# <u>Electricity Within The Heart</u>

**Trevor Dylan Smith** 

Lab: H1

April 25, 2012

### Smith 2

The human heart has enthralled physicians, philosophers, and poets for eons. While it still

eludes the latter two, it has come within the grasp of physicians. Culminating with transplant surgery, internal regulation, and external monitoring, the medical arts have unlocked most secrets of the heart which long lay undiscovered. These advancements were made possible through an improved understanding of nerve signals involving the heart. The complex series of pathways and relays traversed by a signal from one end of the heart to the other with autonomic nervous system regulation is fraught with opportunities for disruption. When these disruptions occur, causing diseases and disorders, physicians utilize the interdisciplinary wonders of modern medicine to diagnose and treat a patient.

Currently, biological scientists have studied the body's physiology well enough to have a general understanding of the cardiovascular system, even though specific diseases and disorders may elude researchers. Although the heart's muscle contractions have automaticity, they are regulated through the nervous system with chemical balances (Huszar 2003, 18). Automaticity is the heart's ability to depolarize cardiac muscle without external nervous stimulation. Depolarization and repolarization of cardiac muscle is the method through which myocardium is made to contract. Depolarization is the decrease of a net negative charge through a transfer of calcium cations into the cell. Heart rate is controlled through the regulation of depolarization and action potential duration by the autonomic nervous system via the sympathetic and parasympathetic nerve fibers. The heart's rate is accelerated and decelerated through efferent innervation – regulatory nerve stimulation – of the sympathetic and parasympathetic nerves, respectively. Going backwards from the actual contraction of the heart, the heart rate is the rate of depolarization of myocardial tissue. This rate increases when calcium channels are opened more. A factor in opening calcium channels is increased cAMP levels of the heart. The

#### evor Smith 25/4/12 12:23

**Comment:** awkward sentence, reword. also change later to latter. Maybe change "remains elusive from" to "eludes". See previous comment for more stuff that relates to this sentence

Trevor Smith 25/4/12 11:37 Comment: unclear sympathetic nerve increases cAMP levels through stimulating the  $\beta$ 1 receptors. The vagus nerve, so named for its origins – the vagal dorsal motor nuclei of the medulla – is seen to counteract this, supplying parasympathetic innervation to the sinoatrial node. The primary method of the vagus nerve is in release of the neurotransmitter acetylcholine, which follows an action potential and binds with muscarinic receptors to decrease heart rate (Anderson & del Castillo 1972, 236-257).

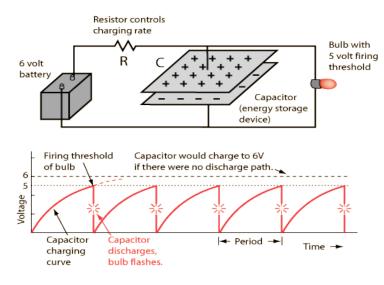
When transmitted along the nervous pathways, signals are composed of an action potential and neurotransmitter. At rest, a nerve's voltage-gated ion channels are closed. As the membrane potential rises, channels open, allowing an inward flow of either sodium or calcium cations. Calcium is more associated with the sinoatrial and atrioventricular nodes than other cardiac tissue. An increase in such ions causes a rise in membrane potential, which creates a spiraling set of events. Once all ion channels are open and the membrane potential has peaked, the voltage-gated ion channels inactivate. In cells utilizing positively charged sodium ions, this maximum plateau is short-lived. In cardiac nodes, which use calcium ions more often, the maximum plateau lasts for a few hundred milliseconds. Active transport removes the sodium or calcium ions. At the same time, potassium channels activate, facilitating a flow of potassium ions out of the cell, returning homeostasis to the cell. An action potential - or rapid firing of electrical signal from one nerve to the next along the axon terminal, synaptic cleft, and dendrite pathway - courses through the nervous system (West 1972, 191-216).

