A TAILORED APPROACH TO CATHETER ABLATION OF PAROXYSMAL ATRIAL FIBRILLATION, by Oral et al.

Paroxysmal atrial fibrillation is a complex arrhythmia initiated by rapid “triggering” foci and likely perpetuated by the triggers and abnormal conduction in regions outside the triggering sites. Although the location of this arrhythmogenic substrate varies among patients, a simple anatomic ablation approach encircling the pulmonary venous regions is often effective, but not always easy to achieve. Oral and colleagues evaluated a tailored approach to ablation, sequentially targeting areas identified as potentially arrhythmogenic on the basis of rapid electrical activity, seeking to achieve an electrophysiological end point of noninducibility of atrial fibrillation. The results are comparable to those of some studies utilizing purely anatomic approaches and show that a tailored approach can achieve efficacy without extensive anatomic ablation. Such an approach can avoid ablation in the high-risk area adjacent to the esophagus. The study illustrates the heterogeneity of this arrhythmia and supports continued evaluation of ablation strategies. See p 1824.

β2-ADRENERGIC RECEPTOR GENETIC VARIANTS AND RISK OF SUDDEN CARDIAC DEATH, by Sotoodehnia et al.

Sudden cardiac death is a major cause of death in developed countries and results in almost one half million deaths per year in the United States. Both experimental and clinical observations suggest that β2 adrenergic receptors partially mediate sympathetic activation. Hence the β2 adrenergic receptor gene (B2AR) represents an attractive candidate gene for susceptibility to sudden cardiac death. Sotoodehnia and colleagues examined the association of two polymorphisms in the B2AR gene (Gly16Arg and Gln27Glu) with sudden cardiac death in the community-based Cardiovascular Health Study. They report that Gln27 homozygotes had a 58% increased risk of sudden cardiac death (nominal P=0.002). They tested both polymorphisms in a second small case-control study, the Cardiac Arrest Blood Study, and replicated the association of Gln27 for sudden cardiac death. Further testing in other samples is required to verify the findings and test for potential modification by sex, ethnicity, medications, risk factors, and preexisting cardiovascular disease. However, this study suggests that genetic polymorphisms may eventually provide information to identify individuals at increased risk of sudden cardiac death. See p 1842.

RETROSPECTIVE DETERMINATION OF THE AREA AT RISK FOR REPERFUSED ACUTE MYOCARDIAL INFARCTION WITH T2-WEIGHTED CARDIAC MAGNETIC RESONANCE IMAGING: HISTOPATHOLOGICAL AND DISPLACEMENT ENCODING WITH STIMULATED ECHOES (DENSE) FUNCTIONAL VALIDATIONS, by Aletras et al.

In trials of reperfusion strategies for acute myocardial infarction, noninvasive assessment of the area-at-risk and final infarct size (and the difference between them—salvage) has been accomplished by the use of serial single-photon emission computed tomography (SPECT) perfusion imaging studies. Incorporating the initial SPECT area-at-risk study is challenging, as isotope must be given prior to intervention, with imaging performed within several hours after acute intervention. Thus, many trials have evolved to using only final infarct size as the surrogate end point, although an index of salvage would be more desirable. In this issue of Circulation, Aletras and colleagues use cardiac magnetic resonance imaging in an animal model of reperfused myocardial infarction to demonstrate that 2 days after the infarct, T2-weighted cardiac magnetic resonance imaging can provide a retrospective “snapshot” of the area at risk from the acute occlusion, followed by late-enhancement gadolinium imaging to assess final infarct size. Thus, one imaging modality, temporally remote from the acute infarct, can potentially provide information on area at risk, final infarct size, and thus salvage from an intervention. Should this result be confirmed in human studies, it would greatly facilitate the study of therapeutic reperfusion strategies, as discussed by Pennell in an accompanying editorial. See p 1865.

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