

The Impact and Role of Serum Electrolytes in Patients with Supraventricular Tachycardia

Hasan Haider Al-Salamy¹ Hedef El-Yassin² Ameen Al-Alwany³

¹Department of Clinical Biochemistry, College of Medicine, University of Kerbala, Iraq

E-mail: hasan.haidar@uokarbal.edu.iq

²Department of Clinical Biochemistry, College of Medicine, University of Baghdad, Iraq

³Dean of the College of Medicine, College of Medicine, University of Baghdad, Iraq



Received: 13/10/2025

Accepted: 22/11/2025

Published: 30/12/2025

DOI:

10.65682/kjnhs.v1.i4.64-74

Abstract

Standardized cardiology tools such as electrophysiological methods and serum biomarkers lack accuracy in predicting which patients are at risk of Supraventricular Tachycardia, and cardiac pathophysiology may sometimes cause sudden death. This study aims to assess the role of electrolytes in supraventricular tachycardia cases and assess their benefit in diagnosing and screening supraventricular tachycardia. This study involved 128 individuals divided into two groups; the first group included 64 healthy individuals, and the second had 64 patients with supraventricular tachycardia. All individuals had an age range of 17-68 years and a body mass index ranging from (30.4-49.9Kg/m²) The study involved 61 females and 60males; the work was carried out between march-September 2021, all patients chosen from a private Nursing hospital and healthy individuals selected from college of medicine Karbala university.

Keywords: Electrolytes, Arrhythmia, Supraventricular Tachycardia



1. Introduction

The term 'SVT' directly refers to a condition called tachycardia, which means having a heart rate of over 100 beats per minute (b.p.m.) while at rest. This type of tachycardia occurs due to the involvement of tissue located at or above the His bundle in the heart's electrical system (Hannun et al., 2019). SVT specifically refers to tachycardia, where the heart beats at a rate exceeding 100 beats per minute (b.p.m.) while the person is at rest. This type of tachycardia occurs when tissue located at or above the His bundle, which is a part of the heart's electrical system, is involved in the mechanism (Page et al., 2016). Historically, SVT has commonly been employed as a term encompassing various forms of tachycardia, with the exception of ventricular tachycardia (VTs) and atrial fibrillation (AF). Therefore, it encompasses tachycardias like atrioventricular (AV) re-entry caused by accessory connections, which fundamentally differ from supraventricular rhythms. The term 'narrow QRS tachycardia' refers to tachycardias with a QRS duration of 120 ms or less. In clinical settings, supraventricular tachycardia (SVT) can present as either narrow or wide QRS tachycardia. While not always the case, the majority of SVT cases exhibit regular rhythms (Lip et al., 2018). These guidelines do not address atrial fibrillation (AF), as it is the topic of distinct clinical guidelines and several consensus documents (Page et al., 2016). Supraventricular tachycardia encompasses a variety of rapid abnormal heart rhythms that arise from a circuit or focal point involving either the atria or the atrioventricular node (Bitter et al., 2015). Electrolytes are charged molecules that have a crucial role in regulating acid-base balance, blood clotting, similarly to bodily fluids, muscle contractions also exhibit a corresponding phenomenon (Bitter et al., 2015). The body contains various electrolytes that fulfil specific and vital functions, and many of them contribute to maintaining the state of equilibrium or stability of fluids between the intracellular and extracellular environments. Maintaining this balance is of utmost importance for essential aspects such as hydration, transmission of nerve impulses, muscle function, and maintaining an optimal (pH level) (Luo et al., 2016). The primary electrolytes present in the body include Potassium, Sodium, Magnesium, Calcium, and Chloride. Among these, Serum Sodium, Potassium, and Chloride are recognized as the key factors that influence the electrophysiological characteristics of the myocardial membrane (Ahmed et al., 2014).

2. Purpose of the Study

To evaluate serum electrolytes in patients and compare its level with apparently healthy individuals.

