CHAPTER 11
ECG Basics

In this chapter we will study the development and distribution of the electrical activity of the heart, which we measure on the body surface as the Electrocardiogram (ECG).

Chapter 11 Learning Objectives

- **Anatomical structures involved in myocardial depolarization**
- **Time delays involved in myocardial depolarization**
- **The basic components of the surface ECG**
- **Understand the basic dipole model**

The heart is, in part, an electro-mechanical device. As we learned in an earlier chapter, the multitude of heart cells behaves electrically as a syncitium, due in most part to the intercalated disks. That is to say that an electrical event in one part of the heart can spread throughout the heart. The electrical activity of the heart normally begins at the junction of the vena cavae and the right atrium – called the sino-atrial node (SA) node. The SA node cells are electrically unstable, in that their membrane potential is always changing due to “leaky” ion channels. The voltage inside the cell is less than the outside of the cell and we say that the trans-membrane potential is negative. As positive ions slowly leak into the cell, the trans-membrane potential reaches a threshold that causes an avalanche of ion cells to open resulting in a large inrush of positive ions, causing an event termed depolarization. Depolarization is the transition of a negative trans-membrane potential into a positive trans-membrane potential. This depolarization which started in the SA node traverses the heart according to Figure 11-1.

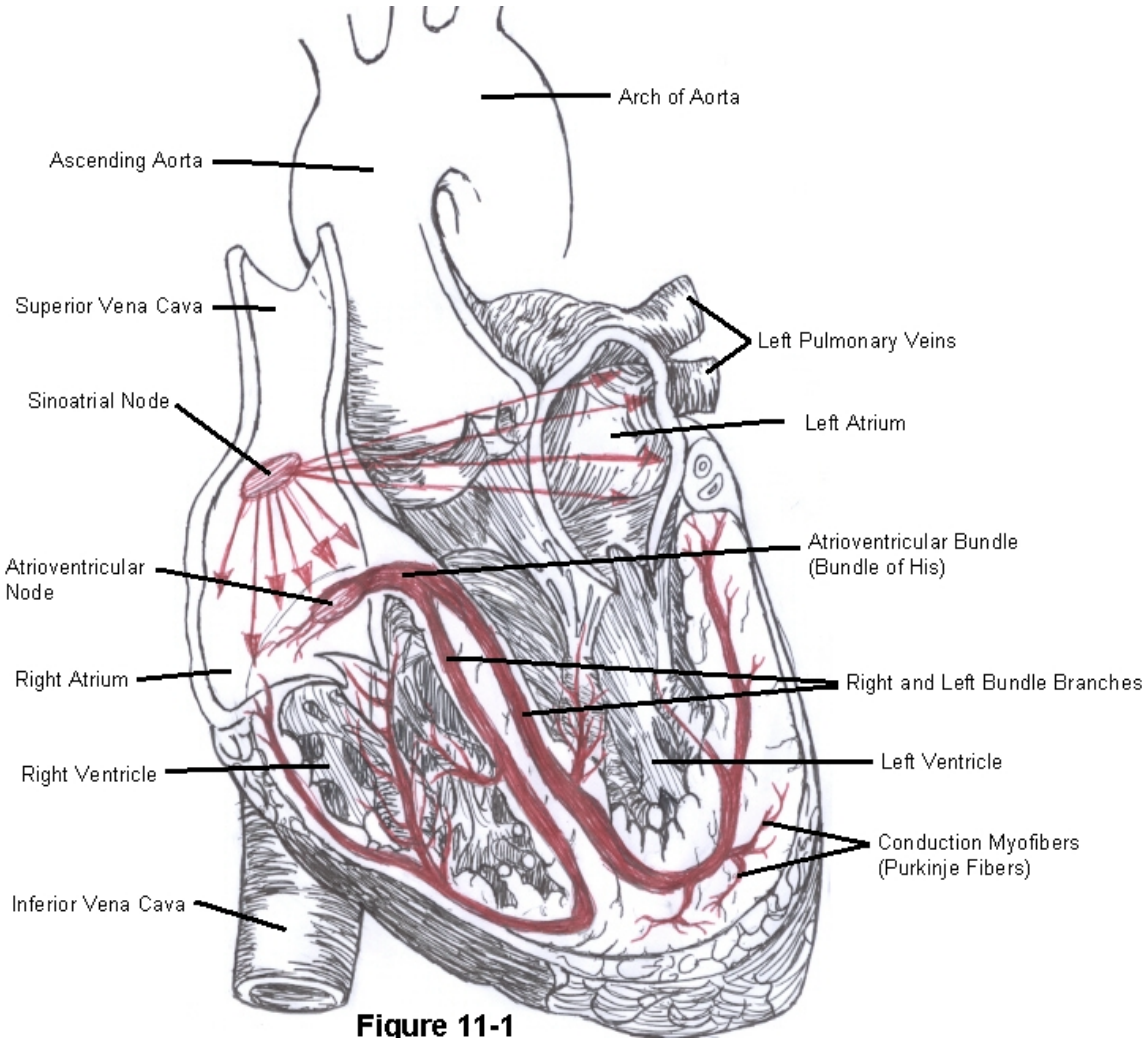


Figure 11-1

After the SA node depolarizes, the depolarization wavefront spreads across the right atrium and then across the left atrium. After the atria depolarize, the wavefronts collide at another region of electrically unstable region, termed the atrio-ventricular (AV) node. Here the cells located within the AV node delay the depolarization event. Nerve fibers connected to these cells, the bundle of His, (sorry no bundle of her) then conduct the depolarization throughout the myocardium of the ventricles, ultimately delivering the depolarization through the Purkinje fibers.

The Bundle of His and Purkinje Fibers are used to speed and optimally direct the depolarization into the much thicker walls of the ventricles. Interestingly, if the SA node fails to depolarize, the AV node has an unstable membrane potential and can act as a back-up pace-maker. If both the SA and AV node fail to depolarize, the ventricles have an unstable membrane potential and can act as a pacemaker. The SA node membrane potential oscillates at a higher frequency (e.g. ~70 bpm) than either the AV node (~50 bpm) or the ventricles (~40 bpm).

Figure 11-2 shows a summary of the times for depolarization of the cardiac cells throughout the heart. Note the large delay in the AV node from (0.04 to 0.16 secs) .

