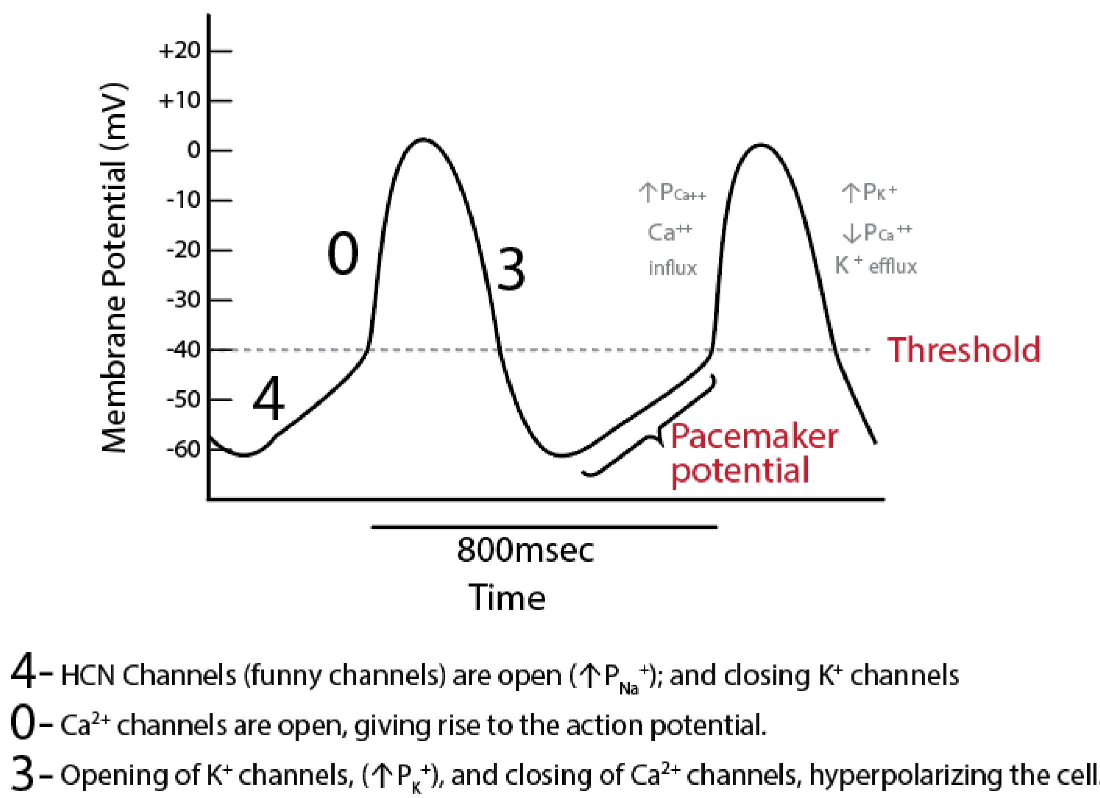
1.2.2

Action Potentials in Cardiac Autorhythmic Cells

One fascinating characteristic of the heart is that it has the ability to beat even if it is out of the body (the scene in *Indiana Jones: Temple of Doom* where the evil priest pulls out the heart and it is still beating is accurate, well mostly accurate). In fact, the heart will continue to beat until the cells die.

