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Editorial Comment

Atrial Flutter: Progress, but No Final Answer*

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Atrial flutter is an arrhythmia whose mechanism has fascinated and puzzled many investigators for several decades (1–3). Currently there is agreement that common (type 1) atrial flutter is based on a reentrant mechanism in the right atrium. The demonstration of an excitable gap and the possibility of entrainment by pacing (4) argue in favor of a reentrant circuit that is mostly functionally determined but uses the unique anatomy and fiber orientation of the right atrium.

Although atrial flutter is a common arrhythmia, it is frequently difficult to treat. Organic heart disease is often present, making termination and prevention of the arrhythmia important. To study the effect of pharmacologic or nonpharmacologic interventions, the ability to initiate the arrhythmia reproducibly would therefore be very helpful. Watson and Josephson (5) showed that in patients with documented atrial flutter, programmed electrical stimulation of the heart allows induction of the arrhythmia in approximately 80% of cases. Fixed rate atrial pacing at rapid rates (300 to 400 beats/min) or shortly coupled atrial extrastimuli introduced during a fast basic pacing rate are usually required to initiate the arrhythmia in the clinical electrophysiology laboratory. In patients, it has also been observed (5) that after pacing, a short period of local atrial fibrillation often precedes the onset of atrial flutter.

The present study. Waldo and his group played an important role in advancing our knowledge of atrial flutter by systematically studying the arrhythmia in the human and animal heart. In this issue of the Journal Shimizu et al. (6) report interesting observations in their animal model, extending our understanding of the initiation of atrial flutter by atrial pacing. They observe that pacing-induced atrial flutter is frequently preceded by transient atrial fibrillation, suggesting an essential role of atrial fibrillation in initiating the reentrant mechanism of atrial flutter by creating local slow conduction and unidirectional block.

The observation that local atrial fibrillation is important for the initiation of atrial flutter is not surprising. To start reentry in the atrial wall, nonhomogeneity in conduction velocity and refractory period duration followed by unidirectional block are required. These circumstances can best be provoked by repetitive atrial activation at the shortest possible interval. This will maximally abbreviate the atrial refractory period, induce slowing in conduction and increase the differences in conduction velocity in the longitudinal and transversal fiber direction (7).

How complete is our understanding of atrial flutter in humans? Much of our knowledge about atrial flutter comes from studies in animal hearts. These range from the model based purely on functional properties of the atrium showing no excitable gap (8) to models based on a combination of functional and anatomic barriers (3,6,9,10) with or without an excitable gap. As clinicians, we need to ask how relevant these observations in animal models are to our understanding and management of atrial flutter in humans. For example, Spinelli and Hoffman (11) recently showed in their animal model rapid termination of atrial flutter by the intravenous administration of class IA, class IC or class III antiarrhythmic drugs. However, the arrhythmia is rarely terminated when these drugs are given intravenously during atrial flutter in humans (12).

Also, in patients with the common (type I) atrial flutter, the electrocardiogram shows an atrial deflection having a very characteristic configuration, rate and regularity, suggesting a similar atrial activation pattern in those patients. This is at variance with the current observations by Shimizu et al. (6), who report that in their animal model the direction of the reentrant movement in the right atrium depended on the site of unidirectional block in the atrium. They found three distinct areas of slow conduction and unidirectional block in their model. Mapping studies in the human heart (13,14) suggest that the area of atrial slow conduction during common atrial flutter is located in the low posteroseptal part of the right atrium. Saoudi et al. (14) showed that a lesion in this area produced by an electrical shock prevented clinical recurrence of atrial flutter in five of eight patients. Surgical interruption of conduction in the low posteroseptal area of the right atrium can also abolish the arrhythmia (15). The duration of the follow-up period in patients with atrial flutter treated surgically or by electrical ablation is still relatively short and we cannot yet exclude the possibility that another area in the right atrium might subsequently behave like an area with slow conduction and unidirectional block.

Conclusions. These two examples demonstrate that there are differences between the human heart with atrial flutter and the animal model. Much progress has been made in our understanding of atrial flutter in humans, and the use of different animal models of atrial flutter has played an important role. Still, some questions remain. We are getting close, but do not yet have a final answer.

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