ORIGINAL RESEARCH

Submitted: 2.01.2023; Accepted: 29.01.2023; Published online: 25.02.2023

Review on Cardiac Arrhythmias with ECG Interpretation on Different Myocardial Regions

Gulothungan G., Vickram A. S.

[1]Department of ECE, Vel Tech Rangarajan Dr. Sagunthala R&D Institute of Science and Technology, Chennai, India.
[2]Department of BioTechniology, Saveetha School of Engineering, Saveetha Institute of Medical and Technical Sciences (SIMATS), Chennai, India.

Abstract

Electrocardiogram (ECG) is one of the major diagnostics tools for Cardiac Arrhythmias (CA). ECG interpretation is more important to provide specific treatment of different arrhythmic myocardial regions. In this article the aim is to focus on detailed normal and abnormal ECG interpretation with different myocardial regions. Contraction and Relaxation Process (CRP) during cardiac cycle generates complex ECG with addition of each conduction node electrical vector activity in cardiac conduction system. Epinephrine stimulates Beta1 adrenergic receptors to produce more G stimulation proteins and thus leads to increase calcium level in the cell that provides frequent production of Action Potential (AP) like sinus tachycardia, Early Afterdepolarization (EAD) and Delayed Afterdepolarization (EAD). Myocardial scar regions, AP cannot move because of blockage, in turns moving rapidly around the dead area in a circular way to lead reentrant arrhythmias. On the other hand of respiration cycle, vagus outflow fluctuates (inspiration) the Sinus Node (SN) to increase heart rate (R-R distance will decrease, more P wave and QRS complex will be produced) and to decrease heart rate during expiration (R-R distance will be more). Finally conclude that different myocardial dysfunction regions are the major cause of multi-classified cardiac arrhythmias.

Keywords

Electrocardiogram, Cardiac Arrhythmias, Myocardial Regions, Action Potential, Early Afterdepolarization and Delayed Afterdepolarization

Imprint

Gulothungan G., Vickram A. S. Review on Cardiac Arrhythmias with ECG Interpretation on Different Myocardial Regions. Cardiometry; Issue No. 26; February 2023; p. 256-265; DOI: 10.18137/cardiometry.2023.26.256265; Available from: http://www.cardiometry.net/issues/no26-february-2023/review-cardiac-arrhythmias

Introduction:

1. Normal Cardiac Rhythm

The electrical activity generation in cardiac myocytes is in the range of 60-100 bpm to be called normal cardiac rhythm (Romanò 2015). Different cardiac electrical events occur at different regions of myocardium during cardiac cycle. Each electrical event plays a key in producing normal cardiac rhythm (Wilkins 2007). In general electrical impulses originating from the SA node (SA) and travel through Atrio-Ventricular (AV), Bundle of His and Purkinje Fibre Cells (PFC) possess normal cardiac output rhythm and normal conduction pathway (Pérez-Riera, Barbosa-Barros, and Baranchuk 2016). SA node provides electrical impulses (stimulation) to the atrium chamber, which in turn is responsible for atrium chamber depolarisation and also to AV node, which acts as conduction pathway between SA and Bundle of His present in between atrium and ventricle (Ashley and Niebauer 2004). First cardiac electrical activity originates from the SA node to process normal conduction with normal moderate conduction velocity at the atrial chamber, passes through AV at very slow conduction velocity and reaches the Bundle of His and PFC with high conduction velocity (Ashley and Niebauer 2004; Kupersmith, Krongrad, and Waldo 1973).

2. Cardiac Arrhythmias

The electrical events originate from other than SA nodes, conduction not in normal pathways and high and low velocity fluctuations in normal cardiac pathways are referred to as Abnormal Cardiac Rhythm (ACR) (6). ACR dysfunction occurs, leading to two stages of Cardiac Arrhythmias (CA), one is Brady Arrhythmias (BA) (rhythm rate lower than 60 bpm) and other is Tachy arrhythmias (TA) (rhythm rate higher than 100 bpm). BA and TA are further produce different rhythm rate that are shown clearly in **Fig. 1**

In TA, the rhythm rate from 100-150bpm is called Simple TA. The electrical impulses suddenly start and disappear somewhere between 150-250bpm are called Paroxysmal TA (Ashley and Niebauer 2004; Kupersmith, Krongrad, and Waldo 1973; Tripathi 2011).

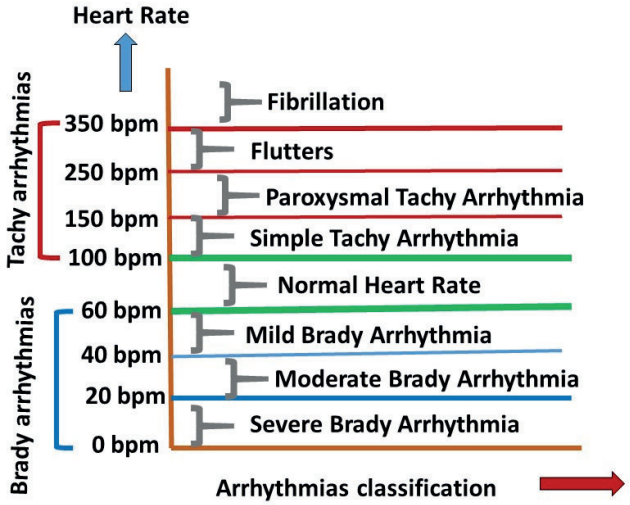

Fig. 1: Different CA based on rhythm rate

The cardiac impulse rate becomes higher compared to normal rhythm lies in the range of 250-350bpm, then this dysfunction is known as Flutters. The flutter activity is present in the atrium region, it is referred to as atrial flutters, likewise in the ventricular region known as ventricular flutters (Nakamura et al. 2022) (Ho and Yen Ho 2022). Sometimes, heart rhythm rate goes above 350bpm is termed as Fibrillation. Like Fluters activity, fibrillation happens in the atrial, called atrial fibrillation and in the ventral region called ventricular fibrillation (Andriulė et al. 2022; Cruz et al. 2022; Aonuma et al. 2022) (Andreadis 2016). Similarly in BA, rhythm rate lies between 40-60bpm referred as Mild BA. The impulse rate lies between 20-40bpm called Moderate BA. The heart rhythm rate activates less than 20bpm known as Severe BA (Miller 2008).

