**ELECTROCARDIOGRAPHY**

**BASIC OVERVIEW OF CARDIAC ANATOMY AND CONDUCTION**

The Heart - It keeps going, and going, and going...

Believe it or not, the heart’s main function is not to decorate Valentine’s Day cards. We all know that by means of its four chambers (two atria and two ventricles), the heart is responsible for circulating blood throughout the lungs and the various tissues throughout the body. Without this circulation, nutrients cannot be delivered, waste products cannot be removed, and the upshot of it all is... you die.

You certainly remember that the cardiac contractions are the result of a well orchestrated electrical phenomenon called **depolarization**. You might even recall that the cell membranes move from their negative resting potential to a more positive threshold which ultimately stimulates them to contract.

In the myocardium there are specialized fibers that are very conductive and allow the rapid transmission of electrical impulses across the muscle, telling them to contract. In order to maximize the force of the contraction there is uniformity in the sequence. That is, the atria contract, then the ventricles contract. This allows both sets to fill properly before ejecting the blood to its next destination. These two sections are independent, yet linked to a single impulse, (in a healthy heart,) initiated by the **sinoatrial, (or sinus) node**. The tissue around the valves helps to channel the impulse from the sinus node through another collection of specialized tissue, the **atrioventricular node**, that is situated between the two sets of chambers. This area allows slightly slower transmission of the impulse to the ventricles, allowing the atria to empty into the ventricles before they contract and force the blood to the lungs or body. This area, the **A/V Node**, slows the impulse down to about 1/25th of the original signal then passes it through to the **atrioventricular bundle**, or the **bundle of His**. This bundle divides itself into two distinct tracts through the ventricles, the **bundle branches**, and on to the **Purkinje fibers**, where the muscle of the ventricle is stimulated to contract from the bottom up, maximizing the force of ejection.

The SA node ticks away at a rate of 60-80 beats every minute. That’s a good thing... Since the SA node is the fastest pacemaker in the heart, all the other cells follow it in synchrony (we call this **syncitium**). This property of beating on their own is called **automaticity**. The really nifty part is that all cardiac muscle possesses automaticity.

It’s important for all cells to be automatic because when/if the SA node fails to depolarize, the next fastest
cells will take over. All of the atrial cells have intrinsic rates of approximately 60/minute. The AV node (conveniently located between the Atria and the Ventricles) intrinsically fires 40-60 times every minute. Even the ventricular cells will eventually depolarize on their own, although each cell has a rate between 20 and 40 times per minute.

The important things to remember here are:

- The fastest pacemaker controls the overall rate.
- Try to remember the intrinsic rates of the different regions.
- Depolarizing cells become more positive.
- Finally, remember that practically everything I just said applies only to a normal, healthy, human heart.

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CARDIAC CONTRACTION AND ECG

Action Potentials - the Key to Contraction

This gets a little tough, so take it slowly...

I spoke of automaticity earlier and mentioned that all myocardial cells possess this wonderful property. Automaticity is the direct result of a cleverly designed "leaky membrane" which regulates the exchange of Sodium, Potassium, and Calcium ions to change the polarization of the cells. The sequence is as follows:

1. Sodium ions enter the cell and begin the depolarization.
2. Calcium ions follow and extend the depolarization even further.
3. Once Calcium stops moving inward, Potassium ions move out and repolarization begins.

In a nutshell, sodium starts the cells’ stimulation. Calcium extends the stimulation thereby allowing the entire muscle to contract before Potassium finally comes along and tells the muscle to relax for a moment and prepare for the next cycle.

The important part of this cycle is the period where the cells reset and prepare for the next wave. This is called the refractory period because the cells are refractory to (or unaffected by) further stimulation.

Actually, there are two portions of the refractory period:

- **Absolute refractory period** - during this period, absolutely no stimulation can cause another action potential. This is the first part of the refractory period.
- **Relative refractory period** - during this portion, it is possible to cause another action potential, but the intensity of the contraction will be relative to the time in this period. So, the further into the period, the better the contraction.
  - An impulse during the relative refractory period may cause a premature contraction. In this situation, the chambers are not filled completely. According to the Frank-Starling Law, this decreased preload will cause cardiac output to decrease.
  - Additionally, serious and life-threatening dysrhythmias can arise if the R-wave of the next beat falls in certain portions of the previous T-wave. It didn't take a genius to call this the "R on T phenomena." -- Just remember... R on T... bad...
If you’re still scratching your head we still have one more chance... (an old paramedic once told this story around a campfire)

Imagine, if you will, a toilet. Please bear with me... I swear this will work!

