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. 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. Studies by Cosio et al 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. 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). Eleven patients had coronary artery disease, but none had clinical evidence of myocardial ischemia at the time of study. Five had cardiomyopathy, with left ventricular ejection fractions ranging from 25% to 45%. Eleven had hypertension. One patient had moderate to severe mitral regurgitation. The remaining patients had no structural heart disease. All had normal thyroid function. Twelve patients were also included in our previous report on DRW. None were taking antiarrhythmic medications or digitalis except 4 who were taking amiodarone for a history of atrial fibrillation. These 4 patients were excluded from data analysis.

All patients had typical AFL. Twenty-seven had chronic AFL (>1 month), and 2 had recurrent paroxysmal AFL. Three patients also had episodes of spontaneous flutter, with flutter wave morphology similar to that of typical counterclockwise AFL but with more rapid and, at times, variable atrial rates.

Electrophysiology Study
After having given informed consent, all patients were brought to the Electrophysiology Laboratory in a postabsorptive state. A coronary sinus catheter was inserted via the right internal jugular vein access with its proximal electrodes placed at the coronary sinus ostium. A 20-pole electrode was inserted via the right femoral vein and placed against the TA (Figure 1). A catheter was placed across the tricuspid valve to record the right anterior septal atrial and His bundle electrograms. All catheters were deployed under fluoroscopy. Pulse oximetry and vital signs were monitored throughout the study.

Twelve-lead surface ECGs and intracardiac electrophographic signals were recorded with a computerized data acquisition system (CardioLab by Prucka Engineering Inc). All signals were filtered with a low-pass filter set at 50 Hz. The mean ± standard deviation of the cycle length of 25 beats was measured from the ECGs.

Circulation is available at http://www.circulationaha.org

© 1999 American Heart Association, Inc.
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. Counterclockwise 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 distinctively 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
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.\(^{18}\) 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, nor 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).

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 alternation (Figure 4).

Figure 7. Reversion of flutter wave polarity associated with reversal of septal activation sequence. Multiple cristal breakthroughs 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 Isthmus and was blocked between isthmus and CS os, as indicated in middle inset. This allowed clockwise wave front in upper lateral RA to propagate further toward septum and resulted in a reversal of septal activation sequence (top). Simultaneous surface ECG (bottom) shows reversal of flutter wave polarity associated with reversal in septal activation sequence. Initial complexes (▲) were recorded during LLR when collision occurred over lateral RA. Subsequent complexes (▼) were recorded with reversal of septal activation sequence. Although the exact mechanism of the arrhythmia is unclear, presence of multiple breakthroughs along the crista terminalis is proposed as depicted in the inset on right. Abbreviations as in previous figures.

of a reentrant circuit that does not involve the TA-ER isthmus.

Anatomic Considerations of the LLR Circuit

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 alternating LLR and typical AFL, consistent with switching between a larger reentrant circuit (typical AFL) and a shorter one (LLR) that share a common pathway in the TA-ER isthmus (Figure 4).

Recently, the septal isthmus of typical flutter was shown to be fixed and present during sinus rhythm, but controversy exists regarding the nature of conduction barrier along the lateral portions of the crista terminalis. A recent elegant study by Shah et al.\(^1\) in patients with typical AFL demonstrated that during pacing from the coronary sinus, there appears to be relatively rapid and homogeneous activation with wave fronts from the posterobasal RA and IVC–TA isthmus fusing (at the lower crista) and ascending upward. The latter observation is in accord with our hypothesis that the crista terminalis, at least distally, is a functional rather than fixed conduction barrier during typical AFL. During 1 episode of LLR, the RA activation became much more complex (Figure 7), which we speculate was a result of conduction breakthrough at multiple sites along the crista terminalis. This episode eventuated in atrial fibrillation soon after the multiple breakthroughs occurred. This raises an interesting possibility that piecemeal breakdown of the conduction barrier along the crista terminalis may form the substrate for multiple smaller reentrant circuits and thereby herald atrial fibrillation.

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 al.\(^2\) 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. Perhaps this oscillation allows impulse breakthrough to the lower segments of the crista terminalis that had a shorter refractoriness. 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 al.\(^3\) and Boyden et al.\(^4\) 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.\(^5,6\) 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 al.\(^7\) 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
sequences in the RA are significantly different during LLR
and typical AFL, the flutter wave morphology is similar to the
clockwise typical AFL when the collision of wave
fronts 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 ac-
counted 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 scruti-
tiny of the surface ECG may yield clues to the presence of LLR.

