

# Non-Invasive Characterization of Atrio-Ventricular Properties During Atrial Fibrillation

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The atrio-ventricular (AV) node is the primary regulator of ventricular rhythm during atrial fibrillation (AF). Hence, ECG based characterization of AV node properties can be an important tool used to monitor and predict the effect of rate control drugs. In this work we present a network model of the AV node, together with an associated workflow for robust estimation of the model parameters from ECG.

The model consists of interacting nodes with separate refractory periods and conduction delays determined by the stimulation history of each node. It has a fast and a slow pathway, interconnected at their last nodes. The refractory period and conduction delay for the nodes in each pathway are described by the minimum values,  $R_{\min}$  and  $D_{\min}$ , the maximum prolongations,  $\Delta R$  and  $\Delta D$ , and two time constants. We also present a workflow utilizing a problem specific genetic algorithm with a fitness function based on the Poincare plot, accounting for dynamics in the data.

The robustness of the parameter estimates was evaluated using simulated data, based on clinical measurements from five AF patients. Further, the methodology was applied to analyzing ECG data from one patient at baseline and during treatment with diltiazem (Fig. 1). Using the simulated data, it was shown that the proposed workflow allowed estimating  $R_{\min}$  and  $\Delta R$  for the slow pathway with an error (mean  $\pm$  std) of  $20 \pm 5$  ms and  $29 \pm 5$  ms respective, and  $D_{\min}$  and  $\Delta D$  with an error of  $2 \pm 7$  ms and  $2 \pm 7$  ms. Corresponding results for the fast pathway were  $48 \pm 12$  ms,  $77 \pm 12$  ms, and  $3 \pm 9$  ms,  $5 \pm 6$  ms. Together, these results suggest that both AV refractoriness and conduction delay can be assessed from ECG during AF with enough precision and robustness to detect effects from different rate control drugs.

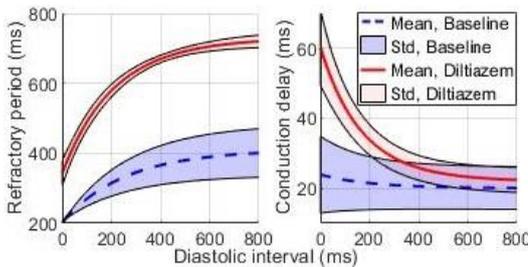


Figure 1. Slow pathway refractory period (left) and conduction delay (right) during baseline and treatment with diltiazem. Note that the total slow pathway conduction delay is the sum of the delay in 10 nodes.