When you pull the handle, (initiate an impulse) water floods the bowl(kinda' like contraction!). This event takes a couple of seconds and you cannot stop it in the middle. Once the bowl empties, the flush is complete. Now the upper tank is empty. If you try pulling the handle at this point, nothing happens (absolute refractory?)

Wait for the upper tank to begin refilling (Potassium moves back). You can now flush again, but the intensity of the flushes increases as the upper tank refills (relative refractory...)

Physiology will never be the same again...

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http://www.drsegal.com/medstud/ecg/Electric.htm
In the mid-1880's, two fellows named Ludwig and Waller discovered that the electrical activity of the heart could be monitored through the skin. Their device, called a capillary electrometer, used sensor electrodes and magnets to generate an electrical field. A capillary tube with fluid was placed in the field. As current passed through the electrodes, the field increased and decreased causing the fluid in the tube to bounce up and down. This device, as cool as they probably thought it was, was far too crude for clinical use.

It took the genius of Mr. Einthoven to produce a function ECG device. Einthoven devised a clever system for recording the same electrical activity on light-sensitive paper. Noticing a recurring pattern of movement, Einthoven named the prominent waves alphabetically, P, Q, R, S, and T

the *P-Wave*, representing the impulse across the atria to the A/V Node;

The *QRS* representing the impulse as it travels across the ventricles;

the *T-Wave*, representing the repolarization of the ventricles.

Modern ECG devices use more sophisticated techniques like amplification, filtering, and digital signal analysis to more accurately and conveniently measure, display, and analyze ECG data. Many of the high-end machines will perform very detailed interpretation of the ECG thus saving the clinician from the tedious and laborious burden of thinking.

In our analysis of these ECG rhythms, we will examine the shape, consistency, and the time between these waveforms to determine the functionality of the heart’s conduction system. Many cardiac conditions have characteristic patterns on the ECG. These *dysrhythmias* are valuable clues to diagnosing and treating cardiac illnesses - acute and chronic.

Through ECG analysis, it is possible to trace the conduction through the heart, estimate the size and orientation of the heart, and even to locate regions of the heart which have suffered injury, ischemia (oxygen deprivation), or necrosis (tissue death).

This tutorial will focus on Lead II ECGs. Lead II implies that the activity is measured from the right arm to the
left leg. The ECG will show activity along the septum of the heart.

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SINUS RHYTHMS

From a clinical standpoint, it is important to focus the examination on the patient and not the ECG. Many dysrhythmias can be completely benign. How does the patient feel? How long has the patient felt this way? A full history can be as useful as a good ECG.

A methodical approach will be much more effective than simply memorizing the appearance of the many dysrhythmias. Always begin your analysis by asking the following questions:

- Is the rate fast or slow? Are the atrial and ventricular rates the same?
- Are the P-P interval and R-R interval regular or irregular? If the rhythm is irregular, is it consistent or irregular irregularity?
- Is there a P-wave before each QRS? Is there a QRS before every P-wave?
- Are the P-waves and QRS complexes identical and normal in configuration?
- Are the P-R and QRS intervals within normal limits?

Sinus Rhythms are a class of rhythms which originate at the SA node. Sinus rhythms generally travel through the entire conduction system without inhibition.

They are characterized by:

- a conducted P-wave
- P-R interval between 0.12 and 0.20 seconds
- The QRS width should be 0.04 to 0.12 seconds and be all QRS's are preceded by a P-wave.

In reality, nothing is normal. Every patient has his own unique ECG patterns. Nevertheless, scientists like to have a baseline for everything. In a perfect world, we'd all have a Normal Sinus Rhythm. If your's doesn't look exactly like this, you probably aren't going to die in the next five minutes. At least read the rest of the tutorial before you panic...

