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Inter-Relationships of Atrial Fibrillation and Atrial Flutter

Mechanisms and Clinical Implications

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There is a close interrelationship between atrial fibrillation (AF) and atrial flutter (AFL). Atrial fibrillation of variable duration precedes the onset of AFL in almost all instances; during AF, the functional components needed to complete the AFL re-entrant circuit, principally a line of block (LoB) between the vena cavae, are formed; if this LoB does not form, classical AFL does not develop. In contrast, there seems to be a spectrum of atrial re-entrant circuits (drivers) of short cycle lengths (CLs) (i.e., AFL). When the CL of the AFL re-entrant circuit is so short that it will only activate portions of the atria in a 1:1 manner, the rest of the atria will be activated rapidly but irregularly (i.e., via fibrillatory conduction), resulting in AF. In short, there are probably several mechanisms of AF, 1 of which is due to a very rapid AFL causing fibrillatory conduction. All of these interactions of AF and AFL have important clinical implications. (J Am Coll Cardiol 2008;51: 779–86) © 2008 by the American College of Cardiology Foundation

A clinical interrelationship between atrial fibrillation (AF) and atrial flutter (AFL) has long been appreciated. Patients who primarily manifest AF commonly also manifest AFL and vice versa (1,2); both commonly occur in the same patient early after open heart surgery (1); and Class IC and IA antiarrhythmic drugs and amiodarone used to suppress AF commonly promote sustained AFL (1,3). These clinical associations are not coincidental but reflect linkage of their pathophysiologies and perhaps even of their mechanism(s). We review the data demonstrating this linkage and emphasize their clinical implications.

Key to this linkage is that classical AFL, a macro-re-entrant rhythm in which the re-entrant wave front travels up the interatrial septum and down the right atrial (RA) free wall or vice versa and includes the cavotricuspid isthmus (CTI) (1,2), almost always develops from antecedent AF of variable duration (4–6). We will develop the theme that in almost all instances antecedent AF is necessary for the development of AFL, because it is during the AF that a critical lateral boundary (i.e., a functional line of block [LoB]) forms between the vena cavae, thereby preventing short circuiting of the AFL re-entrant circuit. Thus, 1 fundamental determinant of whether sustained AF or AFL occurs is development of this LoB. We suggest that in the vast majority of instances, without preceding AF, there can be no AFL, because it is during the preceding AF that this requisite LoB develops. Owing to physiological variability, in some patients, this LoB between the vena cavae is likely fixed (i.e., anatomic) rather than functional. In such patients, antecedent AF may not be necessary to produce AFL.

Another theme is that a fundamental determinant of whether the sustained clinical arrhythmia becomes AFL or AF may be the AFL cycle length (CL). If it is sufficiently short, it will produce fibrillatory conduction, which manifests as clinical AF (7,8). Thus in some and perhaps many instances, without fast AFL, there is no AF. Critical to this consideration, several macro–re-entrant AFLs have been described that are different than those responsible for classical AFL (8–13). Finally, we define AFL and AF as in the statement of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (now Heart Rhythm Society) (8).

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Abbreviations and Acronyms

AF = atrial fibrillation
AFL = atrial flutter
AVNRT = atrioventricular nodal re-entrant tachycardia
AVRT = atrioventricular re-entrant tachycardia
CL = cycle length
CTI = cavotricuspid isthmus
ECG = electrocardiogram
LA = left atrium
LoB = line of block
RA = right atrium

The Mechanism of Classical AFL and the Importance of Block Between the Vena Cavae

On the basis of limited mapping studies in normal canine atria (Fig. 1) and vector analysis of electrocardiograms (ECGs) in humans, Lewis (1,2,14) concluded that AFL resulted from re-entry around the great veins. Importantly, these investigators had great difficulty inducing AFL in these normal atria. Subsequently, Rosenblueth and Garcia-Ramos (1,2) appreciated that, to prevent short circuiting of the AFL re-entrant circuit proposed by Lewis (14), an LoB between the superior and inferior vena cavae seemed necessary. They made an LoB between the vena cavae with a crush lesion in otherwise normal canine atria and then reliably induced AFL. Their limited mapping of induced AFL was interpreted as demonstrating re-entry around the great veins.

Years later, Frame et al. produced an intercaval lesion in canine atria similar to that of Rosenblueth and Garcia-Ramos (1,2) but with an extension of the intercaval lesion toward the RA appendage, creating a Y lesion, and showed that induced AFL resulted from re-entry around the tricuspid valve annulus. They recognized that the Y lesion provided boundaries limiting the re-entrant circuit to the tricuspid ring, and also protected the re-entrant circuit from short-circuiting. Clearly, block between the vena cavae had a critical role in the pathogenesis of AFL, and the Y lesion was simply a variant.

