The new age of atrial fibrillation: Back to the lab

Dawn S. Hui, MD, and Richard Lee, MD, MBA

From the Center for Comprehensive Cardiovascular Care, Saint Louis University, St Louis, Mo.

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Address for reprints: Richard Lee, MD, MBA, 3635 Vista Ave, DT 13th Floor, St Louis, MO 63110 (E-mail: rick.lee@health.slu.edu).

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Richard Lee, MD, MBA, and Dawn S. Hui, MD

Central Message

The canine model offers promise in advancing our understanding of the pathophysiology of AF associated with MR.

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Atrial fibrillation (AF) is a clinical diagnosis, and in the contemporary era, clinical treatment of this disease has progressed in a rapid fashion compared with the several centuries over which the disease concept of AF developed.¹ However, the surgical treatment of AF remains underused and with results that seldom equal those of the gold standard cut-and-sew Maze. A better understanding of the physiologic mechanisms, rather than focusing merely on technological innovation, may be the key to improving surgical outcomes. In this edition of the *Journal*, Ruaengsri and colleagues² report on a canine model of chronic left atrial volume overload. By using a controllable left ventriculoatrial shunt, the hemodynamic and electrophysiologic consequences of a simulated chronic mitral regurgitation (MR) were compared with a sham group. This is a major and important step toward addressing a fundamental knowledge gap.

For perspective on the importance of this work, it is helpful to review some selected historical developments in our understanding of AF. As concerns the adage that “AF begets AF,” Cox and colleagues³ first showed that the *perpetuation* of AF was sustained through macro-reentrant drivers, leading to the development of the Maze procedure. This preceded Haïssaguerre and colleagues’⁴ discovery that the *induction* of AF occurs via focal triggers in and around the pulmonary vein orifices. The distinction between these 2 mechanisms is reflected in the ongoing controversies regarding the optimal lesion set for AF ablation. Not all AF is the same, and the complex and multitude reasons for surgical ablation failure, which may be as high as 20%,⁵ have been elegantly described by Dr Cox.⁶

Ruaengsri and colleagues’² work will be important in determining the details of how MR begets AF. In patients with degenerative MR in normal sinus rhythm, the linearized rate of AF development is 5% annually and associated with increased morbidity and mortality.⁷ Although statistical techniques provide information on associated risk factors, such as age and left atrial dimension, this is no substitute for knowledge of the fundamental processes

that link these 2 disease entities. That said, we note several aspects of the study that may affect its applicability to human physiology. First, the surgical operation simulated immediate MR, which may not mimic the more gradual course that occurs with degenerative disease. Second, the shunt was a nonvalved conduit, creating the potential for bidirectional flow with a vigorous ventricle.

Regardless, it is befitting that the group whose landmark work, not only in foundational knowledge but also surgical innovation and clinical evaluation, may lead us into the future by leading us back into the lab.

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