The EKG—Interpretation of Common Arrhythmias

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Early in this century, Einthoven discovered that he could pick up the electrical activity produced by conduction of impulses through the human heart. He amplified these waves and developed the electrocardiographic machine. Soon it was made to record this activity on a moving strip of paper, the electrocardiogram. In 1918 the EKG was used in studying the response of the heart to anesthesia and surgery.¹ Now, more than 50 years later, this remarkable device is available to all of us.

Some anesthetists, however, feel that this monitor is generally unnecessary. It is my hope that today we will spark an interest in this useful monitor, which has become so sophisticated, yet so simple, that we may constantly observe the heart’s activity on an oscilloscope. It is imperative to be forewarned that the EKG records only the potential for contraction and in no way records the force of myocardial contraction, or whether the ventricles contracted at all.

It has been said that cardiac arrest with no warning is very rare. Some warnings may be palpated by monitoring peripheral pulses, blood pressure, etc. Some, however, may go unnoticed; the EKG, if understood, may well record these potentially unobserved warnings. It is important for the anesthetist to know when EKG changes tell us of impending disaster, for arrhythmias may not wait for specialists to appear. The purpose of this paper is to point out similarities and differences between harmless and alarming EKG changes. This is not a course in interpretation of EKG. Although a brief review is presented, it must be taken for granted that the reader has some background in EKG interpretation as the more serious arrhythmias are demonstrated.

Recently, when discussing electrocardiograms, many authors and speakers have replaced the “K” with “C”. However, I agree with Dr. Dubin, in that it is more logical to use EKG rather than ECG because it avoids confusion with EEG, which records brain waves.² Therefore, we will use EKG throughout this paper.

ELECTROPHYSIOLOGY

The heart is a pump, no more and no less. It is the only means by which blood flow is maintained. Other factors will influence blood flow, but the beginning of flow, pulmonary or systemic, is the sole responsibility of the heart. For effective contractions, one absolute prerequisite is the conduction of impulses by means of depolarization, repolarization and polarity. These three phases or states represent changes of electrical charges, from negative to positive and back to negative again, at the cell membrane. It is the means by which not only the heart, but also the nervous system conducts impulses.

In this explanation of electrophysiology we must consider two compartments: intracellular and extracellular. Intracellular concerns contents within the cell or cytoplasm. Extracellular consists of plasma and

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interstitial or tissue fluid. These compartments are separated by a semipermeable membrane which influences the migration of fluids and substances. The membrane is very selective, permitting some ions to pass and rejecting others. Sodium is found predominantly outside the cell, while potassium is predominantly inside. These positively charged sodium and potassium ions (cations) have corresponding negative charges (anions).

Since the concentration of K+ is greater on the inside of the cell or nerve fiber, the bombardment by kinetic motion is from the inside toward the outside. The membrane is somewhat permeable to K+ and permits some of it to escape. Therefore, a negative charge is left inside the cell and a slight excess of positive charges is outside the cell. Na+, in the meantime, is bombarding from the outside toward the inside, but while at rest, the membrane is impermeable to Na+ (probably caused by the so-called sodium pump) so the Na+

is not permitted to enter. Figure 1-a represents a cell or nerve fiber at rest, or in polarity.

Figure 1-b demonstrates events which occur when a stimulus is applied. The membrane becomes more permeable and sodium ions move intracellularly, leaving their negatively charged ion outside the cell. This causes a change from positive to negative excess outside the membrane; it is said to be depolarized. In this way impulses are conducted from one area to another. If depolarization occurs in a muscle cell, it will, normally, cause contraction. Anything capable of increasing the permeability of a membrane permits depolarization. The hypoxic cell becomes more permeable and will depolarize with ease.

Membranes are eager to return to their "resting state" and must repolarize. To repolarize, ions must shift and return to a slight excess of positive charges outside the mem-

![Diagram](image)

Fig. 1. (a) Cell membrane in polarity.

![Diagram](image)

Fig. 1. (b) Ionic changes during transmission of an impulse.
brane. Since it seems more difficult to move Na+ than K+, potassium immediately moves outside the cell giving a positive charge outside and leaving its anion (−) in the cell. But, for true polarity to occur, the positive charge must come from sodium ions, not from potassium ions. This activates the pump which forces Na+ outside the cell and K+ inside. The cell is now in polarity and resting, but ready for another impulse.