Another classification of Cardiac Arrhythmias (CA) is the region based on heart abnormal rhythms, occurring at SA nodes called sinus arrhythmias, abnormal activity found in atrial myocardial tissue are referred to as atrial arrhythmias (Chahine et al. 2022; Nishikawa et al. 2022). Problems occurring in AV nodes (electrical connection between Atrium and Ventricular region) are known as Nodal or Junctional arrhythmias. All three Sinus, Atrial and Nodal arrhythmias formed above the ventricular tissue are referred as supra ventricular arrhythmias (Andriulė et al. 2022). Abnormal rhythm originated at the ventral region called ventricular arrhythmias. The ventricular tissue acts as a prior function in which it maintains the blood pump flow and gives normal cardiac output, whereas regions have problem in rhythm rate it gives improper cardiac output (contraction and relaxation process will be slow or faster) (Andriulė et al. 2022; Cruz et al. 2022).

3. Action Potential

The normal electrical activity generation Action Potential (AP)) in cardiac cells is due to sodium channel opening, which allows more positive sodium ions into the cell. These incoming sodium ions reduce the resting potential into less negative and reach the threshold potential. After reaching threshold potential, a calcium channel is opened, which allows more positive calcium ions into the cell. This process produces depolarization (Frank, Paul Bianchi, and Keurs 2012). When depolarisation reaches a particular voltage, the potassium channel opens, which sends more potassium ions from the cells that go back to resting potential called repolarisation. This process repeats itself to create a continuous impulse known as AP (Kwek et al. 2022) as shown in **Fig. 2**.

3.1 Mechanism of Cardiac Arrhythmias

The SA nodes have receptors such as Beta1 adrenergic, when epinephrine stimulates Beta1, cellularly it produces G stimulation proteins and this process can lead to addition of calcium in the cell that provides frequent production AP (Kwek et al. 2022; Lv et al. 2022; Ng et al. 2022). That is, the action of epinephrine on an SA node can lead to producing more frequent depolarisation and repolarisation processes based on channels referred as Sinus tachycardia (Increased Automaticity) (Liu, Li, and Yang 2022; Asfaw and Bondarenko 2022). The cell in resting potential takes rest until the sodium-dependent depolarisation process occurs and undergoes a long plateau of potassium leakage and calcium added, because of this process of loss and gain of positive ions is called repolarisation. When the myocytes become ischemic or injured and if the cell is loaded with a number of calcium ions, the resting membrane cannot take rest fluctuating towards threshold potential that produces unwanted triggered automatic impulse that causes tachy arrhythmias (Triggered Automaticity) (Kwek et al. 2022; Lv et al. 2022). In triggered automaticity, the new AP starts at earliest in between repolarization processes known as Early Afterdepolarization (EAD) (Barrio et al. 2022) (Barrio et al. 2022; Lypourlis, Mundisugih, and Chia 2022). When the resting membrane potential fluctuates and reaches the threshold, the depolarisation occurs is somewhat delayed; this activity is known as Delayed Afterdepolarization (DAD) ("The Effects of Autonomic Neurotransmitters on the Delayed Afterdepolarization of Sheep Cardiac Purkinje Fibers"

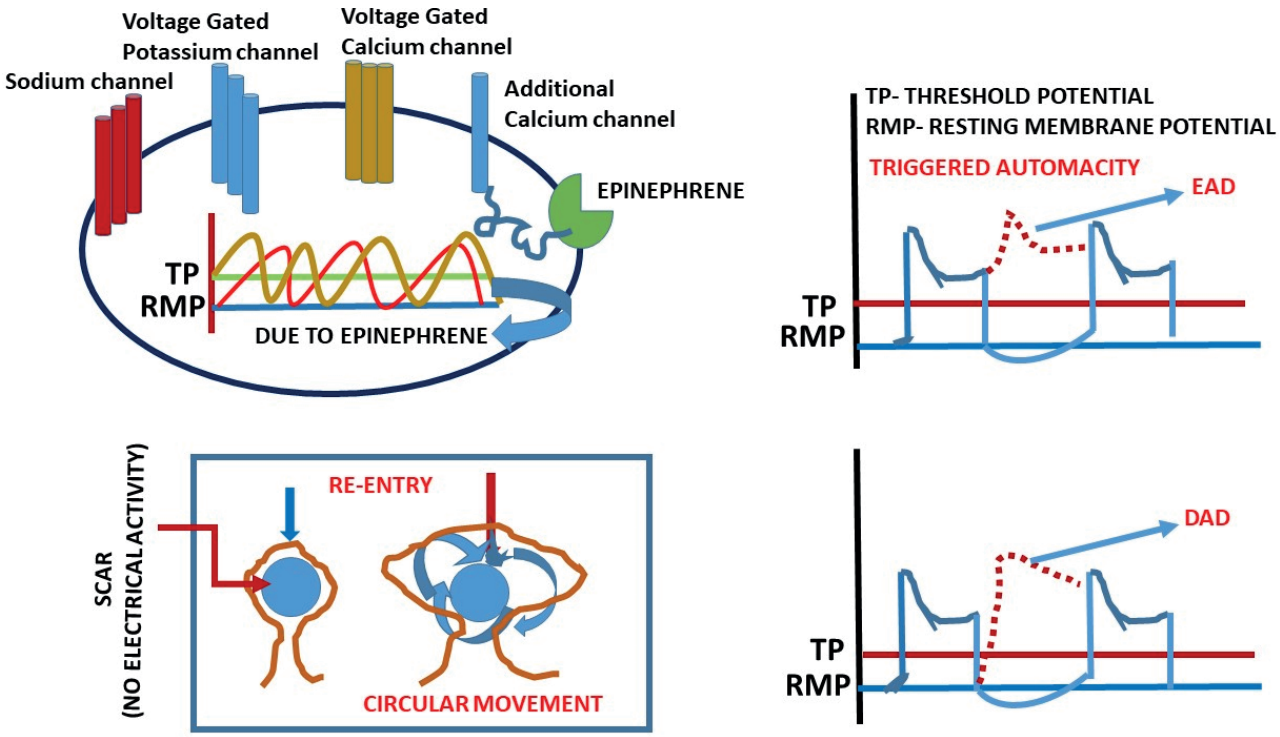

Fig. 2: Types of Cardiac Arrhythmia with ECG Patterns

1988) ("The Effects of Autonomic Neurotransmitters on the Delayed Afterdepolarization of Sheep Cardiac Purkinje Fibers" 1988; Hiraoka 1987).

