TWA magnitude and the area under the corresponding T-wave as he proposed. However, the TWA values at the peak of the T-wave were nearly equal to maximum TWA values in the three patients who developed ventricular tachyarrhythmias, as illustrated in figures 2 and 3 in our paper.[3] Notably, in the patient with non-ST-segment elevation myocardial infarction and sustained ventricular tachycardia, TWA at the peak of the T-wave exactly matched the maximum TWA in all phases (figure 2 of our paper).[3] Thus, our finding that there was no significant positive correlation between TWA magnitude and the corresponding T-wave amplitude is sufficient evidence to prove that the upsurge in TWA was not merely a consequence of increased T-wave amplitude but provided a measure of the degree of cardiac electrical instability. Furthermore, the presence of visible macroscopic TWA before the onset of ventricular tachyarrhythmias is the most decisive proof.

At this stage, given the lack of evidence that the predictive value of TWA index[1] is superior to TWA magnitude itself, adjustment with T-wave amplitude may merely dilute the performance of TWA magnitude for prediction of life-threatening cardiac arrhythmias. Moreover, in monitoring emergency patients who are at high risk of impending lethal arrhythmias, it may be cumbersome to compute TWA index. Based on our experience, we believe that there is no need for correcting visible macroscopic TWA, which signals 'clear and present danger'.

Conflict of interest: Dr Verrier is an inventor of the Modified Moving Average method for TWA analysis with patent assigned to Beth Israel Deaconess Medical Center and licensed by GE Healthcare and Medtronic Inc. The other authors declare no conflicts of interest.

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Where are the T-waves?

It is with great interest that we read the case report by GX Morales *et al.*[1] about giant J-waves caused by iatrogenic hypercalcaemia. Notably, however, Figure 1A only shows one wave after each QRS complex. As a result, this can only be a T-wave—an observation supported also by the fact that the 'giant J-wave' is present in the anterior, lateral, and inferior leads, which would not be typical of Osborn waves. Consequently, this is a fine example of iatrogenic, hypercalcaemia-induced short QT syndrome (QTc 322 ms).

It is important to differentiate between the two: while widespread J-waves predispose to malignant ventricular arrhythmias,[2] there are no human data to suggest that the QT-shortening caused by hypercalcaemia increases the risk of sudden cardiac death.[3]

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Corrigendum

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