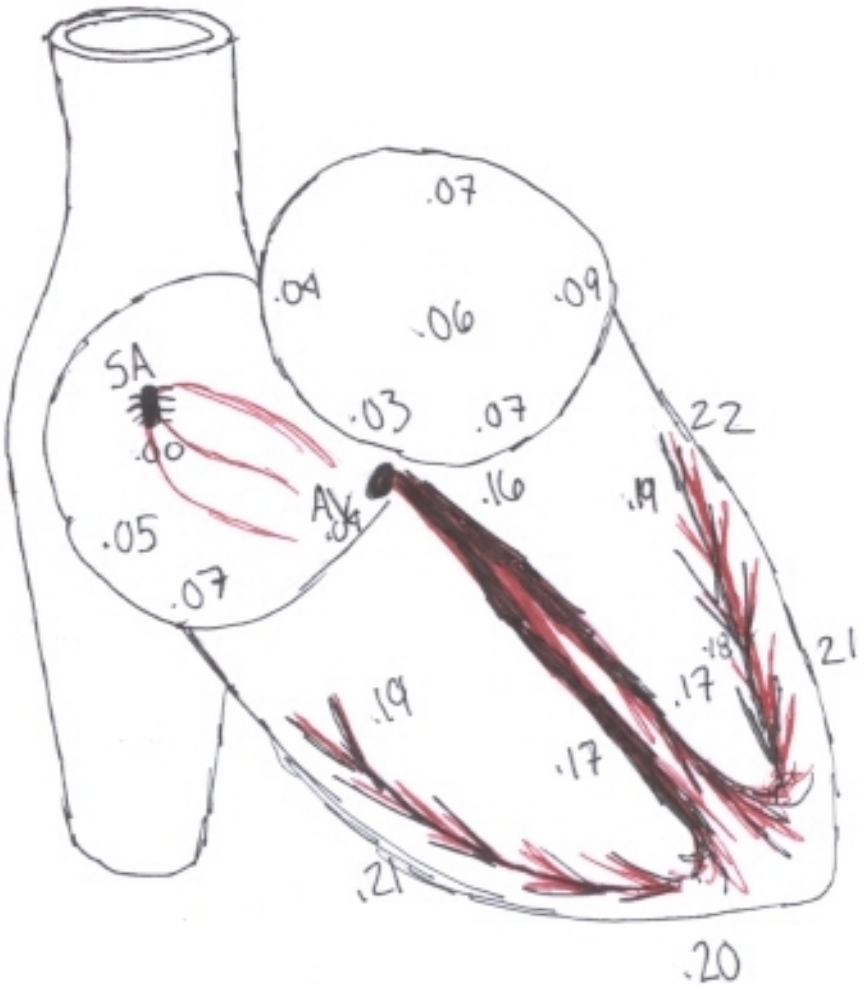


Figure 11-2 Transmission of the cardiac impulse throught the heart, showing the time of appearance (in fractions of a second) of the impulse in different parts of the heart.

Figure 11-3 shows another anatomical view of the depolarization path from the inter-nodal pathways (SA to AV) through the AV node and into the conduction fibers. The Bundle of His splits into two nerve bundles – the right and left. The right bundle branch mostly innervates the right ventricle and the left bundle, the left ventricle.

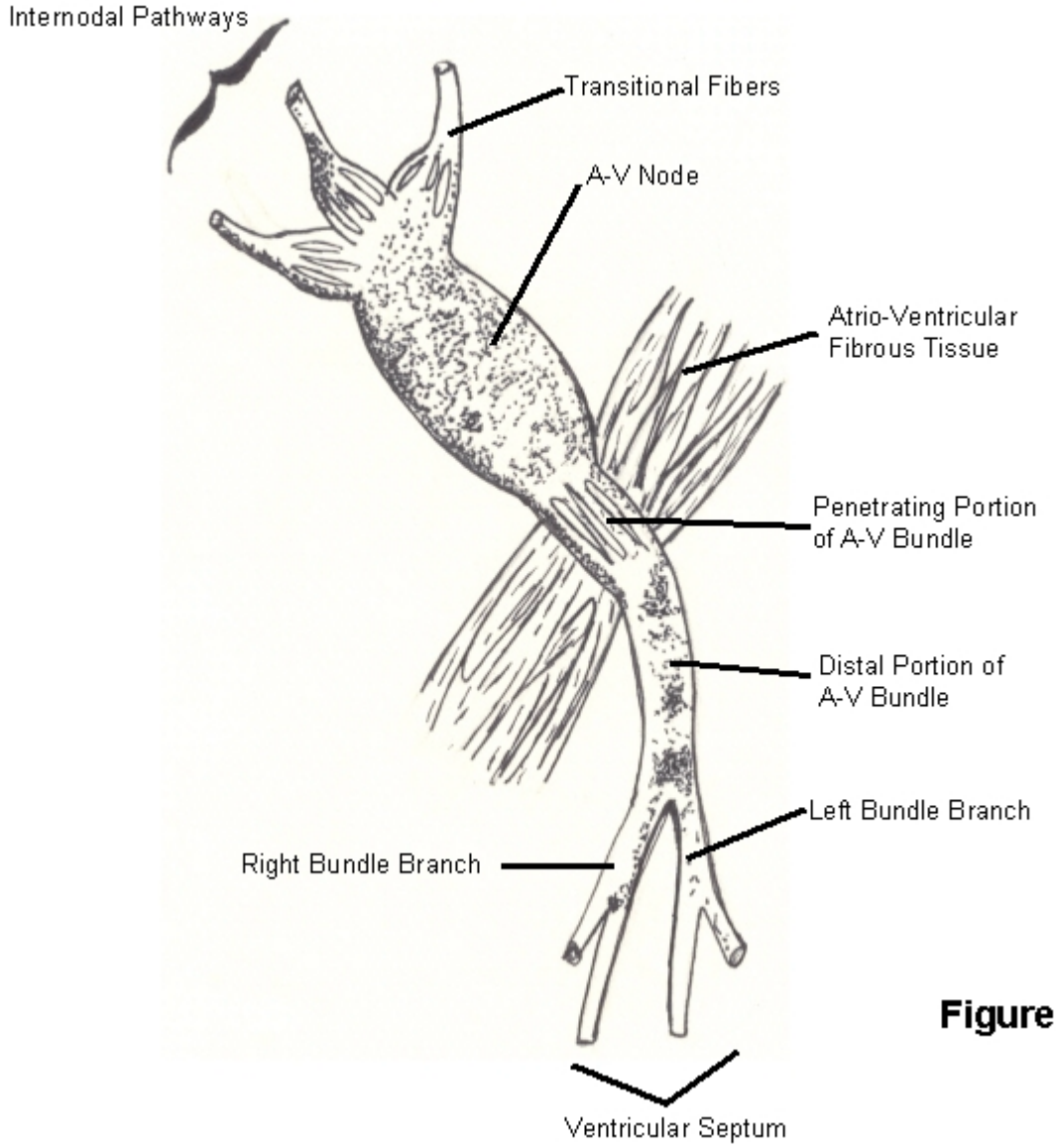


Figure 11-3

Figure 11-4 depicts the depolarization voltage waveforms in time and at different locations. The shapes of these waveforms depend primarily on the number and type of ion channel located in the cardiac cell membranes.

