Right Atrial Flutter Due to Lower Loop Reentry
Mechanism and Anatomic Substrates

Jie Cheng, MD, PhD; William R. Cabeen, Jr, MD; Melvin M. Scheinman, MD

Background—The mechanisms of an atrial flutter (AFL) that is more rapid and at times more irregular than typical AFL are unknown.

Methods and Results—Twenty-nine patients with AFL were studied. Atrial electrograms were recorded from a 20-pole catheter placed against the tricuspid annulus (TA), with its distal electrodes lateral to the isthmus between the TA and the eustachian ridge (ER), and from the His bundle and coronary sinus catheters. Atrial extrastimuli were delivered in the TA-ER isthmus during typical AFL. Episodes of a right atrial flutter rhythm that was different from typical AFL were induced in 3 patients and occurred spontaneously in 3 patients. This sustained AFL, designated as lower-loop reentry (LLR), involved the lower right atrium (RA), as manifested by early breakthrough in the lower RA, wave-front collision in the high lateral RA or septum, and conduction through the TA-ER isthmus. Linear ablation resulting in bidirectional conduction block in the TA-ER isthmus terminated spontaneous LLR in 3 patients and rendered LLR noninducible in all patients. The cycle length of LLR was shorter than that of typical AFL (217±32 versus 272±40 ms, P<0.01). Alternating LLR and typical AFL in 1 patient resulted in cycle length oscillation.

Conclusions—LLR is a subtype of right atrial flutter and depends on conduction through the TA-ER isthmus. (Circulation. 1999;99:1700-1705.)

Key Words: atrial flutter ■ ablation ■ electrical stimulation

The mechanism of typical atrial flutter (AFL) has been well studied.1–8 The isthmus between the tricuspid annulus (TA) and eustachian ridge (ER) has been recognized as a critical portion of the typical AFL circuit and the target site for ablative therapy.5–9–17 Studies by Cosio et al14 elegantly demonstrated that the same reentrant circuit can support both clockwise and counterclockwise circus movement. However, the mechanisms of more rapid and irregular flutter rhythms are unknown. We recently described a subtype of AFL, designated as double-wave reentry (DWR), that is due to the presence of 2 activation wave fronts circulating simultaneously in the original reentrant circuit of typical AFL.18 We showed that DWR is transient and more rapid, with an irregular rate compared with typical AFL. The purpose of the present study is to describe the mechanism of a sustained subtype of right atrial flutter, which we have designated as lower-loop reentry (LLR), that also depends on conduction through the TA-ER isthmus but involves only the lower portion of the right atrium (RA).

Methods

Patient Selection
Twenty-nine patients with AFL were included in the study. There were 27 men, averaging 61.6±14.6 years old (range, 23 to 79 years).

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From the Section of Cardiac Electrophysiology, University of California San Francisco, and the Pacific Heart Institute, Santa Monica, Calif (W.R.C.).
Dr Cheng is now at SUNY Health Science Center at Syracuse, Syracuse, NY. Correspondence to Melvin M. Scheinman, MD, Cardiac Electrophysiology, University of California San Francisco, 500 Parnassus Ave, MU East 4S Box 1354, San Francisco, CA 94143-1354. E-mail scheinman@ep4.ucsf.edu © 1999 American Heart Association, Inc.
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showed negative flutter waves in the inferior leads (II, III, and aVF) associated with counterclockwise endocardial activation around the TA. Among these 28 patients, 3 had both counterclockwise typical AFL and a spontaneous but different flutter rhythm evidenced by a more rapid atrial rate and, at times, cycle length variation. Clockwise typical AFL was induced by atrial overdrive pacing at 200 ms in 1 patient with paroxysmal AFL. The diagnosis of typical AFL was confirmed by intra-atrial recordings from multiple electrodes and by concealed entrainment from the TA-ER isthmus during typical AFL. The baseline cycle length during typical AFL was 272±40 ms. In the 22 patients who had only typical AFL and were not taking amiodarone, the ERP determined from the TA-ER isthmus during typical AFL was 161±19 ms.

