

## Heart Rhythm 101

## By Hans R. Larsen MSC ChE

The membrane (sarcolemma) of a resting heart cell (myocyte) is polarized – that is, the inside (intracellular space) of the cell (cytoplasm) is negatively charged in respect to the outside environment (extracellular space). Responding to an impulse from the sinoatrial (SA) node (the heart's natural pacemaker controlled by the autonomic nervous system) the myocytes depolarize resulting in contraction of the heart muscle. The *depolarization* is caused by a rapid influx of positive sodium (Na<sup>+</sup>) ions followed by a slower influx of calcium ions (Ca<sup>++</sup>). During depolarization the outward leakage of potassium ions (K<sup>+</sup>) is restricted. Atrial depolarization shows up as a P wave on an electrocardiogram (ECG) while ventricular depolarization is identified as the QRS complex – that is, the time period on the ECG during which the ventricles depolarize (contract). The P wave is absent during atrial fibrillation.

Depolarization is followed by *repolarization* (recovery). During this phase, an outflow of K<sup>+</sup> ions is followed by a period during which the intracellular concentrations of K<sup>+</sup> and Na<sup>+</sup> in the myocytes are restored to their resting potential through the action of Na<sup>+</sup>/K<sup>+</sup> ATPase pumps "powered" by magnesium. Magnesium ions (Mg<sup>++</sup>) also play an important role during this phase by slowing down the outward (from intracellular space to extracellular space) flow of potassium ions. At the risk of oversimplification, one could say that while Na<sup>+</sup> and Ca<sup>++</sup> are "excitatory" ions K<sup>+</sup> and Mg<sup>++</sup> ions are "calming". Thus it is not surprising that a deficiency of K<sup>+</sup> and Mg<sup>++</sup> facilitate atrial fibrillation. Repolarization is identified on the ECG as the time period from the end of ventricular depolarization to the peak of the T wave (ST segment).

The *atrioventricular (AV) node* is a specialized conglomeration of myocytes that acts as the speed controller for ventricular contractions (depolarization) just as the SA node does for atrial contractions. Normally, the AV node receives its "instructions" directly from the SA node through a well-defined "wiring circuit"; however, during atrial fibrillation the AV node is bombarded by impulses from rogue atrial cells which, if they are not filtered out by the AV node will cause the rapid, irregular ventricular contractions characteristic of atrial fibrillation.

The period from the start of the QRS complex to the peak of the T wave is of particular interest when it comes to atrial fibrillation. During this period (the *effective refractory period or ERP*) myocyte depolarization can not be triggered by stimulus originating from rogue atrial cells thus preventing afib from being initiated. However, atrial fibrillation can be triggered during the last half of the T wave (*relative refractory period or RRP*) making it highly desirable that the ERP is as long as possible and the RRP as short as possible. Several medications aim to exploit this fact by acting to extend the ERP so that the RRP (the vulnerable period) becomes as short as possible. This is particularly important in the case of the AV node as during the ERP the node can not be stimulated and thus in essence filters out the erratic atrial impulses.

The speed with which an electrical impulse moves across the atrium (normally from the SA node to the AV node) is called the *conduction velocity* and is a measure of the effectiveness of cell to cell depolarization. It is measured in millimeter/millisecond (mm/ms) or in meter/second (m/s). Sympathetic (adrenergic) stimulation increases conduction velocity while parasympathetic (vagal) stimulation reduces it. Slow conduction is associated with the presence of *complex fractionated atrial electrograms* (CFAEs) defined as electrograms (direct measurements of electrical activity) in the atrium with a cycle length less than or equal to 120 ms or shorter than in the coronary sinus or that are fractionated or display continuous electrical activity. CFAEs are believed to be associated with fibrosis and serve as targets in some ablation procedures for atrial fibrillation.

## Electrocardiogram (ECG) of Normal Heartbeat



P-wave: Atrial depolarization (contraction)

QRS Complex: Ventricular depolarization (contraction)

T-wave: Ventricular repolarization (recovery)

ST Segment: From end of QRS complex to peak of T-wave (Repolarization)

ERP: From beginning of QRS complex to peak of T-wave

RRP: Last half of T-wave

The AFIB Report is published 10 times a year by Hans R. Larsen MSc ChE 1320 Point Street, Victoria, BC, Canada V8S 1A5 Phone: (250) 384-2524 E-mail: editor@afibbers.org URL: http://www.afibbers.org ISSN 1203-1933.....Copyright © 2001-2010 by Hans R. Larsen

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