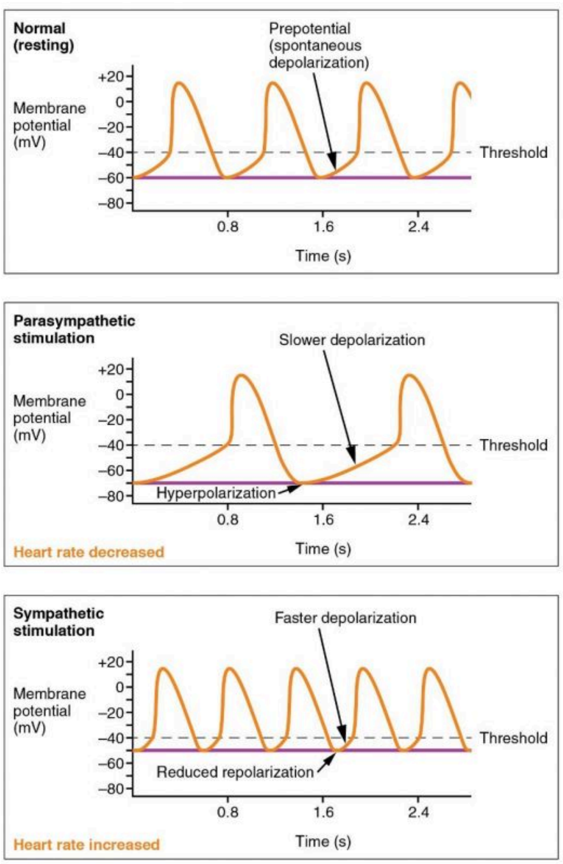
When you studied skeletal muscle, you learned that it is an action potential from a motor neuron that triggers muscle contraction. Cardiac muscle also needs an action potential to stimulate contraction but the heart has the capacity to generate its own action potentials without any neural stimulation. The cells that do this in the heart are special, modified cardiac muscle cells that we refer to as autorhythmic cells. The sinoatrial (SA) node is known as the pacemaker of the heart and is the primary location where the depolarization signal originates from to cause the heart to beat. In this section we will discuss how these cells are able to spontaneously initiate action potentials.



Action Potential in Cardiac Autorhythmic or Pacemaker Cells. The "P" next to any ion in this figure refers to "permeability". Therefore an ↑P means increased permeability

Drawn by BYU-Idaho student Fall 2013

This figure shows action potentials in the autorhythmic cells of the heart. Notice that there are only three phases in these action potentials, phase 4, phase 0, and phase 3. Also notice that there is no real resting phase in these cells. Once the membrane repolarizes it begins to slowly depolarize. These action potentials also lack phases 1 and 2 that were seen in the contractile cells. To explain how these action potentials are generated, we need to introduce an ion channel that we have not encountered previously in our studies. These channels open when the membrane *repolarizes* and close when the membrane *depolarizes.* When these channels were first discovered this behavior seemed odd or funny, therefore, the channels were called **"Funny" channels.** These channels are also known as **HCN channels,** HCN stands for "hyperpolarized activated – cyclic nucleotide-gated" channels because they open when the membrane hyperpolarizes and they can be regulated by the second messenger cyclic AMP, a cyclic nucleotide. We will discuss this regulation later. The funny channels allow Na^+ to slowly enter the cell, resulting in the gradual depolarization of the membrane (phase 4). This gradual depolarization is referred to as the **pacemaker potential.** The pacemaker potential causes the membrane potential to eventually reach threshold. In neurons, skeletal muscle and cardiac muscle, reaching threshold triggers the activation of voltage-gated sodium channels. The autorhythmic cells are different, reaching threshold triggers the activation of **voltage-gated *calcium channels*** and the subsequent influx of calcium results in the depolarization phase of the action potential (phase 0). At the peak of the action potential the calcium channels close and voltage-gated potassium channels open (same as seen in the other excitable tissues). The efflux of potassium results in the repolarization of the membrane (phase 3). Once the potential drops below threshold the potassium channels close, the funny channels re-open and the process begins again. If the heart rate changes, the time between each depolarization wave can decrease or increase depending on how many HCN channels are open and how far below threshold the electrical signal drops before depolarizing again.



Depolarization and change in heart rate.

Author: By OpenStax College [CC BY 3.0 (http://creativecommons.org/licenses/by/3.0)], via Wikimedia Commons, Link: https://commons.wikimedia.org/wiki/File%3A2033_Depolarization_in_Sinus_Rhythm.jpg