During depolarization, a cell or fiber cannot accept another stimulus; neither can it accept a stimulus during the early phase of repolarization, when only potassium ions have changed the electrical charges. The membrane is then said to be refractory. When the ionic balance is produced by the sodium pump or the last phase of repolarization (again refer to Figure 1-b), it gradually becomes more and more susceptible to responding to an impulse. The stronger the impulse, the more likely it is to succeed. Normally the cell or fiber is not expected to respond until it has reached the state of polarity.

Muscle cells forming the myocardium are unique, in that they are each capable of depolarizing independently in given circumstances, and in conducting the impulse from one cell to another. This may be either lifesaving or fatal. Normally there are areas in the heart which set the pace for depolarization, as well as tissue capable of efficient conduction of impulses. The sinoatrial (S-A) node should be the pacemaker (Figure 2-a). Its cells remind us of a caution light which flicks on and off. Why it has the ability to start depolarization is not clearly understood, but its cell membranes seem to become permeable and depolarize very readily.

Once depolarization begins in the S-A node the impulse travels through both atria. We have usually stated that there were no nerve fibers for conduction through the atria; however, researchers have found bundles of specialized conducting tissue which facilitate the spread of impulses through the atria. These special pathways, called the anterior, middle, and posterior internodal tracts (Figure 2-b), conduct the impulse from the S-A node over the right atrium to the A-V node. Another pathway, the anterior interatrial myocardial band (or Bachmann’s bundle), conducts the impulse from the S-A node to the left atrium.
The main impulse, however, seems to direct its path toward the S-A node. Here it is briefly detained while the atria contract. Then the impulse is transmitted through the A-V bundle, down the left and right bundle branches to the Purkinje system, and finally to the ventricular muscle. Now contraction of the ventricles should occur. Usually, wherever depolarization first begins, repolarization first occurs as well. In the ventricles this does not apply. The septum with its bundle branches is the first to depolarize, but repolarization occurs first near the apex of the heart. After contraction, repolarization occurs and muscle cells relax. Cardiac filling begins. In less than a second the cardiac cycle of depolarization, repolarization and polarity repeats itself. It is this electrical activity in the heart which is picked up and amplified by the electrocardiograph. It tells us in which direction the impulse is being conducted. However, to repeat, it is incapable of telling us whether a contraction occurred.

THE ELECTROCARDIOGRAPH

The apparatus for graphing the electric current is somewhat complicated, but may be thought of as a dial indicating whether the current is flowing toward the positive side, the negative side, or not at all (which is indicated by having the arrow point toward zero). Therefore, in visualizing an electrocardiograph (the instrument) the positive terminal is to the right, the negative terminal to the left, and the center, between negative and positive, is zero. The arrow will fluctuate between these points as energy is released and record the fluctuation on a graph or oscilloscope. Refer to the instrument in the upper left-hand corner of Figure 3. By using this same diagram we can demonstrate how we pick up and record the electrical energy by bringing a wire with an attached electrode from the positive side of the instrument and place it on the left arm, and placing another wire and electrode from the negative side on the right arm. If the instrument contained a visible arrow, fluctuations produced would be labeled as recordings from Lead I.

For Lead II, a wire from the positive side of the instrument is connected to the left leg and a wire and electrode from the negative side to the right arm.

To form Lead III, the wire and electrode from the positive side is applied to the left leg, the one from the negative side, to the left arm. Figure 3 and the statements above apply only to the standard limb Leads I, II, and III. They do not apply to chest leads or unipolar limb leads.

For our convenience, the manufacturers have simplified the applica-
tion of these leads by having one electrode each for the left arm, right arm, and left leg. If an apparatus has one for the right leg, it is usually only for the purpose of grounding the instrument.

CARDIAC VECTORS

After applying the electrodes as directed above, we see in Figure 3 an equilateral triangle drawn around the area of the heart. This illustrates that the two arms and the left leg form apexes of a triangle around the heart. The electrode applied to the subject's left arm has a positive electrode in the upper circle and a negative electrode in the lower circle. To complete each apex, you will note that both circles on the right arm will contain negative electrodes. The left leg electrodes are both positive. This is the arrangement when the electrodes are applied on the proper limbs (Figure 4). In the formation of the standard leads we must have the following for:

—Lead I : Positive on the left arm and negative on the right arm
—Lead II : Positive on the left leg and negative on the right arm
—Lead III: Positive on the left leg and negative on the left arm.