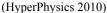
This electrical potential is the difference in concentration of ion across the cell membrane and is measured in millivolts. At rest, the myocardial cell is negatively charged and surrounded by a high concentration of sodium cations. For all cardiac cells, not including those in the Following this electrical event are neurotransmitters, or simple chemicals synthesized from amino acids, monoamines, peptides, and other readily available materials. Otto Loewi, a German researcher of the human body, discovered that neurotransmitters may be initiated by electrical impulse, but have the potential to control nervous function alone (*Nobel Lectures*). In his 1921 experiment, he extracted a chemical which by itself decreased the heart rate. This chemical, which he named Vagusstoff, had already been discovered and named acetylcholine. It was the first neurotransmitter to be discovered. Once at the heart, the signal notifies controlling centers, or nodes, of the heart to operate, which in turn set the heart's muscles into motion. These nodes, controlling the atria and ventricles, repeatedly initiate the beating of a heart, which pumps blood throughout the body.

The commonly termed primary cardiac pacemaker, or sinoatrial node, is situated within the right atrium's upper wall and begins the action potential, which in turn sets atrial contractions into motion through electrical stimulation. The sinoatrial node routinely depolarizes, discharges, and repolarizes without stimulation (but with regulation) from the nervous system in much the same manner as a relaxation oscillator in electronics (Hyperphysics 2010). An oscillator of this type contains a battery, resistor, capacitor, and light (or other electric device) requiring less voltage than the battery's maximum in the configuration below. The capacitor will charge to the battery's voltage, discharging at the 'firing threshold' of the light. This mimics the electrical stimulation from the sinoatrial node, which depolarizes and repolarizes myocytes. This shift in polarity causes muscular activation in much the same manner that nerves are activated. The muscles of the atria themselves conduct this electrical signal. After passing through the atria, the Comment: who?

evor Smith 25/4/12 11:47

**Comment:** reword so it's a sentence. Something like "this neurotransmitter, acetylcholine, was the first to be discovered" signal reaches the atrioventricular node within the lower right atrium. Here the signal is halted so that blood from each atrium might fill its respective ventricle. This is typically a 0.12s delay in adult humans. The electrical action potential is then unleashed again, this time not travelling directly through the muscle fibers.





Through the ventricles, electrical signals travel through the Bundle of His, fascicular branches, and Purkinje fibers (Mendez & Moe 1972, 263-289). The Bundle of His (named for its 1893 discoverer, Swiss cardiologist Wilhelm His, Jr.) conducts current along the ventricular septum, connecting at the apex of the fascicular branches. The fascicular branches in turn carry current to the Purkinje fibers. Purkinje fibers carry signals directly back along the ventricles to force pulmonary circulation of the right ventricle or systemic circulation of the left ventricle through exciting the ventricle's myocardium. Within 0.03-0.04 seconds of an electrical impulse from the atrioventricular node, ventricular muscle is activated. If the sinoatrial node fails to initiate an electrical signal, the heart is formed to rely on the atrioventricular node. Left to its Trevor Smith 25/4/12 11:56 **Comment:** is this an abbreviation? automaticity, the atrioventricular node can set the heart beating at 40-60 beats per minute. This is much slower than the sinoatrial node's 60-100 beats per minute, but is seen as one of the last resorts of the heart. Another evolutionary failsafe that the heart contains is the firing of the Purkinje fibers themselves at 30-40 beats per minute. Just like the atrioventricular node, this is less productive in comparison with the sinoatrial node (Huszar 2002, 16).

When these nerve signals are not sent or received properly, severe disorders may occur. An array of psychological issues can arise from faulty neurotransmitter systems. There are also physical maladies which result from the same problems. When the heart receives signals incorrectly or doesn't receive the normal sequence of signals, it can beat abnormally or not at all. The generic term for an abnormally fast, slow, or irregular heartbeat is dysrhythmia or arrhythmia. Classification of an arrhythmia is based upon speed of heart rate, specific mechanism, and origin. Where an arrhythmia develops can greatly affect its consequences on the heart and body.