3.2 Re Entry or Circus Movement

In a piece of myocardium having area which is electrically not excitable, when an impulse enters the myocardium that surrounds the electrically resistant area and at that time they met each other (conduction velocity was same), those impulses cannot re-enter into the other areas and moves forward because of refractuated (Nogami, Phanthawimol, and Haruna 2022). In another area of the heart having scar, at one side of the scar the impulse moves well but on the other side electric potential cannot move because of blockage. When the moving impulses reaches the other side becomes electrically excitable can enter into the area back and this type of impulses move towards the non-resistant area (dead area) is mostly seen when total time taken by the cardiac impulse around an area is longer than the time required for a blocked area to recover from refractive phase to excitable. And when the impulses started moving rapidly around the dead area in a circular way that generated tachy arrhythmia (Liu, Li, and Yang 2022).

3.4 Sinus Arrhythmia

During the respiration cycle, the vagus outflow fluctuates along with the sinus node altered. During inspiration once vagal inhibition cannot reinhibitate in the sinus node this process can lead to increase in heart rate (R-R distance will decrease, more P wave and QRS complex will be produced).

On the other hand during expiration, vagus stimulation is able to inhibitate the heart rate which results in slight decreases in rhythm (R-R distance will be more). This breathing process is called physiological sinus arrhythmia as shown in **Fig. 3**. Mostly occurs within a diabetic patients (Luthra 2017a). The heart rate will be more than 100 due to increased activity in the SA node. People with fever and thyroid problems lead to an increase in heart rate (depolarisation) produces sinus tachycardia (Nogami, Phanthawimol, and Haruna 2022; Watanabe et al. 2022). In sinus tachycardia the P wave produced more frequently (occurs earlier) in a time because of increased SA nodes that results in increased cardiac stimulation (Morishima et al. 2022). People with sinus bradycardia have slow heart rate less than 60bpm. Patients having hypothyroidism, hypothermia, cholestatic jaundice produce slow cardiac output. P waves appeared less frequently followed by QRS and had a long pass then repeated the complex process (R-R distance will be more) (Lima da Silva et al. 2022) (Luthra 2017b). When sinus nodes become injured it behaves in an irregular manner which results in sudden increased and decreased activity slower than normal pattern at the same time.

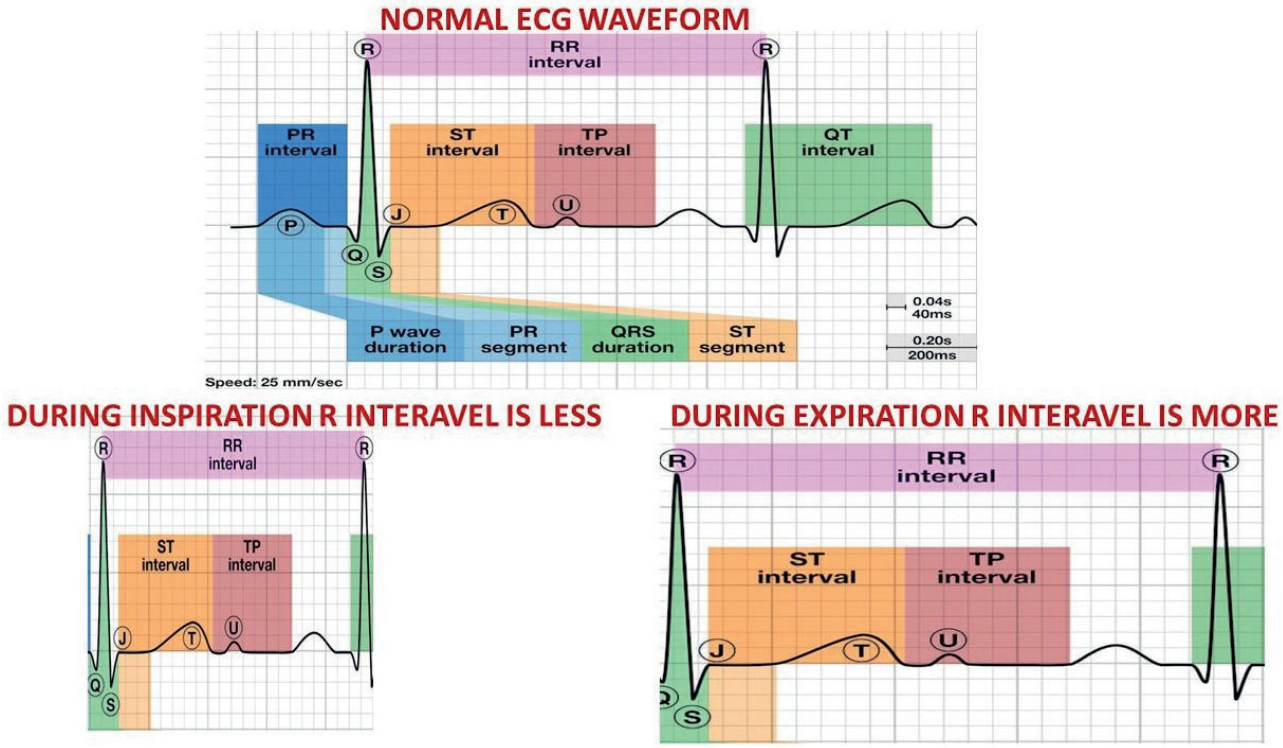

Fig. 3: Normal and Sinus Arrhythmias ECG Patterns during Inspiration and Expiration

This phenomenon is called Sinus tachy brady syndrome or also known as Sick sinus syndrome (Luthra 2017a; Zhang et al. 2022). All these sinus syndrome are clearly mapped with its ECG patterns are shown in **Fig. 4**

3.5 Atrial Tachyarrhythmias

The Atria region is acting as a pacemaker that produces electrical impulse at a rate of 125-250 per minute. This type of cardiac impulse gives ECG pattern as four P waves followed by QRS and T waves because of increases in atrial activity (Multiple atrial activity followed by ventricular electrical impulse) (Baranchuk 2018). The electrical activity in the Atrial region becomes fast at a rate of 250-350 per minute this produces a special type of unrecognized P waves also called flutter waves followed by depolarisation and repolarisation (Klabunde 2005). Foci firing simultaneously takes control of the atrial region, in that case there may be several depolarisation vector present in atrium are

