

Detectability of Regional Ventricular Conduction Slowing from ECGs under Baseline Activation Variability

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Aims: ECG manifestations of localised ventricular conduction slowing may depend on the location, extent, and severity of slowing, and on the underlying baseline activation properties. We aimed to quantify the detectability of regional mid-wall slowing from QRS morphology in representative healthy ventricular models under baseline activation variability.

Methods: Sinus-rhythm activation was simulated in three representative healthy ventricular models using a pure Eikonal model. Three coordinate-defined mid-wall regions (septal-mid, anterior-mid, anterior-apical) were perturbed using calibrated ellipsoidal extents (small, medium, large) and local conduction velocity scaling factors $\alpha=0.8, 0.6$, and 0.4 . Depending on region, these extents affected median myocardial fractions of 3.28–18.50% across anatomies. For each anatomy, 10 plausible healthy inferred baseline solutions were selected from populations calibrated to healthy QRS biomarker and morphology criteria (including QRS duration, axis, R/S-wave progression, transition, and notching), using deterministic farthest-point sampling on centred activation-map RMSE. Detectability was quantified using mean Pearson correlation coefficient (PCC) and worst-lead PCC between matched baseline and perturbed ECGs.

Results: Across representative anatomies and multiple plausible healthy inferred baselines, detectability depended strongly on the location, extent, and severity of slowing. Effects were strongest in anterior-mid, intermediate in anterior-apical, and weakest in septal-mid. Detectability increased monotonically with both extent and slowing severity. In anterior-mid, large perturbations showed a graded response from mean/worst-lead PCC 0.996/0.988 at $\alpha=0.8$ to 0.980/0.934 at $\alpha=0.6$ and 0.931/0.778 at $\alpha=0.4$. For large $\alpha=0.4$ perturbations, mean/worst-lead PCC were 0.931/0.778 in anterior-mid, 0.973/0.885 in anterior-apical, and 0.982/0.941 in septal-mid. Global ΔmaxLAT remained unchanged.

Conclusion: Localised mid-wall ventricular slowing can produce measurable QRS morphology changes in otherwise healthy ventricular models, and detectability depends strongly on the location, extent, and severity of slowing. These findings support evaluation across multiple plausible healthy inferred baselines rather than relying on a single inferred healthy solution.