# SURVEY OF HOW IRREGULARITIES IN THE ELECTRICAL SYSTEM OF THE HUMAN HEART LINK TO DIFFERENT HEART ARRHYTHMIAS

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## **Thesis Approval**

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#### Abstract

This thesis explores the circuital pathway that a signal takes within the heart throughout a full heartbeat and provides a timing analysis. It then surveys the different pathways that could arise in the electrical system of the heart, and the associated heart diseases linked to the location of each pathway. Common heart arrhythmias such as atrioventricular nodal reentrant tachycardia, atrioventricular reentrant tachycardia, atrial flutter, and atrial fibrillation are surveyed, and their pathways are analyzed. Electrocardiograms are presented to aid in understanding of the timing analysis associated with irregularity in heart function. Finally, this thesis explores an avenue of the heartbeat that links it to a circuital pathway, allowing for an electrical model to aid in illustration of the topic. The use of LTSpice is included to aid in a theoretical illustration of both the typical pathway of the heart, as well as the route that blood is forced to take when an additional pathway is created. The model is best served as a learning tool, as the intricacies of the heart resemble an electrical system. Future work on this topic could focus on developing the models features to simulate heartbeats, pressure, and capacitance within the heart.

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#### Introduction

According to the Centers for Disease Control, heart disease is the leading cause of death in American men and women, accounting for nearly one in five deaths [4]. This alarmingly high statistic makes it clear that research and innovation within the fields related to heart disease are incredibly valuable. Current research linking electrophysiology to engineering is minimal, and this research aims to further discussion on the interdisciplinary aspect of the two.

Heart disease commonly shows itself in the form of an arrhythmia, or irregular heartbeat. According to the National Library of Medicine, arrhythmias are defined as an abnormal rhythm of the heart [6]. These irregular heartbeats are an alternative to the normal sinus rhythm of the heart, meaning that the pathway that a heartbeat would take in a normal heartbeat is interrupted. Normal sinus rhythm is the ideal human heartbeat that those without heart conditions possess when at a resting state.

The pathway of the heart is circuital, and in a healthy heart, blood will always follow the same route. This pathway can be modeled through electrical devices such as switches, diodes, and resistors, and connected via wiring to create a figure to illustrate the pathway. Arrhythmias, when analyzed, can be related to a disruption or alternate pathway in the electrical circuit of the heart. Any abnormalities, interruptions, or additional pathways can also be modeled using more of these electrical devices to show the change that differs the heartbeat from normal sinus rhythm.

One goal of this research is to survey different arrhythmias and show how they stray from the pathway of normal sinus rhythm. In addition to this, the timing and position changes that occur with each of the diseases will be presented and discussed. An electrical model designed in

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LTSPICE will be presented to aid in the circuital understanding of the heart. Note that this model is strictly for illustrative purposes, but future research on this topic can work to make this a circuit used for simulations. This model can be used for training purposes, both from an electrophysiology standpoint and from an engineering standpoint.

A healthy heart will beat between sixty and one hundred times per minute; hearts operating at a rate lower than sixty beats per minute are categorized as bradycardias, and hearts operating at a rate higher than one hundred beats per minute are categorized as tachycardias [6]. The work presented in this thesis will focus on tachycardias, which are the more common of these two subcategories. Within the category of tachycardias, there are subcategories that describe different operations occurring within the heart. These categories will be detailed in the following chapters. Table 1 introduces the arrhythmias that will be discussed and the theoretical circuital operation that is discussed throughout this thesis.

Tachycardia	Theoretical Circuital Operation
Atrial Fibrillation	Irregular input waves from the SA node
Atrioventricular Nodal Reentrant Tachycardia	Irregular pathway within the AV node
Atrioventricular Reentrant Tachycardia	Irregular pathway between the atria and ventricles
Atrial Flutter	Irregular pathway within the atria

Table 1: Overview of Arrhythmias and their Circuital Operation

#### **Chapter 1: The Electrical System of the Heart**

The human heart has an electrical system that completes one timing cycle per beat (of the heart). Under normal circumstances, the heart operates in a circuital path, with two main operations – contraction and relaxation. The main purpose of this path is to allow for oxygen-poor blood to oxygenate, thus allowing for a release of oxygen-rich blood into the lungs and throughout the body [8]. The heart is electrically charged and has timing mechanisms – namely, the sinoatrial node and the atrioventricular node that allow for its contraction and relaxation states. The step-by-step procedure of a heartbeat is somewhat complex; the following sections will detail the movement of blood through a single circuital heartbeat. For simplicity, the circuit of the heart will first be listed, and then a more detailed explanation of the process will be included.

#### Circuit of the heart:

- 1. Superior and Inferior Vena Cava
- 2. Sinoatrial Node
- 3. Right Atrium
- 4. Tricuspid Valve
- 5. Right Ventricle
- 6. Pulmonary Valve
- 7. Pulmonary Artery
- 8. Lungs (blood is oxygenated)
- 9. Pulmonary Veins
- 10. Left Atrium

Mitral Valve
 Left Ventricle
 Aortic Valve
 Aorta/Body

#### The Pathway of the Heart

#### Superior/Inferior Vena Cava

The heart has two mechanisms that aid in moving blood to and from it: veins and arteries. These pathways for blood allow for proper movement of the electrical system of the heart. Moreover, as blood moves from the heart into the body, the blood deoxygenates. On the other hand, as blood enters the heart, it (in working conditions) reoxygenates. This is a very simplified explanation of the function of the heart within the body, as oxygenated blood is what allows for proper function of the organs. It should be noted that blockages, clogs, and other diseases within these pathways can lead to heart diseases (not discussed in this thesis), of which valuable research can be done to further this research.

