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Cardiac physiology for biomedical engineering

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Abstract*— *The multidisciplinary team provides research optimization and complementarity of ideas. Engineers working in biomedical engineering can make your work routine easier by sharing them with healthcare professionals who can help you understand physiology. The main objective is to address key points in cardiac physiology and arrhythmia pathophysiology in order to lessen the efforts of biomedical engineering professionals implementing implantable heart rate sensitive devices. It is a descriptive and critical theoretical analysis, which selected the main and current bibliographical references, directed to the fulfillment of the main objective. As a result, we obtained that the most important points are about the electrocardiogram graph and its association with cardiac bioimpedance as a good physiological parameter, also about the respiratory system that can influence cardiac health and the pathophysiology of arrhythmias that help to understand the need. to deploy. Pacemakers. We can conclude that early identification of the gradual ST segment distance may prevent future cardiac arrest and, in frequency-sensitive pacemakers, it is possible to identify from bioimpedance. There is a challenge in implantable devices in differentiating between normal cardiac changes and arrhythmias, and with the ability of biomedical engineering, the possibility of improving the performance of these devices is always real.

Keywords*— *Action potential, bioimpedance, cardiovascular diseases, electrocardiogram, implantable devices.

I. INTRODUCTION

When proposing biomedical devices that influence the functioning of any organ, it is necessary to have a minimum knowledge about its structure and behavior, so that it is possible to identify and differentiate normal physiological changes from pathological changes.

Working in the field of biomedical engineering, there is a need for both physiological and technological knowledge, and therefore the multidisciplinary of the team is justified.

This study addresses the key points selected by a healthcare professional about heart anatomy, physiology, biophysics, and pathophysiology in a simplified manner to facilitate understanding and work of engineering professionals implementing implantable heart devices, particularly those that are frequency sensitive pacemakers.

II. METHODS

To answer the proposed research problem: What does a biomedical engineer need to know about heart functioning in order to implement a system that helps with frequency-sensitive pacemaker? A critical review of the literature was produced, where analyzes and synthesis of the available content in the main scientific publications on the studied subject were performed.

It was used as inclusion criterion of publications, current scientific texts (2015 to 2019) that contain information that help in solving the research problem, being excluded texts without scientific character, from years before 2015 and without relevance to the theme.

III. RESULTS AND DISCUSSION

The heart is the main organ of the circulatory system and has a responsibility to provide nutrients and oxygen to each tissue through the blood. It is divided into four cardiac chambers, where the upper part is composed of right and left atria and the lower part, right and left ventricles (Fig. 1) [1].

Typically, the implantable device electrodes are placed in the right-side chambers. These chambers are separated by fibrous tissue, considered a bad current conductor, forming walls around the atrioventricular (AV) valve openings and preventing the electrical current generated by

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the organ itself from passing rapidly and disorderly through the cardiac tissue [2].

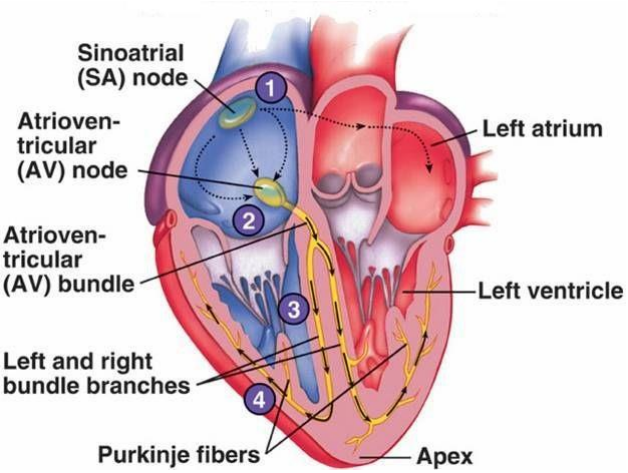

Fig. 1: Anatomy of heart [3]

The left ventricular wall is thicker because it has the function of ejecting the blood for a large circulation and, therefore, the related electrocardiogram (ECG) signal may have a larger wavelength than the right.