3. Method

Six millilitres of venous blood were drawn from each patient before operation. After overnight fast, blood sample collected in gel tube and permitted to form a clot for 30 minutes, then sample was centrifuged at (2000rpm) for ten minutes, the obtained serum was transferred to another tube (Eppendorf safe-lock tubes). Serum electrolytes were measured using ion-selective electrodes (ISE). The quantification of each electrolyte was performed under specific analytical conditions using standard solutions for each element. Additionally, human blood serum samples and a certified reference standard, prepared through a wet digestion process, were also utilized for

analysis. The electrolyte levels determined by constructing 3 (calibration curves) using (5 different con. of standard) acid solutions within the linear range for each electrolyte. The concentrations used were (0.25 mg/L), (0.50 mg/L), (1.0 mg/L), (2.0 mg/L), and (5.0 mg/L) for Sodium (Na) and Magnesium (Mg), and (0.5 mg/L), (1.0 mg/L), (2.0 mg/L), (5.0 mg/L), and (10.0 mg/L) for the other electrolytes.

4. Data Collection and Analysis

Sixty-four patients with SVT (31 female and 32 male) diagnosed by the cardiologists, depending on electrophysiology (EP).

Sixty-four healthy subjects whom don't have any problem of tachycardia or health problem with age rang (20-60 years) (30 female and 36 male).

The patients (with negative history diseases) were selected from electrophysiology unit at the private nursing hospital during March, 2021 to Sep. 2022.

Six milliliters of venous blood were drawn from each patient before operation. After overnight fast blood sample was collected in gel tube and allowed to clot for 30 minutes, then the sample was centrifuged at (2000 rpm) for ten minutes, the obtained serum was transferred to another tube (Eppendorf safe-lock tubes).

Electrolytes including Na^+ , K^+ , Ca^{2+} and Mg^+ , were determined using an atomic absorption while Cl^- , HCO_3^- and PO_4^{3-} were determined by ion-selective electrodes (ISE).