There are two main veins that aid in the electrophysiological method of the heart: the superior and the inferior vena cava. These are the largest veins in the body, and their prime focus is to move oxygen-poor blood from the body into the heart (to allow for reoxygenation, as discussed in the earlier paragraph). These veins are integral to moving blood toward the heart and beginning the circuit of a heartbeat. The inferior vena cava is slightly larger than the superior vena cava (contradictory to their names). One end of the inferior vena cava is located near the

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stomach, and its other end also empties into the right atrium [5]. One end of the superior vena cava is located near the right side of the sternum, and the other end is located at the entrance of the right atrium to allow for the oxygen-poor blood to enter the heart [5]. Moreover, the inferior vena cava is responsible for supplying blood to the heart from the bottom part of the body – namely, the liver, kidney, lower back, and legs [5]. The superior vena cava is responsible for moving blood focused in the top part of the body – namely, the arms, head, neck, chest, and upper back. Figure 1 displays the location of the pathways of these veins (shown in blue), further emphasizing their motive to supply oxygen-poor blood to the body.



Figure 1: Location of Superior/Inferior Vena Cava [3]

The SA node is commonly referred to as the pacemaker of the heart. The main function of the SA node is to generate an electrical signal (thus, a naturally occurring pacemaker). This electrical signal is critical to the timing of the pathway (discussed later in this chapter). Within the pathway of the heart, the function of the SA node is relatively simple – to generate the electrical signal that will allow for the circuit of the heart to begin. The SA node is located within the right atrium of the heart and is composed of a specialized group of tissue that sends signals to electrically stimulate the heart [10]. Once the node fires this signal (about sixty to one hundred times per minute in a healthy adult heart), the superior and inferior vena cava begin to fill the right atrium. Once the SA node fires, a refectory period occurs until its next pulse. This refectory period houses the time in which the rest of the electrical circuit of the heart conducts. The location of the SA node is shown in Figure 2 produced from the Cleveland Clinic, illustrating the parts of the heart that will be discussed during this thesis [5]. The SA node will be discussed in further detail during the Timing Mechanisms section of this thesis.



Figure 2: Illustration of the Pathway of the Heart [5]

#### Right Atrium, Tricuspid Valve, and Right Ventricle

After the sinoatrial node (SA node) fires, the superior and inferior vena cava are signaled to pass blood into the right atrium. Once this occurs, the right atrium begins to pool with oxygenpoor blood collected through the veins that the body has already used (and deoxygenated). The pool of blood in the right atrium will trigger the atria to contract, signaling the tricuspid valve to open [5]. This valve, as well as the other three heart valves, acts as a gate to pass blood through. The tricuspid valve opens, allowing the pathway from the right atrium into the right ventricle, which will now begin to fill. Then, the timing mechanisms in the heart (discussed later in this section) will direct the heart to contract. The location of the right atrium, tricuspid valve, and right ventricle are shown in Figure 2 above [5].

#### Pulmonary Valve/Artery/Vein and Lungs

Picking up from the last section, the heart is in the contraction state. This contraction will open the pulmonary valve, allowing access into the pulmonary artery. The pulmonary valve is similar in nature to an electronic switch, and it opens only during the contraction of the ventricles. Once the blood travels through the pulmonary valve and into the pulmonary artery, the pathway continues into the lungs. In the electrical system of the heart, the lungs have the primary function of reoxygenating the now oxygen-poor blood. The inner workings of the lungs will not be discussed in this thesis but would be valuable to survey during future work. Once the lungs complete their mission of creating oxygen-rich blood, the circuit of the heart begins to return this blood to the body. The lungs will push the blood through the pulmonary vein, which leads into the left atrium. The location of the pulmonary valve, pulmonary vein, and pulmonary artery are shown in Figure 2 above [5]. The location of the lungs is presented in Figure 3 below, showing the pathway that the blood takes from the heart to the lungs for reoxygenation include neighboring organs [11].



Figure 3: Organs Involved in Oxygenating the Blood [11]

#### Left Atrium, Mitral Valve, Left Ventricle

After blood passes through the lungs and back into the heart through the pulmonary vein, it is reoxygenated and ready to be delivered to the body. The next step in the circuit of the heart occurs when the pulmonary vein pours the oxygen-rich blood into the left atrium. Once the left atrium is full, the mitral valve (acting as an electrical gate) opens and pours the blood into the left ventricle. Once this ventricle is filled, the heart is signaled to contract both of its bottom chambers (left and right ventricle) [5]. The location of the left atrium, mitral valve, and left ventricle are shown in Figure 2 above [5].

#### Aortic Valve, Aorta, and Completion of Hearts Circuit Throughout the Body

Once the ventricles contract, the left ventricle will push blood through the aortic valve. The aortic valve is the largest valve in the body and is responsible for delivering oxygen-rich blood to the rest of the body [9]. This valve, similarly to the other three mentioned within the pathway of the heart, will open when the ventricles contract to allow for blood to flow in one direction. Once the ventricles finish their contraction, the aortic valve will close to prevent the blood from flowing in the opposite direction. The aortic valve sends the oxygen-rich blood to the aorta, which is the largest blood vessel in the body and essentially provides blood to the rest of the body [2]. The location of the aortic valve and the aorta within the heart are shown in Figure 2 above [5].

#### **Timing Mechanisms**

The heart operates on a timing cycle that lasts for about 0.6-1.0 seconds. During this cycle, the atria must contract and relax followed by the ventricles contracting and relaxing. Timing is an intuitively important element within the circuitry of the heart, as it is apparent through the history of heart disease in humans that an inconsistency from normal rhythm can be detrimental to a person's health. The following subsections detail the cardiac conduction system, explaining how the heart delegates timing responsibilities to its different components [5]. During examination of these sections, it is important to keep in mind that atria and ventricles are constantly working on their perspective paths to keep blood moving throughout the hearts circuit – thus, the right atrium/ventricle contract with oxygen-poor blood while the left atrium/ventricle

contract with oxygen-rich blood [5]. The following subsections will reference the following diagram, illustrating the location of the SA node, AV node, bundle of His, and Purkinje fibers within the heart [10]. This is also illustrated in Figure 4.