A functional LoB in the region between the vena cavae is also necessary in the canine sterile pericarditis model of induced AFL, whether due to single loop (Fig. 2) or

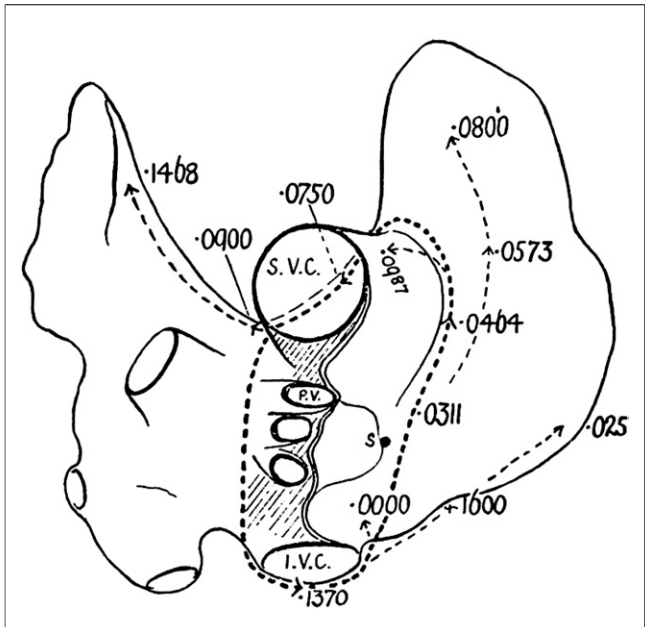

Figure 1 Lewis Model of Atrial Flutter

Induced atrial flutter in a normal canine heart. The **arrows** illustrate the putative re-entrant pathway. I.V.C. = inferior vena cava; P.V. = pulmonary vein; S = site stimulated; S.V.C. = superior vena cava. Reprinted, with permission, from Lewis (14).

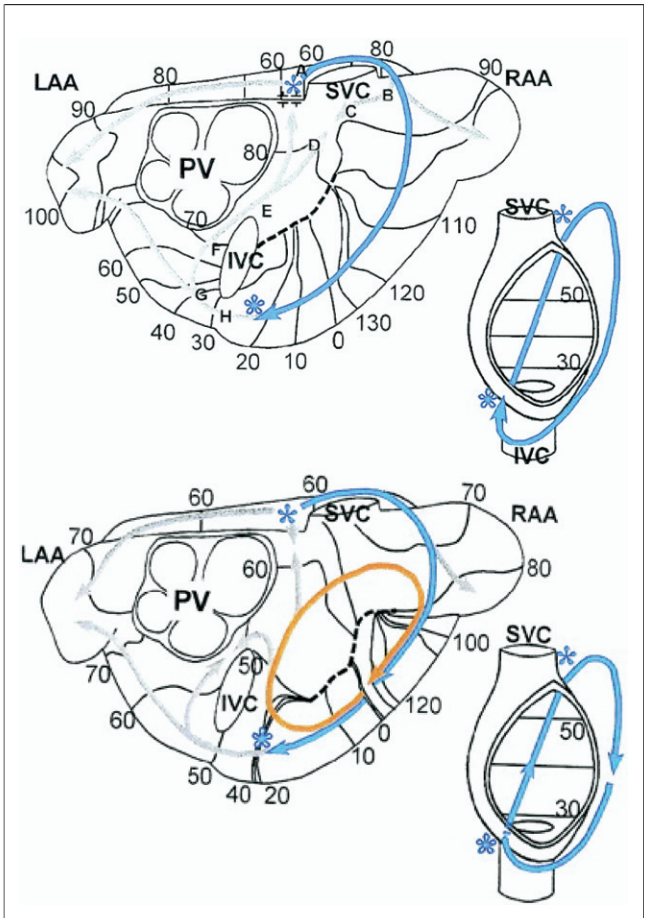

Figure 2 Sterile Pericarditis Model of Atrial Flutter

Activation maps of the atrial epicardium **(left)** and atrial septum **(right)** during sustained atrial flutter in the canine sterile pericarditis model due to a single re-entrant circuit **(top panel)** and figure-of-8 re-entry **(bottom panel)**. **Blue and orange arrows** indicate the re-entrant circuit, **gray arrows** indicate daughter wave fronts generated by the re-entrant circuit, isochrones are at 10-ms intervals, **dashed black line** indicates line of functional block, and **blue asterisks** indicate epicardial breakthrough of septal activation and site of entry from epicardium to atrial septum. Numbers equal activation time in milliseconds. Letters **A to H** are sites from which selected bipolar atrial electrograms were exhibited in the originally published figure. LAA, left atrial appendage; RAA, right atrial appendage; other abbreviations as in Figure 1. Modified, with permission, from Uno et al. (15).

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