# CV 1. HEART ELECTRICAL ACTIVTY

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## LEARNING OBJECTIVES

1. Describe the conduction system of the heart
2. Explain spontaneous electrical activity (pacemaker) in cardiac muscle.
3. Explain action potentials of ventricular cardiac muscle.
4. Explain the cardiac conduction system, pacemakers, and regulation of heart rate by the autonomic nervous system.
5. Explain the ECG and its correspondence to the cardiac action potential (AP).

## EXCITATION IN CARDIAC MUSCLE

The cardiovascular system transports blood containing oxygen, carbon dioxide, nutrients and wastes, between the environment and the cells of the body. It consists of a heart (pump) and blood vessels which deliver nutrients to the tissues (arteries) and ferry waste products away from the tissues (veins).

The heart is a muscular organ (Fig 1) which can contract in a rhythmic manner without direct stimulus from the nervous system. Each heart beat begins with the flow of ions across the plasma membrane of the cardiac muscle cell. This current is generated in specialized cells called **pacemaker cells**. The impulse from the pacemaker cells flows in a unidirectional manner through out the heart via specialized conducting tissue (Fig 1) and into the heart muscle. The electrical impulse results in mechanical contraction of the cardiac muscle through a series of intracellular events involving calcium.

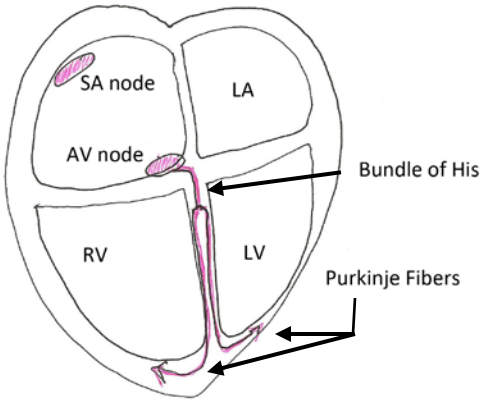


**Figure 1.** Electrical conduction within the heart starts at the sinoatrial (SA) node and passes sequentially to the atriaventricular (AV) node, Bundle of His, left and right bundle branches, and the Purkinje fibers. So the electrical activity moves from the base (A-V junction) to the apex (tip of ventricle) distant from the atria and then sweeps up the sides of the ventricles towards the base.

## PACEMAKER CARDIAC MUSCLE CELLS

**Pacemaker cells** have the unique property of being able to generate action potentials spontaneously (i.e. without input from the nervous system). They can generate an action potential because their resting membrane potential (- 60mV) is unstable. Because the membrane

potential never "rests" it is called a **pacemaker potential.** This potential exists because the pacemaker cells have unusual channels that are permeable to both Na + and K+. These channels are called $I_f$ channels. The "f" is derived from the fact that they were originally called "funny" channels because the $I_f$ channels are Na+ channels with unusual properties. When the $I_f$ channels opens, the **influx of Na+ exceeds the efflux of K** + and the **net influx of positive charges slowly depolarizes the cell.** As the membrane potential becomes more positive, the $I_f$ channels close and the Ca++ (L and T) channels open transiently, which further depolarize the cell. When the threshold potential is reached, a burst of Ca++ L channels open, more Ca++ rushes in, and a steep phase of depolarization occurs (Fig 2). At the peak of the action potential, K+ channels open, K+ rushes out of the cell and the cell repolarizes.

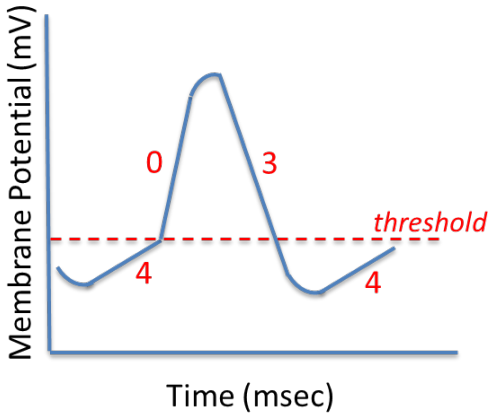


**Figure 2**. Slow action potential has 3 phases (0, 3 and 4).

The pacemaker cells set the **rate of the heart beat**. They are anatomically distinct from the contractile cells because they have no organized sarcomeres and therefore do not contribute to the contractile force of the heart. There are several different pacemakers in the heart but the sinoatrial node (SA) is the fastest. In normal hearts, the SA node is the pacemaker. The other conduction tissue (AV node, bundle of His and Purkinje fibers) will take over as pacemakers in disease states according to their speed of depolarization (AV > bundle of His > Purkinje fibers).