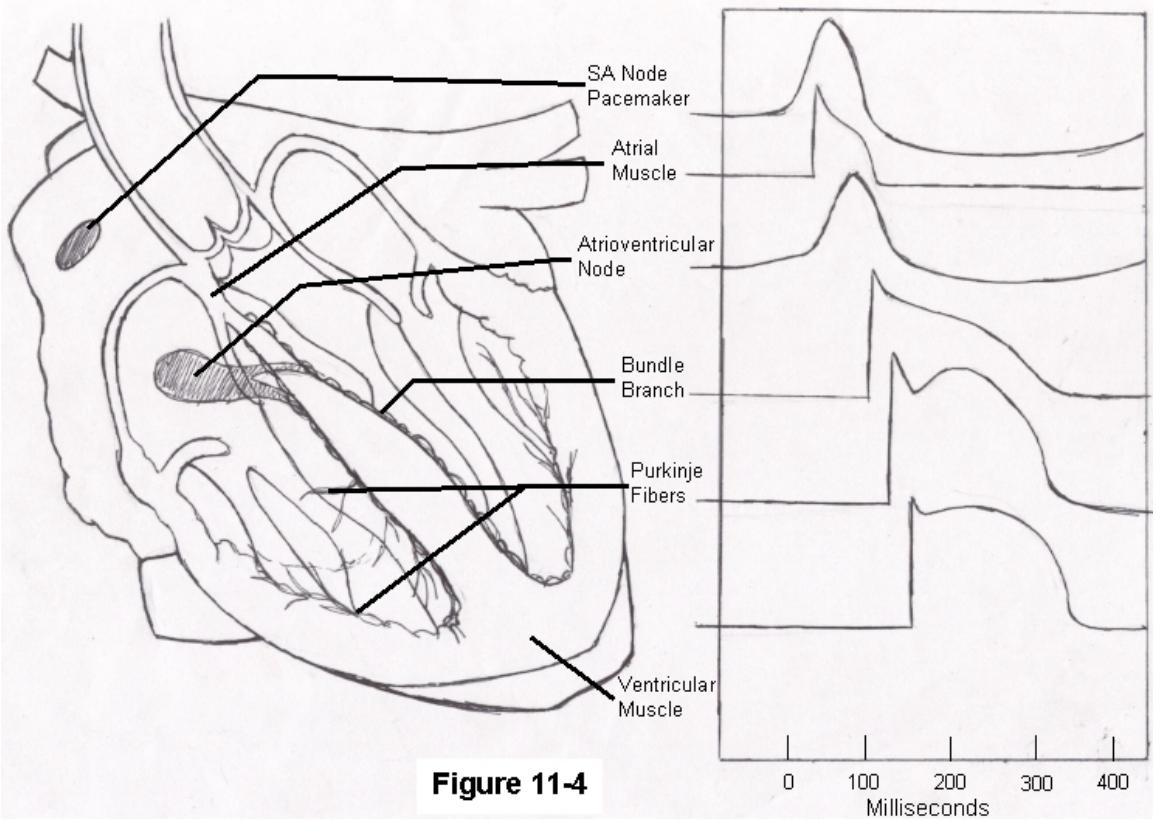


Figure 11-4 Waveforms of Depolarization at Various Locations

Heart rate is controlled, in part, by the autonomic nervous system. Neural input to the cardiac nerves will adjust the properties of the ion channels in the cardiac cell membranes. For example, if the leak channels become more leaky, the heart rate will increase. Figures 11-5 and 11-6 show a simple depiction of this influence. Other factors also effect heart rate such as certain hormones, stretch of the cardiac tissue and a plethora of pharmaceutical agents.