Cardiac behavior from the beginning of one heartbeat to the beginning of the next is called the heart cycle [4], and from it, it is possible to obtain several parameters that, if interpreted correctly, reveal the possible failures in your homeostasis.

The generation of this beat occurs from the electrical current released by the sinoatrial node or sinus node (SA), located above the right atrium, at the entrance of the superior vena cava (Fig. 1). The atrioventricular (AV) node slows the pulse from the upper to the front of the ventricles and the AV beam conducts the current to the lower syncytium, while Purkinje fibers distribute energy equally throughout the heart muscle tissue [5].

Some cardiac fibers are capable of self-excitation, which facilitates the generation of rhythmic automatic discharge with consequent contractions at the same rate. Hence the ease of a single point of current generation (SA node) dictating the rhythm of the entire organ [6].

If there is AV block, for example, the pulse does not pass from the atrial syncytium to the ventricular. The atria remain contracting with normal frequency and the ectopic pacemaker usually developed in the Purkinje fibers leads to ventricular muscle frequencies between 15 and 40 beats per minute (bpm). With this sudden blockage, the Purkinje system takes about 5 to 20 s to react. During this time, the ventricles become bloodless and after 4-5 s due to lack of blood flow to the brain region, fainting and, in more severe cases, death may occur [1].

This property, when decompensated, can generate ectopic excitation points in various parts of the tissue and this is one of the factors that leads to the arrhythmia process. In addition, there is a possibility of failure in the production of electrical current by the SA node, triggering the lack of chronotropism (stable heart rate).

If it occurs again, one of the predisposes is the need for artificial pacemaker implantation. However, there are other possibilities, one being partial or total AV node block, His bundles or Purkinje fibers.

The action potential (AP) recorded in the ventricle is about 105 millivolts (mV). Thus, the intracellular potential is approximately -85mV at rest and reaches about + 20mV during each beat. After BP onset, a membrane remains in depolarization (with electropositive intracellular medium) for 0.2 s, which is recorded as a plateau after repolarization, which is the moment of return of the intracellular medium to the negative pole [6].

Action potential in the atriums is -55 to -60 mV, while in the ventricles -85 to -90 mV. This is because sinoatrial fibers have a better permeability to sodium (Na^+) and calcium (Ca^{2+}) ions and their positive charges counteract some intracellular negativity. As this node is located in the atrium, electronegativity is lower in this region [7].

Potassium ions (K^+) exert a strong influence on membrane potentials and Ca^{2+} act on activation of the muscle contraction mechanism. Combining both information, it can be said that the concentration of each in the extracellular medium is essential for the cardiac pumping process [8].

In excess, K^+ may cause myocardial dilation with consequent sagging, reducing the frequency of beats. It can also block the conduction of the atrial current towards the ventricles, and if this increase is two to three times beyond normal concentration, it is possible that beat weakness is fatal [9].

Excess Ca^{2+} has the opposite action because it induces the myocardium to produce contraction in the form of spasms. Speaking of disability, it precedes a fall similar to lack of K^+, however, the body can maintain its levels more easily without major clinical concerns [8].

Figure 2 shows the cardiac cycle events that were extracted based on the left side of the heart. The curve "a" represents the pressure in the aorta. It can be noted that the aortic valve opens at the time of systole and with its pressure is increased and soon after its closure, the diastole is started and the pressure decreases.

This parameter is not directly perceived by implantable devices, but knowing that this opening depends on the ventricular force, if pacing is not sufficient for the heart's blood ejection process, the ideal pressure will not be reached and the ideal blood volume will not come out of the ventricle.

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Curve "b" represents atrial pressure. It is noted that the amplitude is much smaller than that of the ventricle, because its structure is smaller and its wall less thick [5]. Curve "c" represents ventricular pressure, approaching 120 mmHg for aortic valve opening and blood ejection for large circulation [10]. If current flow to the ventricle is impaired, it will lose its ability to contract, which causes the chamber to empty. In the figure 2, ventricular volume is represented on curve "d".

The electrocardiogram (curve e) is composed of P wave, QRS complex, T wave and eventually U; which are electrical voltages generated in the heart and recorded by the equipment on the body surface. The P wave represents the spread of atrial depolarization. After approximately 0.15s, there is the QRS complex, representing ventricular depolarization, and then the T wave representing ventricular repolarization. The U wave, when identified, corresponds to the repolarization of the papillary muscles [11].

