Review Article

ISSN 2639-8486

Cardiology & Vascular Research

Dimensions and Velocities in the Heart

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Received: 15 Dec 2022; **Accepted:** 05 Jan 2023; **Published:** 09 Jan 2023

Citation: Alejandro S Robalino. Dimensions and Velocities in the Heart. Cardiol Vasc Res. 2023; 7(1): 1-3.

Keywords
Heart, Cardiac Electrophysiology, Chronotropism, Excitability, Dromotropism.

The heart is a muscular organ whose function is to expel blood and oxygenate all the tissues of the economy. It is a marvel of bio-engineering that to perform such a function has as its basis an anatomical structure (valves, conduction pathways, blood vessels) made specifically so that vital functions that maintain life adapt to it or that – in pathological circumstances – adapt to these extreme circumstances in order to fulfill their mission. Therefore, this brief review will treat normal measurements according to the different means of diagnosis used to evaluate cardiac function. As we will see, mathematics and physics are involved in this whole process.

Cardiac Electrophysiology

There are four electrophysiological properties of the heart: chronotropism or automatism, badmotropism or excitability, dromotropism or conductivity and finally inotropism or contractibility.

Chronotropism is the heart's ability to generate impulses that travel through special pathways to activate different cardiac tissues. To transmit an optimal impulse the cardiac cell must have a resting potential as negative as possible this is -80 or – 90 Mv. However to generate momentum they must possess a pre-potential which is a diastole depolarization capable of reaching a threshold potential of – 60 Mv to produce a response. This phase is related to the opening of channels for calcium. The cells that have the most inclined pre-potential phase are those of the sinus node.

Excitability is the ability of the cardiac fiber to respond to a stimulus. This will depend on the level at which the potential threshold is located. Therefore, as the threshold increases, the excitability and life decreases. It is for this reason that in the supernormal phase that correlates with the U wave of the electrocardiogram (EKG), and that lasts 25 msec and in which the threshold potential is 10 to 15% lower than normal, repetitive responses can occur. Therefore, while the intensity of the stimulus that will create a response is lower, the excitability will be greater.

Dromotropism or conductivity is the greater or lesser ease with which a stimulus is conducted. Therefore, by measuring the conduction time in the various structures of the heart we can establish the diagnosis of pathologies. Among the properties on which this conduction speed depends – perhaps the most important – is the level of the transmembrane resting potential at the time of producing a stimulus or at the time, a cell receives this stimulus. The more negative this potential is, the stimuli produced will be the wider and faster. On the contrary, when this potential is less than -80 Mv the response will be decreased and there will be alterations in conduction (blockages).

The transmembrane resting potential depends on the interior of the cell becoming negative by losing K+ ions. During resting, the membrane is more permeable to potassium allowing this ion to accumulate again inside the cell to – in the next action potential – leave the cell again in the repolarization phase. Other ions can go through the membrane, for example Na+, but the permeability of the membrane for this ion is much lower than it has for K+, therefore the concentration of intracellular Na is low, about 10 mM. (The system,c-v). The Na pump removes 3 ions of NA to the outside of the cell while introducing 2 of potassium and the energy source for its operation is ATP [1].

A clear example of this in clinical practice is when you have very early impulses or when the duration of the action potential is very prolonged. In the first case, the impulse will not be driven by falling into the absolute refractory period, and in the second, it may be conducted slowly and aberrantly when falling into the relative

refractory period. The problem will be solved by increasing the distance between the impulses or by shortening the duration of the action potential. This would be one of the causes of long QT arrhythmias in which there is alteration in sodium and potassium currents. The average duration of an action potential considering the supernormal phase is about 325 msec.

Concept of Absolute Refractory Period

According to J.Soler (EFGDLA) a cell cannot create a new action potential until the repolarization reaches a minimum of 50 Mv. This corresponds to phase 3. Therefore a stimulus that reaches the cell with its action potential in phases 1 or 2, will not be able to propagate and this is called absolute refractory period that lasts approximately 275 msec.

Concept of Relative Refractory Period

A suprathreshold stimulus that falls into phase 3 of the action potential (repolarization) may be driven by falling into the relative refractory period between 275 and 300 msec. of action potential [2].

Causes of Reduced Resting Potential

The reduction of the level of transmembrane resting potential can occur in states of anoxia, or hypoxemia (ischemia), clinical states with acid Ph (diabetes, chronic renal failure), or in digitalis poisoning.

Ischemia can create spontaneous diastolic depolarization by modifying the slope of phase 4 in several pacemaker cells, reaching the threshold potential more easily and producing depolarization. Another alteration that can generate ischemia is phenomena of reentry. In which there are areas with depressed excitability and conduction. In this situation an impulse when going through an area with these conditions does so slowly and when leaving it finds the neighboring tissue already recovered electrically and in conditions to be reexcited and may produce a repetitive response [3].

Conduction velocities in cardiac structures

Atria: Lewis showed that the speed of impulse propagation in the atrial wall is 100 cm/second.
Node A-V: has a very slow driving speed between 20 to 200 mm / second.
Beam of His: in its branches the driving speed is 2,500 mm / second.
Purkinje net: 3,000 mm/second.

Inotropism or contractility is the ability of the cardiac muscle to exert contractile force in order to perfuse the tissues of the economy with blood and oxygen. When the stimulus reaches the Purkinje, there is a first activation of the left purkinje 5 milliseconds before the right. This asynchronism is normal.
Contraction is the end result of cardiomyocyte excitation, and calcium is the ion with a very important role in the relaxation-contraction mechanism.