During polarity of the myocardium, the charges at the cell membrane are positive outside and negative inside, and no electric current is generated. The electrocardiograph should register on the zero (or isoelectric) line. The moment a stimulus is received, these electrical charges reverse and the membrane becomes positive inside and negative outside. This is depolarisation and it generates currents of electricity which spread into the tissues surrounding the heart much the same as eddies form when a pebble is dropped into smooth water. These currents (or potentials) are recorded by the electrocardiograph which in turn "writes" an electrocardiogram.

It is necessary to remember that the wave of depolarization sends its peak (or arrowhead) in the direction where the greatest change or potential will be required. Since the apex of the heart contains the largest number of muscle cells, it will also contain the largest number of positive charges outside the cell. Therefore, the longest and strongest arrow formed during depolarization of the ventricles will shoot directly toward the apex. This imaginary arrow formed by the direction of current flow, and always pointing toward the positive charges is said to be a vector. There will be many vectors formed during conduction, but the instrument will record only one vector at a time and will be the "strongest and longest" vector formed at that particular time.

In considering depolarization of the ventricles, we would normally have

![Fig. 4. Transposing a cardiac vector.](image-url)
a vector shooting toward the apex of the heart as shown in Figure 4. In order to transpose this vector (or arrow) on the respective leads, we may draw broken lines from the end (or tail) and head of the vector to each lead as demonstrated in Figure 4. You will note that the transposed vectors of each lead point in the positive direction of the electrodes applied to the subject’s limbs, which formed the imaginary triangle. Normally the total lengths of the vectors forming Leads I and III should equal the length of the vector in Lead II.

Each time an impulse is conducted through the heart, the instrument should record the direction in which the vector is moving. We can now discuss the formation of records or electrocardiograms (hereafter called EKG) in normal conduction, and then discuss how an EKG can tell us of an abnormal condition.

THE ELECTROCARDIOGRAM

On the EKG, voltage is registered on the perpendicular lines; the time required for the spread of depolarization is recorded on the horizontal axis. Time and voltage are expressed as duration and amplitude. The cardiologist, in his accurate interpretation, customarily speaks of duration in seconds and amplitude in millivolts.

Fig. 5. (a) Transposing and recording the normal P wave in Lead II.

As stated previously, depolarization normally starts in the S-A node and causes depolarization of the entire atria with the strongest and longest vector heading toward the A-V node. This vector, if transposed (as shown in Figure 5-a) and then recorded on the EKG, forms the P wave for each lead. After depolarization is complete, the muscle should contract. The effectiveness of contraction is not recorded on the EKG.

The A-V node is apparently able to delay conduction momentarily, for the EKG next records a short span on the zero line. It is quickly followed by transmission or depolarization of both ventricles. This is conducted by the A-V bundle, which branches into the right and left bundles and the Purkinje fibers. These are highly specialized nerve fibers which transmit much more quickly than the conduction tissue of the atria. Because a larger area must be depolarized, and the apex with its numerous cells is located here, the vector will be long. The combination of nerve fibers and many cells will result in a high but narrow wave on the recording. It is named the QRS complex. See Figure 5-b. To repeat once more,

Fig. 5. (b) Transposing and recording the normal QRS complex in Lead II.

after depolarization is complete, the ventricles contract. Both ventricles should contract at the same time.

Depolarization must be followed by repolarization if the cells are to be ready to receive another impulse. Repolarization also generates an electric current, but with lower voltage than depolarization. The atria
probably repolarize during depolarization of the ventricles, and this activity is not usually recorded. Repolarization of the ventricles forms the third wave known as the T wave. Usually, wherever depolarization first began, repolarization will first occur also. But remember, in the ventricles, this does not apply. The septum with the bundle branches is the first to depolarize, but repolarization occurs first at the apex of the heart. Another vector is formed and will register on the EKG (Figure 5-c). Remember that the vector-head always points toward positive charges. This will cause the vector to again point toward the apex, becoming shorter and shorter as repolarization goes to completion.