5. Research Design

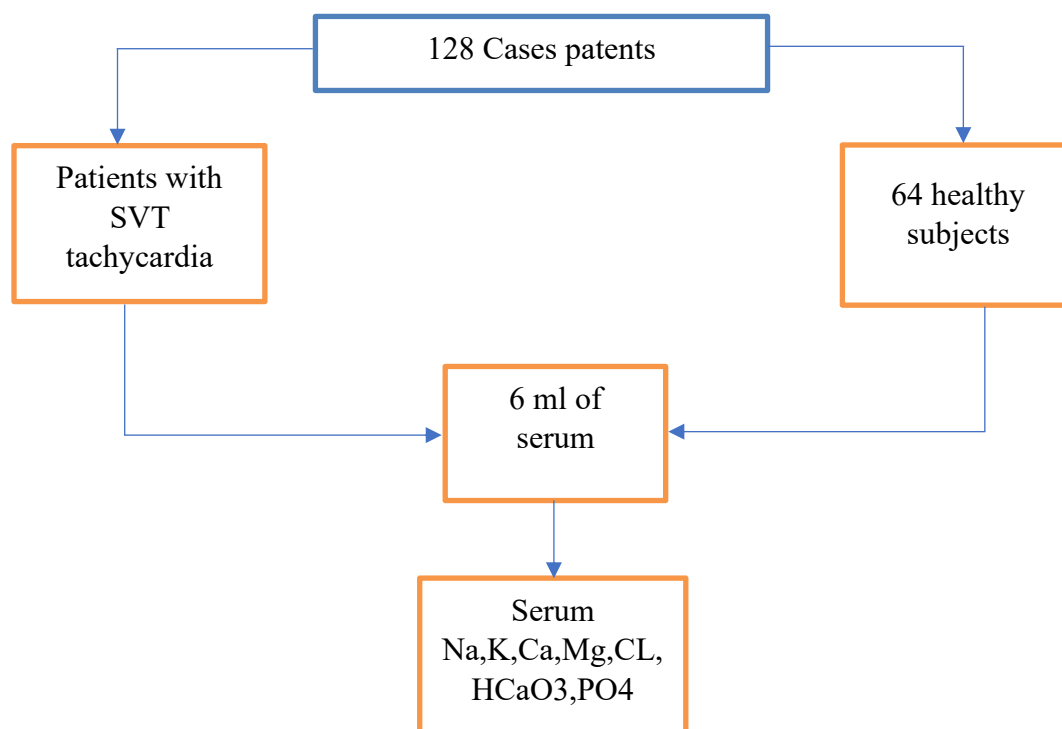


Figure 1: Research Design

6. Findings

supraventricular tachycardia patients showed a significant increase in serum of (Na^+ , Ca^{2+}) with ($P \leq 0.05$) In comparison to individuals who are in good health, while they show a significant decrease in the serum of (Mg^{2+} , PO_4^- , K^+) with ($P \leq 0.05$) when compared with healthy subjects. No significant difference was observed between the two groups ($P > 0.05$) in the sample the degree or extent of something of (Cl^- , HCO_3^-) when compared with each other's.

Table 1: Mean \pm SD of serum electrolytes in healthy subject compared to patients with cardiac arrhythmia

Electrolytes	Healthy subject mean \pm SD	Patients with Supraventricular Tachycardia mean \pm SD	t-test p-value
S. Na^+ (MEq/L)	144.31 \pm 6.49	151.98 \pm 7.47	$P \leq 0.05$
S. K^+ (mg/dL)	4.18 \pm 0.608	3.87 \pm 0.760	$P \leq 0.05$
S. Ca^{2+} (mg/dL)	8.428 \pm 0.75	8.72 \pm 1.02	$P \leq 0.05$
S. mg^{2+} (mg/dL)	1.921 \pm 0.395	1.139 \pm 0.151	$P \leq 0.05$
S. CL^- (meq/L)	102.45 \pm 3.58	113.25 \pm 7.42	$P > 0.05$
S. PO_4^- (mg/dL)	3.698 \pm 0.738	3.207 \pm 0.713	$P \leq 0.05$
S. HCO_3^- (mmol/L)	24.83 \pm 2.67	23.85 \pm 3.65	$P > 0.05$

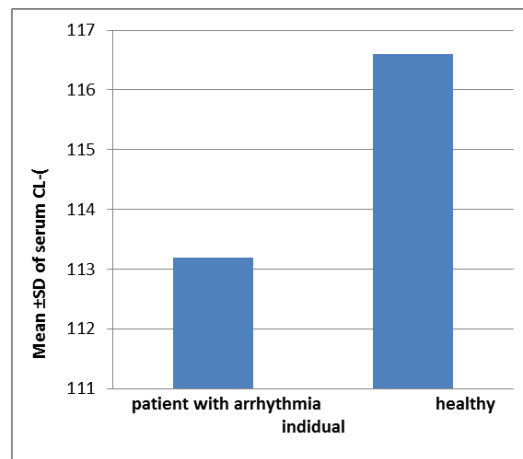


Figure 2: Shows the measurement of serum Chloride in patient with arrhythmia and healthy individual

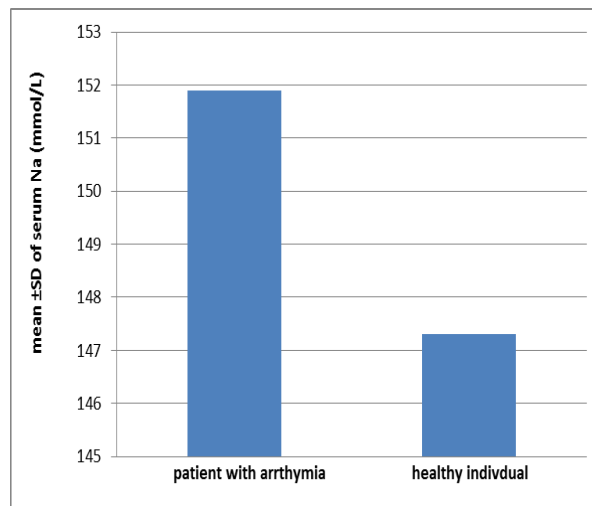


Figure 3: Show the measurement of serum sodium in patient with arrhythmia and healthy individual.