Induction of LLR
In 3 of the 26 patients with typical AFL, another type (LLR) of AFL that was distinctly different from and more sustained than DWR was induced. These episodes of LLR shared a common, distinctive activation pattern characterized by an early breakthrough at the lower lateral RA that generated 2 activation wave fronts in the trabeculated lateral RA, 1 propagating in a caudocranial or clockwise direction along the lateral RA and the other propagating in a counterclockwise direction through the TA-ER isthmus (Figure 2). Consequently, there was collision of the counterclockwise and clockwise wave fronts in the high lateral RA. This finding excluded the upper portions of the RA from the tachycardia circuit. The activation sequence in the left atrium, as reflected in the coronary sinus record-

**Results**

In 28 patients who presented with AFL, the surface ECG showed negative flutter waves in the inferior leads (II, III, and aVF) associated with counterclockwise endocardial activation around the TA. Among these 28 patients, 3 had both counterclockwise typical AFL and a spontaneous but different flutter rhythm evidenced by a more rapid atrial rate and, at times, cycle length variation. Clockwise typical AFL was induced by atrial overdrive pacing at 200 ms in 1 patient with paroxysmal AFL. The diagnosis of typical AFL was confirmed by intra-atrial recordings from multiple electrodes and by concealed entrainment from the TA-ER isthmus during typical AFL. The baseline cycle length during typical AFL was 272±40 ms. In the 22 patients who had only typical AFL and were not taking amiodarone, the ERP determined from the TA-ER isthmus during typical AFL was 161±19 ms.

**Induction of LLR**

In 3 of the 26 patients with typical AFL, another type (LLR) of AFL that was distinctly different from and more sustained than DWR was induced. These episodes of LLR shared a common, distinctive activation pattern characterized by an early breakthrough at the lower lateral RA that generated 2 activation wave fronts in the trabeculated lateral RA, 1 propagating in a caudocranial or clockwise direction along the lateral RA and the other propagating in a counterclockwise direction through the TA-ER isthmus (Figure 2). Consequently, there was collision of the counterclockwise and clockwise wave fronts in the high lateral RA. This finding excluded the upper portions of the RA from the tachycardia circuit. The activation sequence in the left atrium, as reflected in the coronary sinus record-
ings, remained the same as during typical AFL. The reentrant circuit of LLR appears to involve only the base of the RA, ie, the TA-ER isthmus, the portions of the smooth or posterior RA, and across the lower segment of the crista terminalis. Therefore, we designated this type of AFL as LLR.

Two sustained episodes (>30 seconds) of induced LLR were recorded after block of 1 of the double wave fronts of DWR in the TA-ER isthmus (Figure 2). These findings were reproducible in that multiple nonsustained episodes of LLR were induced after termination of DWR in these 3 patients.

Spontaneous LLR

Multiple spontaneous episodes of LLR were observed in 3 patients. The duration of these episodes ranged from a single beat, as in alternating LLR and typical AFL (see below), to more than several minutes. All 3 also had spontaneous episodes of sustained counterclockwise typical AFL. Examples of sustained episodes of typical AFL and LLR in the same patient are shown in Figure 3. In all 3 patients, there were similar flutter wave morphologies but at 2 different cycle lengths, with the longer cycle length associated with the counterclockwise typical AFL and the shorter cycle length associated with LLR. In 1 patient, alternating LLR and typical counterclockwise AFL resulted in oscillation in the cycle length that coincided with changes in the RA activation sequence (Figure 4). However, the isthmus conduction time, measured from TA 1 to proximal CS, remained unchanged during the alternans. This finding is consistent with alternation between a large circuit (typical AFL) and a small circuit (LLR) that shared a common isthmus. However, in 1 patient, the difference in local activation time between TA 1 and proximal CS was shorter during LLR than during typical AFL (Figure 5). We postulate that in this particular case, the early breakthrough occurred more distal to TA 1 and generated 2 activation wave fronts, 1 counterclockwise to the CS and the other clockwise to TA 1, resulting in simultaneous activation of the proximal CS and lower lateral RA. In all 3 patients, changes in flutter cycle length coincided with corresponding transitions between typical AFL and LLR.

**Electrophysiological Characteristics of Induced and Spontaneous LLR**

In all episodes of induced LLR, the breakthrough of activation at the lower lateral RA was preceded by a short-long sequence resulting from block of 1 of the 2 wave fronts during DWR in the TA-ER isthmus (Figure 2). In contrast to
posterobasal RA at the lower segment of the crista terminalis suggested block of the activation wave front from the sinus occurred with the loss of the early breakthrough, which may represent increasing functional conduction block that led to conversion. Difference in local activation times between TA 1 and Isthmus was also increased. This can be explained by a breakthrough site during LLR between TA 1 and Isthmus that led to simultaneous activation wave fronts, orthodromically to Isthmus and antidromically to TA 1. On conversion to typical AFL, Isthmus was activated sequentially by wave front propagated from TA 1, resulting in increased difference in local activation time. Stars indicate site of breakthrough. Abbreviations as in previous figures.