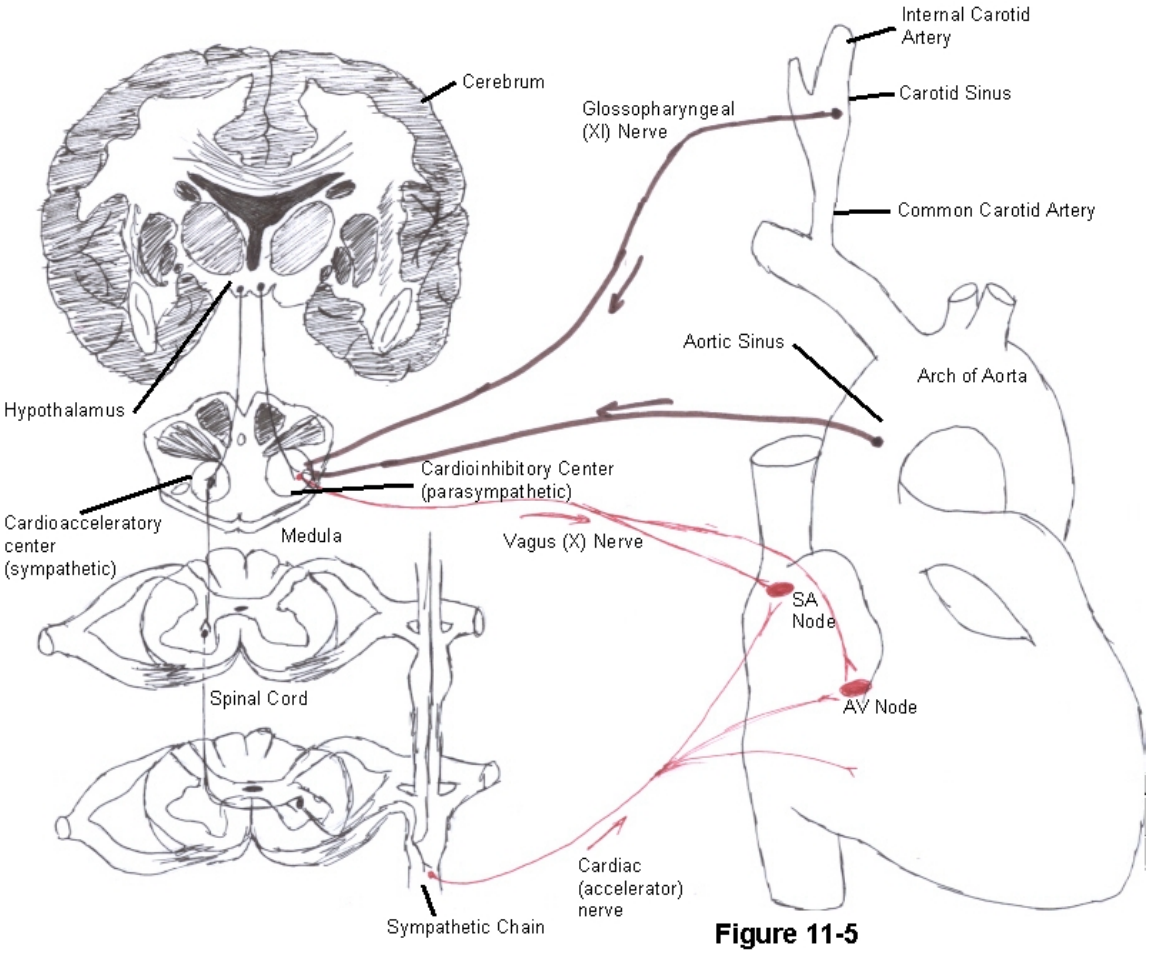


Figure 11-5 Neural Input to Cardiac Tissue

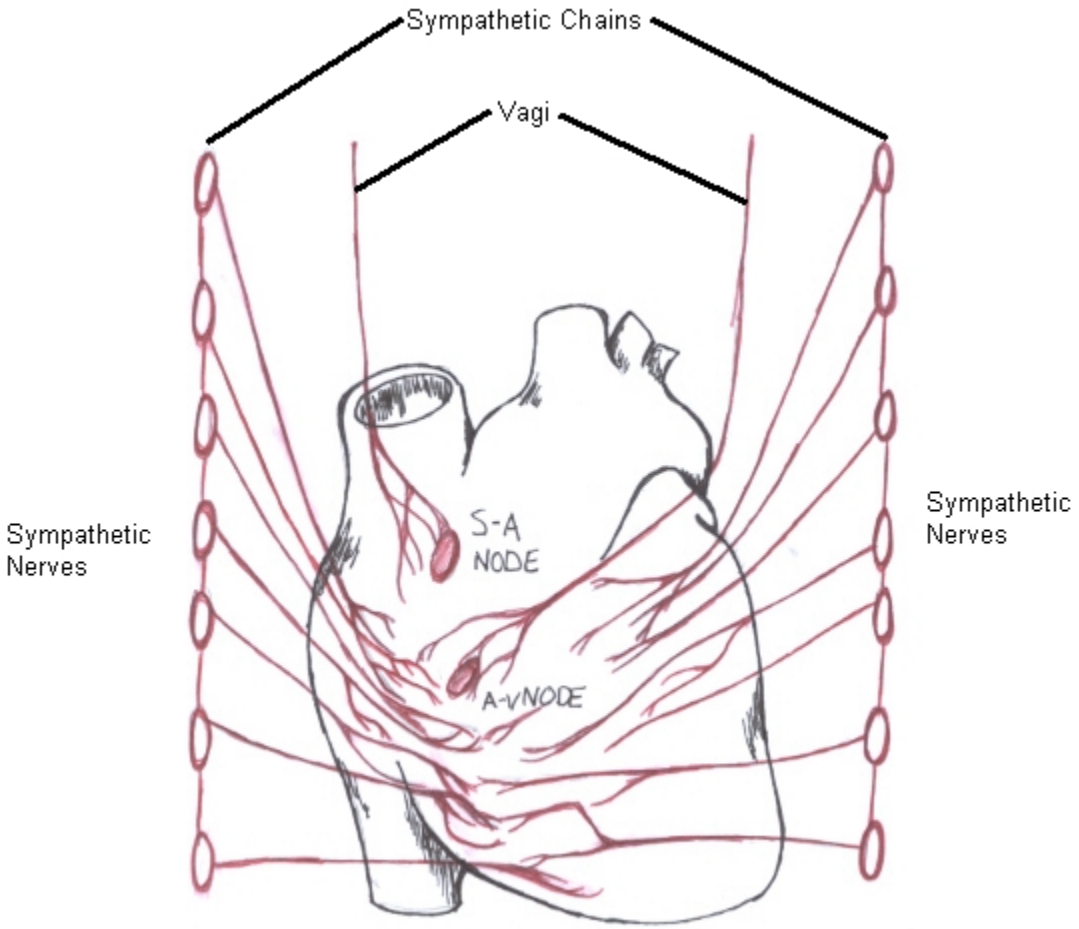


Figure 11-6

Next, we will develop a simple model of how the depolarization wavefront propagates through the body and is recorded on the surface with electrodes. To begin, let's examine a very simple case as shown in Figure 11-7(a-e). Here we have a large cell with electrodes on either end. The inside of the cell is more negative than the outside, depicted by + and – ions. At first we have a negative membrane potential and since there is no difference in the distribution of charge along the length of the "cell", no potential difference on the surface is discerned and the meter reads 0 volts. Now as depolarization begins on the left side, the negative electrode will see more negative ions and the meter will read positive voltage.