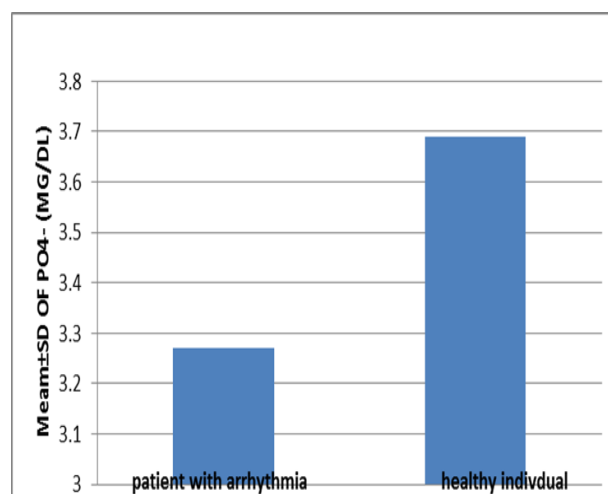


Figure 4: Shows the measurement of phosphate in patient with arrhythmia and healthy individual.

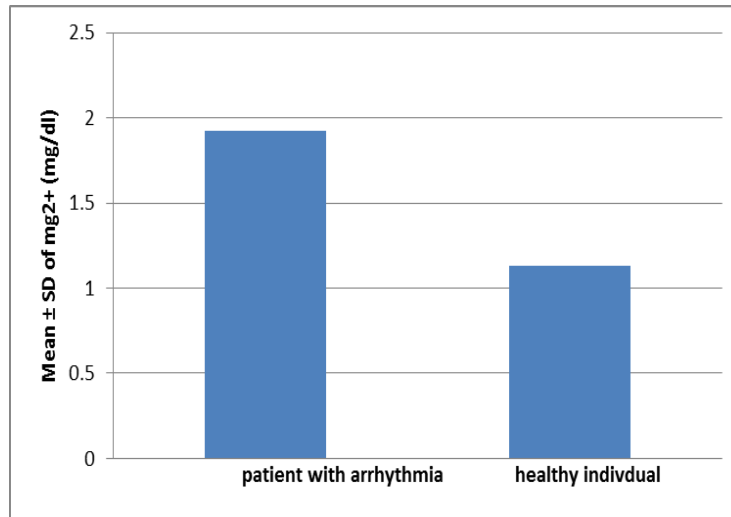


Figure 5: Show the measurement of magnesium in patient with arrhythmia and healthy individual.

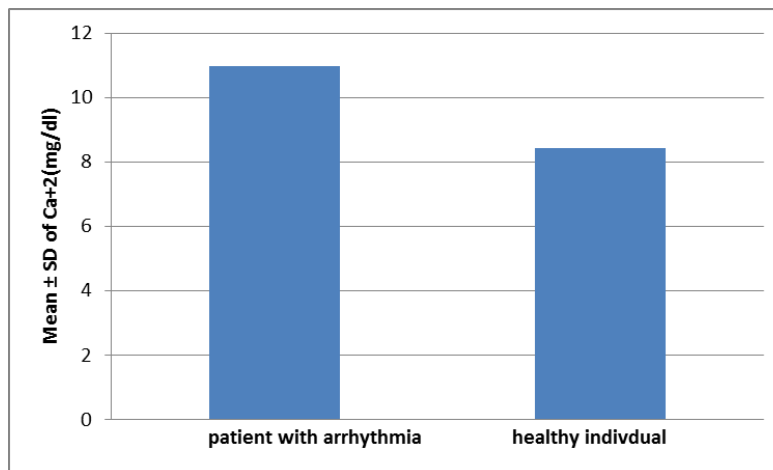


Figure 6: Show the measurement of calcium in patient with arrhythmia and healthy individual.