Such a case can be seen in the difference between atrial and ventricular fibrillation. Within the atria, fibrillation may be either transient or permanent. In many cases, atrial fibrillation is treated as the effect and symptom of a greater malady. Medications have been developed to control both the heart's rate and rhythm. The greatest danger from atrial fibrillation is an increase in the risk of death when paired with chronic conditions (Huszar 2002, 122-125). On the contrary, ventricular fibrillation cannot be permanent. Within minutes of a continuing episode of fibrillation situated in the ventricles, blood circulation can cease and cerebral hypoxia may result. To reverse the condition, CPR, defibrillation, or adrenaline must be administered (Huszar 2002, 155-158, 168). Trevor Smith 25/4/12 11:58 Comment: no comma

Trevor Smith 25/4/12 11:59 **Comment:** one word?

#### Smith 6

Slight arrhythmias are not uncommon and cause no undue strain upon the body. A mild acceleration or deceleration of the heart rate termed sinus arrhythmia can occur with respiration, most noticeably in children and during meditation exercises. Wolff-Parkinson-White Syndrome is a more pronounced example of a non-threatening arrhythmia. This specific arrhythmia is due to presence of the Bundle of Kent (named for its discoverer, British physiologist Albert Frank Stanley Kent), an abnormal series of paths through which electrical signals may bypass the atrioventricular node to excite the ventricles prematurely. Normally, this syndrome is benign, but rarely sudden cardiac death may occur from atrial tachydysrhythmia being conducted into the ventricles (Conover 2002, 275). There are, however, extreme cases of arrhythmia where the body's level of oxygen decreases enough to harm the brain. Cardiac arrest resulting in a slackening in blood circulation will lead to hypoxia if left untreated. When this happens, the heart must be forced to resume its regular beating or serious brain injury and death will result.

Atrioventricular heart blocks are a form of arrhythmia resulting from an irregular operation of the atrioventricular node. There are three degrees of heart block described by Huszar (2002, 171-187), with first being the least destructive and third the most serious. Firstdegree heart block develops from the atrioventricular node delaying an electrical signal beyond 0.20 seconds and is commonly brought on by diseases of the atrioventricular node, myocarditis, acute myocardial infarction, medications, electrolyte disturbance, and enhanced vagal tone. Unless the heart block was brought on by a more serious occurrence, treatment typically consists of modifying medications or electrolyte balance. Second-degree heart block types 1 and 2 arise when some of the sinoatrial node's electrical signal is conducted to the ventricles, but not all. Type 1 is the more benign of the pair, identified by an increasingly greater interval of electrical signal delay at the atrioventricular node followed by a 'skipped' ventricular contraction. No treatment is required in this instance if the atrial rhythm was not the root cause of an arrhythmia. Type 2 is much more serious, characterized by electrical signals being infrequently not conducted. This can be often traced to a disease within the Bundle of His or Purkinje fibers and is treated through implantation of a cardiac pacemaker/defibrillator. A greater risk stems from the possibility of progression to third-degree heart block.

Third-degree heart block is the term for a complete failure of electrical signals from the sinoatrial node to propagate into ventricular tissue. To continue circulating blood throughout the body, the heart relies on its failsafe mechanisms. Such are cardiac automaticity of the atrioventricular node and Purkinje fibers, depending on where the heart block is situated. When relying upon a secondary stimulation of the ventricles, bradycardia will often result with a clear disjunction between atrial and ventricular rhythm. Rarely, Lyme disease and birth defects are the cause of complete heart block. More often, an acute myocardial infarction, coronary ischemia, or degeneration of any portion of the electrical system will be the cause. Two forms of an acute myocardial infarction damage the heart and can be the onset of a complete heart block. Anterior wall myocardial infarctions yield permanent damage to the Bundle of His or Purkinje fibers, requiring implantation of a pacemaker/defibrillator. Inferior wall myocardial infarctions affect the atrioventricular node, but recovery usually occurs. Both forms of myocardial infarctions can be brought on by coronary ischemia. The progression of damage from various sources to the cardiac electrical conductance system can eventually lead to failure and a third-degree heart block.

