e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(8); 1560-1563

Original Research Article

Relationship between Serum Electrolytes and ECG Alterations Pre- and Post-Exercise

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Received: 01-05-2025 / Revised: 15-06-2025 / Accepted: 21-07-2025

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Conflict of interest: Nil

Abstract

Background: Exercise induces several biochemical and electrophysiological changes that reflect the cardiovascular and muscular adjustments of the body. Monitoring these shifts helps assess physiological adaptation and cardiac safety.

Material and Methods: A study on 60 participants compared serum sodium, potassium, calcium, and magnesium levels along with ECG parameters before and after exercise using paired analysis.

Results: Statistically significant increases in all electrolytes and various ECG parameters including heart rate, P wave, QRS complex voltage, ST segment, RR interval, and QTc interval were observed post-exercise.

Conclusion: Exercise elicits measurable changes in both biochemical and ECG parameters that are essential to consider in fitness assessments and cardiovascular risk evaluation.

Keywords: Electrolytes, Exercise, ECG Changes, QTc Interval.

Introduction

Electrolytes such as sodium (Na^+), potassium (K^+), calcium (Ca^{2+}), and magnesium (Mg^{2+}) are vital to cardiac excitability and conduction, and their concentrations are tightly regulated to maintain electrocardiographic stability [1]. Physical exercise induces dynamic shifts in fluid and ion distribution, which can transiently alter serum electrolyte levels and affect ECG parameters even in healthy individuals [2]. A prospective study evaluating pre- and post-exercise serum concentrations revealed statistically significant changes in Na^+, K^+, Ca^{2+}, and Mg^{2+}, suggesting that acute physical activity impacts electrolyte homeostasis [3].

Potassium dynamics are particularly critical: even modest hyper- or hypokalemia can produce ECG changes, including peaked T waves, PR prolongation, and QRS widening, which may predispose individuals to arrhythmias if not monitored [4]. Magnesium, often considered the "forgotten electrolyte," stabilizes the myocardial cell membrane, and hypomagnesemia is associated with QT prolongation and ventricular arrhythmias [5]. Serum calcium influences the action potential plateau phase, with hypocalcemia causing QT prolongation and hypercalcemia shortening it [6]. Furthermore, exercise-related sweating and increased renal excretion may contribute to sodium and potassium losses, prompting compensatory shifts in serum calcium and magnesium, which can be reflected in subtle ECG changes like ST-segment variations and heart rate variability [7][8]. Advanced AI-driven approaches are also emerging, linking ECG waveform characteristics to real-time estimates of electrolyte levels—most notably predicting serum potassium with high accuracy during dynamic physiologic states such as exercise [9].

Despite these insights, most literature focuses on single electrolyte–ECG correlations or clinical populations, with limited integrated analysis of multiple electrolytes and their electrocardiographic repercussions in the context of exertion. This study addresses that gap by simultaneously assessing serum Na^+, K^+, Ca^{2+}, and Mg^{2+} levels before and after exercise, and correlating them with ECG changes to better understand physiologic electro physiologic adaptations.

Material and Methods

This observational study was conducted over a defined period within a controlled clinical setting to evaluate the correlation between serum electrolyte

levels and electrocardiographic (ECG) changes before and after exercise. A total of 60 healthy adult participants were enrolled in the study after obtaining informed written consent. The inclusion criteria involved participants aged 18–45 years, free from known cardiovascular, renal, or metabolic diseases, and not on any chronic medications known to affect electrolyte balance or ECG parameters. Individuals with a history of electrolyte imbalance, endocrine disorders, or abnormal baseline ECG were excluded from the study.

Each subject underwent a thorough clinical evaluation, including demographic data collection, anthropometric measurements, and baseline vitals. Blood samples were collected after 12 hours of fasting to assess baseline serum concentrations of sodium (Na^+), potassium (K^+), calcium (Ca^{2+}), and magnesium (Mg^{2+}) using standardized biochemical methods. A 12-lead ECG was also recorded at baseline using a calibrated digital electrocardiograph to evaluate heart rate, rhythm, PR interval, QRS duration, QT interval, and ST segment morphology.