![Figure 5. (c) Transposing and recording the normal T wave in Lead II.](image)

A typical EKG of a complete cardiac cycle consists of a group of waves which Einthoven named the P wave, the QRS complex, and the T wave. The P wave represents depolarization of the atria; the QRS complex depolarization of the ventricles, and the T wave is the wave of ventricular (not atrial) repolarization. The intervals between these waves are also significant and are represented in Figure 6. The P-R interval indicates the electrical activity in the atria, and the Q-T interval indicates ventricular activity. The only communication between the atria and ventricles is the A-V bundle, which provides an excellent dividing point. Figure 6 also demonstrates why abnormalities occurring before the QRS involve the atria, whereas those of the QRS and the area following involve the ventricles.

Because we are concerned with the shapes, rhythm, and frequency of these three waves, we must measure time and amplitude. The height (or depth) of a wave and the horizontal axis for time (duration) are both measured by small millimeter squares. Every fifth millimeter square is drawn in bold black lines. Our measure then consists of large bold squares and small light squares. Going up or down from the isoelectric line one bold square represents 0.5 millivolts; two bold squares would be one millivolt (Figure 7). Five bold squares along the horizontal axis measure one second. Therefore, one bold square represents 0.2 second.

![Figure 6. Relationship of EKG to atria and ventricles.](image)

![Figure 7. Horizontal lines indicate time. Perpendicular lines indicate voltage.](image)
and one small square only 0.04 second. Although many oscilloscopes do not display this graphic picture, we should develop an eye for these norms for they tell us the duration of any part of the cardiac cycle. Time and voltage are of considerable importance in interpretation.

NORMAL SINUS RHYTHM (NSR)

Sinus rhythm simply means that the heart is receiving its impulse from the normal pacemaker, namely the S-A node. If, then, conduction follows in the normal manner, we would expect a normal tracing as seen in Figure 8. Generally we say it “fires off” about 70 times each minute. Therefore we should see two QRS complexes between five bold squares. The S-A node is under the influence of vagal and sympathetic impulses, and bradycardia or tachycardia may occur.

![Fig. 8. Normal sinus rhythm. Rate 74/min.](image)

Every anesthetist should develop key points in observation of the EKG complexes as they race across the screen. Because they are here one minute and gone the next, this must be accomplished by practice, preferably many times each day. Although there are many things to keep in mind, the following suggestions will sharpen perception.

1. Regarding the QRS complex
   a. Are the R-R intervals in rhythm?
   b. What is the duration of the R-R intervals?
   c. Is the QRS wide or narrow?
   d. Is the QRS preceded by a P wave?
   e. Is the main deflection above or below the isoelectric line?

2. Regarding the P Wave
   a. Is it in rhythm?
   b. What is its rate?
   c. Is it above or below the isoelectric line?

3. Regarding the P-R interval
   a. Is there a normal time interval or is it delayed?
   b. Is there correlation between the P wave and the QRS complex?

It takes time to develop a quick eye in detecting unusual deflections or rhythms at a glance. A person who drives a car has learned to observe numerous danger signals; likewise, one can learn EKG signals. To begin with, watch the peaks, viz. the R waves. Sing the rhythm to yourself, R-R-R, and fix it in your mind. In this way you will quickly detect bradycardia or tachycardia, although they may stay “in rhythm.” If an R wave appears out of rhythm much too early or much too late, immediately follow its line to the isoelectric line and automatically notice whether it is wide or narrow and see whether it is preceded by a P wave. Next, notice the deflection of the T wave. This routine will certainly help you become proficient in recognizing arrhythmias as they occur, and will also give you terminology which will be meaningful when communicating with those assisting you in interpretation and therapy.

ATRIAL ARRHYTHMIAS

Only a few atrial arrhythmias will be mentioned. Causes and therapy will not be discussed, as time does not permit. Many excellent texts are available for this purpose, some of which are listed in the bibliography.
Bradycardia may normally be present in athletes. Under anesthesia and surgery it may occur due to drugs or reflexes which activate the vagus nerve or depress sympathetic response. Figure 9 shows about seven bold squares between the R-R intervals. Its rate would be about 45 per minute. In bradycardia the R-R waves may or may not be in rhythm.

Fig. 9. Bradycardia. Rate 45/min.

