e-ISSN: 2988-3237

Sriwijaya Journal of Internal Medicine (SJIM)

Journal website: https://phlox.or.id/index.php/sjim

Overview of the Cardiac Conduction System: A Narrative Literature Review

Rachmat Hidayat[1*]

[1]Department of Biology, Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia

ARTICLE INFO

Keywords:
Conduction system
Electricity
Cardiac

***Corresponding author:**
Rachmat Hidayat

E-mail address:
rachmathidayat@fk.unsri.ac.id

The author has reviewed and approved the final version of the manuscript.

https://doi.org/10.59345/sjim.v1i1.18

A B S T R A C T

The conduction system of the cardiac initiates and coordinates the electrical signals that cause rhythmic and synchronous contraction of the atria and ventricles. This literature review aims to describe the electrophysiology of the cardiac conduction system. Electrical impulses usually arise in the sinoatrial (SA) node. The SA node is innervated by sympathetic and parasympathetic nerves. Each action potential moves rapidly from cell to cell and through the myocardium of the atria, onto the atrioventricular node (AV node), and causes both atria to contract and begin systole. The AV node, located on the right atrial wall superior to the tricuspid valve and anterior to the coronary sinus ostium, conducts action potentials into the ventricles. From the AV node, the conducting fibers converge to form the bundle of His (atrioventricular bundle) within the posterior border of the interventricular septum.

1. Introduction

Electrical impulses usually arise in the sinoatrial (SA) node. The SA node is located at the junction of the right atrium and superior vena cava, just above the tricuspid valve. The SA node lies only about 1 mm below the visceral pericardium, making it susceptible to injury and disease, especially pericardial inflammation. The SA node is fed by the sinus node artery, which passes through the center of the node. The SA node is innervated by sympathetic and parasympathetic nerves. In resting adults, the SA node generates about 60 to 100 action potentials per minute, depending on age and physical condition. Each action potential moves rapidly from cell to cell and through the myocardium of the atria, onto the atrioventricular node (AV node), and causes both atria to contract and begin systole. The AV node, located on the right atrial wall, superior to the tricuspid valve and anterior to the coronary sinus ostium, conducts action potentials into the ventricles. It is innervated by nerves from the autonomic parasympathetic ganglia, which serve as receptors for the vagus nerve and cause the slowing of the conduction of impulses through the AV node.[1-3]

From the AV node, the conducting fibers converge to form the bundle of His (atrioventricular bundle) within the posterior border of the interventricular septum. File His later bring up right and left bundle branch. The right bundle branch (RBB) is thin and runs without much branching to the apex of the right ventricle. The thinness and relative lack of RBB branches make them susceptible to impaired impulse conduction by damage to the endocardium. The left bundle branch (LBB) in some cardiac divides into two branches or fascicles. The left anterior bundle branch (LABB) passes to the left anterior to the papillary muscle and floor of the left ventricle and crosses the

aortic outflow tract. LABB is susceptible to damage to the aortic or left ventricular valves. The left posterior bundle branch (LPBB) travels posteriorly and traverses the left ventricular entry duct to the base of the left posterior papillary muscle. These branches spread diffusely through the inferior posterior wall of the left ventricle. Because of the relatively non-turbulent flow of blood through this part of the left ventricle, the LBB is somewhat protected from wear-and-tear injury.

The terminal branches of the RBB and LBB are Purkinje fibers. They extend from the ventricular apex to the fibrous ring and penetrate the cardiac wall to the outer myocardium. Part of the ventricular interventricular septum is the first area to be excited. Both RBB and LBB activate the septum. The rapid spread of impulses to the ventricular apex is achieved by the extensive network of Purkinje fibers. Last to be activated are the basal and posterior parts of the ventricles.[4-6]

Cardiac excitation

From the SA node, the impulses that initiate systole spread throughout the right atrium at a conduction velocity of about 35 cm/s. Because impulses from the SA node arrive at the AV node very quickly, researchers have proposed that these nodes are connected by internodal pathways, called the anterior, middle, and posterior internodal pathways. However, the existence of this pathway is controversial; not all experts agree that they exist.