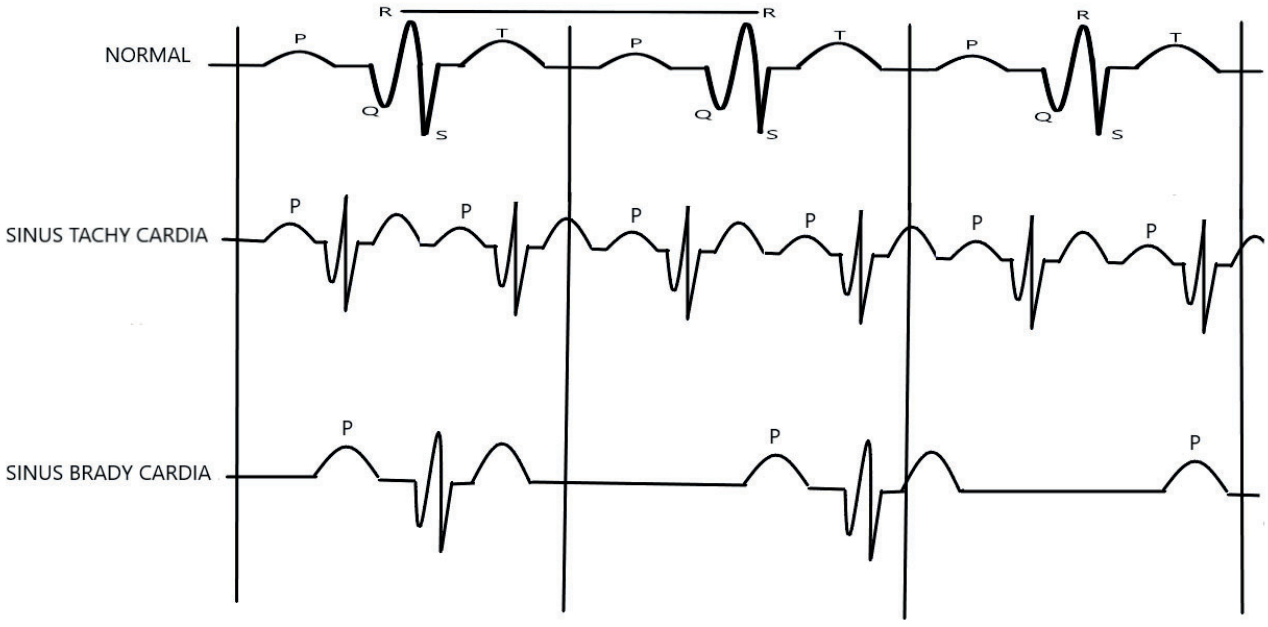

Fig. 4: Sinus Arrhythmias with ECG Patterns During Tachy and Brady Condition

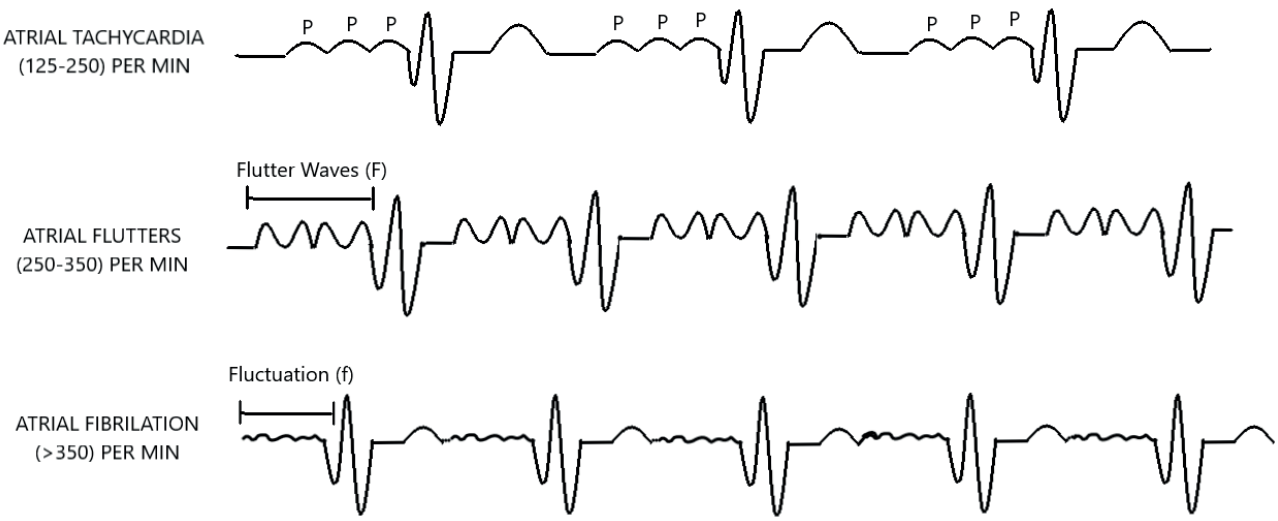

Fig. 5: Atrial Arrhythmias with ECG Patterns on Different Conditions

directed in a different directions at a given time produce electrical activity that are not synchronized because of this process there is no well-defined electrical power in the atrial activity. The ECG pattern performs as the P wave possesses some fluctuates from the atrial region, conducted down to AV nodes which gives QRS complex processes. The electrical activity of this type is more than 350 per minute (Goldberger 2006). All these atrial syndrome are clearly mapped with its ECG patterns are shown in **Fig. 5**

3.6 Junctional or Nodal Arrhythmia

The connection between the atrial and ventral region is called Nodal. This process empties the blood in the atria and allows blood into the ventricular region by conduction pathway. When there is a problem at this junction either the tissue in the junction becomes too fast leading to junctional tachyarrhythmia or the node process becomes slow indicates junction bradyarrhythmia (Tan et al. 2022). This describes the arrythmias problem in the junction area at the centre point. There is a fibrous area in which the atrial is mounted at above end and the ventricular is mounted at below end. The fibrous acts as an electrical insulator which means current cannot pass through it. So, the only area where the current can pass nodes from the atria through the ventricle is at the centre AV node. This is why we can introduce the AV node as a special electrical window or specialized electrical connection between the atria and the ventricle (Soltani et al. 2022).