Acknowledgment
Dr Cheng was supported in part by a Mark C. Lidwill Traveling
Fellowship from the North American Society of Pacing and
Electrophysiology.

References
1. Wells JL Jr, MacLean WAH, James TN, Waldo AL. Characteristics of
atrial flutter: studies in man after open heart surgery using fixed atrial
2. Waldo AL, MacLean WAH, Karp RB, Kouchoukos NT, James TN.
Entrainment and interruption of atrial flutter with atrial pacing: studies in
3. Lewis T, Drury A, Illiescu C. A demonstration of circus movement in
clinical flutter of the auricles. Heart. 1921;8:341–357.
5. Klein G, Guiraudon G, Sharma A, Milstein S. Demonstration of macro-
reentry and feasibility of operative therapy in the common type of atrial
6. Cosio FG, Lopez GM, Goicoea A, Arribas F. Electrophysiologic studies
7. Olgin JE, Kalman JM, Fitzpatrick AP, Lesh MD. Role of right atrial
diaphragmatic structures as barriers to conduction during human type I atrial
flutter: activation and entrainment mapping guided by intracardiac echo-
8. Kalman J, Olgin J, Lee RJ, Saxon LA, Lesh MD. The anterior barrier in
9. Saoudi N, Derumeaux G, Cribier A, Letac B. The role of catheter ablation
techniques in the treatment of classic (type I) atrial flutter. Pacing Clin
10. Feld GK, Fleck RP, Chen PS, Boyce K, Bahnson TD, Stein JB, Calisi
CM, Ibarra M. Radiofrequency catheter ablation for the treatment of human
type I atrial flutter: identification of a critical zone in the reentrant
circuit by endocardial mapping techniques. Circulation. 1992;86:
1233–1240.
Catheter ablation by low energy DC shocks for successful management of
12. Cosio FG, Lopez-Gil M, Goicoea A, Arribas F, Barroso JL. Radiofre-
cency ablation of the inferior vena cava-tricuspid valve isthmus in
S, Pitha JV, Becker AE, Arruda M, Gonzalez MD, Widman LE, Rome M,
Neuhauser J, Wang X, Calame JD, Goudeau MD, Jackman WM. Role of
the tricuspid annulus and the eustachian valve/ridge on atrial flutter:
relevance to catheter ablation of the septal isthmus and a new technique
for rapid identification of ablation success. Circulation. 1996;94:
407–424.
Atrial endocardial mapping in the rare form of atrial flutter. Am J Cardiol.
Electrophysiologic and electrophysiographic characterization of atypical
atrial flutter in man: use of activation and entrainment mapping and
implications for catheter ablation. J Cardiovasc Electrophysiol. 1997;8:
121–144.
16. Olgin JE, Kalman JM, Saxon LA, Lee RJ, Lesh MD. Mechanism of
initiation of atrial flutter in humans: site of unidirectional block and
17. Lesh MD, Kalman JM, Olgin JE. An electrophysiologic approach to
cessation of atrial flutter and tachycardia: from mechanism to
practice. In: Singer I, ed. Interventional Electrophysiology. Baltimore,
18. Cheng J, Scheinman MM. Characteristics of double-wave reentry induced
by programmed stimulation in patients with typical atrial flutter. Circu-
lation. 1998;97:1589–1596.
Garrigue S, Clémenty J. Three-dimensional mapping of the common
20. Frame LH, Edward KR, Berstein RC, Fei H. Reversal of reentry and
acceleration due to double-wave reentry: two mechanisms for failure to
terminate tachycardias by rapid pacing. J Am Coll Cardiol. 1996;28:
137–145.
21. Pinto JM, Graziano JN, Boyden PA. Endocardial mapping of reentry
around an anatomical barrier in the canine right atrium: observations
during the action of the class IC agent, flecainide. J Cardiovasc Electro-
physiol. 1993;4:672–685.
22. Boyden PA, Graziano JN. Activation mapping of reentry around an
anatomical barrier in the canine atrium: observations during the action of
the class III agent, d-sotalol. J Cardiovasc Electrophysiol. 1993;4:
266–279.
23. Cheng J, Karch MR, Scheinman MM. Electrophysiologic effects of
Right Atrial Flutter Due to Lower Loop Reentry: Mechanism and Anatomic Substrates
Jie Cheng, William R. Cabeen, Jr and Melvin M. Scheinman

Circulation. 1999;99:1700-1705
doi: 10.1161/01.CIR.99.13.1700
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1999 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/99/13/1700

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/