Sinus tachycardia signifies that the S-A node is responsible for the increase in rate. There are other types of tachycardia, some of which will be discussed shortly. In sinus tachycardia, as the R-R waves rush across the screen, immediately determine whether it is preceded by a P wave. Sinus tachycardia is usually more easily corrected than other tachycardias. Figure 10 demonstrates sinus tachycardia. Its rate, with about 2.5 bold squares between the R-R interval would be about 120/minute. Ventricular filling occurs during the rest period between the T wave of one cycle and the P wave of the next. It is obvious that cardiac filling, and thereby cardiac output, is curtailed according to increased rate.

Fig. 10. Sinus Tachycardia. Rate 120/min.

Sinus arrest indicates failure of the S-A node to initiate an impulse, and one or more dropped beats are evident (Figure 11). Usually the second pacemaker, the A-V node, or the ventricle will set the pace. It is possible for total arrest to occur and resuscitative measures must be instituted. Usually this block is preceded by sudden vagal stimulation to a very susceptible S-A node.

Nodal rhythm usually occurs when the S-A node fails to set the pace and the A-V node becomes the pacemaker. This may occur only on occasion, or the entire rhythm of the heart may be controlled from this area for some time. The ventricles receive their impulse in the normal manner and the QRS complex should be unchanged. The A-V node exhibits a slower rate than the S-A node. Usually its rate is about 60/minute. The abnormality is evident in the P wave because the deflection will be below the line. This occurs because the vectors are shooting through the atria in the opposite direction, and when transposed on Einthoven’s triangle, will point toward the negative electrodes (Figure 12-a). As a pacemaker, the A-V node may present three possible changes.

Fig. 11. Sinus Arrest.

Fig. 12 (a) Transposed vector.
in the P wave; (1) The inverted P wave may appear just before the ventricular complex, and the absence of the P-R interval is obvious (Figure 12-b); (2) depolarization of the atria may coincide with depolarization of the ventricles. Figure 12-c shows that since the electrocardiograph will only record the longest and strongest vector, a normal appearing QRS appears, but no P wave is evident; (3) depolarization of the atria may occur after depolarization of the ventricles. Then the inverted P wave occurs after the QRS (Figure 12-d.) In nodal rhythm the atria do not have time to empty the blood they contain into the ventricle before the ventricle contracts. This will decrease cardiac output to a small degree. Although the cause and significance of nodal rhythm is not clearly understood, it occurs frequently during anesthesia. It may warn of increased vagal tone.

_Nodal tachycardia_ is tachycardia initiated by the A-V node, whereas _atrial tachycardia_ indicates that the focus starting depolarization is found in areas other than the A-V or S-A nodes. On an oscilloscope, it is almost impossible to differentiate between the two. For convenience the term _supraventricular tachycardia_ is often applied.\(^1\)

![Fig. 13. Wandering Pacemaker.](image)

_A wandering pacemaker_ is illustrated in Figure 13. The pacemaker shifts between the A-V and S-A nodes and causes constant changes in P-R intervals.

![Fig. 14. Premature atrial contraction.](image)

_Premature atrial contractions_ (PAC) are also called auricular extrasystoles. They are out of rhythm. The impulse or pacemaker may be in some part of the atria other than the S-A node, then spread over the muscle and stimulate the A-V node. This may result in a normal QRS and T wave, although it will be out of rhythm. Figure 14 illustrates a premature atrial contraction. You will note that by the time the S-A node would normally initiate an impulse, the ventricles are in refractory and do not accept the impulse. Therefore, we often see a longer pause between the T wave of the premature complex and the next P wave.

All hearts, either normal or abnormal, may show occasional PAC’s. Lack of sleep, excessive intake of coffee, or smoking in excess may be
the recording makes it very obvious. The cause. It is necessary for the
anesthetist to distinguish PAC's from ventricular premature contractions
which may be much more serious.