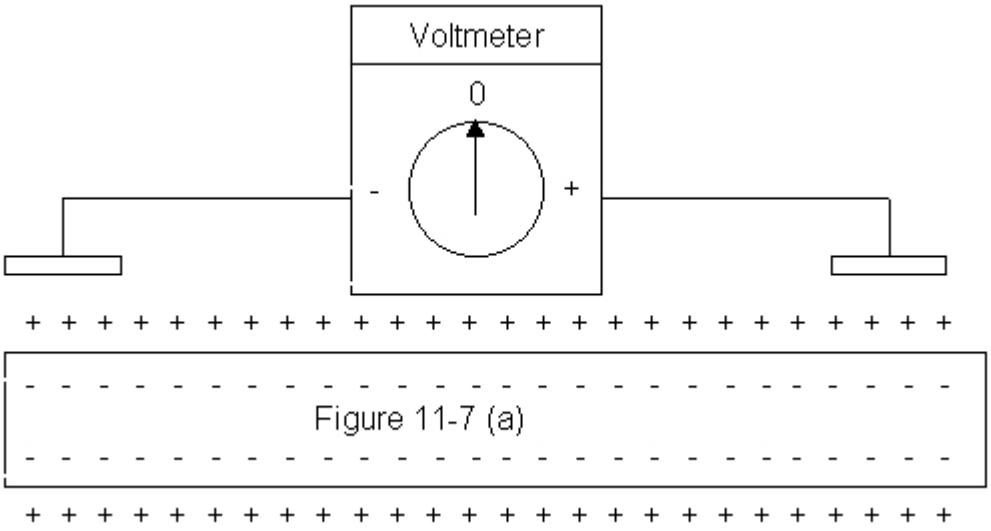


Figure 11-7 (a)

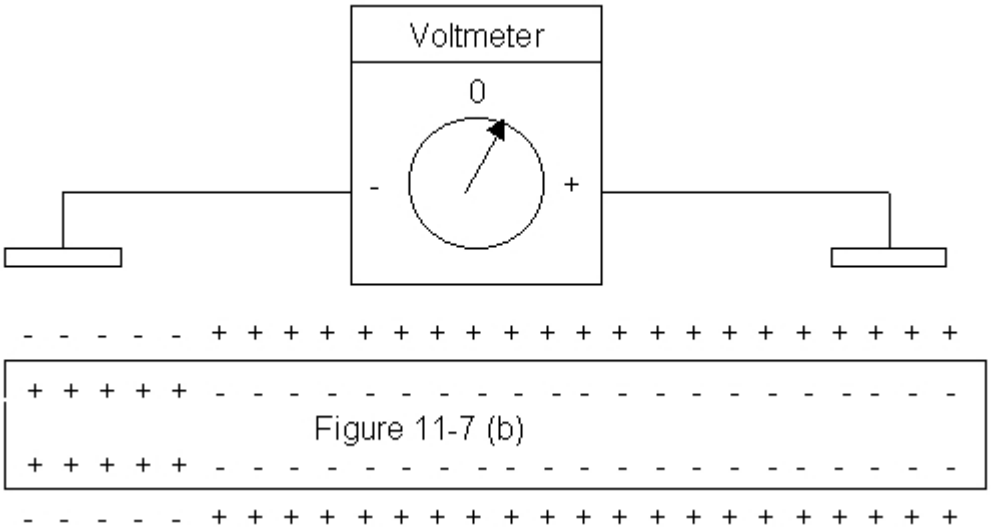


Figure 11-7 (b)

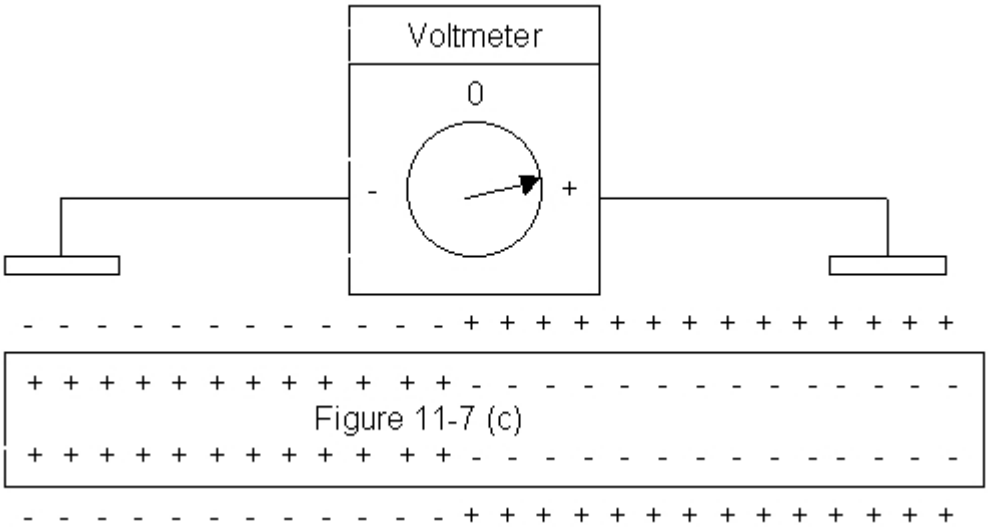


Figure 11-7 (c)

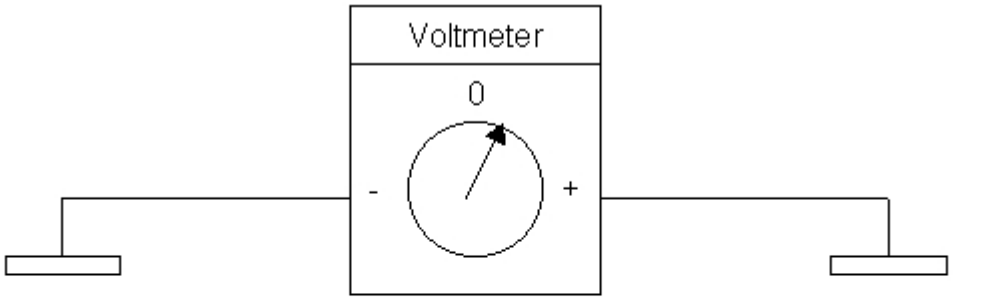


Figure 11-7 (d)

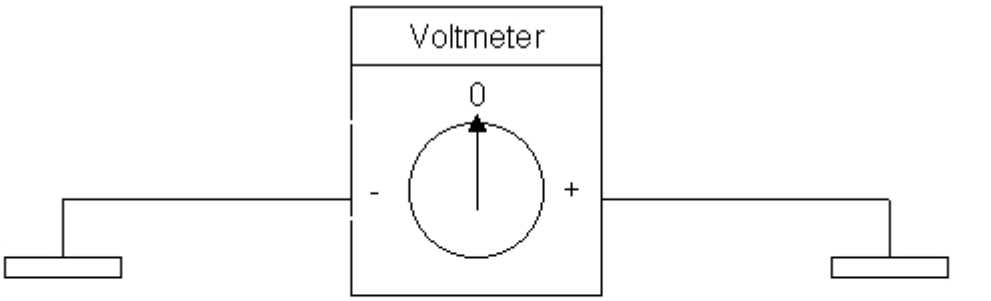


Figure 11-7 (e)

In the last figure (e) of the series, the cell has completely depolarized and its trans-membrane potential is positive, but its surface potential is now zero. Figure 11-8 shows what the surface voltage as a function of time might look like. At first, the surface potential from one end of the cell to the other is zero. But as ions start to invade the cell, there are a few negative ions under the negative electrode. As the number of ions swells, so does the magnitude of the surface voltage until a maximum is reached when there are equal concentrations of negative ions under the negative electrode and positive ions under the positive electrode. After this, the surface voltage starts to diminish until only negative ions

exist under both surface electrodes, at which time the surface voltage is zero again.