The criteria for a Normal Sinus Rhythm is:

- P-wave before each QRS with an interval of 0.12 to 0.20 seconds in duration.
- A QRS width of 0.04 to 0.12 seconds
- Q-T interval of less the 0.40 seconds.
- The rate for a normal sinus rhythm is 60 to 100 beats a minute.
### Normal Sinus Rhythm

<p>| | |</p>
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>P-R interval</td>
<td>0.12 to 0.20 seconds</td>
</tr>
<tr>
<td>QRS duration</td>
<td>0.04 to 0.12 seconds</td>
</tr>
<tr>
<td>Rate</td>
<td>60 to 100 beats a minute</td>
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</table>

If the rate is below 60 beats a minute but the rest is the same it is a **Sinus Bradycardia**.

*Brady-* means slow. Like your brain after you watch the Brady Bunch!

### Sinus Bradycardia

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<tbody>
<tr>
<td>P-R interval</td>
<td>0.12 to 0.20 seconds</td>
</tr>
<tr>
<td>QRS duration</td>
<td>0.04 to 0.12 seconds</td>
</tr>
<tr>
<td>Rate</td>
<td>less than 60 beats a minute</td>
</tr>
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</table>

Yes... the QRS complexes look completely different... That's okay. I already said that we're all different. Relax.

If the rate is between 100 to 150 beats a minute with the same intervals it is a **Sinus Tachycardia**.

*Tachy-* means fast. (Think of what happens to your heart rate when you sit on a tack)

### Sinus Tachycardia

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<tbody>
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<td>0.04 to 0.12 seconds</td>
</tr>
<tr>
<td>Rate</td>
<td>100 to 150 beats a minute</td>
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When the pattern becomes irregular with normal intervals it is a **Sinus Arrhythmia**

### Sinus Arrhythmia

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<td>P-R interval</td>
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</tr>
<tr>
<td>QRS duration</td>
<td>0.04 to 0.12 seconds</td>
</tr>
<tr>
<td>Rate</td>
<td>60 to 100 beats a minute, regular rhythm with <strong>periodic irregularity</strong></td>
</tr>
</tbody>
</table>

Clinically, these could all be perfectly fine. Any of them could also be deadly. It is possible to show a regular Sinus Rhythm on the ECG with no contraction of the heart! Check your pulse. Is it there? Good. Now quit worrying so much.
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**ATRIAL RHYTHMS**

It is possible for the the atria to depolarize a bit earlier than normal. This is actually quite common (in the sense that it happens to a lot of people, not that one person does it all the time!). If you’re willing to go back to the toilet analogy, it would be like your toilet flushing by itself right before you pushed the handle... Yeah, I know it’s a stretch but we were on a roll!

If the premature impulse falls in the relative refractory period or later, an atrial contraction may occur. This, logically, is called a **Premature Atrial Contraction (PAC)**.

Examine the PAC and notice some characteristics of **atrial complexes**:

- *Upright normal P-wave*
- *Narrow QRS complex (0.04 - 0.12 ms)*

If the SA node or the internodal pathways suffer stress or damage, the atria can frequently initiate impulses from other portions of the atrial tissue. PACs are one possible outcome. Other rhythms which may arise in atrial tissue are fibrillation and flutter.

**Atrial Fibrillation** describes a condition in which the atrial tissue randomly generates action potentials from many different regions. Physically, the atrial muscle appears to quiver (it looks like Jell-o). There are no noticable p-waves, and the overall rhythm is irregularly irregular.

The reason you cannot see P-waves is that the atrial activity is about as scrambled as a breakfast omelette. The key to recognizing A-fib are the narrow QRS’s and the irregularly irregular rhythm.

**Atrial Flutter** is recognized by the distinct "saw tooth" pattern of P-waves.

The QRS complexes can appear at different intervals and frequencies. Naming conventions for A-flutter depend on these relations.

**Atrial Flutter, 2:1 Block**

http://www.drsegal.com/medstud/ecg/atrial.htm
The 2:1 block indicates there are 2 P-waves followed by 1 QRS.

