

Mapping Simulated ECG to Early Acute Kidney Injury Based on Electrolyte Disturbances

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Introduction: Acute kidney injury (AKI) leads to imbalances in blood K^+ and Ca^{2+} levels, affecting cardiac electrophysiology and potentially causing arrhythmia. However, the specific ECG features associated with early-stage AKI development remain unclear. There is a lack of non-invasive biomarkers for warning of cardiac injury during the initial phases of AKI.

Methods: Serum K^+ and Ca^{2+} levels in mice were measured at 1, 3, 6, and 12 hours post-AKI onset. These electrolyte values were input as parameters into cardiac computational model to simulate the ECG during the early stages of AKI. A one-dimensional transmural ventricular model of the mouse was constructed, incorporating endocardial, middle, and epicardial layers. The middle layer cell model was designed to simulate the long plateau phase, consistent with previous experimental observations. The ECG of the fiber model was simulated by superimposing electrical signals of these three layers in one heartbeat.

Results: Experimental results show that serum K^+ and Ca^{2+} levels decrease initially in 1-hour post-AKI, followed by a continuous increase over the next 12 hours. In silico study, low serum K^+ leads to hyperpolarization, resulting in increased R and S wave amplitudes during the 1-hour post-AKI phase. In the 12-hour post-AKI phase, high serum K^+ causes electrical conduction delay and depolarization of the resting voltage, manifesting as prolonged QRS duration and decreased R and S wave amplitudes. The T wave peak, corresponding to the action potential duration plateau phase, is delayed.

Conclusion: Electrolyte alterations within 12 hours of AKI onset induce observable changes in the ECG, demonstrating that ECG may serve as a valuable non-invasive clinical biomarker for identifying cardiac injury in the early stages of AKI.

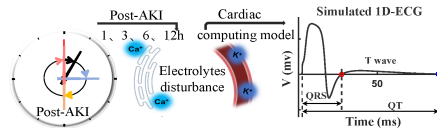


Figure 1. Measuring both serum $[K^+]_o$ and $[Ca^{2+}]_o$ to input cardiac model and simulate ECG in early AKI.

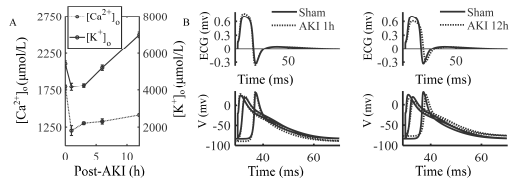


Figure 2. Electrolytes (A), Simulated ECG and transmembrane potential (B) in post AKI