Focus, reentry, or “focal” reentry?

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THE QUESTION WHETHER tachyarrhythmias are caused by enhanced impulse formation (“focus”) or by reentrant excitation (“reentry”) has been the subject of debate for more than a century. In 1887 McWilliam (12) was the first to suggest that disturbances in impulse propagation could be responsible for fibrillation, and he clearly envisaged the possibility that myocardial fibers could be reexcited as soon as their refractory period had ended. Some 30 years later, the work of Mines (13, 14) and Garrey (5) firmly established the role of reentry as a cause of tachyarrhythmias. Garrey demonstrated that a minimal mass of tissue is required for maintenance of fibrillation and showed that fibrillation is not due to a single rapidly firing focus. During ventricular fibrillation, there are “blocks of transitory character and shifting location,” and “it is in these ‘circus contractions,’ determined by the presence of blocks that we see the essential phenomena of fibrillation” (5). Mines (13, 14), using ring-like preparations of cardiac muscle, formulated the essential requirements of reentry, such as unidirectional block and the wavelength concept, where reentry is most likely to occur when conduction velocity is low and the duration of the refractory period is short.

Still, around the beginning of the 20th century, most investigators assumed that arrhythmias, including fibrillation, were caused by a rapidly firing ectopic focus (9, 17), and this view was still held by Scherf and Schott (19) in 1953. In the words of Bozler (3), “oscillatory afterpotentials provide a simple explanation for extrasystoles and paroxysmal tachycardia.” The pendulum between focus and reentry kept oscillating. Lewis et al. (9) “leaned to the view that irritable foci in the muscle underlay tachycardia and fibrillation,” and this view was also expressed in the first edition of his famous book, The Mechanism and Graphic Registration of the Heart Beat (10). However, in this book, an addendum dated May 20, 1920, was added: “In observations recently completed and as yet unpublished, we have observed much direct evidence to show that atrial flutter consists essentially of a single circus movement. . . . The hypothesis which Mines and Garrey have advocated now definitely holds the field” (10). Reentry held the field for a long time, until in 1947 Scherf revived the focus theory. He proposed that fibrillation is caused by a reentrant excitation (“focal” reentry) or to spiral waves. The difference between the two is that, in the former, the core is kept permanently refractory, whereas, in the latter, the core is excitable but not excited. Most of the evidence favors spiral wave reentry (8). Spiral waves, also called rotors or vortices, have been observed both in atrial and ventricular fibrillation (4, 6, 7) where they act as drivers to activate the rest of the atria or ventricles by fibrillatory conduction.

In this issue of American Journal of Physiology-Heart and Circulatory Physiology, Massé et al. (11) describe rotors on the endocardial and epicardial surface of human hearts during the initial phase of ventricular fibrillation induced by burst pacing. They studied two patients with right ventricular myopathy secondary to Tetralogy of Fallot and three patients with severe left ventricular dysfunction due to a previous anterior infarct. All patients underwent anti-ventricular tachycardia surgery. An important new element of this study is that both endocardial mapping, using an intracavitary balloon electrode, and epicardial mapping were performed. In several patients, rotors on both endocardium and epicardium were found; those on the endocardium had shorter cycle lengths than those on the epicardium. Although there were no intramural recordings, this finding makes the presence of a three-dimensional scroll wave (21) unlikely. In some cases, no epicardial rotor could be found, and the epicardial activation pattern resembled that of multiple wavelet reentry, whereas on the endocardium a stable rotor was present. This underscores the importance of simultaneous epicardial and endocardial mapping. Mapping was performed 1 s after the beginning of ventricular fibrillation and lasted 7 s. Therefore, it is unknown whether in later stages of fibrillation it was still a single rotor, or two rotors, that acted as drivers. It is possible that in later stages multiple wavelets were responsible for fibrillation. Still, this is an important study showing that, in the myopathic human heart, endocardial and
epicardial rotors are responsible for the initial phase of ventricular fibrillation, induced by burst pacing.

REFERENCES