Current has to pass through the Bundle of Hiss and Ventricular system. There are many reasons why current passes through this slower. Firstly, AV nodal cells are special myocardial cells and very small in size so the current has to jump many numbers of membranes in a short distance (Davis 1997)). Secondly, gap junctions in between the AV nodal cells are very less. These gap junctions are specialized electrical windows in between the myocardial cells which involve the transfer of current from one cell to the next cell. Thus, the AV node becomes a low resistance area (33). Third, Diameter of the cell is small, cells with smaller diameter conduct slowly and cells with larger diameter conduct fast (Levine 2013) (Fisch and Knoebel 2000). Fourth, In case of depolarization of current there can be two types of channels involved: Voltage gated Sodium Channel and Voltage gated Calcium Channel. Resting membrane potential of the AV node is – 60mV. At this point the fast-conducting sodium channels are permanently closed. That is why SA Node and AV Node depolarization depend on Calcium Channel which acts slowly compared to Sodium Channel (Das and Zipes 2012). Fifth, the electronegativity of the AV node is less negative so the positive Calcium Channel attracts slowly. The morphological arrangement of AV node is shown in **Fig. 6**

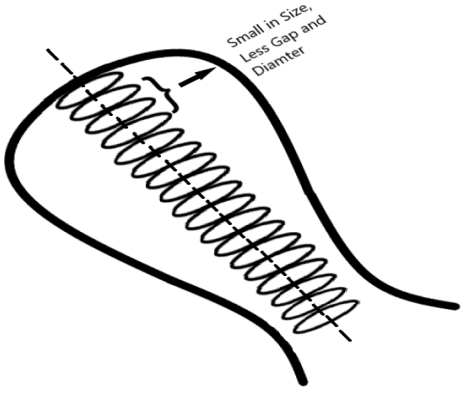

Fig. 6: Morphological Arrangement of Node Cell in AV Node

Normal Cardiovascular System

Cardiac system is an electromechanical pump which helps to supply essential nutrients to the tissue and remove unwanted by products through blood circulation. Cardiac output is based on the efficient mechanism of Contraction and Relaxation Process (CRP) during cardiac cycle. CRP in turn synchronizes and coordinates by Cardiac Conduction System (CCS). CCS is a specialized cardiac myocyte, which produces normal cardiac rhythm originating from the Sino-Atrial (SA) node via Atrio -Ventricular (AV) node and Bundle of His to purkinje fibre cells (PFC). In a normal healthy heart, there will be only one electrical passage. Since atrial depolarisation led to atrial contraction which means atrial electrical activity leads to atrial mechanical activity and this mechanical activity will be slower. During this process the current is held for a while at the AV node which is a tissue specialized in slow conduction with very slow velocity (Dorlet and Brembilla-Perrot 2015). Which means there will be a delay of about 0.1s. This delay is very important for the mechanical function of the heart. Importance of AV node delay is to introduce a delay between atrial and ventricular excitation and Gives time for the atrial contraction to empty the blood into the ventricles (Gussak et al. 2003). In the ECG this delay between the atrial and ventricular gives an interval between the PR. In the case of humans generally this interval will be 120 – 200ms (Fotiadis and I. 2015) (Kenny 2011). Myocardial node cell conduction characteristics are given in Table **1.**

Table 1

Myocardium Node Cell Characteristics in Conduction

NODE	SPECIALIZATION
SA Node	High Automaticity
Atrial and Ventricular	Contraction
AV Node	Slow Conduction
Bundle of Hiss, Bundle Branches, Parkinson System	Fast Conduction

5. Electrical Events in the Cardiovascular System

5.1 Atrial Depolarization – First Electrical Event

In a Normal Heart electrical activity starts from depolarization of SA Node. A wave of Depolarization is generated by the SA node. Initially cells near the SA node are stimulated and undergo depolarization and this again occurs to the second layer then to third layers and so on. The wave of depolarization from the SA node is passing through the cell layers within the Atrial Myocardium towards downward and leftward because the region of the SA node is present in Atria on the right and upper side. All the depolarizing current can be added together which makes a single vector which passes left and downward. This vector represents the electromagnetic force which is produced when both the Atria undergo the process of depolarization (Wilkins 2007) (Huang and Wood 2011). This vector is termed as Atrial Depolarization Vector are clearly shown in **Fig. 7.**

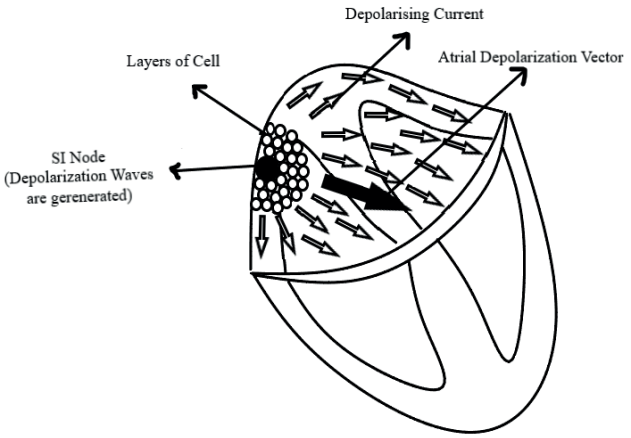

Fig. 7: Atrial Depolarization Electrical Event Vector Force Representation

Atrial Myocardium is thin in size, because of its thin structure the vector is smaller. Atria does not have a very specialized and fast conducting system; it undergoes depolarization moderately. When both Atria complete the depolarization. The depolarising waves hitting the fibrous tissue present between the Atria and Ventricle is not a good conductor. Because of this most of the depolarising current which hits the fibrous tissue dies out. Which does not allow the depolarisation to pass through the Atria and Ventricle.

5.2 Stimulation of AV Node -Second Electrical Event

The Specific point where the depolarisation can pass through is the AV node. This is termed as the Electrical window. Only, the depolarisation which hits the AV node passes through the ventricle while the other dies (Michael 2017). AV node is a special modified myocardium, we know that AV node is specialized in slow conduction of electricity it takes about

0.1sec. It's because initially atria should contract and later ventricle contracts. The characteristics of AV node and Purkinje system are shown in **Table 2 and Fig. 8.**

Table 2

Myocardium AV Node and PFC Characteristics in Structure

AV NODE	PURKINJE SYSTEM
Slow Conduction	Fast conduction
Small Cell Size	Large Cell Size
Less Number of Electrical Windows	More Number of Electrical Windows
Less Diameter Cells	Large Diameter Cells
Arranged in a Right angle pattern from the direction of current flow	Arranged in a Linear pattern from the direction of current flow
Depolarization depends on Calcium which works slowly	Depolarization depends on Sodium which works fast
Resting Membrane potential is -60mv which is less electronegative compared to Purkinje System.	Resting Membrane potential is -90mv which is more electronegative compared to AV node which attracts the cations more.