*Atrial Flutter, Variable Block*

This is extremely common among A-flutter patients. There can be as few as a single P-wave or as many as 6 or more P-waves between each QRS complex.

The clinical significance of A-fib and A-flutter is two fold:

- First, because the atria generally don't have time to fill completely, the preload to the ventricles is reduced and cardiac output suffers.
- Perhaps even more dangerous is the erratic and turbulent blood flow caused by the erratic contraction and relaxation of the atrial walls. In the turbulence, blood can form small clots which may lodge in small vessels. If a clot (or thrombus) is released into the cerebral vessels, it can cause a stroke (often called CVA, cerebrovascular accident).

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ATRIOVENTRICULAR NODE BLOCKS

For various reasons, conduction through the AV node may become impeded even more than usual (remember that the AV node is supposed to slow conduction down). This may be entirely harmless, or depending on the degree of resistance, could prove to be a fatal dysrhythmia. AV-blocks are rated with any of three degrees of severity.

1st Degree Heart Block

1st degree blocks are generally benign. They are characterized by a constant PR interval greater than 0.2 seconds. The rhythm is otherwise normal. Rates may range from bradycardias to tachycardias with a full degree of variation in between. Ordinarily, there will be no symptoms associated with a 1st degree block.

2nd Degree Heart Block

2nd degree blocks are subdivided into two types:

- **2nd Degree - Type I (Wenkebach)**

  This rhythm is distinguished by a repeating cycle of increasing PR intervals. As the interval get longer, a P-wave is either not conducted (ie; there is no QRS) or the P-wave is simply dropped. The cycle then repeats again. Typically, RR intervals become shorter until the dropped beat.

  Type I blocks are generally not dangerous. The patient may complain of palpitations, or skipped beats.

- **2nd Degree - Type II (Mobitz)**

  This rhythm can be recognized by a consistent PR interval and frequently non-conductive P waves. QRS complexes may appear widened depending on the location of the block. Wide QRS complexes indicate
that the ventricles are depolarizing from an action potention in the ventricular tissue, rather than from or above the AV junction.

Generally speaking, Type II blocks are not a good sign. They have a tendency to worsen, leading to 3rd degree blocks.

**3rd Degree Heart Block**

The 3rd degree block is by far the most dangerous. There is absolutely no conduction through the AV node. Due to the automaticity of each region of the heart, the atria beat at there intrinsic rate (60-80 bpm) and the ventricles, which are **completely isolated from the atria** beat at their slower rate of 20-40 bpm.

The QRS complexes will often be wide, but depending on the origin the ventricular action potential, they may remain narrow.

The P-P interval and R-R interval will each be regular and consistent. The P-P interval will be faster than the R-R and there will be no relation between the two.

A 3rd degree block is also called *Atrioventricular dissociation*.

The danger of these high-degree blocks should be obvious. Ventricular contraction will not always be preceded by an atrial contraction. Hence, the ventricles are not guaranteed to contain enough blood for a detectable contraction.

Clinical treatment for high degree heart blocks can be pharmacological, or invasive. Autonomic drugs, such as Atropine, can be used to inhibit vagal stimulation and increase the bradycardic rates typically associated with heart blocks. If conduction is not improved with medication, artificial pacemakers can be installed to stimulate either the atria, ventricles, or both, in a synchronized rhythm.

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JUNCTIONAL RHYTHMS

Premature Junctional Contractions

The AV node, just like the other cardiac tissue, has automaticity, the AV node is stimulated before it fires by itself. Occasionally, however, an extra impulse may develop in the junction, thereby spreading up to the atria, and down to the ventricles. Just like the PACs described earlier, these Premature Junctional Contractions occur periodically.

A PJC may be seen in a patient with respiratory difficulty. The poor gas exchange irritates myocardial tissue and causes abnormal activity. PJCs are generally not treated because they are harmless.

PJCs are distinguished from PACs by the P-wave. A PAC has a fairly normal looking P-wave... upright and round. PJCs, however, do not originate in the atria, therefore, the atrial depolarization moves up toward the base of the heart, rather than downward toward the apex. P-waves from PJCs will be inverted (look at the 3rd and 5th complexes in the picture). Often, the P-wave will occur along with the monstrous QRS complex, and will therefore be hidden from view.