DWR, induced LLR was a more stable rhythm that persisted for as long as 4 minutes, compared with DWR, which lasts only for 2 to 12 beats. The duration of spontaneous LLR also varied from 1 beat to sustained episodes (>30 seconds).

The cycle length of LLR ranged from 170 to 250 ms, which was significantly shorter than that of typical AFL (217±32 versus 272±40 ms, P<0.01). During sustained episodes of induced LLR (>30 seconds), there was little if any change in flutter cycle length (<10 ms) except, before termination of sustained LLR, a significant cycle length variation was seen as a result of conduction variation in the posterior limb of the circuit (Figure 5).

Patients with inducible LLR had a shorter atrial ERP determined in the TA-ER isthmus than those without LLR (138.8±13.4 versus 168.5±13.9 ms, P<0.005). Intravenous ibutilide (2 mg over 15 minutes) was administered before RF ablation in 2 of the 3 patients with inducible LLR, and the ERP increased (from 132 to 180 ms and from 144 to 247 ms, respectively). No further episodes of LLR were inducible after ibutilide infusion. In all 6 patients with either inducible or spontaneous LLR before RA ablation, neither typical AFL, DWR, or LLR could be induced, despite aggressive pacing protocol after bidirectional conduction block in the TA-ER isthmus.

Termination of LLR

Most episodes of the induced LLR reverted spontaneously to typical counterclockwise AFL (Figures 4 and 5). Such reversion occurred with the loss of the early breakthrough, which suggested block of the activation wave front from the posterobasal RA at the lower segment of the crista terminalis and allowed for resumption of counterclockwise typical AFL. One episode persisted for >4 minutes and deteriorated into atrial fibrillation. In all 3 patients with spontaneous LLR, RF energy applied to the TA-ER isthmus during LLR led to termination of LLR with bidirectional block (Figure 6).

Surface Flutter Wave Morphology of LLR

In all patients with LLR, either induced or spontaneous, the surface ECG showed negative flutter waves in the inferior leads and a positive deflection in V1, similar to that of typical counterclockwise flutter (Figures 3 and 4). However, the terminal portion of the flutter wave lost its positive deflection in the inferior leads during LLR in association with wavefront collision along the lateral trabeculated RA wall during LLR. In 1 patient, multiple breakthroughs occurred along the crista terminalis in association with a blocked premature atrial depolarization in the TA-ER isthmus that led to nearly simultaneous cristal activation and allowed for reversal of both the sequence of septal activation and surface flutter wave (Figure 7).

Discussion

Our most important observation was to define the mechanism and probable substrate of a subtype of more rapid right atrial flutter, which we designate as LLR. Our data suggest that during LLR, the activation wave front traverses the TA-ER isthmus, activates the posterior RA, and then skirts the IVC with breakthrough along the low RA. The latter generates 2 wave fronts, which collide and are extinguished in the high lateral or septal RA while the tachycardia is driven by the circuit at the base of the RA. Termination of LLR by linear RF lesions delivered in the TA-ER isthmus provides definitive proof for the involvement of the TA-ER isthmus and the strongest evidence against a focal mechanism. Moreover, an aggressive pacing protocol after ablation failed to provoke the tachycardia, providing evidence against alternative mechanisms such as abnormal automaticity or triggered activity or...
be fixed and present during sinus rhythm, but controversy (Figure 4). LLR that share a common pathway in the TA-ER isthmus to a larger reentrant circuit (typical AFL) and a shorter one ending LLR and typical AFL, consistent with switching between the TA-ER isthmus terminated LLR (Figure 6). In addition, we have shown that the interruption of conduction through the same isthmus must also be involved in the LLR. The TA-ER isthmus forms the critical component of the AFL posterior barrier, whereas the TA forms the anterior barrier.

Compelling evidence indicates that typical flutter circuits in humans depend on the crista terminalis and ER as its posterior barrier, whereas the TA forms the anterior barrier. The TA-ER isthmus forms the critical component of the AFL circuit. The same isthmus must also be involved in the LLR. We have shown that the interruption of conduction through the TA-ER isthmus terminated LLR (Figure 6). In addition, the isthmus conduction time remained identical with alternat-
ing itself may have resulted from a breakthrough between TA 1 and another observation relates to the finding of DWR termination introducing LLR. Termination of 1 of the DWR wave fronts produces a pause after a period of rapid rate, which may facilitate conduction through the lower lateral cristal region. Previous studies by Pinto et al21 and Boyden et al22 also suggested that premature action potential recordings obtained from atrial tissue in an isolated tricuspid ring model of AFL showed marked oscillation of action potential duration at more rapid tachycardia rates. Regardless of the precise mechanism of the lower cristal breakthrough, LLR appears to be perpetuated by a linking phenomenon in which the upper portions of the RA are excluded from the circuit by repetitive collision. We also found that patients with induced LLR had a significantly shorter ERP than those without. In addition, in the patients with induced LLR, this arrhythmia could not be initiated after intravenous ibutilide. The effects of ibutilide during AFL were to increase atrial refractoriness and to decrease the excitable gap in the TA-ER isthmus.23,24 We hypothesize that the lengthening of the refractory period in the TA-ER isthmus precluded initiation of this rapid reentrant tachycardia.