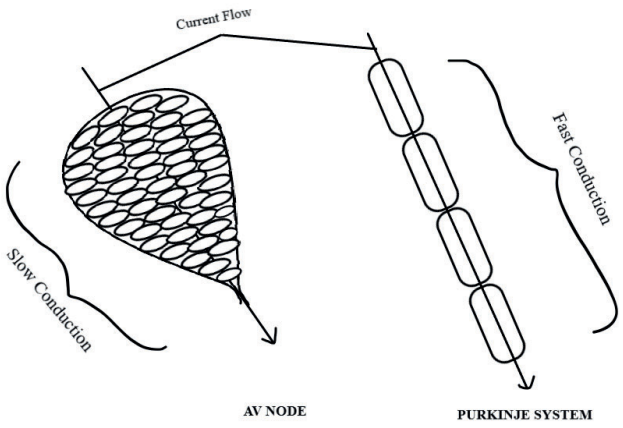

Fig. 8: Structural Arrangement of AV Node And PFC in Myocardium

As depolarising current passes through the AV node, it passes through the bundle of hiss and bundle branches initially it depolarises the septum of the ventricle secondly then the major part of the ventricle and thirdly to the basal ventricle.

5.3 Ventricular Septum Depolarisation – Third Electrical Event

In Septum Myocardium, the upper part is fibrous and the lower part is muscular. Fibrous tissue allows it to pass through the bundle branches which is acting as an insulator like a rubber in the electrical events. Septum Myocardium is stimulated by the left bundle branch and not by the right bundle branch because there is a small connection between the left bundle branch and Septum Myocardium whereas the right bundle branch passes the current downwards directly (Jastrzębski et al. 2020) (Havranek et al. 2015). This connection is also at the lower part of the septum, so the wave of depolarisation is from the left to right and bottom to top. There is a small vector which moves from right to upward. During this Ventricular Septum Depolarisation small vector but fast conduction takes place.

5.4 Major Parts of Ventricular Depolarization- Fourth Electrical Event

Purkinje Cells will be connected to myocardial cell one another. Staring from inner myocardium and then to the outer myocardium. Left ventricle is thicker than the Right ventricle so the depolarizing vector will be strong in the left which is called Left Ventricular Depolarization Vector and weak in the right which is called Right Ventricular Depolarization Vector (Havranek et al. 2015; Carpio et al. 2019). All these vectors will occur simultaneously which means they can be added which creates another vector which represents the depolarization of a major part of both ventricles are shown in **Fig. 9**.

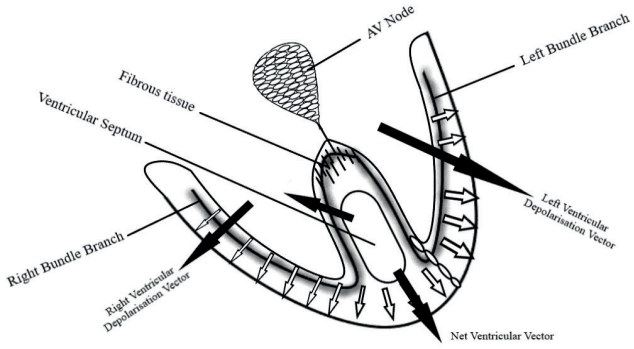

Fig. 9: Electrical Event Vector Force Representation in Myocardium

- Left Ventricular Depolarization Vector – Downward and Leftward
- Right Ventricular Depolarization Vector – Downward and Rightward
- Net Ventricular Vector (Left + Right) – Downward and Leftward

5.5 Ventricular Basal Depolarisation- Fifth Electrical Event

After the depolarization of major parts, it reaches to the basal area. Here the current movement will be upward and rightward and these small and fast vec-

tors represent ventricular basal depolarization. This is the last stage of ventricular depolarization represents neatly in **Fig. 9** (Hiroi et al. 2004).

Conclusion:

ECG is one of the major diagnostics tools for CA. ECG interpretation is more important to provide specific treatment of different arrhythmic myocardial regions. CRP during cardiac cycle (systole and diastole) generates complex ECG with addition of each conduction node electrical vector activity in cardiac conduction system. Increase calcium level in the cardiomyocytes that provide frequent production of AP like sinus tachycardia, EAD and DAD. Myocardial scar regions, AP cannot move because of blockage, in turns moving rapidly around the dead area in a circular way to lead reentrant arrhythmias. On the other hand of respiration cycle, vagus outflow fluctuates (inspiration) the SA node to increase heart rate and to decrease heart rate during expiration (R-R distance will be more). Finally conclude that different myocardial dysfunction regions are the major cause of multi-classified CA.

References:

1. Andreadis, Emmanuel A. 2016. Hypertension and Cardiovascular Disease. Springer.
2. Andriulė, Inga, Dalia Pangonytė, Asfree Gwanyanya, Dainius Karčiauskas, Kanigula Mubagwa, and Regina Mačianskienė. 2022. "Detection of TRPM6 and TRPM7 Proteins in Normal and Diseased Cardiac Atrial Tissue and Isolated Cardiomyocytes." International Journal of Molecular Sciences 23 (23). https://doi.org/10.3390/ijms232314860.
3. Aonuma, Kazuhiro, Dongzhu Xu, Nobuyuki Murakoshi, Kazuko Tajiri, Yuta Okabe, Zixun Yuan, Siqi Li, et al. 2022. "Novel Preventive Effect of Isorhamnetin on Electrical and Structural Remodeling in Atrial Fibrillation." Clinical Science 136 (24): 1831–49.
4. Asfaw, Tesfaye Negash, and Vladimir E. Bondarenko. 2022. "A Compartmentalized Mathematical Model of the β- and β-Adrenergic Signaling Systems in Ventricular Myocytes from Mouse in Heart Failure." American Journal of Physiology. Cell Physiology, December. https://doi.org/10.1152/ajpcell.00366.2022.
5. Ashley, Euan A., and Josef Niebauer. 2004. Cardiology Explained. Remedica.
6. Baranchuk, Adrian. 2018. Brugada Phenocopy: The Art of Recognizing the Brugada ECG Pattern. Academic Press.
7. Barrio, Roberto, M. Ángeles Martínez, Sergio Serrano, and Esther Pueyo. 2022. "Dynamical Mechanism for Generation of Arrhythmogenic Early Afterdepolarizations in Cardiac Myocytes: Insights from in Silico Electrophysiological Models." Physical Review. E 106 (2-1): 024402.
8. Carpio, Edison F., Juan F. Gomez, Rafael Sebastian, Alejandro Lopez-Perez, Eduardo Castellanos, Jesus Almendral, Jose M. Ferrero, and Beatriz Trenor. 2019. "Optimization of Lead Placement in the Right Ventricle during Cardiac Resynchronization Therapy. A Simulation Study." Frontiers in Physiology 10 (February): 74.
9. Chahine, Yaacoub, Fima Macheret, Karen Ordovas, Joonseok Kim, Patrick M. Boyle, and Nazem Akoum. 2022. "MRI-Quantified Left Atrial Epicardial Adipose Tissue Predicts Atrial Fibrillation Recurrence Following Catheter Ablation." Frontiers in Cardiovascular Medicine 9 (December): 1045742.
10. Cruz, Inês, Sara Lopes Fernandes, Sílvia O. Diaz, Francisca Saraiva, António S. Barros, João Primo, Francisco Sampaio, Ricardo Ladeiras-Lopes, and Ricardo Fontes-Carvalho. 2022. "Epicardial Adipose Tissue Volume Is Not an Independent Predictor of Atrial Fibrillation Recurrence after Catheter Ablation." Revista Espanola de Cardiologia , November. https://doi.org/10.1016/j.rec.2022.11.006.
11. Das, Mithilesh Kumar, and Douglas P. Zipes. 2012. Electrocardiography of Arrhythmias: A Comprehensive Review: A Companion to Cardiac Electrophysiology. Elsevier Health Sciences.
12. Davis, Dale. 1997. Differential Diagnosis of Arrhythmias. Saunders.
13. Dorlet, Sarah, and Béatrice Brembilla-Perrot. 2015. "0049: Long-Term Follow- up of AV Conduction Disturbances after Slow Pathway Ablation in Patients with AV Node Reentrant Tachycardia." Archives of Cardiovascular Diseases Supplements. https://doi.org/10.1016/s1878-6480(15)71700-3.
14. Fisch, Charles, and Suzanne Knoebel. 2000. Electrocardiography of Clinical Arrhythmias. Wiley-Blackwell.
15. Fotiadis, and Dimitrios I. 2015. Handbook of Research on Trends in the Diagnosis and Treatment of Chronic Conditions. IGI Global.
16. Frank, George B., C. Paul Bianchi, and Henk Keurs. 2012. Excitation-Contraction Coupling in Skeletal, Cardiac, and Smooth Muscle. Springer Science & Business Media.

17. Goldberger, Ary Louis. 2006. Clinical Electrocardiography: A Simplified Approach. Mosby.
18. Gussak, Ihor, Charles Antzelevitch, Stephen C. Hammill, Win K. Shen, and Preben Bjerregaard. 2003. Cardiac Repolarization: Bridging Basic and Clinical Science. Springer Science & Business Media.
19. Havranek, Stepan, Tomas Palecek, Tomas Kovarnik, Ivana Vitkova, Miroslav Psenicka, Ales Linhart, and Dan Wichterle. 2015. "Arrhythmogenic Substrate at the Interventricular Septum as a Target Site for Radiofrequency Catheter Ablation of Recurrent Ventricular Tachycardia in Left Dominant Arrhythmogenic Cardiomyopathy." BMC Cardiovascular Disorders 15 (March): 18.
20. Hiraoka, Masayasu. 1987. "Characteristics of Triggered-Activity and Delayed Afterdepolarization in Responses to the Electrical Stimulation." Japanese Circulation Journal. https://doi.org/10.1253/jcj.51.176.
21. Hiroi, Yukio, Katsuhito Fujiu, Shuhei Komatsu, Makoto Sonoda, Yasunari Sakomura, Yasushi Imai, Yumi Oishi, et al. 2004. "Carvedilol Therapy Improved Left Ventricular Function in a Patient with Arrhythmogenic Right Ventricular Cardiomyopathy." Japanese Heart Journal 45 (1): 169–77.
22. Ho, S. Yen, and S. Yen Ho. 2022. "Normal and Abnormal Atrial Anatomy Relevant to Atrial Flutters." Cardiac Electrophysiology Clinics. https://doi.org/10.1016/j.ccep.2022.03.001.
23. Huang, Shoei K., and Mark A. Wood. 2011. Catheter Ablation of Cardiac Arrhythmias. Elsevier Health Sciences.
24. Jastrzębski, Marek, Paweł Moskal, Agnieszka Bednarek, Grzegorz Kiełbasa, Aleksander Kusiak, Tomasz Sondej, Adam Bednarski, Pugazhendhi Vijayaraman, and Danuta Czarnecka. 2020. "Programmed Deep Septal Stimulation: A Novel Maneuver for the Diagnosis of Left Bundle Branch Capture during Permanent Pacing." Journal of Cardiovascular Electrophysiology 31 (2): 485–93.
25. Kenny, Tom. 2011. The Nuts and Bolts of Paced ECG Interpretation. John Wiley & Sons.
26. Klabunde, Richard E. 2005. Cardiovascular Physiology Concepts. Lippincott Williams & Wilkins.
27. Kupersmith, Joel, Ehud Krongrad, and Albert L. Waldo. 1973. "Conduction Intervals and Conduction Velocity in the Human Cardiac Conduction System." Circulation. https://doi.org/10.1161/01.cir.47.4.776.
28. Kwek, Xiu-Yi, Andrew R. Hall, Wei-Wen Lim, Khairunnisa Katwadi, Poh Loong Soong, Elina Grishina, Kun-Han Lin, et al. 2022. "Role of Cardiac Mitofusins in Cardiac Conduction Following Simulated Ischemia-Reperfusion." Scientific Reports 12 (1): 21049.
29. Levine, Glenn N. 2013. Arrhythmias 101. JP Medical Ltd.
30. Lima da Silva, Gustavo, Nuno Cortez-Dias, Afonso Nunes Ferreira, Elad Nakar, Raquel Francisco, Mariana Pereira, Javier Moreno, Raphaël P. Martins, Fausto J. Pinto, and João de Sousa. 2022. "Impact of Different Activation Wavefronts on Ischemic Myocardial Scar Electrophysiological Properties during High-Density Ventricular Tachycardia Mapping and Ablation." Journal of Cardiovascular Electrophysiology, November. https://doi.org/10.1111/jce.15740.
31. Liu, Qifang, Jun Li, and Long Yang. 2022. "The Short-Long-Short-Short Sequence and Polymorphic Ventricular Tachycardias Storm." Annals of Noninvasive Electrocardiology: The Official Journal of the International Society for Holter and Noninvasive Electrocardiology, Inc, December, e13034.
32. Luthra, Atul. 2017a. "Interesting Cases Diagnosed by ECG." ECG. https://doi.org/10.5005/jp/books/12933_26.
33. ———. 2017b. "Nomenclature of ECG Deflections." ECG. https://doi.org/10.5005/jp/books/12933_2.
34. Lv, Shichao, Yunjiao Wang, Wanqin Zhang, and Hongcai Shang. 2022. "The Chemical Components, Action Mechanisms, and Clinical Evidences of YiQiFuMai Injection in the Treatment of Heart Failure." Frontiers in Pharmacology 13 (November): 1040235.
35. Lypourlis, Dimitrios, Juan Mundisugih, and Yongcheng Victor Chia. 2022. "Early Afterdepolarizations and Electrical Storm after Cardioversion for Atrial Fibrillation." HeartRhythm Case Reports 8 (4): 254–58.
36. Michael, Kevin. 2017. Interpreting Cardiac Electrograms: From Skin to Endocardium. BoD – Books on Demand.
37. Miller, John M. 2008. Ventricular Arrhythmias. W B Saunders Company.
38. Morishima, Itsuro, Yasunori Kanzaki, Yasuhiro Morita, Koichi Inoue, Atsushi Kobori, Kazuaki Kaitani, Toshiya Kurotobi, et al. 2022. "Catheter Ablation for Paroxysmal Atrial Fibrillation With Sick Sinus Syndrome: Insights From the Kansai Plus Atrial Fibrillation Registry." Heart, Lung & Circulation, October. https://doi.org/10.1016/j.hlc.2022.09.007.