Following this, all participants were subjected to a standardized exercise protocol on a treadmill according to the modified Bruce protocol.

The duration and intensity of exercise were individualized to ensure that each participant achieved at least 85% of their predicted maximum heart rate, confirmed through real-time monitoring.

Immediately after cessation of exercise, a second blood sample was drawn within 5–10 minutes and another 12-lead ECG was performed under identical conditions.

All data were compiled and analyzed to evaluate the changes in serum electrolyte levels and ECG parameters pre- and post-exercise. Statistical analysis was carried out using SPSS software version XX. Paired t-tests were applied to compare pre- and post-exercise values of electrolytes and ECG parameters. Correlation coefficients were calculated to assess the degree of association between changes in electrolytes and changes in ECG features. A p-value of less than 0.05 was considered statistically significant.

Results

In Table 1, the comparison of serum electrolytes before and after exercise in 60 participants revealed statistically significant changes across all parameters. The sodium levels showed a slight but consistent rise from 141 ± 3.10 mmol/L to 142 ± 3.51 mmol/L after exercise, with a highly significant p-value (<0.001), indicating a meaningful shift. Potassium levels also increased significantly, moving from 4.1 ± 0.32 to 4.15 ± 0.29 mmol/L ($p < 0.001$), suggesting muscle activity may influence potassium mobilization. Similarly, magnesium levels rose from 1.84 ± 0.09 to 1.87 ± 0.10 mmol/L ($p < 0.001$), while calcium increased marginally from 4.58 ± 0.23 to 4.60 ± 0.24 mmol/L ($p < 0.001$), reinforcing the subtle yet important biochemical effects of physical exertion.

Table 2 highlights the electrocardiographic responses to exercise. Heart rate significantly increased from a resting mean of 80.10 ± 10.80 bpm to 104.10 ± 12.54 bpm post-exercise ($p < 0.001$), reflecting expected sympathetic activation. The P wave voltage showed a meaningful rise from 0.14 ± 0.04 to 0.16 ± 0.04 mV ($p < 0.001$), whereas the increase in P wave duration from 0.08 ± 0.02 to 0.11 ± 0.13 ms was not statistically significant ($p = 0.108$). The PR interval remained unchanged at 0.14 ms, indicating no conduction delay ($p = 0.960$). QRS complex voltage rose significantly from 1.09 ± 0.29 to 1.14 ± 0.32 mV ($p < 0.05$), though the duration shift was not significant ($p = 0.106$). Notably, both ST segment voltage and duration increased significantly ($p < 0.001$), demonstrating the heart's repolarization response to exertion. The RR interval shortened significantly ($p < 0.001$), corresponding with increased heart rate. The QR interval showed a significant reduction from 0.35 to 0.32 ms ($p < 0.001$), and QTc interval rose slightly from 0.41 to 0.42 ms ($p < 0.001$), indicating adaptation of ventricular repolarization to increased physical demand.

Table 1: Various Parameters in Pre and Post Exercise Group (n = 60)

Parameter	Exercise Group	Mean ± SD	p-Value
Sodium	Pre	141 ± 3.10	<0.001
	Post	142 ± 3.51	
Potassium	Pre	4.1 ± 0.32	<0.001
	Post	4.15 ± 0.29	
Magnesium	Pre	1.84 ± 0.09	<0.001
	Post	1.87 ± 0.10	
Calcium	Pre	4.58 ± 0.23	<0.001
	Post	4.60 ± 0.24	

Table 2: ECG Parameters in Pre and Post Exercise Group (n = 60)