Figure 4: Timing Mechanisms Within the Electrical System of the Heart

#### Sinoatrial Node

The first step in the operation of a heartbeat revolves around the SA node. This node, often referred to as the anatomical pacemaker of the heart, is located within the right atrium. It has two main objectives: to set the rate and the rhythm of the heart, which should be anywhere from sixty to one hundred beats per minute (BPM) for a healthy adult at rest [8]. Thus, this signal sets the timing constraint for the rest of the circuit – the path of one heartbeat must be complete before the SA node fires again. Irregularities in this firing (such as the circuit not completing in time or the SA node firing faster/slower than normal), is an indication of a heart disease. Figure 4 above shows the location of the SA node within the electrical system of the heart.

#### Atrioventricular Node

The second component of the timing mechanism of the heart is the atrioventricular node, (AV node), located in the center of the chambers of the heart between the atria and the ventricles, in an area referred to as the triangle of Koch. This node can be figuratively illustrated as a gate that can open at a rising clocks edge – its main objective is to slow down the electrical signal until the atria are empty. This delay, which consumes only a fraction of a second, allows for the contraction in progress to have an appropriate amount of time to empty the atria before contraction of the ventricles begins. Figure 4 above shows the location of the AV node within the electrical system of the heart.

An important feature that the AV node relies on is its two pathways. There is a slow pathway (which is associated with a fast refractory period), as well as a fast pathway (which is associated with a slow refractory period). During normal sinus rhythm, the signal will travel down both pathways; the fast pathway will conduct through to the bundle of His. Then, this fast pathway will go into a slow refractory period. While it is in refractory, the slow pathway will finish conduction and terminate, as there is no place for it to conduct through to (the fast pathway is in refractory). Figure 5 shows an illustration of this concept [17].



Figure 5: Illustration of AV Node During Normal Sinus Rhythm

#### Bundle of His / Bundle Branches

Next, the timing of the heartbeat will focus on the bundle of His, located in the septum of the ventricles and branching off into two areas: the left and right bundle branches. The left bundle

branch sends signals to the left ventricle, and the right bundle branch sends signals to the right bundle branch, both respectively attempting to communicate to the ventricles that it is their time to contract. The signal will travel through these branches to their ends, where the Purkinje fibers will proceed with the next step in the timing of the heart. Figure 4 above shows the location of the bundle of His within the electrical system of the heart, pointing at the right and left bundle branches.

#### Purkinje Fibers

As mentioned previously, the Purkinje fibers are located at the end of the branches of the bundle of His. Moreover, the location of these nerve cells is within the ventricle walls, underneath the surface of the inner layer of the heart, called the endocardium [9]. These pathways finger off into small trails that connect to the ventricles for proper and seamless communication of the signal. They are composed of nerve cells that specialize in quick transmission from the bundle of His to the ventricles. Once the signal is passed from the Purkinje fibers to the ventricles, the ventricular contraction will begin, allowing for the oxygenated blood to flow through the aorta and into the rest of the body, thus ending the cycle of the heartbeat. At this point, the heart will await another signal firing from the SA node. Figure 4 above shows the location of Purkinje fibers within the electrical system of the heart.

## The Electrocardiogram

One of the most powerful tools used to diagnose heart disease is the electrocardiogram (ECG - also widely referred to as the EKG, which comes from the German spelling of the word, elektrokardiogramm). The ECG is applied to the skin and the electrical signals in the heart can be sensed to read the rhythm of the heart. Note that the pathway of the heart, with its timing mechanisms, are the basis of the recording that an ECG makes. The ECG of a patient in normal sinus rhythm is shown in Figure 6.



Figure 6: ECG of a Heart in Normal Sinus Rhythm [7]

The ECG shown in Figure 6 shows a heart in normal sinus rhythm. Normal sinus rhythm consists of three main waves: the P wave, the QRS wave, and the T wave. All of these waves correspond to a specific function that the heart is performing. Along with these waves, there are also intervals that are used to determine the heart's functioning powers, namely the PR interval, the ST interval, and the QT interval. It should be noted that Figure 6 is a generalized/ideal version of the ECG and is representative of a healthy adult heart.

The first noticeable wave shown in the ECG simulation in Figure 6 is the P wave. The rise and fall of the P wave corresponds to the depolarization of the atria in the heart. Depolarization is the excitement of polarized (resting) cells. This depolarization begins when the SA node fires and ends when the signals are excited to the entrance of the AV node (and throughout the atria). This process of depolarization throughout the atria usually lasts about 0.08 to 0.10 seconds (80-100 ms) in duration during normal sinus rhythm [7]. This timing is critical to proper operation of the heart, as faster or slower heart beats can lead to chaotic contractions of the atria and ventricles.

Once the P wave settles, there is a period of isoelectric (zero voltage) time in the waveform - this is the AV node operating as intended [7]. The AV node has a function that calls it to slow down the signal, so as to allow time for the ventricles to fill before they contract. The time from which the P wave begins to signal upward to the time in which the QRS wave begins is called the PR interval. This interval can range between 0.12 to 0.20 seconds (120 to 200 ms) in duration [7]. The entire interval from the beginning of the P wave to the start of the QRS wave (or the beginning of atrial depolarization to the beginning of ventricular depolarization) is represented in the PR interval.