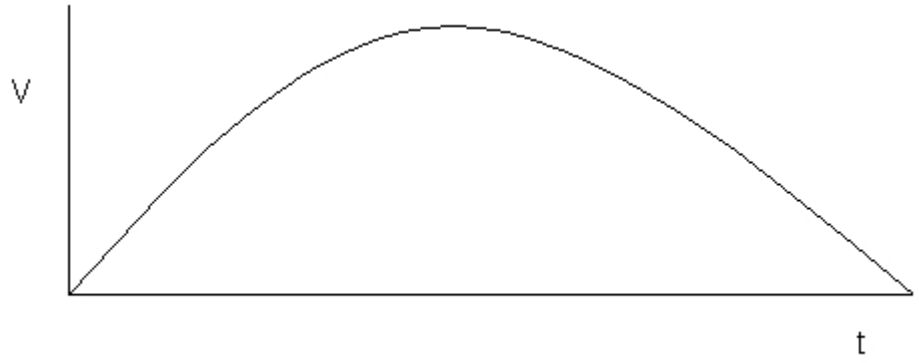
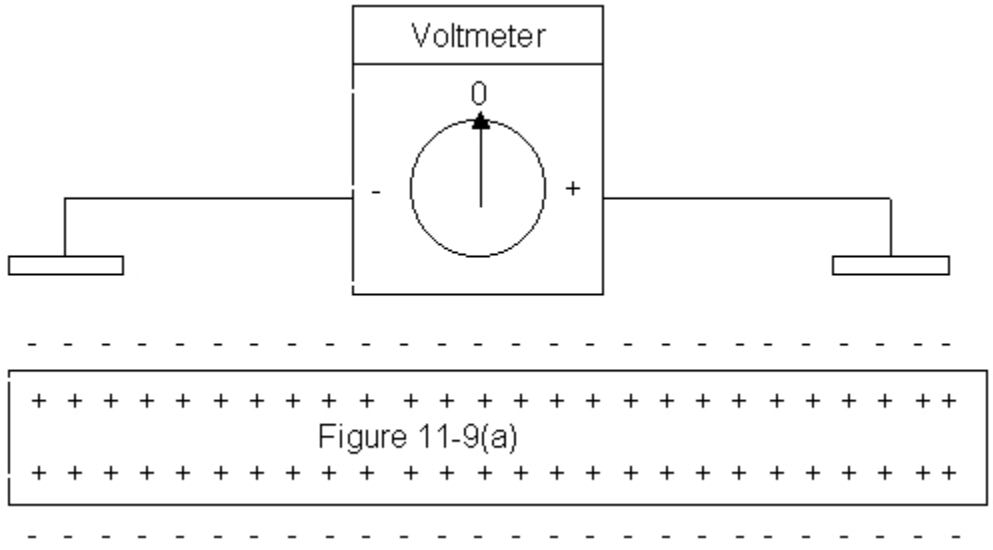
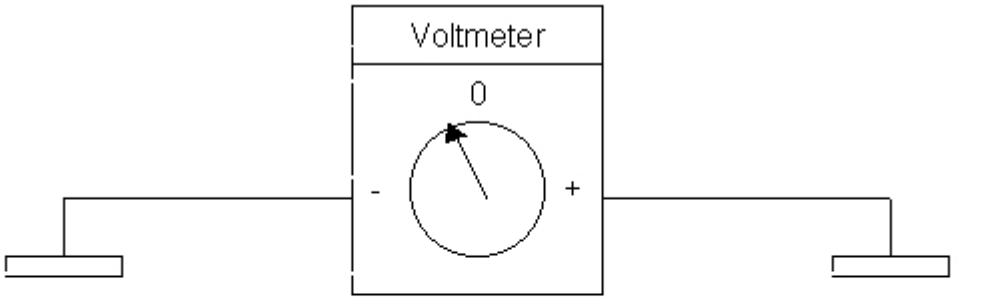


Figure 11-8 Surface Voltage Waveform

Now the repolarization of the cell occurs. This is when the negative ions are moved inward and the positive ions are moved outward. Figures 11-9 (a-e) illustrate the resulting surface voltage readings for a repolarization.





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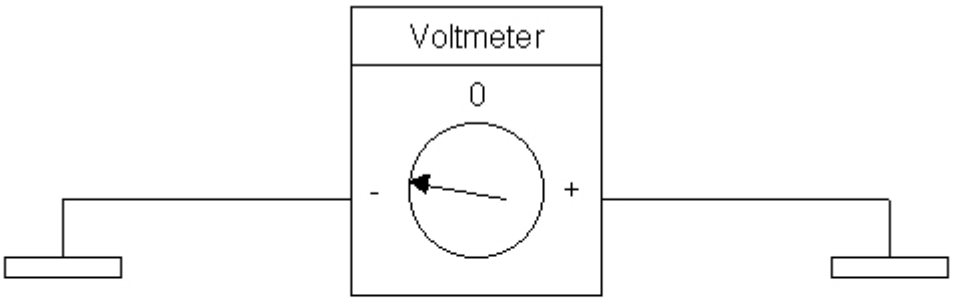
- - - - +

Figure 11-9 (b)

- - - - +

+ + + + -



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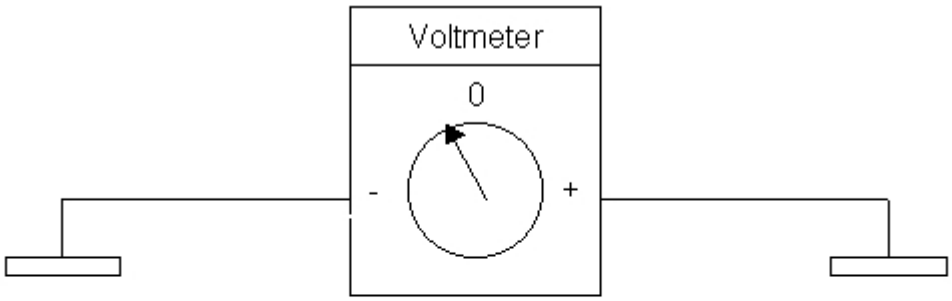
- - - - - - - - - - - - - + + + + + + + + + + + + +