Physiological Considerations of the LLR Circuit

The chief paradox relating to our hypothesis is that all patients with LLR also had typical AFL. If a short circuit between the exit point from the TA-ER isthmus to the low lateral RA exists, why is it not manifest all the time? The answer to this question is not clear from our data, but certain points may help explain the paradox. Frame et al20 showed that monophasic action potential recordings obtained from atrial tissue in an isolated tricuspid ring model of AFL showed marked oscillation of action potential duration at more rapid tachycardia rates. Regardless of the precise mechanism of the lower cristal breakthrough, LLR appears to be perpetuated by a linking phenomenon in which the upper portions of the RA are excluded from the circuit by repetitive collision. We also found that patients with induced LLR had a significantly shorter ERP than those without. In addition, in the patients with induced LLR, this arrhythmia could not be initiated after intravenous ibutilide. The effects of ibutilide during AFL were to increase atrial refractoriness and to decrease the excitable gap in the TA-ER isthmus.23,24 We hypothesize that the lengthening of the refractory period in the TA-ER isthmus precluded initiation of this rapid reentrant tachycardia.

Determination of Flutter Wave Morphology During Right Atrial Flutter

Our data support the observation by Okumura et al25 from canine experiments that the flutter wave morphology is largely determined by the activation sequence of the septum and left atrium (Figures 3, 4, and 7). Although the activation

Figure 7. Reversion of flutter wave polarity associated with reversal of septal activation sequence. Multiple cristal break-throughs occurred during LLR, and a premature atrial depolarization (<) was seen in TA-ER isthmus. Premature depolarization itself may have resulted from a breakthrough between TA 1 and another observation relates to the finding of DWR termination introducing LLR. Termination of 1 of the DWR wave fronts produces a pause after a period of rapid rate, which may facilitate conduction through the lower lateral cristal region. Previous studies by Pinto et al21 and Boyden et al22 also suggested that premature activation of a site in the flutter circuit could be due to the breakdown of lateral boundaries. Regardless of the precise mechanism of the lower cristal breakthrough, LLR appears to be perpetuated by a linking phenomenon in which the upper portions of the RA are excluded from the circuit by repetitive collision. We also found that patients with induced LLR had a significantly shorter ERP than those without. In addition, in the patients with induced LLR, this arrhythmia could not be initiated after intravenous ibutilide. The effects of ibutilide during AFL were to increase atrial refractoriness and to decrease the excitable gap in the TA-ER isthmus.23,24 We hypothesize that the lengthening of the refractory period in the TA-ER isthmus precluded initiation of this rapid reentrant tachycardia.
sequences in the RA are significantly different during LLR and typical AFL, the flutter wave morphology is similar to the counterclockwise typical AFL when the collision of wavefronts occurs along the lateral RA, with the activation of the interatrial septum remaining in a caudal-to-cranial direction. However, there was a subtle difference in the terminal portion of the flutter wave, ie, the loss of the terminal positive deflection of the flutter wave in the inferior leads (Figures 3 and 4). This temporally coincided with and could be accounted for by the wave-front collision along the lateral trabeculated RA wall. Furthermore, the flutter wave polarity changed to that of clockwise typical AFL when the interatrial septum was activated in a cranial-to-caudal direction (Figure 7).

Limitations
The chief limitation of our study is lack of sufficient intracardiac recording sites, especially in the posterior RA, to define the course of the entire LLR circuit. Entrainment during LLR to prove that the TA-ER isthmus was an integral part of the tachycardia circuit was not successful because of frequent conversion of LLR to typical AFL. However, TA-ER isthmus conduction block always terminated LLR.

Clinical Significance
Existing data in the literature suggest that only typical AFL, either counterclockwise or clockwise, is amenable to ablation in the TA-ER isthmus. The important practical significance of our observations relates to the findings of a rapid and, at times, irregular flutter rhythm (LLR) that may be TA-ER isthmus–dependent and curable by RF ablation. Close scrutiny of the surface ECG may yield clues to the presence of LLR.

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References
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