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The next waves shown in the ECG of the heart in normal sinus rhythm in Figure 6 above are the Q, R, and S waves, commonly referred to together as the QRS complex. The QRS complex shows the ventricular depolarization of the heart. The Q wave recognizes the movement within the interventricular septum, causing a negative voltage on the ECG because it conducts from left to right. This initializes contraction of the ventricles. Next, the R wave shows up on the ECG. This is the tallest peak in the waveform, and also accounts for the smallest amount of time. The R wave represents the powerful contraction of the ventricles, and the muscle contraction allows for the blood to be pushed through the aorta and into the body. This is a very quick movement, despite being a critical part of the heart beating process. Finally, the S wave represents the last step in ventricular repolarization, where a few small activations within the ventricles occur, and the contraction finishes. The QRS wave corresponds to the full repolarization process of the ventricles and takes about 0.06 to 0.10 seconds (60 to 100 ms) to complete in a healthy adult heart [7].

After the QRS wave completes, a brief isoelectric period occurs again called the ST interval. This period represents a still movement in the heart before refractory occurs. The last wave shown in Figure 6 is the T wave, which represents ventricular repolarization. It should be noted that this wave is generally oriented in the opposite direction (positive or negative) in comparison to the depolarization waves (QRS complex) because the cells move in opposite directions for these actions. During this repolarization, the heart muscles relax and prepare to contact the atria again. This period is slightly longer than the duration of the QRS wave, and the isoelectric period that follows before the next P wave is approximately 0.20 seconds (200 ms) in a healthy adult heart. The interval from the beginning of the QRS wave to the end of the T wave is called the QT wave, representing the start of ventricular depolarization to the end of

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repolarization. This period lasts approximately 0.20 to 0.40 seconds depending on the state of the heart [7].

As noted previously, the rate of a healthy heart with stay around sixty to one hundred beats per minute when at rest. This leads to the calculation of another interval, the RR ratio, which finds the amount of time between each heartbeat shown on an ECG. The formula to calculate this interval is

$$RR\ (ms) = \frac{1000ms}{1s} * \frac{60s}{BPM}$$

The following table shows some of the characteristics of a healthy heart that were discussed in this section.

Heart Characteristic	Healthy Heart Data
PR Interval	120 to 200 ms
QRS Interval	60 to 100 ms
ST Interval	80 to 120 ms
QT Interval	350 to 450 ms
RR Interval	600 to 1000 ms
Heart Rate	60 to 100 bpm
Rhythm	Regular Rhythm

Table 2: Timing Intervals for ECG of Healthy Heart

#### **Chapter 2: Electrical Heart Model**

The electrical system of the heart has similar properties to electrical circuits. For this reason, a model of the heart can be designed. The circuit designed in this thesis is purely theoretical and is only intended as an illustration. Future research on this topic can surround finding optimal resistor/capacitor values and finding models for the components used, as well as creating a simulation to model the electrical system. It is also important to note that the model is only modeling the blood flow during one full heartbeat, from the start of the blood path to the end. The full model is shown in Figure 7, and the reasoning for each component is explained in the following paragraph.

Each component in the circuit model used to illustrate the pathway of the heart was considered. To model the four main valves in the heart (tricuspid, pulmonary, mitral, aortic), a switch was the first choice of device to use. Since these valves have an open-and-close operation, using a switch was a fitting choice. For similar reasoning, a switch was also used to model the superior/inferior vena cava. To model the four chambers of the heart (left atrium, right atrium, left ventricle, right ventricle), a capacitor is used. To model the heartbeat, a pulse is sent through the superior/inferior vena cava. These components, along with the arbitrary resistors shown in the figure that can be used to illustrate any resistances within the heart, make up the basic circuit model shown in Figure 7. This will be the base model for the model used to illustrate the arrhythmias shown in the next section.



Figure 7: Basic Circuit Illustration Modeling the Heart

#### **Chapter 3: Atrioventricular Nodal Reentrant Tachycardia**

#### Atrioventricular Nodal Reentrant Tachycardia Conduction Pathway in the Heart

Atrioventricular Nodal Reentrant Tachycardia (AVNRT) another supraventricular tachycardia that is centralized to the AV node (and its surrounding tissue). Contrary to the similarity that the names suggest, AVNRT and AVRT are vastly different arrhythmias; AVNRT does not rely on an accessory pathway to be created. This section will reference the operation of the AV node during normal sinus rhythm, discussed in Chapter 1.

The AV node has two conduction pathways - the slow and the fast pathway. The slow pathway is associated with a fast refractory period, and the fast pathway is associated with a slow refractory period. This leads to normal conduction down the fast pathway - as this is the path of least resistance - which will lead to a quicker conduction down through the bundle of His. Once this signal reaches the bottom of the AV node, the fast pathway goes into refractory, while the slow pathway finishes its conduction. Once this slow pathway reaches the bottom of the AV node, its pathway is terminated, as its surroundings are in refractory, and it has no pathway to take. Moreover, AVNRT arises with the presence of a premature beat (this does not always lead to AVNRT, only under certain circumstances).

There are some variations of AVNRT, but the most common are called slow-fast (also called common AVNRT, accounts for about ninety percent of AVNRT cases) and fast-slow (about ten percent of AVNRT cases). For simplicity in this thesis, the slow-fast case will be discussed. Slow-fast AVNRT is reliant on a premature beat occurring. If this premature beat occurs while the fast pathway is still in its (slow) refractory period and while the slow pathway has finished its refractory period, the signal will travel down the slow pathway and reach the

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bottom of the AV node before the fast pathway. Then, if by this time the fast pathway has finished refractory, the signal can travel retrograde (back up) this fast pathway, initiating the reentrant circuit that encompasses AVNRT. Moreover, the circuit travels antegrade down the slow pathway, stimulates the ventricles, and then travels retrograde toward the atrium. This circuit rapidly activates the ventricles as it flows around the AV node (oftentimes much quicker than the normal SA node firing). The following illustration demonstrates the activation of this reentrant circuit. As shown, the signal goes down the fast pathway and then goes both through the bundle of His and retrograde up the node.