Figure 11-9 (c)

- - - - - - - - - - - - - + + + + + + + + + + + + +

+ + + + + + + + + + + + + - - - - - - - - - - - - -



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- + + + + + +

Figure 11-9 (d)

- + + + + + +

+ - - - - - -

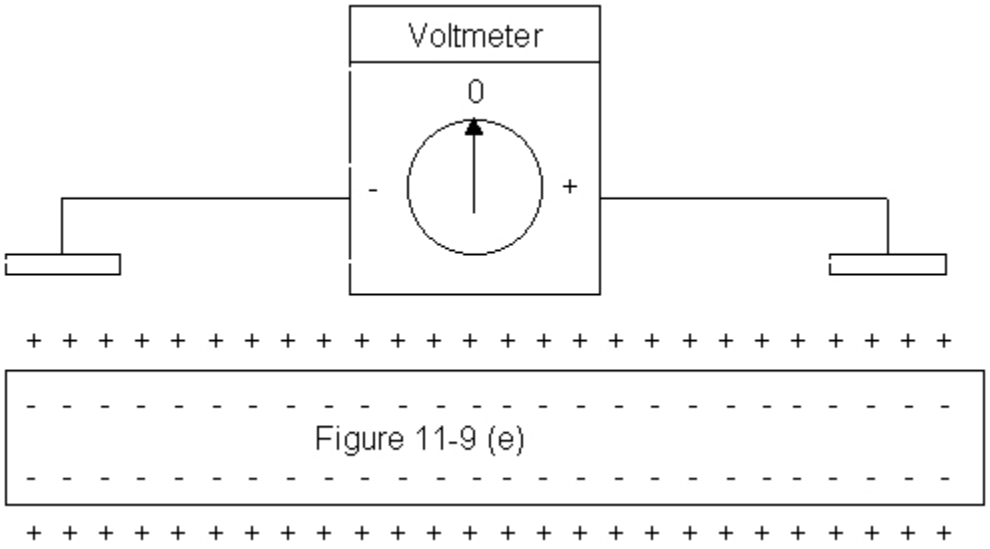

Figure 11-10 depicts what the surface voltage waveform might look like for a repolarization event.

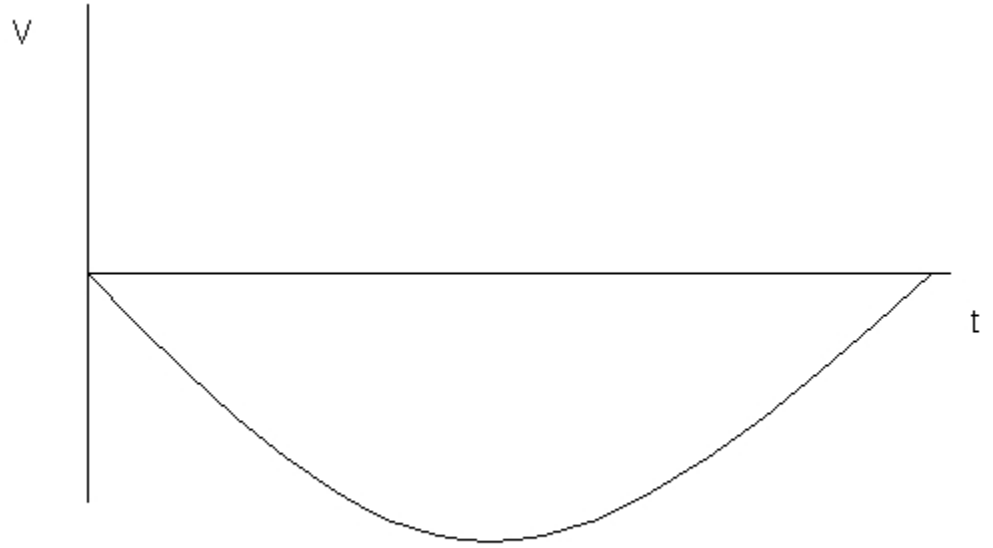

Figure 11-10 Surface Voltage for Repolarization

We can view the moving boundary of charge imbalance as a dipole (Figure 11-11). The dipole has a positive and negative charge. If the positive end is moving toward the positive electrode, we'll see that as a positive going voltage, whose magnitude depends on the amount of charge. If the positive end is moving away from the positive electrode, we'll see that as a negative voltage, whose magnitude depends on the amount of charge.

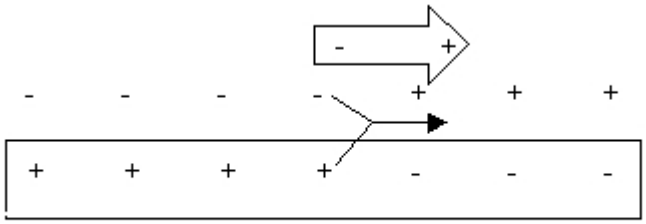

Figure 11-11 Dipole Model for Depolarization Wavefront

Because the body is highly conductive due to so much saline, these small voltage changes on the surface of the heart will be conducted to the surface of the body. Here with proper instrumentation we can measure these small electrical changes and learn important things about the conduction properties of the heart. For current purposes, imagine an electrode attached to the skin of the right shoulder (-) and another electrode positioned on the left leg (+). These represent the two electrodes of the earlier examples. Under normal conditions, the surface voltage waveform (ECG) looks like Figure 11-12.

