Anisotropic Conduction and Re-entry in the Heart

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Following the seminal paper by Madison Spach and Mark Josephson, clinicians have been aware of anisotropic re-entry as an established mechanism of arrhythmogenesis, although the exact mechanisms responsible remain uncertain.[1] Nevertheless, changes in microanatomical structures, such as cellular coupling, gap junction distribution and function and fibre disarray, lead to anisotropic conduction, i.e. dependence of myocardial velocity on myocyte orientation.[2] Anisotropic conduction was initially attributed to conduction tissue, such as the atrioventricular (AV) node, but we know now that in cardiac tissue, in general, conduction velocity is anisotropic. Particularly in disease states, such as postinfarction myocardium, anisotropic conduction and spatial inhomogeneity of refractoriness may be implicated in the genesis of re-entrant, or even focal, arrhythmias. The length of the re-entrant pathway is determined by subtle electrophysiological–anatomical changes, and there may be an excitable gap.

In this issue of *Arrhythmia & Electrophysiology Review*, Kotadia et al. present an elegant update on the topic, providing very interesting perspectives. Anisotropy is the property of directional dependence, in that the orientation of myocardial activation and velocity are determined by myocyte direction. Thus, the speed of conduction is greatest in the direction parallel to the longitudinal orientation of myocytes. However, myocyte orientation may be identified using diffusion tensor MRI in explanted hearts, and multisite pacing protocols have been proposed to estimate myocyte orientation and anisotropic conduction *in vivo*. These tools have the potential to contribute to the understanding of the role of myocyte disarray and anisotropic conduction in arrhythmic states. If identifiable during clinical procedures, areas of enhanced anisotropic conduction may represent novel targets in which ablative therapy could be trialled if demonstrated to promote fibrillation.

This is an exciting hypothesis that, if proven to have clinical utility, may contribute to our efforts towards substrate characterisation and subsequent modification of arrhythmias difficult to eradicate, such as ventricular tachycardias and, perhaps, AF. Unravelling the mysteries of anisotropic conduction may also provide further insights into arrhythmias apparently unrelated to structural heart disease, such as atrioventricular nodal re-entry tachycardia and its enigmatic circuit. This really is a brave new world in the study and therapy of arrhythmias. ■

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