Does APD Gradient Indicate Dispersion of Repolarization?

To the Editor:

The article by Glukhov et al.1 is a very important study, providing, as it does, direct measurement of action potential durations (APD) transmurally in the human heart. We commend the authors for providing the first evidence for M cells in the human heart. However, we dispute the assumption throughout the article that APD gradient is a measure of dispersion of repolarization (DOR). Gradient of APD is an artificial measurement with no physiological significance, because the action potential in different regions begins at different times.2 For example, if the transmural activation time were 50 ms and the APD gradient were 60 ms, with the earliest activated regions having the longest APDs, the dispersion of repolarization times would be only 10 ms, because the “gradient” of APD, in fact, seeks to nullify activation gradient and reduce DOR. In a patient with heart failure and impaired activation–repolarization coupling with the same activation gradient, the APD gradient could be nil but result in a DOR of 50 ms. Therefore, DOR must be measured from the gradient of total repolarization time, which is the sum of activation time and APD. Dispersion of total repolarization time would indicate the spatial dispersion of the repolarization moment, which is physiologically linked to arrhythmogenesis.

This could, at least partially, explain the surprising conclusion of the authors that dispersion of repolarization was less in the heart failure group. For example, in Figure 3D of the article, at least at the more physiological pacing cycle lengths (≤1000 ms), DOR may in fact be higher in the heart failure group if measured as the dispersion of total repolarization time. We believe that reanalysis of DOR with this definition would provide more accurate and valuable information.

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