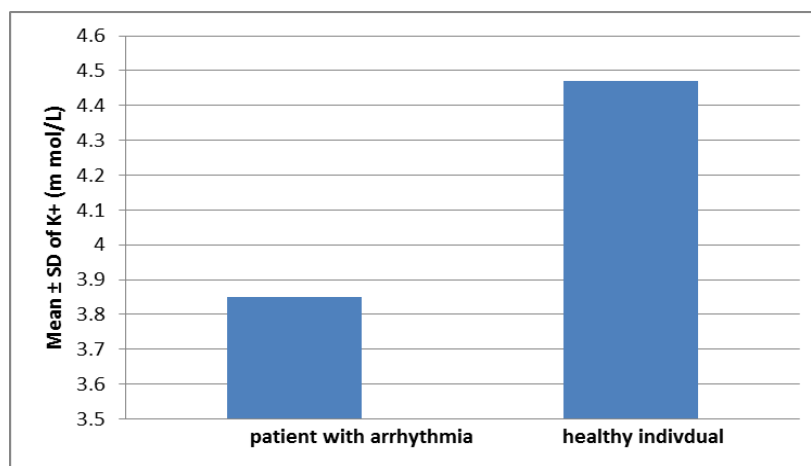


Figure 7: Show the measurement of potassium in patient with arrhythmia and healthy individual.

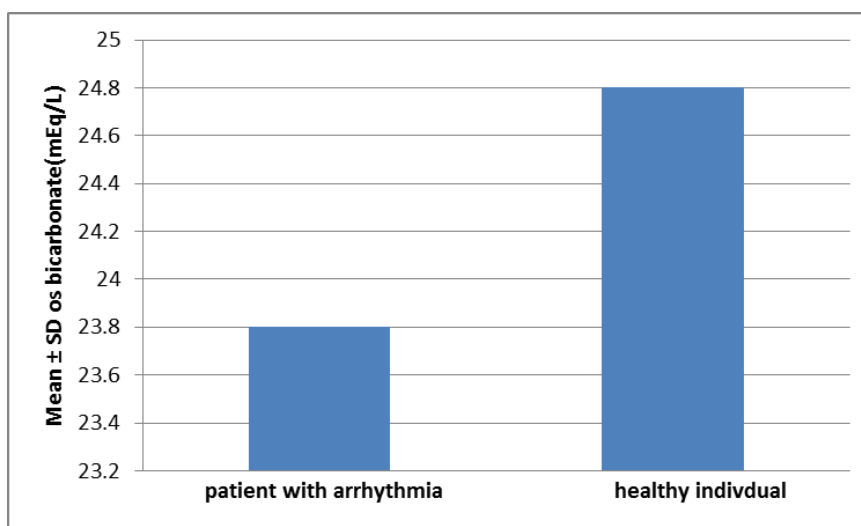


Figure 8: Show the measurement of bicarbonate in patient with arrhythmia and healthy individual.

7. Discussion and Conclusion

Study's results showed disturbances with hypernatremia and hypokalemia in patient with supraventricular tachycardia, that an irregular heartbeat can cause the heart to Facilitate the flow of blood inefficiently causing to poor blood circulation in the body (Palmer & Schnermann, 2015). Sodium is the Primary positively charged ion found in the extracellular fluid. Its movement across cell membranes plays a critical role in initiating depolarization and influencing an amplitude of action potentials (Viera & Wouk, 2015). In a study examining the impact of sudden changes in blood sodium levels on electrocardiogram (ECG) patterns, the rapid development of hypernatremia resulted in prolonged QT waves and a Dampening of the amplitude of (P) waves and (QRS) complexes. Similar ECG changes were observed in our patient (Wang et al., 2016). The decrease in the amplitude of P waves and QRS complexes was associated with changes in blood conductivity, leading to a short-circuiting effect on myocardial potential (Al-Alwany, 2015). The prolongation of the QT interval indicates an elongated duration of the action potential, which is caused by elevated extracellular sodium levels leading to an increase in the transmembrane gradient (Catterall, 2018). The results showed a significant decrease in potassium level, this is in line with the result of the hypernatremia as it regulated by aldosterone as normal response (Wisedchaisri, 2019). Study's results showed significant decreased in level of potassium this is in line with the result of the hypernatremia as it regulated by aldosterone as normal response (Catterall et al., 2017). Studies suggest that deviations towards both low potassium levels (hypokalemia) and high K levels (hyperkalemia) are Correlated with an elevated risk of tachycardia Specifically, potassium levels below 3.0 mmol/L can lead to significant prolongation of the QT interval, which carries a subsequent Probability of developing a particular form of tachycardia known as torsade des pointes (Lenaeus, 2017). Potassium plays a key role in regulating heart rate. if levels are too low lead to heart arrhythmia (Jiang, 2020). Study suggests that potassium help arrhythmia including enough potassium-rich food in daily diet is important for a steady heart rhythm (Gamal El-Din et al., 2019). Individuals diagnosed with supraventricular tachycardia (SVT) exhibit notable alterations in their calcium (Ca²⁺) levels.

