills.

The Family Physician, urban or rural, is always on the front line when dealing with a patient's fresh ECG. Whether ECG's are routinely referred to an expert or not, all Family Physicians must continuously work to improve their ECG interpretation skills.

See Page 26 for a description of the planned Maintenance of Competence Workshop.

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Appendix: Examples of 12-lead Electrocardiograms

Example 1: Normal recording.

Example 2: Acute inferior myocardial infarction.

Example 3: Left bundle branch block.

Normal Electrocardiogram.

Acute Inferior Myocardial Infarction.

Left Bundle Branch Block.