Atrial Flutter: A Review of Its History, Mechanisms, Clinical Features, and Current Therapy

Ken W. Lee, MD, MS, Yanfei Yang, MD, and Melvin M. Scheinman, MD

Introduction

While atrial flutter (AFL) was first recognized shortly after the birth of electrocardiography, its mechanism and therapy were hotly debated until recently. Drug therapy proved to be notoriously poor for those with recurrent AFL in that drugs failed to prevent recurrences and large doses of AV nodal blockers were often needed for rate control. Over the past decade monumental shifts have occurred with respect to better definition of the arrhythmia mechanism and the remarkable efficacy of catheter ablative therapy. It is especially appropriate for this essay to review and take stock of where we have been, where we are, and where we hope to be in the future.

Historical Perspectives

The story of AFL begins more than a century ago.[1,2] The first published description of AFL dates back to 1886 when McWilliam described observing regular, rapid excitations of the atrium in an animal.[3] In 1906 Einthoven made an electrocardiographic recording of AFL.[4] Characteristic sawtooth waves in the inferior ECG leads were described by Jolly and Ritchie[5] in 1911. These authors were the first to distinguish AFL from atrial fibrillation (AF). In 1913 Lewis and coworkers[6] also described the distinctive sawtooth waves. Lewis and his colleagues were the first to investigate the mechanism of this arrhythmia.[7,8] Using a combination of epicardial maps and ECG recordings from a canine model of AFL induced by rapid atrial pacing, they showed that constant activation of at least some part of the atrium resulted in the flutter waves seen in the surface ECG. They also showed that the activation sequence was orderly, ie, the wavefront circulated in either a cranial-caudo or a caudo-cranial

Curr Probl Cardiol 2005;30:121–168.
0146-2806/$ – see front matter
doi:10.1016/j.cpcardiol.2004.07.001

direction in the right atrium.[7] From this groundbreaking experimental work, Lewis and his colleagues concluded that AFL was due to intra-atrial circus movement around the vena cavae.[8]

Subsequent works that supported the notion that flutter was due to intra-atrial reentry included those of Rosenbleuth and Garcia-Ramos who constructed a crush injury model of this arrhythmia by creating a lesion between the vena cavae.[9] Based on the epicardial maps, the authors deduced that the reentry loop circled around the atrial crush lesion. Interestingly, they also noted that when the crush lesion was extended from the inferior vena cava (IVC) to the AV groove, the arrhythmia disappeared and could not be induced. This important finding suggests that the true circuit may have included the cavotricuspid isthmus.

Intra-atrial macro-reentry as the mechanism of AFL was not universally accepted. Goto et al[10] and Azuma et al[11] had shown that aconitine caused abnormal automaticity at rapid rates in the rabbit atria. It was thought that if the atrial aconitine site fired fast enough, either flutter (1:1 conduction) or fibrillation (fibrillatory conduction because the atrial rate was too fast and 1:1 conduction could not be supported) occurred.[12] Based on these and other works with aconitine,[10,11,13-16] Scherf felt that flutter was due to abnormal automaticity.

Building on the work of Rosenbleuth and Garcia-Ramos, Frame et al[17-19] showed that the flutter reentry loop could exist outside of an atrial crush lesion. They created a "Y" lesion in the canine right atrium by extending the intercaval crush lesion to the right atrial free wall. The "Y" lesion produced a circuit that rotated around the tricuspid annulus. Similar flutter circuits may exist in patients who have undergone right atriotomies during repair of congenital heart defects.[20,21]

Over a span of nearly two decades, detailed experiments in various animal models and clinical studies have not only confirmed that the mechanism of flutter was due to intra-atrial macro-reentry but also set the stage for the development of curative catheter ablation therapy.[22-30] Of particular importance were the elegant works of Waldo et al,[27] Inoue et al,[28] and Stevenson et al,[29,30] all of described techniques of manifest and concealed entrainment. The latter allowed for identification of a site for catheter ablation.

AFL as an arrhythmia that could be successfully ablated with radiofrequency (RF) energy depended on the identification of a vulnerable, critical zone in the reentrant circuit. In 1986 Klein et al[31] reported their findings on intra-operative mapping studies of two patients with persistent flutter. They found that the narrowest part of the circuit had relatively slow conduction and localized to the low right atrium, between the IVC