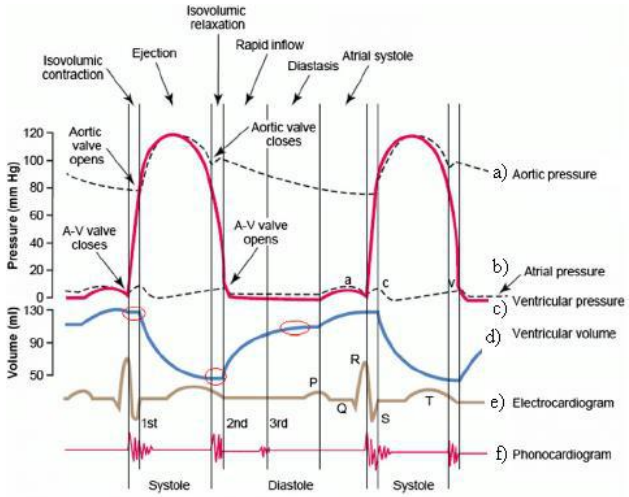

Fig. 2: In a) Graph of aortic pressure during the left cardiac cycle, b) Left atrial pressure in systole and diastole, c) Left ventricular pressure in systole and diastole, d) Ventricular volume in the period of relaxation and contraction, in e) Signal electrocardiogram with the waves P, QRS and T, and in f) the left cycle phonocardiogram [1]

There is a delay of 0.1 s in the passage of this impulse from the atriums to the ventricles, allowing atrial systole, which brings blood to the ventricles and then ventricular systole, to take blood to the organs. This delay is essential so that systole does not occur at the same time in all chambers [2].

Electrocardiogram graph is indispensable when it comes to heart health research and, in frequency-sensitive pacemakers, its parameters can be used. The bioimpedance signal, for example, is a condition that can be read by this type of device as an ideal or pathological indicator of functioning, mainly because it behaves synchronously with the ST segment.

ST segment provides information on the delay time between systole and ventricular diastole and the progressive distance between it is indicative of future cardiac arrest [12]. Implantable devices do not read the ECG directly; However, by perceiving the cardiac bioimpedance signal, this parameter can be extracted and used as a pathological alert.

In addition, there are other associated physiological conditions, such as heart rate (HR), number of beats in one minute, and cardiac output (CO), which correspond to the volume of blood that passes through the heart in one minute [2].

At rest, a healthy individual can pump 4-6 liters of blood per minute and, in intense physical activity, has the ability to increase this value up to seven times. This volume control is based on the intrinsic cardiac regulation of the variation in the amount of fluid reaching the heart and the control of the heart rate, as well as its pumping force, which are the responsibility of the Autonomic Nervous System (ANS) [1].

The sympathetic and parasympathetic (or vagus) nerves innervate myocardial tissue and are part of the pumping effectiveness. Sympathetic stimulation can increase cardiac output by 100%, beats usually 70 per minute can reach 250 and increase systolic strength by up to two times in cases of physical activity or stressful situations such as fear.

The parasympathetic can slow down and even stop beating for a few seconds, and then tap again 20 to 40 times per minute while vagal stimulation is stimulated, for example, by meditation. It is important to report that, as parasympathetic fibers are more concentrated in the atria, their stimulation is able to reduce HR, but not considerably the contraction force [11], considering that variations in cardiac output (CO) resulting from nerve stimulation imply changes in HR and contraction force [2].

When a frequency-sensitive pacemaker has the ability to extract ST segment information associated with CO and HR, it increases its reliability of accuracy when acting, considering the property of interpreting physiological changes by disease process or physical activity, as an example.

Intense physical stress requires more circulatory system activity due to the need to increase blood flow to the muscles currently being exercised. Non-athletes may have an increase in cardiac output up to five times above normal, whereas in a top athlete it reaches up to seven times [2].

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In order to perform this special nourishment of the active tissues, the heart is stimulated by the sympathetic nervous system to considerably increase its frequency and pumping force, and in turn inhibits the parasympathetic nervous system [3].

