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Research Article

ECG Based Serum Potassium Estimation

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Abstract

Potassium is the most commonly found cation inside the human body and is more concentrated intracellularly than in extracellular components. The homeostasis of Potassium in the blood is maintained by dietary intake and excretion through urine, stool and sweat. In between this, it is primarily stored in muscles and bones. When the blood levels of Potassium start to fluctuate, its effects are found all over the body including skeletal, gastrointestinal and cardiac muscles. However, blood reports usually detect the serum level changes quite late, the process being time consuming, minimally invasive and requires the expertise of phlebotomy every time. We have identified a method of estimating Serum Potassium levels from 12-lead ECGs. Every 0.2mV (2 small squares) increase in T-wave height in a 12-lead ECG corresponded with approximately 0.7mEq/L increase in Serum Potassium levels; conversely 0.2mV (2 small squares) decrease in T-wave height corresponded with approximately 0.7mEq/L decrease in Serum Potassium levels. These equations were tested with 695 and 611 patients respectively, showing a p value of <.00001 (significance interval = 0.01) in both cases. This method of calculating Serum Potassium levels from T-wave heights in ECGs is not only extremely accurate but also non-invasive and less time-consuming. Utilizations of these equations can be most useful in cardiac monitored beds (eg. ICU, CCU, HDU etc) where Potassium levels can be continuously displayed without any extra hardware, any expertise or any invasive procedures. This can help clinicians assess very subtle changes in Serum Potassium levels as and when they occur.

Keywords: Potassium; fluctuate; phlebotomy; repolarization

Background

Potassium is the most abundant cation found in the human body with concentration levels of approximately 55mEq/Kg body weight [1]. A major portion of this Potassium is found in intracellular compartments (most generously in muscle and bone cells) with concentrations of approximately 150 mmol/L while the concentration of potassium in the extracellular fluid is strictly maintained between 3.5 and 5.5 mmol/L [2]. The exclusive source of Potassium is dietary (approximately 100mEq per day) and counterbalanced by excretion through urine (90%), stool and sweat (10%) [3]. Though the homeostasis of Serum Potassium is maintained heavily by the kidneys [4], the effects of its fluctuations can be felt across the body, most commonly affected tissues being skeletal, cardiac and gastrointestinal muscles [5]. One of the reasons for these widespread effects to Potassium level fluctuations is its involvement as the main ion responsible for the repolarization phase of action potentials in all cells of the body [6].

ECG involvement

Due to the effects of Potassium fluctuations in the cardiac muscles, widespread ECG changes are often noted in both Hypokalemia and Hyperkalemia patients. These changes include (Table 1).

Table 1:

ECG changes in Hypokalemia	ECG changes in Hyperkalemia
Increased P wave amplitude	Peaked T waves
Prolongation of PR interval	P wave widening/flattening, PR prolongation
Widespread ST depression and T waveflattening/inversion	Bradyarrhythmia's: sinus bradycardia, high-grade AV block with slow junctional and ventricular escape rhythms, slow AF
Prominent U waves (best seen in the precordialleads V2-V3)	Conduction blocks (bundle branch block,fascicular blocks)
Apparent long QT interval due to fusion of T andU waves	QRS widening with bizarre QRS morphology

Problem Statement

However, these changes only occur after the levels of Serum Potassium have gone up or down, there is no indication to predict when these changes will occur and by extension when and how much of variations in Serum Potassium levels the patient will suffer. Even when the patients start experiencing symptoms, blood tests often take too long to report the fluctuations.

Aim

The aim of this paper is to present a method of Serum Potassium level estimation from ECG tracings.

Methods

A multi-centric clinical trial was carried out across 4 hospitals in India with a total patient count of 1306.

Inclusion Criteria

a) Adult patients above 14 years of age.

b) 611 patients of Hyperkalemia.

c) 695 patients of Hypokalemia.

d) In-patient admissions (for ease of follow-ups).

Exclusion Criteria

a) Patients below 14 years of age.

b) Patients without pathologies known to cause Hyperkalemia or Hypokalemia.