Heart rate (HR) can be modulated by autonomic nervous stimulation. Increased parasympathetic stimulation of muscarinic receptors on the heart slows the firing of the SA node. Parasympathetic stimulation does so by delaying the closing of K+ channels (efflux). The increased K+ efflux further hyperpolarizes the cells and slows the opening of the $I_f$ channels. In contrast, sympathetic stimulation speeds heart rate by shortening the time to threshold. Sympathetic stimulation increases Na influx via the $I_f$ channels and closes the K channels.

**Myocardial contractile cells** are tightly linked to one another by **intercalated disks**, specialized adhesive junctions, which ensure transmission of force from one myocardial cell to an adjacent cell. The cells also contain **gap junctions** that facilitate transmission of electrical impulses from cell to cell. Myocardial contractile cells have a resting membrane potential of approximately -85 millivolts (mV). Depolarization occurs when the permeability to sodium (PNa +) increases, and sodium flows into the cell (Phase 0, Fig 3). As the membrane potential reaches about +20 mV,

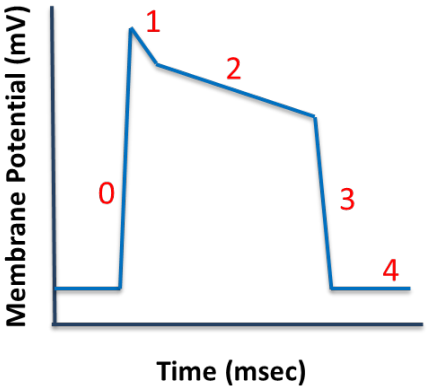


**Figure 3.** Fast action potential of cardiac contractile cell has four phases 0-4.

the voltage gated sodium channels inactivate. The muscle cell begins to repolarize as K+ leaves the cell through open voltage gated K+ channels (Phase 1). At this membrane potential, voltage gated Ca++ channels open causing the action potential to flatten as the $K^+$ efflux balances the Ca++ influx. The plateau (Phase 2) ends when Ca ++ channels close and $K^+$ efflux exceeds Ca++ influx. In Phase 3, K+ efflux repolarizes the muscle cell. The resting membrane potential is maintained by the activity of the Na-K ATPase (Phase 4).

## ELECTROCARDIOGRAM (ECG also known as EKG)

The electrical activity of the heart can be recorded by the electrocardiogram (ECG). Electrodes placed on the surface of the body can measure electrical activity of the heart because the fluid of the body is a good conductor.

An ECG recording is the sum of all of the electrical potentials generated by all the cells of the heart at any instance in time. Each deflection (wave) of the ECG represents either depolarization or repolarization of the specific parts of the heart. Because depolarization occurs before mechanical contraction, the waves of depolarization can be associated with contraction and relaxation of the atria and the ventricles.

A typical ECG recording and the waves are shown below (Fig 4). The **P wave** corresponds to depolarization of the atria. The **QRS complex** corresponds to depolarization of the ventricles. The **T wave** corresponds to repolarization of the ventricle. Why is repolarization of the atria not seen?

