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ORIGINAL ARTICLES

A Physiological Model of the Effect of Hypoglycemia on Plasma Potassium

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Abstract

Background:

Adrenaline release and excess insulin during hypoglycemia stimulate the uptake of potassium from the bloodstream, causing low plasma potassium (hypokalemia). Hypokalemia has a profound effect on the heart and is associated with an increased risk of malignant cardiac arrhythmias. It is the aim of this study to develop a physiological model of potassium changes during hypoglycemia to better understand the effect of hypoglycemia on plasma potassium.

Method:

Potassium counterregulation to hypokalemia was modeled as a linear function dependent on the absolute potassium level. An insulin-induced uptake of potassium was modeled using a negative exponential function, and an adrenaline-induced uptake of potassium was modeled as a linear function. Functional expressions for the three components were found using published data.

Results:

The performance of the model was evaluated by simulating plasma potassium from three published studies. Simulations were done using measured levels of adrenaline and insulin. The mean root mean squared error (RMSE) of simulating plasma potassium from the three studies was 0.09 mmol/liter, and the mean normalized RMSE was 14%. The mean difference between nadirs in simulated and measured potassium was 0.12 mmol/liter.

Conclusions:

The presented model simulated plasma potassium with good accuracy in a wide range of clinical settings. The limited number of hypoglycemic episodes in the test set necessitates further tests to substantiate the ability of the model to simulate potassium during hypoglycemia. In conclusion, the model is a good first step toward better understanding of changes in plasma potassium during hypoglycemia.

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Abbreviations: (DIN) difference in nadir, (NRMSE) normalized root mean squared error, (OGTT) oral glucose tolerance test, (RMSE) root mean squared error

Keywords: diabetes, hypokalemia, physiological model, potassium

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