Impaired function of the sarcoplasmic reticulum (SR), a vital component in cardiac myocyte contraction, is a prominent feature observed in cases of human heart failure. This impaired SR function is believed to be, at least partially, a manifestation of the diminished calcium cycling within the cardiac myocyte (Jiang, 2020). In the context of failing human hearts, there is a reduced uptake of Ca into the (SR) caused by decrease the levels of sarco(endo)plasmic reticulum Ca ATPase (SERCA) relative to its inhibitory protein, phospholamban (PLB) (Balk et al., 2017). (Ca²⁺) ions Facilitate different types of cardiac arrhythmias Expanding inventory of inherited gene abnormalities, can cause potentially lethal cardiac arrhythmia syndrome as Incorporating conditions such as catecholaminergic polymorphic ventricular tachycardia, congenital long QT syndrome, and hypertrophic cardiomyopathy (De Lucia et al., 2018). Calcium ions interact with and activate additional calcium channels situated on (SR) in cardiac cells. This activation facilitates the release of Ca from (SR), leading to contraction of heart (Feridooni et al., 2015). Patients with supraventricular have a significant decrease in there serum mg⁺² levels, hypomagnesemia is known to cause cardiac arrhythmia, increased digitals sensitivity, (ECG) changes Incorporating, widening (QRS) , prolonged PR/QP intervals, T wave change (Hamilton & Terentyev, 2018). This is agreement with research showed that Mg⁺² deficiencies or restricted magnesium causes arrhythmia by other words research Studies have indicated a potential connection between inadequate blood magnesium levels and an increased susceptibility to cardiovascular disease (Eisner et al., 2017). Another possible explained, that in the gut, Ca and Mg intake influence each other's absorption, a high Ca intake may decrease, and a low Mg intake may increase Ca absorption (Boyden & Smith, 2018). The serum phosphorus shows a significant reduction in its level in arrhythmia patients. That serum phosphorus has been linked to cardiovascular risk factor, an important cause which leading to increase pulse wave velocity increases cardiac work and decrease coronary artery blood flow (Uehara et al., 2017). Depletion of phosphorus lead to arrhythmia and elimination of ATP synthase, resulting in reversible myocardial dysfunction. A study postulated that hypophosphatemia as a cause of new cardiac arrhythmia in the intensive care population (Terentyev & Hamilton, 2016). Nevertheless, phosphorus has received less research attention. While it is recognized that low levels of phosphorus (hypophosphatemia) can disrupt myocardial contractility and diminish cardiac function, its potential to induce cardiac arrhythmia remains relatively unexplored(Hegyí et al., 2019). Patients with supraventricular tachycardia showed evaluation of their level of chloride but didn't reach the significant level, although chloride currents, such as the CAMP protein kinase a dependent current (Haghighi et al., 2014). calcium activated currents and swelling – induced currents (Scardigli et al., 2018). Although calcium ions have the ability to modify cardiac electrical activity, their precise impact on the pathogenesis of cardiac arrhythmias remains uncertain. However, it is understood that chloride currents influenced by calcium can contribute to shortening of the action potential and depolarization of the resting membrane potential. Consequently, these effects may increase the vulnerability to arrhythmias (Landstrom et al., 2017). Serum bicarbonate showed decrease in its level when compared with healthy individual, a study suggests that HCO₃⁻ administration resulted in a significant and substantial fall in atrial oxygen tension, myocardial oxygen consumption, and a systemic oxygen consumption (Ghauri et al., 2019). Low bicarbonate level in blood is a sign of metabolic acidosis, heathy kidney help keep bicarbonate level in balance (Kalani & Raphael, 2019). This may be main causes that reduction in its level didn't reach the significant level.