Treatment for many arrhythmias can include medications, movement, electric shock, and cauterization (Huszar 2002, 188-239). Several antiarrhythmic drugs actually have the potential to instigate arrhythmia, requiring close supervision. Other medications are used to control heart rate

**Comment:** is there a reason why this is capitalized?

#### Smith 8

or prevent the potential blood clots resulting from arrhythmias. Internal and external electrodes can be utilized to apply electric shock to the heart. Cardiac pacing is used for instances of bradycardia, either through temporarily external or permanently implanted pacemakers. Electric shock varies in whether the application is synchronized with the heartbeat or not. When electric shock is synchronized with the heartbeat, this is termed cardioversion. Medication can also achieve this effect. With cardioversion via electricity, patients are usually suffering from supraventricular tachycardias and will be mildly sedated. Electric shock applied without synchronization to the heartbeat because of its absence is termed defibrillation. More electricity is used for defibrillation with no anesthetic for the unconscious patient. Many public facilities now have access to an automated external defibrillator; these are designed to aid persons unfamiliar with medical technology through instructions in print, audio, or video. Cauterization remains an inconsistent form of treatment for certain arrhythmias, while others are curable. Faulty electric pathways of the heart are eliminated by freezing them or burning them with heat, a laser, or electricity.

After suffering a myocardial infarction, it isn't uncommon for a cardiologist to recommend implantation of an internal defibrillator (Antman 2004, 618). Such devices monitor the heart's beating and initiate an electrical impulse if there happens to be a lapse in beating. Most often, an internal defibrillator is offered as an implantable cardioverter-defibrillator (ICD). An ICD is used when a patient requires a permanent heart monitor and may experience any number of cardiac dysfunctions from myocardial infarction to ventricular fibrillation, either of which can result in death otherwise. Another commonly used device is an internal pacemaker, meant to either replace or augment the heart's electric conduction of signals. Several pacemakers are produced ranging from temporary to permanent; atrial or ventricular; and constant to heart Trevor Smith 25/4/12 12:05 Comment: aid

evor 3111(11 23/4/12 12.07

**Comment:** incomplete/confusing as to what you mean

rate-responsive pacing. The benefit of a pacemaker controlling both the atria and ventricles is a coordinated heartbeat, maximizing blood circulation and preventing arrhythmias. Although it would seem in a patient's best interest to have the ICD or pacemaker in full control of his/her heart rate, the right ventricle can suffer damage from constant manipulation. The development of rate-responsive pacing relieves a great deal of this danger.

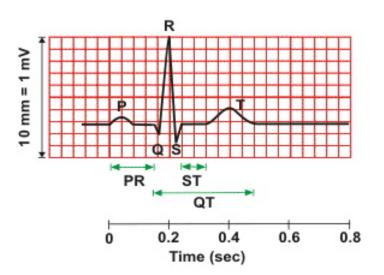
Externally, monitor devices such as the electrocardiograph (ECG) have been developed. An ECG utilizes several monitoring points on the body to produce an informative set of data concerning the heart over a period of time. Detection of drug or device induced change, arrhythmia, heart structure/damage is possible through reading an electrocardiograph. Electrocardiographs can be printed, digital, and computer-monitored. To achieve an accurate reading in a 12-lead ECG, there is typically a set of ten electrodes placed across key areas of the body; six on the abdomen/chest cavity, one on each wrist, and one on each ankle to monitor the heart, radial arteries, and pedal arteries, respectively (each pair of limbs creates one lead). Additional leads may be used on the back or the limb leads can be resituated nearer to the torso as long as they remain symmetrical. A 12-lead ECG is typically used to detect serious heart disorders and is printed for future study. An ECG may also be conducted using only 3-5 leads. Such a reading is useful for surgeries, transport, and other instances where the constant monitoring of a patient's heart is necessitated. In an electrocardiograph, the electric charge of the heart is monitored. The muscle is depolarized from its negative charge by an influx of positive sodium or calcium ions. The unique points are related to differences in charge of the atria and ventricles (Conover 2003).