Figure 8: Illustration of AVNRT Reentrant Circuit [17]

#### Electrocardiogram of Heart with Atrioventricular Nodal Reentrant Tachycardia

When an ECG is performed on a heart that is actively in AVNRT, the first sign of irregularity will be in the rate of heart beats. In most cases, the heart rate will rise to between 140 to 280 beats per minute; there may also be a narrower QRS complex [12]. Though the heart can be beating sometimes double the rate it should be, the beating is normally a constant rhythm. The corresponding RR intervals can be calculated using the following equations:

$$RR (ms) = \frac{1000ms}{1s} * \frac{60s}{140BPM} \approx 430 ms$$
$$RR (ms) = \frac{1000ms}{1s} * \frac{60s}{280BPM} \approx 215 ms$$

In slow-fast AVNRT, an ECG may show a P wave closely coupled after QRS (or inside the QRS) because the circuit is going down the slow pathway and up the fast pathway (multiple things happening simultaneously). The P wave may also appear inverted, which would occur because it is traveling backwards [8]. This creates confusion on the ECG and is a telltale sign of an arrhythmia. Additionally, the P wave may have a smaller amplitude than normal, as the constant stimulation from the retrograde path of the AV node is stimulating it too quickly for it to complete its full contraction. Note that an EP study would be the next step in diagnosis after the ECG is conducted for proper analysis. Figure 9 shows an idealized ECG of a patient with normal sinus rhythm vs. a patient with AVNRT. Note the obvious increased heart rate and absence of the P wave.



Figure 9: ECG of a Patient with Normal Sinus Rhythm vs. a Patient with AVNRT

AVNRT is reliant on the timing mechanism of the heart centralized around the AV node. For this reason, an electrical model of this arrhythmia was left out of this research. Future work on this topic could add timing mechanisms to the electrical model in order to simulate and better model timing issues. It is clear that AVNRT presents an additional conduction pathway within the AV node, causing a reentrant circuit to affect the timing mechanism of the heart. This type of circuit would be an interesting contrast to that of AVRT, discussed in Chapter 4, as the two are often confused. The following table summarizes common intervals and data associated with AVNRT.

	Healthy Heart Data	Heart in AVNRT Data
PR Interval	120 to 200 ms	Obstruction of P interval (inverted or inside QRS) (>70 ms)
QRS Interval	60 to 100 ms	Prolonged QRS interval (>120 ms)
ST Interval	80 o 120 ms	>80 ms (loss of isoelectric time)
QT Interval	350 to 450 ms	>300 ms (loss of isoelectric time)
RR Interval	600 to 1000 ms	215 to 430 ms
Heart Rate	60 to 100 bpm	140 to 280 bpm
Rhythm	Regular Rhythm	Regular

Table 3: Comparing Healthy Heart to Heart with AVNRT [13][8]

#### **Chapter 4: Atrioventricular Reentrant Tachycardia**

#### Atrioventricular Reentrant Tachycardia Conduction Pathway in the Heart

Atrioventricular Reentrant Tachycardia (AVRT) is a macro reentrant arrhythmia that occurs when an additional pathway is introduced in the heart, particularly between the atrium and ventricles. From an electrical standpoint, this means that there is an additional pathway that arises in the heart in addition to the pathway that conducts during normal sinus rhythm. This additional, or accessory pathway, links the atria to the ventricles, confusing the conduction of the heart by allowing for unwanted communication.

AVRT requires an accessory pathway to arise. This supraventricular tachycardia occurs when the accessory pathway links the atrium and ventricles in addition to the pathway of normal sinus rhythm. This accessory pathway allows for communication confusion because as the SA node fires electrical impulses, these impulses will conduct down the pathway of the AV node (normal sinus rhythm), while simultaneously following a pathway down the accessory pathway. As discussed in Chapter 1, the AV node acts as a gateway to the ventricles to allow for their proper filling of blood. However, the pathway down the accessory pathway does not have this timing mechanism. Therefore, the ventricles are stimulated prior to their proper filling, and prior to when the normal conduction pathway would have stimulated them. Figure 10 shows an example of an accessory pathway.

There are two main types of AVRT: orthodromic (which accounts for approximately 90-95 percent of AVRT cases), and antidromic AVRT (which accounts for approximately 5 to 10 percent of AVRT cases [12]. During orthodromic AVRT, conduction through the heart occurs in the antegrade direction (from the atrium to the ventricles, back to the atrium). This is called the

manifest pathway [17]. Then, the signal travels back up the ventricles to the atrium (oftentimes resulting in a retrograde P wave after the QRS wave shown on the ECG). During antidromic AVRT, conduction through the heart occurs in the retrograde direction (from the ventricles to the atrium, and back to the ventricles). This is called the concealed pathway, as it commonly requires an electrophysiology study to be conducted before it can be diagnosed [17].



Figure 10: Example of Accessory Pathway Taken During AVRT [1]

The most common condition to show AVRT is called Wolff-Parkinson-White (WPW) syndrome (AVRT and WPW are sometimes even used interchangeably, though AVRT is just the most

common arrhythmia seen with WPW) [12]. This occurs with orthodromic operation of AVRT. For simplicity, the WPW occurrence of AVRT will be analyzed during discussion of this thesis, but it should be noted that other occurrences can (less commonly) occur. In WPW, the accessory pathway that links the atrium to the ventricles is stimulated by the SA node (at the same time that the signal is performing its normal conduction pathway, through the AV node). The following image [14] will be used as a tool to explain the pathways within the heart.