Action potentials are delayed in the region of the AV node, possibly due to electrophysiological differences in the cells that make up the region of the AV. The velocity of conduction within the nodes is about 10 cm/sec, much slower than conduction through the atria. The delay between atrial and ventricular excitation allows an additional boost to ventricular filling through atrial contraction (atrial kick). From the AV node, the impulse travels from the AV bundle and via bundle branches to the Purkinje fibers. The velocity of conduction in the AV and Purkinje fibers is the fastest in the cardiac.

Ventricular activation occurs sequentially in three phases: (1) septal activation, (2) apical activation, and (3) basal (top) and posterior activation. The first area of the ventricles to be excited is part of the interventricular septum. The septum is activated from the RBB and LBB, although the impulses run from left to right. The extensive network of Purkinje fibers promotes the rapid spread of impulses to the ventricular apex. Activation crosses the cardiac wall from the inside out. The basal and posterior portions of the ventricles are the last to be activated. Deactivation, which begins in diastole, occurs in the opposite direction, spreading from outside to inside (epicardium to endocardium). All areas of the ventricles recover at about the same time.[7-10]

Propagation of cardiac action potential

Electrical activation of muscle cells, called depolarization, is caused by the movement of ions, including sodium, potassium, calcium, and chloride, across the cardiac cell membrane. The deactivation, called repolarization, occurs in a similar way. The movement of ions in and out of the cell creates an electrical difference (voltage) across the cell membrane, which is called the membrane potential. The resting membrane potential of myocardial cells is between -80 and -90 millivolts, that of the SA node is between -50 and -60 millivolts, and that of the AV node is between -60 and -70 millivolts. During depolarization, the inside of the cell becomes less negatively charged as positive ions move inward. In cardiac cells, the difference between the resting membrane potential (in millivolts) and the decrease in negative charge caused by depolarization is the cardiac action potential. The various phases of the cardiac action potential are associated with changes in the permeability of the cell membrane to sodium, potassium, chloride, and calcium. The threshold is the point at which the selective permeability of the cell membrane to these ions is temporarily impaired, leading to depolarization. If the resting membrane potential becomes more negative as a result of a decrease in the extracellular potassium concentration (hypokalemia), the phenomenon is called hyperpolarization.

Table 1. Intracellular ion concentration and extracellular in the myocardium.

Ion	Intracellular concentration	Extracellular concentration
Sodium (Na+)	15 mM	145 mM
Potassium (K+)	150 mM	4 mM
Chloride (Cl-)	5-30 mM	120 mM
Calcium (Ca++)	107 M	2 mM

Notes: M, Mol; mM, milimol per kilogram.

Normal myocardial cell depolarization and repolarization occur in five phases numbered 0 to 4 (Figure 1). Phase 0 consists of depolarization. This phase lasts 1 to 2 milliseconds (ms) and represents a rapid influx of sodium into the cell. Phase 1 is the initial repolarization, during which calcium slowly enters the cell. Phase 2, also called a plateau, is a continuation of repolarization, with the slow influx of calcium and sodium into the cell. Potassium is removed from the cell during phase 3, with a return to the resting membrane potential in phase 4. The time between action potentials corresponds to diastole.