Parameter	Phase	Mean ± SD	p-Value
Heart Rate (bpm)	Pre	80.10 ± 10.80	<0.001
	Post	104.10 ± 12.54	
P Wave Voltage (mV)	Pre	0.14 ± 0.04	<0.001
	Post	0.16 ± 0.04	
P Wave Duration (ms)	Pre	0.08 ± 0.02	0.108 (NS)
	Post	0.11 ± 0.13	
PR Interval (ms)	Pre	0.14 ± 0.02	0.960 (NS)
	Post	0.14 ± 0.01	
QRS Voltage (mV)	Pre	1.09 ± 0.29	<0.05
	Post	1.14 ± 0.32	
QRS Duration (ms)	Pre	0.10 ± 0.13	0.106 (NS)
	Post	0.073 ± 0.01	
ST Segment Voltage (mV)	Pre	0.01 ± 0.04	<0.001
	Post	0.04 ± 0.04	
ST Segment Duration (ms)	Pre	0.09 ± 0.02	<0.001
	Post	0.09 ± 0.02	
RR Interval (ms)	Pre	0.14 ± 0.02	<0.001
	Post	0.10 ± 0.07	
QR Interval (ms)	Pre	0.35 ± 0.01	<0.001
	Post	0.32 ± 0.01	
QTc Interval (ms)	Pre	0.41 ± 0.01	<0.001
	Post	0.42 ± 0.01	

Discussion

The observed changes in serum electrolytes and ECG parameters before and after exercise reflect the dynamic physiological responses associated with physical exertion. The statistically significant rise in serum sodium, potassium, magnesium, and calcium levels post-exercise in this study aligns with known mechanisms of electrolyte regulation. These shifts are primarily attributed to fluid shifts, hormonal influences, and muscle activity during exertion. Sodium and potassium are critical for action potential propagation in excitable tissues, and even small variations can substantially influence cardiac electrophysiology. Consistent with this, our findings of increased potassium post-exercise corroborate earlier reports indicating transient hyperkalemia during physical activity, largely due to potassium efflux from contracting muscles [11].

The increase in magnesium and calcium may be secondary to hemoconcentration and increased neuromuscular demand. Magnesium acts as a co-factor for several enzymatic reactions and helps stabilize cardiac rhythm, while calcium plays a central role in excitation-contraction coupling. Literature indicates that prolonged or intense exercise can modulate these ions, and their homeostasis is essential to avoid arrhythmias or muscle fatigue [12]. The current results fall within expected physiological norms, reinforcing that moderate exercise induces measurable but safe changes in electrolyte profiles. From an electrocardiographic standpoint, increased heart rate and shortened RR intervals reflect heightened sympathetic tone, a well-documented autonomic response to exercise. The ST segment showed statistically significant elevation in both voltage and duration, supporting previous observations that exercise can induce early repolarization patterns, particularly in healthy individuals [13]. This physiological ST elevation must be distinguished from pathological patterns to avoid overdiagnosis of myocardial ischemia in clinical settings. Interestingly, the QTc interval showed a modest but statistically significant increase post-exercise. QTc prolongation post-exercise can be benign in healthy individuals; however, excessive prolongation is a known risk factor for ventricular arrhythmias. Thus, understanding these trends is vital for identifying individuals with potential underlying repolarization abnormalities [14]. The increase in QRS complex voltage and P wave voltage suggests augmented cardiac output and atrial depolarization demands, respectively, as previously documented in exercise physiology studies [15]. Overall, this study emphasizes the importance of monitoring both electrolyte changes and ECG responses during exercise. These parameters not only provide insight into the physiological adaptations to physical activity but also serve as crucial indicators for identifying at-risk individuals, especially in clinical exercise testing or cardiac rehabilitation programs.

Conclusion

This study demonstrated that exercise leads to statistically significant changes in serum electrolytes and ECG parameters in healthy

individuals. The elevations in sodium, potassium, magnesium, and calcium levels post-exercise coincide with physiological adaptations to increased muscular and cardiovascular demands. Corresponding ECG changes, including increased heart rate, altered P wave and ST segment characteristics, and prolonged QTc, reflect cardiac autonomic and repolarization responses. Monitoring these biochemical and electrical shifts can aid in early detection of abnormal patterns and guide safe exercise prescriptions in clinical settings.

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