8. Conclusion

Disturbances in level of electrolytes were associated with supraventricular case by other words arrhythmic causes' electrolytes disruption.

9. Acknowledgements

I would like to express my thanks to "ALLAH" the most Gracious and most merciful, and to His prophet "Mohammad "blessings and peace be upon him.

I am deeply thankful to my supervisor's prof. Dr. Hedef El-yassin & Assist. prof. Dr. Ameen Al-Alwany who has faithfully worked hard to bring this work into big success. May Allah bless them for their guidance and great efforts they had done. This thesis wouldn't have been possible without their sincere help and assistance.

I am extremely grateful all staff of the cardiothoracic electrophysiology unit in medical city hospital in Baghdad –Iraq and all staff of the Toxicology laboratories in the Medical City of Baghdad- Iraq.

I would also like to thank all the sick and healthy people for their cooperation in obtaining the study samples.

References

- Ahmed, S. S., Nur, F., Ullah, R., Al-Mamun, A., Laila, T. R., & Khan, A. M. (2014). Electrolyte imbalance in admitted diabetic patients: Patterns and factors responsible. *KYAMC Journal*, 5(1), 458–466.
- Al-Alwany, A. A. H. (2015). Radiofrequency ablation of typical atrioventricular nodal reentry. *Medical Journal of Babylon*, 12(3), 745–754.
- Balk, E. M., Adam, G. P., Langberg, V. N., Earley, A., Clark, P., & Dawson-Hughes, B. (2017). Global dietary calcium intake among adults. *Osteoporosis International*, 28(12), 3315–3324.
- Bitter, T., Fox, H., & Gaddam, S. (2015). Sleep-disordered breathing and cardiac arrhythmias. *Canadian Journal of Cardiology*, 31(9), 28–34.
- Boyden, P. A., & Smith, G. L. (2018). Ca²⁺ leak—What is it? Why should we care? Can it be managed? *Heart Rhythm*, 15(4), 607–614.
- Catterall, W. A. (2018). *Voltage-gated sodium channels and electrical excitability of the heart BT - Cardiac Electrophysiology: From Cell to Bedside*, 1–11.
- Catterall, W. A., Wisedchaisri, G., & Zheng, N. (2017). The chemical basis for electrical signaling. *Nature Chemical Biology*, 13, 455–463.
- De Lucia, C., Eguchi, A., & Koch, W. J. (2018). New insights in cardiac β -adrenergic signaling during heart failure and aging. *Frontiers in Pharmacology*, 9, 904.
- Eisner, D. A., Caldwell, J. L., Kistamás, K., & Trafford, A. W. (2017). Calcium and excitation-contraction coupling in the heart. *Circulation Research*, 121(2), 181–195.
- Feridooni, H. A., Dibb, K. M., & Howlett, S. E. (2015). How cardiomyocyte excitation, calcium release and contraction become altered with age. *Journal of Cardiology*, 83, 62–72.
- Gamal El-Din, T. M., Lenaeus, M. J., Ramanadane, K., Zheng, N., & Catterall, W. A. (2019). Molecular dissection of multiphase inactivation of the bacterial sodium channel NaVAb. *Journal of General Physiology*, 151(2), 174–185.
- Ghauri, S. K., Javaeed, A., Mustafa, K. J., Podlasek, A., & Khan, A. S. (2019). Bicarbonate therapy for critically ill patients with metabolic acidosis: A systematic review. *Cureus*, 11(3), 4297.
- Haghighi, K., Bidwell, P., & Kranias, E. G. (2014). Phospholamban interactome in cardiac contractility and survival: A new vision of an old friend. *Journal of Molecular and Cellular Cardiology*, 77, 160–167.
- Hamilton, S., & Terentyev, D. (2018). Proarrhythmic remodeling of calcium homeostasis in cardiac disease; implications for diabetes and obesity. *Frontiers in Physiology*, 9, 1517.
- Hannun, A. Y., Rajpurkar, P., Haghpanahi, M., Tison, G. H., Bourn, C., Turakhia, M. P., & Ng, A. Y. (2019). Cardiologist-level arrhythmia detection and classification in ambulatory electrocardiograms using a deep neural network. *Nature Medicine*, 25(1), 65–69.
- Hegyí, B., Bers, D. M., & Bossuyt, J. (2019). CaMKII signaling in heart diseases: emerging role in diabetic cardiomyopathy. *Journal of Molecular and Cellular Cardiology*, 127, 246–259.
- Jiang, D. (2020). Structure of the cardiac sodium channel. *Cell*, 180, 122–134.
- Kalani, L., & Raphael. (2019). Metabolic acidosis in CKD: Core curriculum. *American Journal of Kidney Diseases*, 74(2), 263–275.