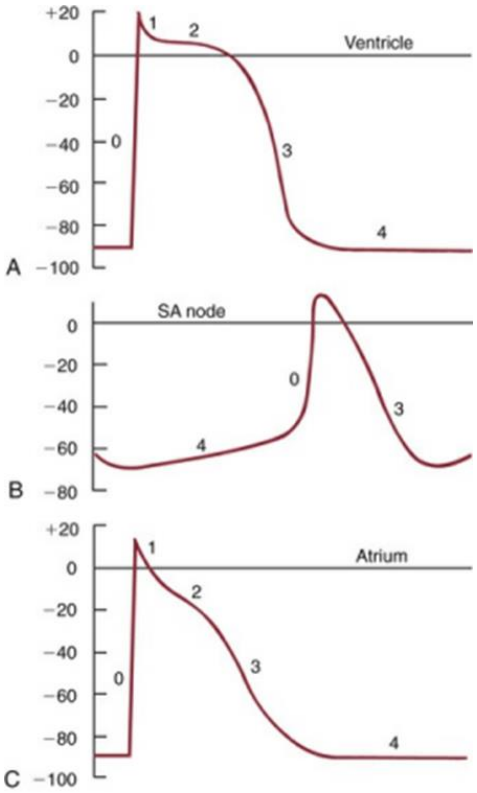

Figure 1. Cardiac action potential. (A) Ventricles; (B), Sinoatrial node (SA); (C) Atrium. The sweep speed at B is half that at A or C.

The depolarization and repolarization phases occur somewhat differently in SA and AV node cells, differences that allow these cells to generate cardiac action potentials independently. Purkinje fiber cells, atria, and ventricles begin with negative resting membrane potential and progress to rapid upstroke or depolarization (phase 0), early rapid repolarization (phase 1), plateau (phase 2), and later rapid repolarization (phase 1). phase 3). The rapid influx of phase 0 is mediated by sodium ions flowing through "fast channels" in the cell membrane and causing rapid increases in action potentials in the Purkinje

fibers, atria, and ventricles. In contrast, SA and AV node cells start with a less negative resting membrane potential, progress to a slow upstroke (phase 0), and usually have no plateau (phase 2). Slow inflows, mediated by calcium through transient and long-lasting channels and sodium ions flowing through the cell membrane "slow channels", are responsible for action potentials of the SA node and AV node. Therefore, drugs that block calcium have a profound effect on slow inflow and can alter cardiac rate. Slow channel-blocking drugs, such as verapamil, are used to treat various cardiovascular disorders.

A refractory period, during which no new cardiac action potentials can be initiated by a stimulus, follows depolarization. This effective or absolute refractory period corresponds to the time required for channels to reopen to allow sodium and calcium influx (phase 0 to half of phase 3). The relative refractory period occurs towards the end of repolarization, following the effective refractory period. During this time, the membrane can depolarize again, but only with a greater-than-normal stimulus. Abnormal refractory periods due to disease can lead to abnormal cardiac rhythms or dysrhythmias, including ventricular fibrillation and cardiac arrest.[11-15]

Electrocardiogram

The origin of a normal electrocardiogram is from the electrical activity recorded by skin electrodes, which is the sum of all the cardiac action potentials. The P wave represents atrial depolarization. The PR interval is a measure of the time from the start of atrial activation to the onset of ventricular activation, usually ranging from 0.12 to 0.20 seconds. The PR interval represents the time it takes to travel from the sinus node through the atrium, AV node, and His-Purkinje system to activate ventricular myocardial cells. The QRS complex is the sum of all the depolarizations of the ventricular muscle cells. The configuration and amplitude of the QRS complex vary greatly between individuals. The duration is usually between 0.06 and 0.10 seconds. During the ST interval, the entire ventricular myocardium is depolarized. Interval QT is sometimes called electric systole from the ventricles. This lasts about 0.4 seconds but is inversely proportional to the cardiac rate.[16,17]