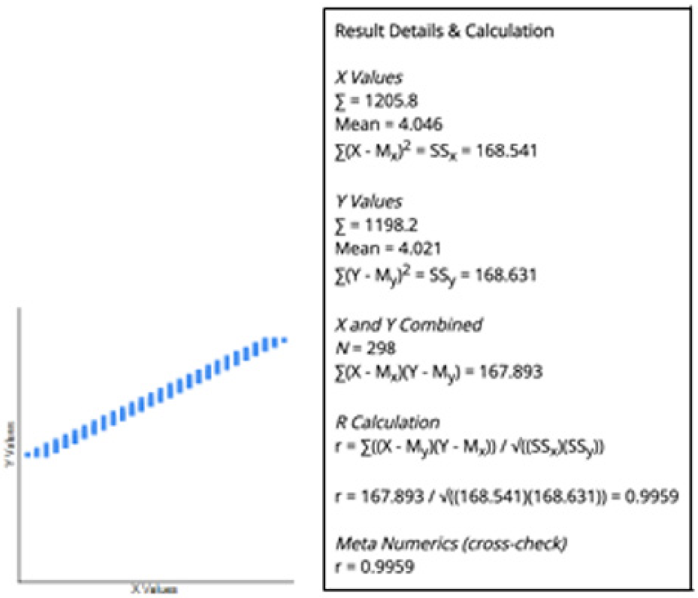
c) Patients treated in outpatient departments.

For each patient, we reported blood tests for Serum Potassium and 12-lead ECGs for each patient during admission and after their Potassium levels have stabilised.

Results

Rising Potassium levels

For a rising Serum Potassium (patients being treated for Hypokalemia), every 0.2mV (2 small squares) increase in T-wave height in a 12-lead ECG corresponded with approximately 0.7mEq/L increase in Serum Potassium levels. From this equation, once the initial Potassium level: T-wave proportion was mapped, we were able to calculate the patients' Potassium levels and verify it with the blood reports later (Figure 1).



From this, we calculate

a) Value of R = 0.9959.

b) P-Value is < .00001 (significance interval = 0.01).

Figure 1: We again plotted the values with Serum Potassium levels from blood tests being the X values and Serum Potassium estimation from 12-lead ECGs being Y values and used Pearson Correlation Coefficient to judge the accuracy of this theory.

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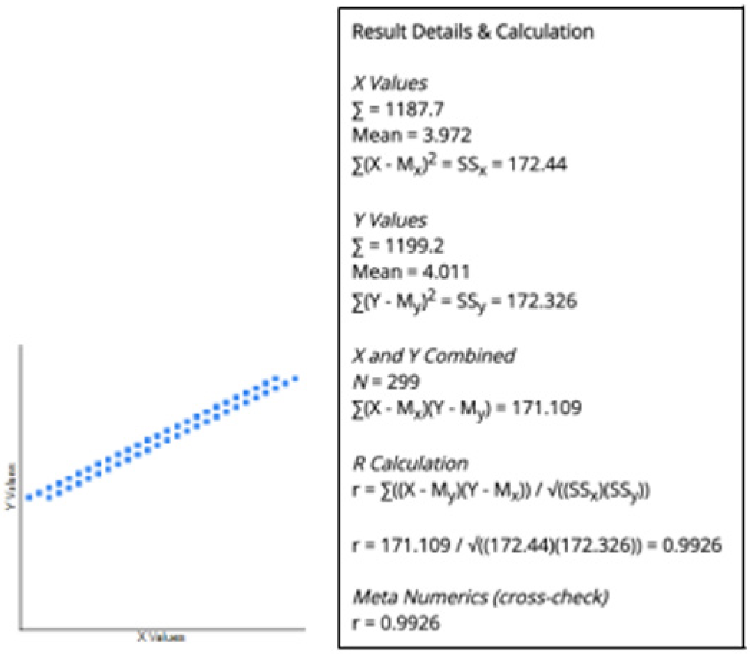
Falling Potassium levels

Again, for a reducing Serum Potassium (patients being treated for Hyperkalemia), 0.2mV (2 small squares) decrease in T-wave height corresponded with approximately 0.7mEq/L decrease in Serum Potassium levels. Here also we mapped the initial Potassium level: T-wave proportion and calculated the patients' Potassium levels and verified it with the blood reports (Figure 2).



Here, we calculate

a) Value of R = 0.9926.

b) P-Value is < .00001 (significance interval = 0.01).

Figure 2: Similar to the previous section, here also we plotted the values with Serum Potassium levels from blood tests being the X values and Serum Potassium estimation from 12-lead ECGs being Y values and used Pearson Correlation Coefficient to judge the accuracy of this theory.

Conclusion and Discussion

From the above calculations it is very obvious that the method of calculating Serum Potassium levels from T-wave heights in ECGs is not only extremely accurate but also non-invasive and less time-consuming. Utilizations of these equations can be most useful in cardiac monitored beds (e.g. ICU, CCU, HDU etc.) where Potassium levels can be continuously displayed without any extra hardware, any expertise or any invasive procedures. This can help clinicians assess very subtle changes in Serum Potassium levels as and when they occur.

Acknowledgement

None.

Conflict of Interest

No conflict of Interest.

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