There is therefore a displacement of cytoplasmic Ca towards the sarcoplasmic reticulum during ventricular relaxation through the action of the CaATPase of the reticulum and this matches with the low calcium levels observed in the diastole [4].

Electrocardiography

It is the external record of electrical potentials originating in the heart. He studies cellular electrophysiology and the anatomy and physiology of the heart's conduction system.

The cardiac impulse is generated in the sinus node, which by having the largest number of cells that have diastolic depolarization phase (pre-potential), assumes the function of being the normal pacemaker of the heart. The range of impulse generation goes from 60 to 90 per minute and below or above these figures we have pathologies such as bradyarrhythmias or tachyarrhythmias.

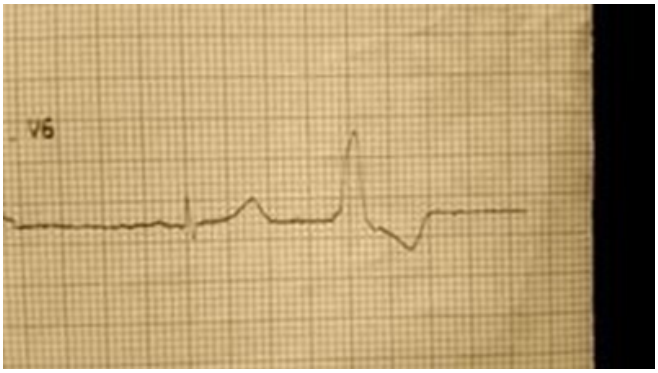
Once the impulse is created, it propagates radiately through the atria at a speed of 100 cm / second giving way to the P wave of the electrocardiogram (EKG) that lasts 0.12 "and is better appreciated in the D2 lead. This activation produces a resulting vector that in the frontal plane is located at +60º. Then the impulse passes and goes through the AV node, which is a structure with a slow conduction speed. In the EKG this conduction through the node is plotted in P-R space with a duration of 0.12 to 0.20". When the AV node generates impulses – when the sinus node suffers some pathology – these oscillate between 40 and 59 per minute.

Taking into account the pathological duration of the P-R space, we have pre-excitation syndromes when P-R is below 0.12" and 1st degree AV blocks when it exceeds 0.20". If we relate this peripheral P-R space with the electrogram of His we have 2 intervals: the P-A with an approximate duration of 27 msec and representing the time between the beginning of the P wave of the peripheral EKG and the first deflection of the atrial electrogram (A), and the A-H with a duration of 92 msec and which represents the time between the atrial deflection A and the rapid deflection H of the beam of His.

Once the AV splice is activated, the impulse goes through to the His, its branches, to the Purkinje and finally to the ventricular muscle. This is represented in the QRS deflection of the EKG with a normal duration between 0.08 and 0.09" and in the electrogram of His by the interval H-V with a duration of 43 msec and representing conduction in the beam of Hiz and the Purkinje tissue (depolarization). Then the activation proceeds through the free walls of the ventricles until finally activating the basal portions of the heart, completing the QRS enrollment of peripheral KG [5]. Increased abnormal durations of the QRS complex are seen in branch blockages and ventricular extrasystoles.

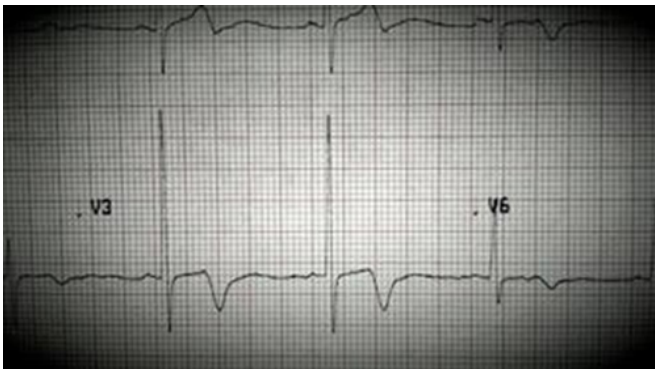
Extrasystoles have a QRS similar to complete branch blockages with a complex duration exceeding 0.12" and with secondary changes in the T wave as seen in the FIGURE. Which corresponds to an isolated ventricular extrasystole. These extrasystoles are due to post-depolarizations which are oscillations in the membrane potential during repolarization [1].

Figure taken from the author's file



Then will come the repolarization or phase 3 of the action potential that corresponds to the T wave of the EKG. When there are alterations in the polarity of this T wave we can make the diagnosis of myocardial ischemia as in the figure No. Which corresponds to a subepicardial ischemia of the septal region.

Figure taken from the author's file



The action potentials of Purkinje cells are very different from those of automatic cells (sinus node, AV node) and that is due to their different functions. The conduction speed in the Purkinje is high about 3000 mm / second and to achieve that they have a very vertical phase 0 and a very negative resting transmembrane potential (-90 Mv). They do not have pre-potential because their function is not to originate impulses (automatism), but to lead them in the most effective and fast way.

Drugs such as amiodarone, propranolol and procainamide can prolong the duration of action potential correlating with the QT interval of peripheral EKG. Non-pharmacological causes of QT prolongation are hypocalcemia and hypokalemia [3].

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