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The Reply

We thank Dr Reiffel for his interest in our case description[1] and his comments. Indeed, the presence of precordial T-wave inversions in the setting of pulmonary embolism has been described periodically in the literature. The cases described by Dr Reiffel supplement the cases we referenced in our report to reinforce this point. Two theories are often cited as an explanation for this finding. The first is heightened sympathetic tone caused by an autonomic nervous system−mediated response triggered by acute pulmonary embolism. The other is myocardial ischemia caused by reduced cardiac output in the setting of acute right ventricular dilatation.[2] Although these theories have been postulated, the mechanism remains unclear. Dr Reiffel's observations regarding QT prolongation and the T peak to T end (TpTe) interval are instructive. Taken together, T-wave inversion, QT prolongation, and TpTe interval prolongation all have diagnostic and prognostic implications in the evaluation of pulmonary embolism, as highlighted in this case. Furthermore, these are a few among many electrocardiographic findings that have been associated with pulmonary embolism, including but not limited to ST depression, ST elevation, atrial tachyarrhythmias, peaked p waves, low voltage, QT dispersion, and QRS fragmentation.[3] Given the heterogeneity in the electrocardiographic manifestations of pulmonary embolism, we highlight the importance of careful electrocardiogram analysis in patients in whom the diagnosis of pulmonary embolism is being considered.

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