39. Nakamura, Toshihiro, Koji Fukuzawa, Takeshi Aiba, and Seiko Ohno. 2022. "Case Report of a Ventricular Fibrillation Storm with a Cardiac Conduction Disorder and HCN4 Variant 18 Years after Ablation of Atrial Flutter." European Heart Journal. Case Reports 6 (11): ytac431.
40. Ng, Kwong-Man, Qianqian Ding, Yiu-Lam Tse, Oscar Hou-In Chou, Wing-Hon Lai, Ka-Wing Au, Yee-Man Lau, et al. 2022. "Isogenic Human-Induced Pluripotent Stem-Cell-Derived Cardiomyocytes Reveal Activation of Wnt Signaling Pathways Underlying Intrinsic Cardiac Abnormalities in Rett Syndrome." International Journal of Molecular Sciences 23 (24). https://doi.org/10.3390/ijms232415609.
41. Nishikawa, Yusei, Hiroyuki Takaoka, Tomonori Kanaeda, Haruhiro Takahira, Sakuramaru Suzuki, Shuhei Aoki, Hiroki Goto, et al. 2022. "A New Composite Indicator Consisting of Left Ventricular Extracellular Volume, N-Terminal Fragment of B-Type Natriuretic Peptide, and Left Ventricular End-Diastolic Volume Is Useful for Predicting Reverse Remodeling after Catheter Ablation for Atrial Fibrillation." Heart and Vessels, December. https://doi.org/10.1007/s00380-022-02220-x.
42. Nogami, Akihiko, Wipat Phanthawimol, and Tetsuya Haruna. 2022. "Catheter Ablation for Ventricular Tachycardia Involving the His-Purkinje System: Fascicular and Bundle Branch Reentrant Ventricular Tachycardia." Cardiac Electrophysiology Clinics 14 (4): 633–56.
43. Pérez-Riera, Andrés R., Raimundo Barbosa-Barros, and Adrian Baranchuk. 2016. Left Septal Fascicular Block: Characterization, Differential Diagnosis and Clinical Significance. Springer.
44. Romanò, Massimo. 2015. Text Atlas of Practical Electrocardiography: A Basic Guide to ECG Interpretation. Springer.
45. Soltani, Danesh, Bayan Azizi, Roja Rahimi, Azita H. Talasaz, Hossein Rezaeizadeh, and Ali Vasheghani-Farahani. 2022. "Mechanism-Based Targeting of Cardiac Arrhythmias by Phytochemicals and Medicinal Herbs: A Comprehensive Review of Preclinical and Clinical Evidence." Frontiers in Cardiovascular Medicine 9 (September): 990063.
46. Tan, Hong-Wei, Wei-Dong Gao, Xin-Hua Wang, Zhi-Song Chen, and Xue-Bo Liu. 2022. "A Four-Stepwise Electrocardiographic Algorithm for Differentiation of Ventricular Arrhythmias Originated from Left Ventricular Outflow Tract." Journal of Clinical Medicine Research 11 (21). https://doi.org/10.3390/jcm11216398.
47. "The Effects of Autonomic Neurotransmitters on the Delayed Afterdepolarization of Sheep Cardiac Purkinje Fibers." 1988. Journal of Molecular and Cellular Cardiology. https://doi.org/10.1016/s0022-2828(98)90094-3.
48. Tripathi, Onkar Nath. 2011. "Cardiac Ion Channels and Heart Rate and Rhythm." Heart Rate and Rhythm. https://doi.org/10.1007/978-3-642-17575-6_1.
49. Watanabe, Tomonori, Yasuhiro Yokoyama, Hitoshi Hachiya, Takafumi Okuyama, Hiroaki Watanabe, Ayako Yokota, Masashi Kamioka, et al. 2022. "Electrogram Characteristics at Successful Cryoablation Sites in Slow-Fast Atrioventricular Nodal Reentrant Tachycardia." Journal of Electrocardiology 75 (September): 44–51.
50. Wilkins, Lippincott Williams &. 2007. ECG Interpretation. Lippincott Williams & Wilkins.
51. Zhang, Xin 'ai, Yong Zhao, Yutong Zhou, Jiayu Lv, Jiaran Peng, Haiyan Zhu, and Ruxiu Liu. 2022. "Trends in Research on Sick Sinus Syndrome: A Bibliometric Analysis from 2000 to 2022." Frontiers in Cardiovascular Medicine 9 (November): 991503.