Genetically Encoded Voltage Indicators
Mapping Cardiac Electrical Activity Under a New Light

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Since the days of Einthoven, graphical representations of cardiac electric activity have greatly advanced our understanding of arrhythmias and their mechanisms. Throughout the 20th century, technological advances led to improvements in the temporal and spatial resolution with which electric signals were detected. Optical mapping of electric activity was made possible by the discovery of voltage sensitive dyes that report changes in light emission as a function of transmembrane potential. A major breakthrough came with the use of high-speed cameras that can simultaneously detect the optical signal from thousands of neighboring sites, adding orders of magnitude to the spatial resolution. As in many other fields of science, visualizing what before had only been imagined led to a rapid advance in the understanding of function. Optical mapping continues to progress and with the work of Chang Liao et al, published in this issue of Circulation Research, the field takes a new leap forward. Instead of using voltage-sensitive dyes, the authors have recorded cardiac electric activity using a genetically encoded voltage indicator (GEVI) with no apparent toxicity and a good signal/noise ratio. The data show that the emitter is a reliable reporter of activation waves at fast rates, such as those seen in the mouse ventricle during atrial or ventricular tachycardia. This is not to reduce the importance of the present article; only to emphasize that this new approach is a major step forward that remains complementary of others. Just like the first camera-based optical mapping study opened a huge door of investigation even if constrained by technical limitations of its time, this study has set a major cornerstone from where to build ahead. Faster GEVIs are already being used in the neuroscience field and are likely to be followed by others. The new approach described by Chang Liao et al opens numerous new paths of investigation, such as the possibility of developing GEVIs that, by use of specific promoters, report from individual elements of the cardiac conduction system or even from nonmyocyte cells populating the neighborhood of surviving tissue after injury. There is much to be known that remains veiled by technical constrains now lifted through the use of GEVIs. Exciting times are ahead, illuminated from within the genome of the cardiac cells.

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References


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