



The comments of Dr Chhabra and colleagues regarding the dynamic variability of PQ-segment depression (PQD) and the timing of the electrocardiograms (ECGs) are appreciated. In acute pericarditis, the PQ segment reflects the evolving pathophysiology of the disease and it changes accordingly.^{1,2} Although the exact mechanism of PQD in short QT syndrome (SQTS) is not known, one should not expect a major variability in PQD because the pathology remains unchanged in these patients. This assertion is also concordant with our observation that PQD persists with only slight deviations during follow-up. Nevertheless, the retrospective nature of the study does not allow us to accept this observation as a solid “finding.” Another concern pointed out by Dr Chhabra and colleagues was the timing of the ECG tracings. The ECG tracings were from patients diagnosed with SQTS (from the European SQTS registry),³ and none of the ECG tracings used for the analysis were taken from the immediate post-resuscitation phase. Furthermore, patients were free from antiarrhythmic drugs that may affect cardiac action potentials and thus possibly the PQ segment.

Cardiac arrest secondary to myocardial infarction may also cause PQD owing to pericarditis during acute myocardial infarction or post-resuscitation. However, cardiac arrest occurred in SQTS in the absence of acute ischemia (ST-elevation myocardial infarction and non-ST-elevation myocardial infarction were ruled out).

Another concern raised by the authors was regarding the analysis of PQD in patients with atrial fibrillation/flutter. As correctly pointed out by Dr Chhabra and colleagues, the analysis of the PQ segment is possible only in sinus rhythm. All the patients who developed atrial fibrillation—either paroxysmal, persistent, or permanent—had at least 1 ECG with sinus rhythm, enabling the analysis of PQD in these patients.

In general, we also agree that further studies are needed to better understand the underlying mechanism and clinical significance of PQD in SQTS.

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