Junctional Escape Complexes

If no stimulus reaches the AV node, the cells assume that the SA node never fired. The AV junction will reach it's automatic threshold and generate an action potential. Unlike PJCs, the escape complexes will appear late in the rhythm (which is why they occurred, and why they are called escape beats!). Otherwise, they posess the same deformed, inverted, or absent P-waves of any other junctional beat. The QRS complex will remain narrow, because the impulses are originating above the ventricles.

Clinically, PJCs are not usually treated. They are one of the many quirks that we all experience at one time or another. Escape complexes, on the other hand, are not such a good thing. The complexes themselves are good (because without them, you're heart would simply not beat). Rather, the clinician must investigate why they are occuring:
• Is the SA node damaged?
• Has a myocardial infarction damaged the atrial tissue, or the internodal pathways?
• Is there a high-degree AV block?

As always, we use the ECG as a diagnostic tool, but we **examine and treat the patient!!**

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VENTRICULAR RHYTHMS

We're almost done! Before you go watch "E.R." we'll cover the most serious portion of this tutorial.

The ventricles represent the largest, strongest portions of the heart, and therefore lead to the most serious problems when they fail (what good is a hockey team if nobody can shoot?)

We already learned that the purkinje fibers permit impulses to speed through the ventricular muscle in 0.04 to 0.12 seconds. In a typical atrial or junctional complex, we see narrow QRS complexes for this very reason.

If the fibers become blocked, though, the QRS time will increase to longer than 0.12 seconds.

Premature Ventricular Contractions

On occasion, a ventricular cell may initiate an impulse and cause a contraction. A single occurrence probably won't cause any serious problems. PVCs can be caused by a variety of conditions including respiratory problems and stress (and taxes).

Ordinarily, there will be a compensatory pulse with a PVC. If there is no pulse, we say the PVC is interpolated. If you have a patient with PVCs, be sure to check for a corresponding pulse. This has a large part in deciding whether the PVCs are dangerous or not.

PVCs are classified on the basis of their origin

- **Unifocal PVCs** originate from the same *focus.*
  
  They all have the same shape, or *morphology.*

- **Multifocal PVCs** arise from multiple *foci.*
  
  Each focus has a unique morphology. Often, there will be a repeating sequence, indicating several specific foci. Make an effort to note the number and sequence of foci.

PVCs are also classified by their frequency

- **Bigeminy, trigeminy,** etc...
If each normal contraction is followed by a single PVC, we call this bigeminy. If two normal contractions are followed by a single PVC, we have trigeminy. Be smart... what do you think quadrigeminy is?

Notice in this example, that the PVCs are unifocal... bigeminy tends to arise from a single focus.

- **Couplets**

  Exactly two PVCs in a row is called a couplet.

**R on T Phenomena**

We discussed R on T when we spoke about the refractory periods. R on T can lead to a fatal dysrhythmia called ventricular tachycardia. Obviously, we'd like to avoid this. R on T can occur with very fast rates, but also with ectopic beats like PACs, PJC, and PVCs.

**Ventricular Tachycardia**

V-Tach is a rapid dysrhythmia in which the ventricles depolarize very quickly and without regard for the atria. V-Tach is actually said to happen whenever three or more PVCs occur in a row (which is why we don't name anything beyond a couplet). Regarding pulses, any of the following could happen:

- pulse for every complex - the pulses will be weak and cardiac output low.
- pulse for some beats - this is ominous
- no pulse - there may or may not be any contraction at all, but if there's no pulse, you're patient is in bad shape.

We'll discuss the treatment for this a little later.

**Ventricular Fibrillation**

V-fib is the most common fatal dysrhythmia in adult patients. You see it "E.R." every week. V-fib represents a chaotic depolarization of random ventricular cells. A heart in V-fib literally looks like jiggling Jell-o. There is no pulse associated with this rhythm. CPR won't do much good either, nor will most drugs. You'd better hope the defibrillator works!

V-Fib is usually described as coarse (above) or fine (below). Generally, as the tissue dies, the voltage decreases. Hence, coarse is a little better than fine V-Fib.