In addition, most peripheral circulation arterioles are very contracted except active muscle arterioles, as they are very vasodilated. This is exactly why the heart needs to respond by sending more of its supply through pumping blood, and the body can still redirect fluid from where it is most needed at the moment. Interestingly enough, too, is the considerable increase in systemic filling pressure that occurs because the vein walls contract with a very powerful force [2].

Sympathetic stimulation is important for the consequent increase in blood pressure, resulting in vasoconstriction of arterioles in the body. For active muscles, there is increased frequency of beats and increased blood ejection pressure in the body [12].

When an individual comes out of rest, the oxygen-rich blood supply must be increased to nourish the muscles used. The more circulation needed, the greater the cardiac output (how much blood circulates in the heart per minute) and pulmonary ventilation [1]. Heart rate changes isolated from ventilatory behavior indicate pathological process that should be researched.

Cardiac contraction force can be increased by elevating the temperature by physical activity. However, in states where the condition continues, such as fever, HR can bend and deplete metabolic systems, causing weakness. If hypothermia occurs, consequently there is a reduction in this frequency, which can lead to death [12].

Outside of physical activity, there is still the possibility of the heart manifesting inappropriate behavior. When such events occur frequently and are correctly diagnosed, the cardiologist may consider it necessary to implant an artificial pacemaker to assist in the process of induced activity regularization and, consequently, to increase life expectancy.

Cardiovascular disease is the leading cause of death from disease worldwide, with an estimated 17.7 million deaths in 2015 (31% worldwide) and 17.9 million in 2016 (44% worldwide). In addition, more than 75% of these occurred in low-income and middle-income countries [13].

The main clinical features of arrhythmias required for pacemaker implantation are: hypersensitive carotid syndrome, neurocardiogenic syncope, SA node disease, non-drug-responsive bradycardic syndrome, ventricular control atrial fibrillation, AV-grade AV block, and second-degree type. II AV, advanced AV block, fiber bundle injury, alternative bundle branch block and other related consequences [3].

The general pathophysiology of arrhythmias is divided into three, namely: hyperautomaticity; triggered activity; and reentry process.

Hyperperaeromaticity is a process represented by the activation and acceleration of cardiac cells that results in spontaneous depolarizations. This hyperactivity can be derived from nervous system dysfunction or even fever, shock, acidosis and endocrinopathies [3]. In the triggered activity, the changes are generated from the variation of the membrane potential, which generates new action potentials and, consequently, arrhythmias. This may be due to decreased ion efflux in the cell or even increased positive ion influx. The reentry process is the main reason for ventricular and supraventricular arrhythmias. Occurs by the presence of an obstacle in the anatomical structures through which the electric current is conducted [2].

It is possible, by observing the action potential wavelength (Fig. 3), to identify if there are pathological changes in the current conduction. The formula consists of: Wavelength = Conduction Speed x Refractory Period. Thus, when the wavelength exceeds the anatomical size of the circuit, the arrhythmia is extinguished. And if the wavelength is shorter than the arrhythmic circuit, the arrhythmia will be maintained [6].

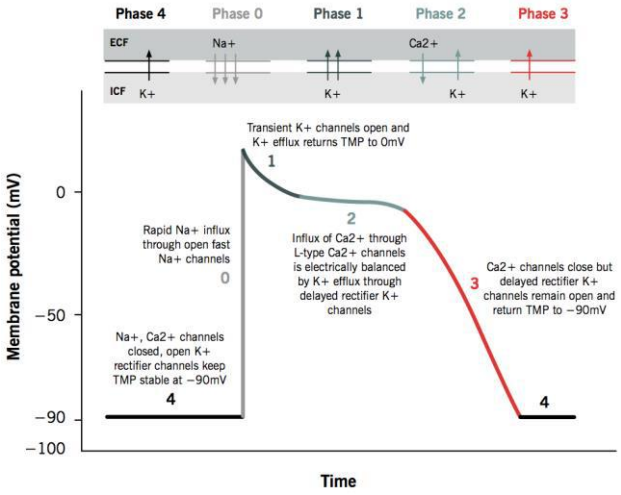

Fig. 3: Heart action potential

With this graph it is possible to visualize the moments of depolarization (0), rapid and incomplete repolarization (1), plateau moment (2), depolarization (3) and electrical diastole (4), thus completing a cardiac cycle.