Figure 11: Conduction Path Taken During AVRT with WPW Syndrome [14]

As shown in Figure 11, the accessory pathway that connects the atrium and ventricles creates a means for the reentrant circuit. Note that the presence of this accessory pathway alone does not always mean that AVRT will occur, as a premature beat is usually necessary to initiate the arrhythmia. Additionally, AVRT is reliant on the refractory periods of both the accessory pathway and the normal conduction pathway. To explain step, by step, first, the SA node fires a signal. Then, the signal travels to the entrance of the normal conduction pathway as well as the entrance of the accessory pathway. The heart then analyzes the refractory periods of the pathways - if the normal conduction pathway is ready to conduct again (as shown in the image), it will, and vice versa with the accessory pathway. WPW syndrome occurs when the normal conduction pathway has completed its refractory period, but the accessory pathway has not. Thus, the next step is that the signal will conduct through the AV node (normally), and travel down the bundle of His. Things will operate seemingly as normal, until the signal is attempting to depolarize through the Purkinje fibers. The end of the normal conduction pathway will present the accessory pathway, which has now completed its refractory period and is ready to conduct again. Thus, similar to a typical circuit, the signal will continue to pass through this pathway. As shown in Figure 11, once that signal travels through the accessory pathway, it enters the atrium again, where the normal conduction pathway has finished its refractory period (and is ready to conduct again. Thus, this same signal has the ability to reenter the AV node and excite the ventricles again. This creates the unwanted circuital pathway shown in the Figure 11, known as the WPW syndrome case of AVRT.

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#### Electrocardiogram of Heart with Atrioventricular Reentrant Tachycardia

The electrocardiogram that a patient in AVRT shows can vary. For brevity, the one discussed in this thesis will detail an ECG that shows a heart in antidromic AVRT (with WPW syndrome) traveling in the retrograde direction. Note that an EP study would be necessary for proper diagnosis.

Perhaps the most common suggestion of AVRT is shown through the presence of a delta wave on the ECG. A delta wave is a prolongation of the QRS complex that arises when an accessory pathway is conducting a signal from the atria to the ventricles. This is a slow process, but contraction of the ventricles will occur faster than the normal conduction pathway because there are no timing mechanisms (AV node) to slow it down. It is shown as a slow uprising in comparison to the normal QRS complex because it has a different conduction pathway. An ECG showing a heart in AVRT with a highlighted delta wave is shown in Figure 12 [1], with comparison to a healthy heart for comparison.



Figure 12: ECG of Heart in Normal Sinus Rhythm Vs. Heart showing WPW syndrome AVRT [1]

In addition to the presence of a delta wave on the ECG, there are other timing signs that the test can show that indicate a linkage to AVRT. For example, a heart rate from 150 to 250 beats per minute would be an indicator of AVRT [12]. This occurs because the accessory pathway is constantly conducting in addition to the normal conduction pathway. The corresponding RR intervals can be calculated using the following equations:

$$RR(ms) = \frac{1000ms}{1s} * \frac{60s}{150BPM} = 400 ms$$
$$RR(ms) = \frac{1000ms}{1s} * \frac{60s}{250BPM} = 240 ms$$

Also, a complete or partial obstruction of the P wave may occur, as this normally represents atrial depolarization. Moreover, this P wave may occur at the end of the QRS duration, as the atria may be the last mechanism to contract. The following table summarizes common intervals and data associated with AVRT.

	Healthy Heart Data	Heart with AVRT Data
PR Interval	< 120 ms	Obstruction of P interval (>70 ms)
QRS Interval	60 to 100 ms	Prolonged QRS interval (delta wave) (>120 ms)
ST Interval	80 to 120 ms	80 to 120 ms
QT Interval	350 to 450 ms	350 to 450 ms
RR Interval	600 to 1000 ms	240 to 400 ms
Heart Rate	60 to 100 bpm	150 to 250 bpm
Rhythm	Regular Rhythm	Regular

Table 4: Comparing Healthy Heart to Heart with AVRT [13][8]

#### Atrioventricular Reentrant Tachycardia LTSpice Circuit Model

A circuital model can be used to illustrate the pathway that a heart in AVRT is forced to take. Note that Chapter 2 explains the basic model that was used, and this section will explain the changes. As explained earlier, AVRT (particularly, WPW syndrome) is reliant on an accessory pathway that connects a ventricle to its corresponding sides atrium. Thus, an additional conduction pathway can illustrate this arrhythmia. For the model, the most common left side connection was made, but note that this could happen on either side of the heart. Figure 13 shows this addition to the circuit, highlighting the theoretical area of the heart surrounding the mitral valve.

The current in this circuit will be forced down this pathway, taking current (which is modeling blood) that is going through the mitral valve. Note that timing mechanisms are not modeled here, but this extra pathway would interfere with the internal timing of the heart (as the blood in the accessory pathway will reach the ventricles at a different time than the blood that is going through the mitral valve). The full circuit is shown in Figure 14.



Figure 13: Modeling of AVRT in the Pathway of the Heart



Figure 14: Modeling the Pathway of the Heart with AVRT

#### **Chapter 5: Atrial Flutter**

#### **Atrial Flutter Conduction Pathway in the Heart**

Atrial Flutter is a paroxysmal arrhythmia that is centered around the atria of the heart. This can be seen as a macro reentrant circuit throughout the left or right atria. There are two types of flutter: typical atrial flutter and atypical atrial flutter. Typical atrial flutter is localized in the right atrium and is the more common type [10]. Atypical atrial flutter is localized to the left atrium and is less common [10]. For simplicity, this thesis will model and address typical flutter. Atrial flutter causes an increased heart rate, with upper chambers beating at up to 350 beats per minute. This causes quicker conduction through the AV node, but because of its built in timing mechanism, the lower ventricles are beating at up to 150 beats per minute. It should be noted that unlike atrial fibrillation (discussed in Chapter 6), the beating of atria suffering from flutter are electrically regular [5]. This means that though there is an increased rhythm, the rhythm is conducting in a nonchaotic way.