In *Understanding Electrocardiography*, Mary Conover gives a thorough explanation for each component of the ECG wave. As is shown below, there are five basic portions of the entire

**Comment:** extra gap in the space above between paragraphs

wave; each contributes to a complete assessment of the heart. Those portions of the wave travelling in the positive direction are moving left across the heart and those moving negatively are representing right-directed electric currents. The P wave corresponds to the activation of first the right atrium, then the left atrium and atrioventricular node, by the sinoatrial node. When imbalanced, the cause can be an abnormality in one of the atria. The PR segment is the nearly straight line representing the bundle of His and Purkinje fibers being activated.

**Comment:** supposed to be capitalized?



(Cardiovascular Physiology Concepts 2007)

As the first portion of either ventricle to be activated, the interventricular septum conducts electricity from left to right, resulting in a negative spike, Q. The large QRS complex represents the electrical activation and contraction of the ventricles. The mild T wave is a representation of repolarization of the ventricles. When accessory pathways are developed across the heart between the atria and ventricles, a delta wave can develop, eliminating the Q spike and smoothing out the shape from the P and R waves. This is indicative of Wolff-Parkinson-White Syndrome with development of the bundle of Kent.

Although biomedical engineering has broadened the abilities of modern physicians, there is still much that can be done towards miniaturizing technology and minimalizing invasive procedures. The major issues of both are the size and limitations of implanted cardioverterdefibrillators, pain associated with cauterization of myocardial muscle or nerves, and problems of arrhythmia medication. Major disorders, such as failure of the sympathetic or parasympathetic nerves, sinoatrial node, atrioventricular node, and all other portions of the heart have developed solutions from within the heart itself only to be improved by modern physicians. The cardiac muscles are unique among all other cells of the human body in their ability to both conduct electrical signals and contract to circulate blood.

## Works Cited:

- Anderson, Margaret & del Castillo, J. 1972. "Cardiac Innervation and Synaptic Transmission in [the] Heart." In *Electrical Phenomena in the Heart*, ed. Walmor C. De Mello, 236-257. New York: Academic Press.
- Antman, et al. 2004. "ACC/AHA Guidelines for the Management of Patients with ST-Elevation Myocardial Infarction." *Circulation* 110: 588-636.
- Conover, Mary Boudreau. 2003. Understanding Electrocardiography. St. Louis: Mosby.

Huszar, Robert J. 2002. Basic Dysrhythmias: Interpretation & Management. St. Louis: Mosby.

- Klabunde, Richard E. "Electrocardiogram (EKG, ECG)." 2007. Cardiovascular Physiology Concepts. Accessed April 15, 2012. <u>http://www.cvphysiology.com/Arrhythmias/A009.htm</u>
- Mendez, Carlos & Moe, Gordon K. 1972. "Atrioventricular Transmission." In *Electrical Phenomena in the Heart*, ed Walmor C. De Mello, 263-289. New York: Academic Press.
- Nave, C. R. "Relaxation Oscillator Concept." 2010. HyperPhysics. Accessed April 18, 2012. http://hyperphysics.phy-astr.gsu.edu/hbase/electronic/relaxo.html#c2
- "Otto Loewi Biography." 1965. In *Nobel Lectures, Physiology or Medicine 1922-1941*. Amsterdam: Elsevier Publishing Company.
- West, Theodore C. 1972. "Electrophysiology of the Sinoatrial Node." In *Electrical Phenomena* in the Heart, ed. Walmor C. De Mello, 191-216. New York: Academic Press.