IV. CONCLUSION

It is a fact that there is much more complex information, from the anatomy to the pathophysiology of arrhythmias, however, with the information present in this text, after a practical observation of the difficulties

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encountered by biomedical engineers in the development of this type of project, it is believed to be sufficient to facilitate understanding of heart function and propose technologies that can solve pathological problems.

One of the main points that deserves due attention is the early identification of ST segment detachment on the electrocardiogram, as it may prevent future cardiac arrest. When it comes to frequency-sensitive pacemakers, this behavior can be identified by reading the bioimpedance.

Getting the implantable device to identify when cardiac change occurs due to normal day conditions or pathological disorders is always a challenge. However, with the ability of biomedical engineering, the possibility of improving the performance of these devices is always real.

REFERENCES

[1] Guyton, A.C. e Hall, J.E. (2017). Tratado de fisiologia médica, 13rd ed. Rio de Janeiro: Elsevier, 1168p.
[2] Berne, R.M e Levy, M.N. (2018). FIsiologia. 7th ed, vol. 7. Rio de Janeiro: Elsevier, 880p.
[3] Mc Graw-Hill. Available in: https://br.pinterest.com/. Accessed in October 11, 2019.
[4] Vanputte, C.L., Regan, J.L. e Russo, A.F. (2016). Anatomia e fisiologia de seeley.10th ed. Porto Alegre: Artmed, 1264p.
[5] Boron, W.F. e Boulpaep, E.L. (2017). Medical physiology. 3st ed. Philadelphia: Elsevier, 1312p.
[6] Duran, J.E.R. (2015). Biofísica conceitos e aplicações.2nd ed. Pearson, 320p.
[7] Gandon-Renard, M., Bedioune, I., Karam, S., Varin, A., Lechène, P., Bichali, S., Leroy, J., Algalarrondo, V., Stratakis, C., Mercadier, J.J., Benitah, J.P., Gomez, A.M., Fischmeinster, R. e Vandecasteele, G. (2019). The cAMP-dependent protein kinase type, I regulates cardiac excitation-contration coupling. Archives of Cardiovascular Diseases Supplements (ISSN 1878-6480), 11(2), 261-266. https://www.sciencedirect.com/journal/archives-of-cardiovascular-diseases-supplements
[8] Catterall, W, Lenaeus, J, El-Din, T.M. (2019). Structure and pharmacology of voltage-gated sodium and calcium channels. Annual Review of Pharmacology and Toxicology (ISSN 1545-4304). https://doi.org/10.1146/annuarey-pharmtox-010818-021757.
[9] Guerri, G., Krasi, G., Precone, V., Paolacci, S., Chiurazzi, P., Arrigoni, L., Cortese, B., Dautaj, A., Bertelli, M. (2019). Cardiac conduction defects. Acta Biomed (30(10), 20-29). DOI: 10.23750/abm.v90i10-S.8751
[10] Silverthorn, D.U. (2017). Fisiologia humana: uma abordagem integrada. 7th ed. Porto Alegre: Artmed, 960p.
[11] Santos, J.L.F. (2016). Eletrocardiograma ao alcance de todos. 3rd ed. São Paulo: Phorte, 176p.
[12] Timperley, J., Leeson, P., Mirchell, A. R. J., Betts, T. (2019). Cardiology pacemakers and ICDs. 2nd ed. Oxford Medical Publications, 7109p.
[13] OPAS. Doenças cardiovasculares. 2017. Disponível em: https://www.paho.org/bra/index.php?option=com_content&view=article&id=5253:doencas-cardiovasculares&Itemid=1096. Accessed in October 11, 2019.
[14] Ikonnikov, G., Yelle, D. (2013). Physiology of cardiac contraction and contractility. Available at: http:// www.pathophys.org/physiology-of-cardiac-conduction-and-contractility/#Electrophysiology. Accessed in October 11, 2019.