#### **Electrocardiogram of Heart with Atrial Flutter**

The ECG of a patient with atrial flutter will differ from patient to patient, but an example is shown in Figure 15 [13]. A ratio is commonly used to specify the amount of atrial contractions to ventricular contractions (such as 2:1, where there are two atrial beats for every ventricular beat, or 3:1, where there are three atrial beats for every ventricular beat) [13]. Since flutter commonly has a regular rhythm, the ECG can show this ratio. As seen in Figure 15 below, there is a clear repetition to the beat. The two sawtooth waves resemble the atrial contractions, and the tall spike represents the ventricular contraction. The two (possibly three) atrial contractions here are due to the reentrant circuit that is circling the right atrium. The additional P waves shown per QRS

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complex on the ECG of a heart with flutter are commonly called flutter waves. Additionally, there is commonly a loss of the isoelectric time periods that are found on the ECG of normal hearts.



Figure 15: ECG of Patient with Atrial Flutter [13]

Note the multiple sawtooth waves that are associated with each QRS complex. Though the heart can be beating at up to triple the rate it would be during normal sinus rhythm, the beating is normally a regular rhythm. A theoretical RR interval can be calculated using the following equation, assuming that the heart is beating at a constant rate of less than three hundred beats per minute:

$$RR(ms) = \frac{1000ms}{1s} * \frac{60s}{300BPM} \approx 200 ms$$

The following table summarizes common intervals and data associated with atrial flutter discussed in this chapter. Note that the P and T waves are oftentimes concealed by flutter waves, making them difficult to measure.

	Healthy Heart Data	Heart with Atrial Flutter Data
PR Interval	120 to 200 ms	Multiple P waves per heartbeat (flutter waves)
QRS Interval	60 to 100 ms	60 to 100 ms
ST Interval	80 to 120 ms	< 0 ms (oftentimes concealed)
QT Interval	350 450 ms	< 0 ms (oftentimes concealed)
RR Interval	600 1000 ms	> 200 ms
Heart Rate	60 to 100 bpm	> 300 bpm
Rhythm	Regular Rhythm	Regular Rhythm

 Table 5: Comparing Healthy Heart to Heart with Atrial Flutter [13][8]

## **Atrial Flutter LTSpice Circuit Model**

A theoretical example circuit of a heart with atrial flutter is shown in the following LTSpice circuit. As explained previously, atrial flutter occurs when there is an additional conduction pathway within the right atrium, causing a reentrant circuit to affect the rhythm of the heart. Similar to AVRT, this can be modeled as an additional pathway. Since typical flutter is the most common flutter, this is what was chosen to be modeled. The circuital addition shows a second pathway that surrounds the right atria. The model is shown in Figure 16 and further explained below.



Figure 16: Modeling Atrial Flutter within the Pathway of the Heart

Typical flutter can be illustrated by creating a path within the right atria – or, in other words, creating a new path for the current to pass through. This is modeled with an additional wired path capable of conduction that pass around the right atria. Thus, the current will path

through the R15 resistor and the C8 capacitor that is modeling the right atria, and then follow the pathway through the tricuspid valve; simultaneously, the current will flow through the R2 resistor (modeling resistance outside the right atrium). These two pathways will allow for the atria to be stimulated by two different mechanisms. Figure 17 shows the modeling within the system of the heart.



Figure 17: Modeling the Pathway of the Heart with Atrial Flutter

#### **Chapter 6: Atrial Fibrillation**

#### Atrial Fibrillation Conduction Pathway in the Heart

Atrial fibrillation, commonly known as AFib or A-Fib, is the most common arrhythmia seen in humans. An estimated twenty two percent of strokes are related to A-Fib [15]. The disease is wildly predicted to increase its appearance in the coming years, thus making understanding its effects even more important. Having atrial fibrillation also makes the chances of a person having a stroke increase by five times [15].

In normal sinus rhythm, the heart follows the pathway and timing of the heart discussed in Chapter 1. A heart undergoing atrial fibrillation will experience a chaotic firing of the SA node. This chaotic firing is a direct result of an improper electrical signal firing, mostly presenting itself in a rapidly occurring heartbeat. This acceleration in the heartbeat does not allow enough time for blood to complete its circuit throughout the heart and can lead to pooling/clotting [5]. For example, if the SA node fires one signal and it follows for the atria to contract, and then fires another signal before the ventricles to contract, chaos erupts in the heart as the atria and ventricles are signaled to contract in nonuniform ways. This chaos is illustrated in Figure 18 [10]. The yellow signals represent the confusion of the atria and depict why this would put stress on the heart.



Figure 18: Illustration of Heart in Atrial Fibrillation [10]

A-Fib occurs as a result of either an ectopic foci or a reentry circuit [8]. When an ectopic foci point occurs, there are pacemaker cells accumulating in an additional point to the SA node. This confuses the heart, as there are two different points of circuit initialization. Thus, the heart will conduct as normal, as in the SA node will fire, go through the AV node, and the atria and ventricles will contract. However, in addition to this occurring, there is an additional point (usually in the right atrium or an additional point in the left atrium) where a signal is generated and also sent through the AV node, where the atria and ventricles are signals to contract again. Intuitively, it follows that this would cause chaos and irregularity in the circuit of the heart. For an electronic simile, it would be as if an additional clock was inputted to the middle of a circuit, randomly creating high and low points.

In addition to an ectopic foci point, A-Fib can also arise due to the development of another reentry circuit, some of which are discussed in sections (AVRT, AVNRT). These reentrant circuits can cause chaos in the atria and the firing of pacemaker cells, also leading to the appearance of A-Fib in the heart. Thus, A-Fib can be diagnosed in addition to another heart disease.

