

# Beyond Diabetes Classification: Interpreting Vectorcardiographic Repolarization Geometry as a Cardiometabolic Electrical Phenotype [Letter]

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## Dear editor

We read with great interest the study by Lv and Zhao, which investigated diabetes-related alterations in vectorcardiographic (VCG) repolarization geometry using physiological analysis and interpretable machine learning. The authors reported that spatial QRS-T angle, T-wave magnitude, systolic blood pressure, T-loop area, and body mass index were among the most influential contributors to diabetes classification. Their work is valuable because it suggests that metabolic dysfunction may be associated with subtle alterations in ventricular repolarization geometry detectable from reconstructed VCG. However, several methodological and interpretative issues should be clarified before these findings can be considered physiologically or clinically informative.<sup>1</sup>

First, the clinical meaning of the machine-learning model requires careful qualification. Diabetes is diagnosed using glycemic criteria rather than electrocardiographic features; therefore, the value of VCG-based analysis is unlikely to lie in identifying diabetes itself. A more clinically relevant interpretation is that repolarization geometry may reflect a subclinical cardiometabolic electrical phenotype. In this context, spatial QRS-T angle and T-loop morphology should not be regarded merely as classifiers of diabetes status, but as potential markers of autonomic, myocardial, and electrophysiological remodeling in patients with metabolic disease. This distinction is important because previous studies have already linked spatial QRS-T angle with type 2 diabetes, left ventricular performance, glycemic control, and cardiac autonomic neuropathy.<sup>2,3</sup> A larger QRS-T angle has also been shown to predict myocardial infarction and all-cause mortality in diabetic populations.<sup>4</sup> Thus, the novelty of the present study may reside less in diabetes classification and more in the integration of VCG geometry with interpretable machine learning.

Second, the mechanistic interpretation of altered repolarization geometry remains incomplete. Diabetes may influence ventricular repolarization through several overlapping pathways, including cardiac autonomic neuropathy, myocardial fibrosis, microvascular ischemia, oxidative stress, impaired calcium handling, reduced potassium currents, and diminished repolarization reserve. These mechanisms may lead to spatial discordance between depolarization and repolarization vectors, manifesting as widening of the QRS-T angle or changes in T-wave magnitude and T-loop area. Previous evidence has shown that diabetes is associated with prolongation and spatial dispersion of ventricular repolarization, which may contribute to greater ventricular electrical instability.<sup>5</sup> However, the present study did not include diabetes duration, HbA1c, hypoglycemic exposure, renal function, electrolyte status, cardiac autonomic testing, echocardiographic parameters, or medication data. Without these variables, it remains difficult to determine whether the observed VCG differences are driven by diabetes per se, cumulative glycemic burden, autonomic dysfunction, structural myocardial remodeling, or unmeasured cardiovascular comorbidities.

Third, clarification is needed regarding the role of spatial QRS-T angle in the machine-learning model. The methods section states that continuous QRS-T angle values were used for physiological analysis but not as predictors in the



machine-learning models. However, the results and SHAP analysis identify spatial QRS-T angle as the most influential feature. Because SHAP interpretation depends directly on the variables entered into the model, this apparent inconsistency should be resolved. If QRS-T angle was included as a predictor, the final feature set should be explicitly reported. If it was not included, the reported SHAP ranking requires correction or further explanation.

Fourth, the use of inverse Dower transformation deserves additional caution. Although reconstructed VCG from standard 12-lead ECG is practical, it is not equivalent to Frank-lead VCG. The authors appropriately acknowledge that inverse Dower transformation may underestimate absolute vector amplitudes.<sup>1</sup> This limitation is particularly relevant because magnitude-based variables, including T-wave magnitude, QRS magnitude, and loop areas, contributed to the model. Differences in thoracic impedance, body habitus, cardiac position, and myocardial mass may influence amplitude-derived features, even when average BMI appears similar between groups. Therefore, angular measurements and amplitude-derived parameters should be interpreted separately, and future studies should consider validating reconstructed VCG metrics against Frank-lead VCG or other established transformation methods.

Finally, the study design limits causal and prognostic inference. The cross-sectional nature of the analysis precludes conclusions about whether diabetes causes repolarization remodeling or whether VCG abnormalities predict future arrhythmia, heart failure, myocardial infarction, or mortality. Although the model achieved a relatively high ROC-AUC, the PR-AUC was more modest, and the absence of external validation limits generalizability.<sup>1</sup> Future work should therefore move beyond diabetes classification and test whether VCG-derived repolarization geometry improves risk stratification among patients with diabetes. Longitudinal studies incorporating HbA1c, diabetes duration, cardiac autonomic function, echocardiography or cardiac magnetic resonance, kidney function, medication exposure, and hard cardiovascular outcomes would be particularly informative.

In conclusion, Lv and Zhao's study provides an intriguing hypothesis-generating observation that diabetes is associated with altered VCG repolarization geometry. The key clinical implication, however, may not be the identification of diabetes, but the detection of a subclinical cardiometabolic electrical phenotype. Clarifying the model inputs, accounting for relevant metabolic and cardiovascular confounders, and validating these findings in longitudinal cohorts will be essential before VCG-based repolarization geometry can be considered a robust marker of metabolic cardiac involvement.

## Data Sharing Statement

Data sharing is not applicable to this communication as no data were created or analysed in this communication.

## Author Contributions

XYZ: Conceptualization, Writing – original draft, Writing – review and editing. YL: Investigation, Writing – review & editing. All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the communication; gave final approval of the version to be published; have agreed on the journal to which the communication has been submitted; and agree to be accountable for all aspects of the work.

## Funding

The authors declare that no funding was received for this communication.

## Disclosure

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this communication.

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