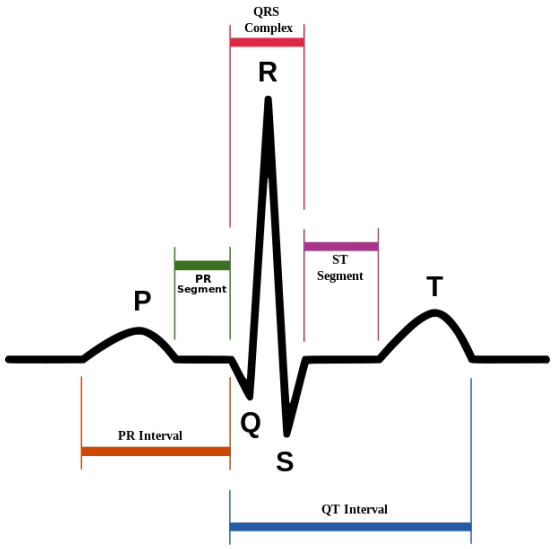


**Figure 4.** ECG corresponds to the contractile myocyte action potential. Phases 0 and 1 are the QRS complex. Phase 2 is the ST segment. Phase 3 is the T wave. [http://en.wikipedia.org/wiki/User:Agateller].

## ELECTRICAL ACTIVITY PRECEEDS CONTRACTION

In the heart, electrical activity (depolarization and repolarization) proceeds in a sequential manner. The initiation for a heart beat arises in the pacemaker cells of the sinoatrial (SA) node located in the right atria. From the SA node, the wave of depolarization moves through both atria (P wave), resulting in **atrial contraction**. The impulse then passes through the intranodal pathways connecting the SA node with the atrioventricular node (AV node). At the AV node the impulse slows allowing the atria to contract before the ventricles depolarize. The impulse then passes from the AV node through specialized conducing tissue known as the Bundle of His. The Bundle of His branches (left and right) within the septum that separates the ventricles and then into the Purkinje fiber system, which carry the impulse through the ventricular walls (QRS complex). This specialized conduction system ensures that the ventricles contract in a synchronized fashion and results in a contraction that begins at the apex (tip) of the heart. This is important because blood is ejected through the valves (pulmonic and aorta) that are located at the base of the heart (at the A-V junction).

**Heart rate and rhythm** can be obtained from the ECG. **Heart rate** is calculated from peak to peak of the QRS complex (i.e., from R wave to R wave). A normal resting heart rate is 60-100 beats per minute. Rates faster than this are called **tachycardia**; slower rates are **bradycardia**.

A normal heart **rhythm** occurs at regular intervals and includes a P wave, QRS complex, and T wave. An irregular rhythm is called an **arrhythmia**. Arrhythmias can occur if there is an extra beat, a missed beat, or a condition known as **fibrillation**, when the atria or ventricles are contracting in an uncoordinated fashion.

Problems within the electrical conductance system can change the ECG. For example:

1. No P waves are evident, if the SA node fails to fire.
2. P waves occur independently of the QRS complex, if there is a complete block of conductance through the AV node. In this condition, ventricular depolarization is driven by pacemakers within the ventricle (either the Bundle of His or the Purkinje fibers).

## KEY CONCEPTS

1. The heart is a muscular organ which can contract in a rhythmic manner without direct stimulus from the nervous system because of the activity of pacemaker cells. The action potentials of the pacemaker and contractile cells of the heart differ. The pacemaker cells have an unstable resting membrane potential. In the heart, the SA node is the fastest pacemaker cells and sets the rate of beating. Other pacemakers are found within the electrical conduction system and include the AV node, bundle of His and Purkinje fibers.

2. Heart rate is determined by the input from the parasympathetic (PNS) and sympathetic (SNS) nervous systems. PNS activity slows heart rate; SNS speeds heart rate. At rest the normal heart rate is 70-80 beats per min.

3. The ECG is the sum of the electrical activity of the entire heart. The P wave correlates to atrial depolarization. The QRS complex correlates to ventricular depolarization. The T wave is the repolarization of the ventricles. Rhythm is determined by the SA node.

4. Disease of the electrical conduction system in the heart is manifested by change(s) in the ECG.

## QUESTIONS

1. What portion of the ECG would change if there is a delay in the conduction between the SA node and the AV node?
2. Will the ECG change when heart rate increases?

## ANSWERS

1. PR interval increases.
2. Increased sympathetic drive to the heart increases heart rate. This will be cause a decrease in the R-R interval, Q-T interval, and P-R interval.