- Landstrom, A. P., Dobrev, D., & Wehrens, X. H. T. (2017). Calcium signaling and cardiac arrhythmias. *Circulation Research*, *120*(12), 1969–1993.
- Lenaeus, M. J. (2017). Structures of closed and open states of a voltage-gated sodium channel. *Proceedings of the National Academy of Sciences USA*, *114*, 3051–3060.
- Lip, G. Y. H., Collet, J. P., Haude, M., & Huber, K. (2018). Management of antithrombotic therapy in AF patients presenting with ACS and/or undergoing PCI. *European Heart Journal*, *39*, 2847–2850.
- Luo, J., Brunelli, S. M., Jensen, D. E., & Yang, A. (2016). Association between serum potassium and outcomes in patients with reduced kidney function. *Clinical Journal of the American Society of Nephrology*, *11*(1), 90–100.
- Page, R. L., Joglar, J. A., Caldwell, M. A., Calkins, H., Conti, J. B., Deal, B. J., Estes, N. A. M., Field, M. E., Goldberger, Z. D., Hammill, S. C., Indik, J. H., Lindsay, B. D., Olshansky, B., Russo, A. M., Shen, W. K., Tracy, C. M., & Al-Khatib, S. M. (2016). ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: Executive summary. *Journal of the American College of Cardiology*, *67*(2), 1575–1623.
- Page, R. L., Joglar, J. A., Caldwell, M. A., Calkins, H., Conti, J. B., Deal, B. J., ... & Al-Khatib, S. M. (2016). 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *Journal of the American College of Cardiology*, *67*(13), e27-e115.
- Palmer, L. G., & Schnermann, J. (2015). Integrated control of Na transport along the nephron. *Clinical Journal of the American Society of Nephrology*, *10*(4), 676–687.
- Scardigli, M., Ferrantini, C., Crocini, C., Pavone, F. S., & Sacconi, L. (2018). Interplay between sub-cellular alterations of calcium release and t-tubular defects in cardiac diseases. *Frontiers in Physiology*, *9*, 1474.
- Terentyev, D., & Hamilton, S. (2016). Regulation of sarcoplasmic reticulum Ca²⁺ release by serine-threonine phosphatases in the heart. *Journal of Molecular and Cellular Cardiology*, *101*, 156–164.
- Uehara, A., Murayama, T., Yasukochi, M., Fill, M., Horie, M., Okamoto, T., Matsuura, Y., Uehara, K., Fujimoto, T., & Sakurai, T. (2017). Extensive Ca²⁺ leak through K4750Q cardiac ryanodine receptors caused by cytosolic and luminal Ca²⁺ hypersensitivity. *Journal of General Physiology*, *149*(2), 199–218.
- Viera, A. J., & Wouk, N. (2015). Potassium disorders: Hypokalemia and hyperkalemia. *American Family Physician*, *92*(6), 487–495.
- Wang, P., Huang, G., Tam, N., Wu, C., Fu, S., Hughes, B. P., Wu, L., & He, X. (2016). Influence of preoperative sodium concentration on outcome of patients with HBV cirrhosis after liver transplantation. *European Journal of Gastroenterology & Hepatology*, *28*(10), 1210–1215.
- Wisedchaisri, G. (2019). Resting-state structure and gating mechanism of voltage-gated sodium channel. *Cell*, *178*, 993–1003.