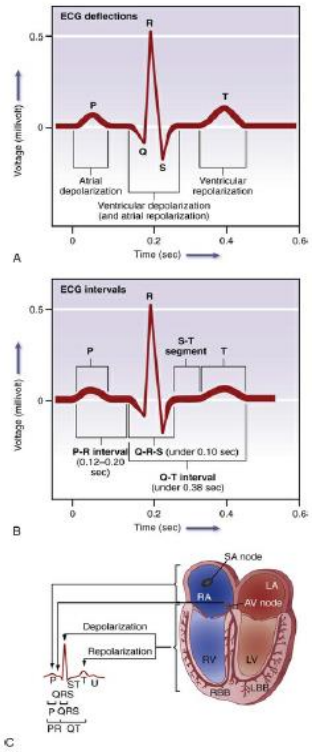

Figure 2. Electrocardiogram (EKG) and the electrical activity of the cardiac. (A) Normal ECG. Depolarization and repolarization; (B) ECG interval between QRS P, and T waves; (C) Schematic representation of the EKG and its relation to the electrical activity of the cardiac; Atrioventricular (AV); left atrium (LA); Left bundle branch (LBB); left ventricle (LV); right atrium (RA); right bundle branch (RBB); right ventricle (RV); sinoatrial node (SA node).

Automation

Automaticity, or the property of generating spontaneous depolarizations to the threshold, allows the SA and AV nodes to generate cardiac action potentials without any stimulus. Cells capable of spontaneous depolarization are called automatic cells. The automatic cells of the cardiac conduction system can stimulate the cardiac to beat even when the cardiac is transplanted and, thus, has no innervation. Spontaneous depolarization is possible in automating cells because the membrane potential does not "rest" during phase 4. Instead, it slowly creeps toward the threshold during the diastole phase of the cardiac cycle. Because the threshold is approached during diastole, phase 4 in automatic cells is called diastolic depolarization. Electrical impulses usually begin in the SA node because their cells depolarize more rapidly than other automatic cells.[18,19]

Rhythm

Rhythm is the orderly generation of action potentials by the cardiac conduction system. The SA node determines the speed because it usually has the fastest depolarization rate. The SA node undergoes spontaneous depolarization 60 to 100 times per minute. If the SA node is damaged, the AV node can become a pacemaker at a rate of about 40 to 60 spontaneous depolarizations per minute. Eventually, however, the conducting cells in the atrium usually take over from the AV node. Purkinje fibers are also capable of spontaneous depolarization but at a slower rate. The Purkinje fibers, therefore, only serve as a pacemaker when the SA and AV nodes are diseased or there is a disturbance in the movement of electric current through the cardiac.[20]

2. Conclusion

Electrical impulses usually arise in the sinoatrial (SA) node. The SA node is innervated by sympathetic and parasympathetic nerves. Each action potential moves rapidly from cell to cell and through the myocardium of the atria, onto the atrioventricular node (AV node), and causes both atria to contract and begin systole. The AV node, located on the right atrial wall superior to the tricuspid valve and anterior to the coronary sinus ostium, conducts action potentials into the ventricles.

3. References

1. Dickstein K, Normann C, Auricchio A, Bogale N, Cleland JG, Gitt AK, et al. CRT survey II: An ESC survey of cardiac resynchronization therapy in 11088 patients—who is doing what to whom and how? Eur J Cardiac Fail 2018; 20: 1039–51.
2. Lund LH, Benson L, Ståhlberg M, Braunschweig F, Edner M, Dahlström U, et al. Age, prognostic impact of QRS prolongation and left bundle branch block, and utilization of cardiac resynchronization therapy: findings from 14 713 patients in the Swedish Cardiac Failure Registry. Eur J Cardiac Fail. 2014; 16: 1073–81.
3. Maisel WH, Stevenson LW. Atrial fibrillation in cardiac failure: epidemiology, pathophysiology, and rationale for therapy. Am J Cardiol. 2003; 91: 2D–8D.
4. Huizar JF, Tan AY, Kaszala K, Ellenbogen KA. Clinical and translational insights on premature ventricular contractions and PVC-induced cardiomyopathy. Prog Cardiovasc Dis. 2021; 66: 17–27.
5. Vernooy K, Verbeek XA, Peschar M, Crijns HJGM, Arts T, Cornelussen RNM, et al. Left bundle branch block induces ventricular remodelling and functional septal hypoperfusion. Eur Cardiac J. 2005; 26: 91–8.
6. Zupan I, Rakovec P, Budihna N, Brecelj A, Kozelj M. Tachycardia induced cardiomyopathy in dogs; relation between chronic supraventricular and chronic ventricular tachycardia. Int J Cardiol. 1996; 56: 75–81.
7. Huizar JF, Kaszala K, Potfay J, Minisi AJ, Lesnefsky EJ, Abbate A, et al. Left ventricular systolic dysfunction induced by ventricular ectopy: a novel model for premature ventricular contraction-induced cardiomyopathy. Circ Arrhythm Electrophysiol. 2011; 4: 543–9.
8. Dosdall DJ, Ranjan R, Higuchi K, Kholmovski E, Angel N, Li L, et al. Chronic atrial fibrillation causes left ventricular dysfunction in dogs but