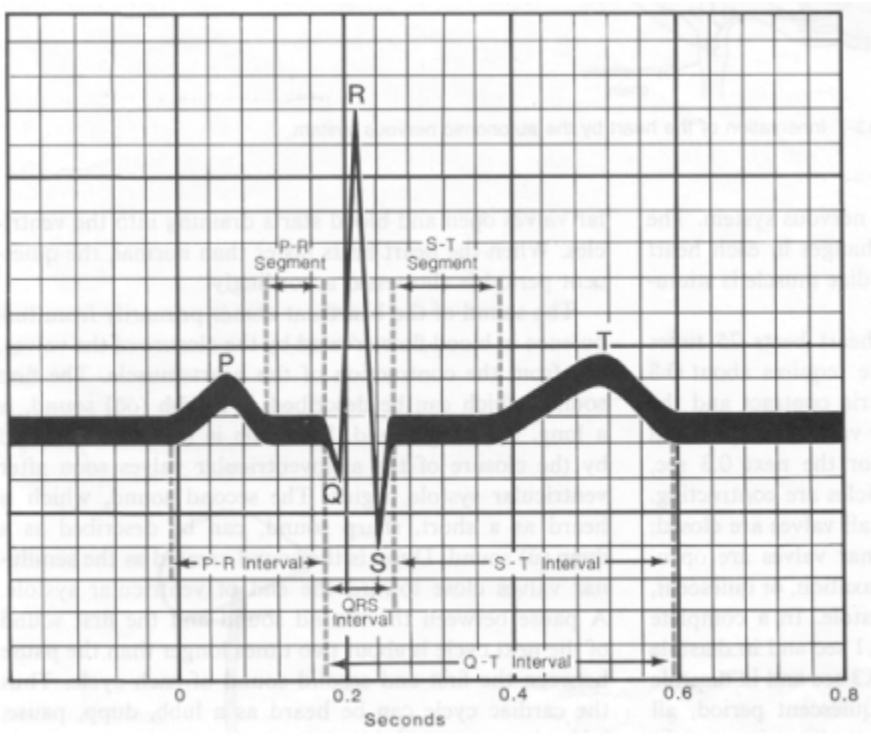

Figure 11-12 Normal ECG Waveform (Lead II)

We can see that a Pwave occurs first, followed by a large QRS wave and finally a smaller T wave. The Pwave is produced by the depolarization of the atria and we also can see that the “dipole” is moving roughly in a direction from the right shoulder (-) to the left leg (+). The QRS, due to depolarization of the ventricles, is much larger owing to the fact that there is much more mass (more charge) involved, but the movement of the “dipole” is still towards the left leg (+). We do not see the repolarization of the atria as it occurs during the massive QRS wave, however the T wave is the repolarization of the ventricle. Note that unlike our earlier example, it is not negative, but rather the “dipole” is moving away from the left leg, indicating that the repolarization takes place from the tissue closest to the left leg and continues towards the tissue closest the right shoulder. Figure 11-13 illustrates the dipole motion.

In summary, we have learned of the anatomical structures that create the pace of the heart, SA node, AV node and ventricles. We’ve learned that the SA node dominants the pace under normal conditions. Further, we’ve learned about the pathways that help conduct the depolarization events. We’ve created a simple dipole model to help us interpret surface voltage waveforms (ECG). Finally, we can begin to interpret the basic components of the ECG.

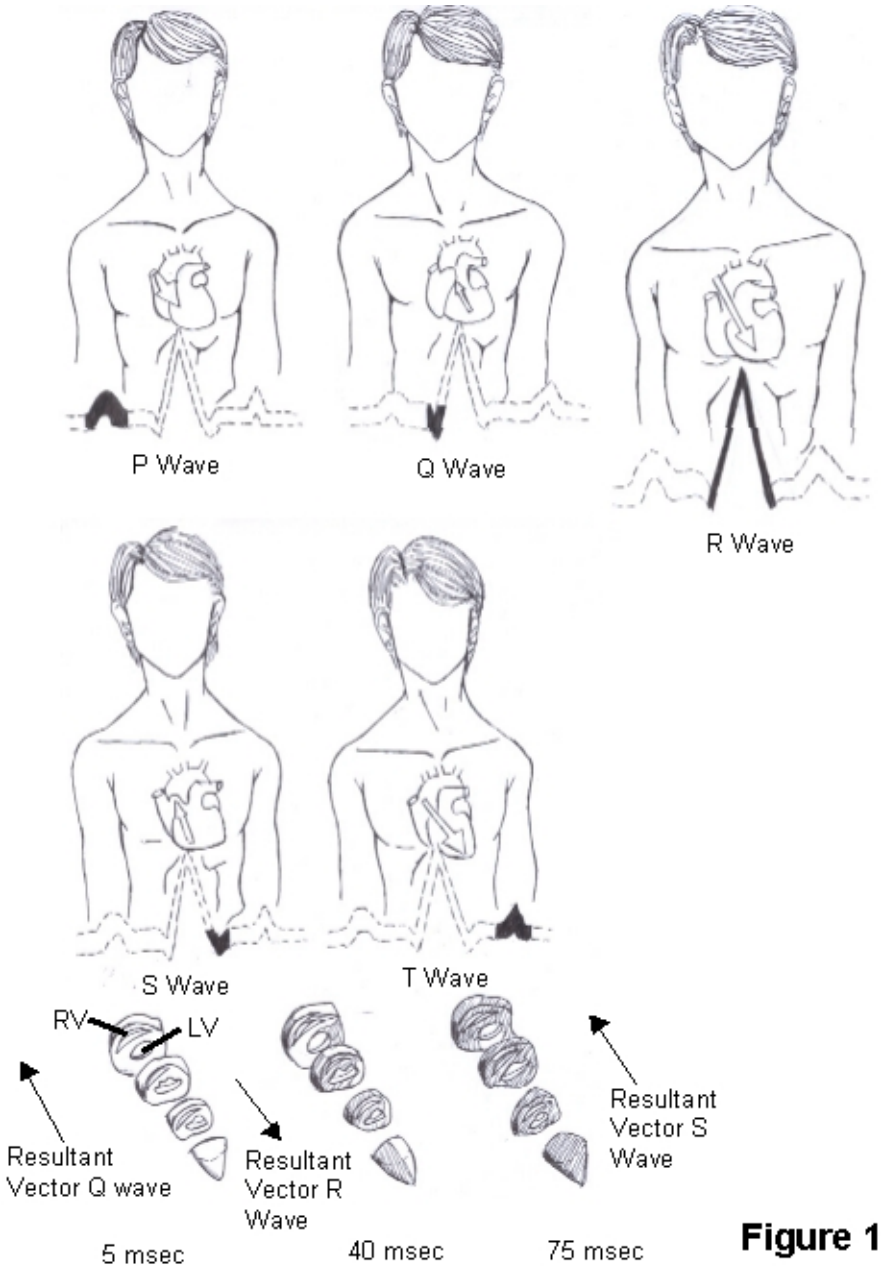
P Wave
Q Wave
R Wave
S Wave
T Wave
RV
LV
Resultant Vector Q wave
Resultant Vector R Wave
Resultant Vector S Wave
5 msec
40 msec
75 msec

Figure 11-13