#### **Electrocardiogram of Heart with Atrial Fibrillation**

The appearance of A-Fib is most easily explained by using the ECG reading as a tool. Figure 19 makes the appearance of an irregular heartbeat somewhat obvious - the heart in A-Fib has no clear pattern to it. The QRS complex is most easily recognizable as an incorrect/irregular pattern of the heart. The chaos occurring within the heart is shown in comparison to a heart in normal sinus rhythm, and it can be seen that there are typically several QRS complexes occurring in a heart in A-Fib for every single heartbeat that occurs in a healthy heart. In addition to the chaotic QRS complexes, the figure shows that there is no clear P wave, T wave, or continuity of the electrical system. This also indicates that there is no clear and consistent timing study for a heart in A-Fib. Though the heart rate is commonly increased in comparison to normal sinus rhythm, it is not normally as drastic as other arrhythmias, such as AVRT and Atrial Flutter. Heart rate will normally range from about 100 to 175 beats per minute. The corresponding RR intervals can be calculated using the following equations:

$$RR(ms) = \frac{1000ms}{1s} * \frac{60s}{100BPM} \approx 600 ms$$

$$RR(ms) = \frac{1000ms}{1s} * \frac{60s}{175BPM} \approx 350 \, ms$$

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Figure 19: ECG Comparison of Heart in Normal Sinus Rhythm Vs. Heart in A-Fib [16]

	Healthy Heart Data	Heart in A-Fib Data
PR Interval	120 to 200 ms	Irregular per heartbeat
QRS Interval	60 to 100 ms	60 to 100 ms
ST Interval	80 to 120 ms	Irregular per heartbeat
QT Interval	350 to 450 ms	Irregular per heartbeat
RR	600 to 1000 ms	350 to 600 ms
Heart Rate	60 to 100 bpm	100 to 175 bpm
Rhythm	Regular Rhythm	Irregular

 Table 6: Comparing Healthy Heart to Heart with A-Fib [13][8]

#### **Atrial Fibrillation LTSpice Circuit Model**

Atrial fibrillation is a heart disease that interferes with the normal sinus rhythm pathway of the heart, alluding that it can be modeled by an electrical circuit. The following images show the addition to the pathway of the heart that can model A-Fib. Since this heart disease is characterized by a chaotic firing of signals from the SA node (oftentimes due to an ectopic foci point), the additional point can be illustrated using a voltage source in series with the SA node with differently timed pulses. These pulses in series will create a chaotic input signal for the rest of the pathway of the heart. Thus, there will not be regularity about the timing of the pathway. This will cause irregular stimulation of the heart. An example of the input signals to the heart from an electrical standpoint can be seen in Figures 20a and 20b. This simulation shows the way the input signal of the heart may theoretically fire when in atrial fibrillation.



Figure 20a: Circuit Modeling of Theoretical Input Signals Associated with A-Fib



Figure 20b: Simulation of Theoretical Input Signals Associated with A-Fib

By applying this information to the base illustration of a healthy heart, a model of how A-Fib may look in the pathway of the heart can be created. Note that it should be assumed that a rising positive voltage in the circuit would resemble a singular heartbeat. Also note that a behavioral voltage source with a random voltage setting could also be used to simulate the chaotic pulses that A-Fib presents. The circuital model shown in Figure 21 illustrates this idea of atrial fibrillation in the pathway of the heart.



Figure 21: Modeling the Pathway of the Heart with Atrial Fibrillation

#### **Chapter 7: Future Work and Conclusion**

#### **Future Work**

Future work on this topic can take a few different avenues. The first realm where future work can be helpful surrounds heart diseases that were not discussed in this paper. This can include bradycardias, ventricular fibrillation, or other less common supraventricular tachycardias. Surveying the link that the pathway in the heart creates and comparing it to that of normal sinus rhythm can aid in understanding the heart from an electrical standpoint. Additionally, creating circuital models that illustrate the disease in comparison to that of a healthy heart can be especially helpful to those with an electrical engineering background entering the electrophysiology field, as knowing the differences in the healthy and unhealthy heart is integral to understanding electrophysiology.

The second avenue of future work that can be beneficial is in simulation of the circuits. Since this research focused on making an illustration of the heart through electrical components, simulations of the heart have been left untouched by this modeling. Research into modeling and simulating heartbeats, capacitance, and pressure can be especially helpful with electrophysiology training. Furthermore, research into modeling and simulating the timing mechanisms discussed in this research would aid in understanding of the timing of heartbeats from an electrical point of view. This can also be helpful in simulation, as a real-time wave form can be created with further research in this realm.

When considering future work regarding the circuit models presented in this thesis, it is important to note that these are idealized, illustrative representations of a single heartbeats' pathway. Moreover, the pathway begins with the SA node firing and vena cava filling the right

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atrium and ends with the blood being returned to the body. To review, the capacitor that is illustrated at the end (C5, after the aortic valve switch) of the circuit is representing the termination of one heartbeat. Understanding this is critical for future work in the realm of simulations, and it should be noted that the oxygen in this blood would then be absorbed by the body and returned to the heart through the vena cava. Future models may benefit from exploring a circuital connection from the body to the vena cava (with the addition of timing mechanisms). This will model the passing of blood from the heart, to the body, and back into the heart (oxygenation, deoxygenation, back to oxygenation). This can aid in modeling ECGs and timing more accurately and clearly.

#### Conclusion

This research has surveyed four common arrhythmias associated with the human heart and analyzed their electrocardiograms to aid in understanding. Atrioventricular reentrant tachycardia, atrioventricular nodal reentrant tachycardia, atrial flutter, and atrial fibrillation were discussed, and their pathways analyzed and compared. Additionally, timing mechanisms were discussed and charted for clear understanding of the timing that a healthy heartbeat will have; this is then presented in comparison to hearts with AVRT, AVNRT, atrial flutter, and A-Fib. An LTSpice circuit model was created to aid in illustrating the pathway of a healthy heart and compare it to examples of the pathway of an unhealthy heart. Finally, future work was discussed to present ideas for this research to continue.

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