VENTRICULAR ARRHYTHMIAS

Premature ventricular contractions should be recognized by every anes-
thesist. Normal persons may have occasional PVC's, and although they
are a nuisance they may not be danger-ous. When they occur on the oscil-
loscope as we monitor patients under anesthesia, they should alert us to the
possibility of approaching fibrillation. PVC's may arise from any point
(focus) in the ventricular muscle, and on occasion fire off with regulari-
ty from that focus. On the other hand, there may be many foci firing
off in different areas of the ventricles, showing multifocal premature con-
tractions; this demonstrates an ex-
remely irritable heart and calls for
immediate therapy. In order to rec-
ognize these dangerous contractions on
the EKG, it will be helpful to use the
diagrams in Figure 15. First of
all, two normal complexes are
recorded. Then an irritable focus in the
upper right ventricle initiates prem-
ture depolarization. The longest
and strongest vector will still aim for
the apex. Conduction will not move
through the bundle nerve fibers of
the conducting system, but will la-
boriously move from one muscle cell
to another. This takes time and the
QRS will be broad and tall. Increased
time is not demonstrated in the

transposed vectors in Figure 15-a but
Repolarization should begin in the
apex, but since it took a long time
for the apex to become depolarized,
the area where depolarization first
occurred will begin the process of
repolarization. This will cause an
inverted T wave to the QRS because
vectors always direct their point
toward the area of positive charges.
The next complex is normal, and all
seems well.

Fig. 15. (b) P.V.C. — Focus in the apex.

In Figure 15-b a premature con-
traction developed in the apex of the
heart. Depolarization will now move
up toward the base of the heart, and
therefore the vector will point in that
direction. It, too, will be conducted
through muscle and produce a wide
QRS. In transposing this vector on
the triangle, it is obvious that the
vector in each of the three leads
points toward the negative electrode.
This will cause the electrocardiograph
to record below the isoelectric line
and the deflection is negative. In
PVC's, as stated before, repolariza-
tion usually begins in the same area
where depolarization began; likewise
in this instance, the T wave will be
inverted to the QRS which is in the
positive direction. The T wave may
be used to verify that a PVC occurred
if we remember that when the QRS
is above the line the T wave will be
below the line and vice versa.

Not only during anesthesia, but at
any time, we would probably consider
that PVC's are of clinical significance:

Fig. 15. (a) P.V.C. — Focus in the base of
the heart.
Fig. 16. Multifocal P.V.C.'s.

Fig. 17. A salvo of three P.V.C.'s.

Fig. 18. P.V.C. on the descending part of the T wave.

Fig. 19. Bigeminy.
(1) if there is more than one every five minutes.
(2) if they are multifocal as illustrated in Figure 16.
(3) if there are salvos of 2 or 3 beats as demonstrated in Figure 17.
(4) if they occur on the descending part of the previous T wave as in Figure 18.
(5) if they appear in bigeminy, Figure 19.
(6) if they appear in trigeminy, Figure 20.

Generally speaking, any of the above need to be reported and treated, for any one of them may be the fore-runner of ventricular tachycardia, which is extremely serious. It gives no rest to ventricular muscle, does not permit filling of the ventricles, and contractions are most ineffective. Ventricular tachycardia may be the result of one focus (as in Figure 21) or of many foci. The QRS is wide and is often the only complex visible on the EKG. The QRS complexes may be above or below the line. With multiple foci, some are above and some below the line. As cardiac output decreases, the myocardium becomes ischemic and fibrillation follows. See Figure 22.

Ventricular fibrillation is often a terminal event because the impulses keep spreading from one muscle cell to another. There is no contraction as such. The muscles "squirm," and the myocardium resembles a "bag of worms." Polarity, depolarization and repolarization occur here, there, and everywhere. No descriptive waves
can be identified and soon, because no activity is present, we have a “flat EKG.”

If, however, an alert team is at hand, some of these disasters may be successfully reversed, bringing a happy ending to the drama. Treatment of the irritable ventricle may have been effective while it exhibited PVC’s, or it may have been performed during ventricular tachycardia. If, however, it progressed to ventricular fibrillation, defibrillation will be needed. The purpose of defibrillation is to throw every muscle cell into depolarization at once, thereby encouraging repolarization to follow in the normal manner. Needless to say, in order to be successful, the stage must be set. There must be adequate oxygen, CO₂ elimination as well as proper electrolyte balance, which includes acid-base balance. If possible, the original cause for the fibrillation must be eliminated.