not goats: experience with dogs, goats, and pigs. Am J Physiol Cardiac Circ Physiol. 2013; 305: H725–H731.

9. Seferovic PM, Ponikowski P, Anker SD, Bauersachs J, Chioncel O, Cleland JGF, et al. Clinical practice update on cardiac failure 2019: pharmacotherapy, procedures, devices and patient management. An expert consensus meeting report of the Cardiac Failure Association of the European Society of Cardiology. Eur J Cardiac Fail. 2019; 21: 1169–86.
10. Aiba T, Hesketh GG, Barth AS, Liu T, Daya S, Chakir K, et al. Electrophysiological consequences of dyssynchronous cardiac failure and its restoration by resynchronization therapy. Circulation. 2009; 119: 1220–30.
11. Jiang M, Zhang M, Howren M, Wang Y, Tan A, Balijepalli RC, et al. JPH-2 interacts with Cai-handling proteins and ion channels in dyads: contribution to premature ventricular contraction-induced cardiomyopathy. Cardiac Rhythm. 2016; 13: 743–52.
12. Wang Y, Eltit JM, Kaszala K, Tan A, Jiang M, Zhang M, et al. Cellular mechanism of premature ventricular contraction-induced cardiomyopathy. Cardiac Rhythm. 2014; 11: 2064–72.
13. Torrado J, Kowlgi GN, Ramirez RJ, Balderas-Villalobos J, Jovin D, Parker C, et al. Eccentric hypertrophy in an animal model of mid- and long-term premature ventricular contraction-induced cardiomyopathy. Cardiac Rhythm. 2021; 2: 80–8.
14. Gupta S, Figueredo VM. Tachycardia mediated cardiomyopathy: pathophysiology, mechanisms, clinical features, and management. Int J Cardiol. 2014; 172: 40–46.
15. Mueller KAL, Heinzmann D, Klingel K, Fallier-Becker P, Kandolf R, Kilias A, et al. Histopathological and immunological characteristics of tachycardia-induced cardiomyopathy. J Am Coll Cardiol. 2017; 69: 2160–72.
16. Cooper MW. Postextrasystolic potentiation. Do we really know what it means and how to use it? Circulation. 1993; 88: 2962–71.
17. Huizar JF, Ellenbogen KA, Tan AY, Kaszala K. Arrhythmia-induced cardiomyopathy: JACC state-of-the-art review. J Am Coll Cardiol. 2019; 73: 2328–44.
18. Takada H, Takeuchi S, Ando K, Kaito A, Yoshida S, Hisada S, et al. Experimental studies on myocardial contractility and hemodynamics in extrasystoles. Jpn Circ J. 1970; 34: 419–30.
19. Cooper MW, Lutherer LO, Lust RM. Postextrasystolic potentiation and echocardiography: the effect of varying basic cardiac rate, extrasystolic coupling interval and postextrasystolic interval. Circulation. 1982; 66: 771–6.
20. Ling L-h, Khammy O, Byrne M, Amirahmadi F, Foster A, Li G, et al. Irregular rhythm adversely influences calcium handling in ventricular myocardium. Implications for the interaction between cardiac failure and atrial fibrillation. Circ Cardiac Fail. 2012; 5: 786–93.