Since ventricular fibrillation and cardiac arrest are not unknown entities during anesthesia and surgery, we need to be on the alert for factors which may cause these serious arrhythmias. This is not the time or place to divert into causes, but it seems appropriate to call attention to the importance of recognition of a relatively innocuous atrial premature contraction and the ability to distinguish it from the possibly serious premature ventricular contraction. Figure 23 illustrates a PAC where the QRS is out of rhythm (note irregular R-R-R intervals). As the eye follows the QRS to the isoelectric line, we note that it is not high or wide and is preceded by a P wave. This P wave may at times be inverted. We also note that the T wave is not inverted to the QRS. However, in a premature ventricular contraction as shown in Figure 24, we again have an R wave out of rhythm; it is very tall and wide and is not preceded by a P wave (the wave directly in front of it is the T wave of the previous complex). We also note immediately that the T wave is inverted to the QRS. These few facts make it possible for us to distinguish between atrial and ventricular premature contractions. Monitoring peripheral pulses is important; however, it is usually difficult to identify the more serious arrhythmias in this manner.

**Fig. 24. Two Premature Ventricular contractions.**

**ATRIOVENTRICULAR BLOCK**

Heart block or atrioventricular block may occur during anesthesia and surgery, and although treatment may or may not alter the condition, we should be familiar with the terminology. Heart block is a term used to indicate a delay of impulses through the usual conducting system from the A-V node to the ventricles. It may be incomplete (partial) or complete. Often, incomplete heart block is divided into first degree block and second degree heart block. Complete heart block is called third degree heart block.

In heart block of the **first degree** there is an increase in the P-R interval. Occasionally this interval may be as long as 0.48 seconds, but a ventricular contraction does follow. Usually there are normal P waves and
normal QRS complexes. If the P-R interval is longer than 0.20 seconds, it is considered to be heart block.\(^9\) On the EKG strip in Figure 25, the P-R intervals of the first three complexes are normal, and no block exists. The last three complexes demonstrate first degree block. The P-R duration is about 0.28 seconds.

![Fig. 25. First degree heart block.](image)

**In second degree heart block** there is also a delay in the P-R interval, but dropped beats occur. There may be a dropped beat on occasion, or conduction may fail with regularity and frequency, as seen in Figure 26, where every other impulse is blocked.

![Fig. 26. Second degree heart block.](image)

In this instance ventricular rate is one-half of the atrial rate. Wenckebach's phenomenon (Figure 27) is also a second degree block not uncommonly seen in the operating room. Here the P-R intervals become progressively prolonged on each succeeding beat, until one atrial impulse is not conducted to the ventricles at all. Some second degree blocks may have no definite ratio and may demonstrate an extremely alarming EKG tracing, when the anesthetist can only ask whether there can possibly be a greater emergency than the one demonstrated on the screen of the oscilloscope.

![Fig. 27. Second degree as in the Wenckebach’s.](image)

**Third degree or complete heart block** is seen in Figure 28. Here the atria continue to contract at their own rate, but the conduction pathway to the ventricles is totally blocked, and the ventricles establish their own rhythm by regulation of their own pacemaker below the block. The rate of the ventricular pacemaker is slow, usually between 30 and 40 beats per minute. In the classic complete heart block, there are regular P waves and often relatively normal-appearing QRS complexes, but each of these is totally unrelated to the other. In Figure 25, the P waves have been labeled. They appear about every third bold square. If they do not appear at the appointed time, it is because the QRS or the T wave occurred during that time. It is notable that the QRS complexes appear about every eighth bold square. This gives it a rate of 38 per minute, which is very typical for complete heart block. In this strip the QRS's appear wider than usual. They are suggestive of an enlarged heart, showing that additional muscle cells have developed and conduction is slightly prolonged. The T waves are not inverted to the QRS. Having these facts at our disposal, we may conclude that we have complete heart block.

![Fig. 28. Third degree or complete heart block.](image)
SUMMARY

This paper has only touched on some of the arrhythmias and conduction anomalies which the EKG may demonstrate. We have only utilized the three standard leads, but from these we may expect about 80 to 90 percent accuracy in diagnosis. In summary it is obvious that the EKG is of value to the anesthetist for observing rate, rhythm, conduction of impulses, the origin of the pacemaker, and the origin and frequency of premature contractions.

If the anesthetist has made these observations and evaluated their significance, she is in a better position to respond in the best interest of human physiology by "maintaining" the patient throughout surgery.

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