Atypical Atrial Flutter Originating in the Right Atrial Free Wall

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Background—Data from experimental models of atrial flutter indicate that macro-reentrant circuits may be confined by anatomic and functional barriers remote from the tricuspid annulus–eustachian ridge atrial isthmus. Data characterizing the various forms of atypical atrial flutter in humans are limited.

Methods and Results—In 6 of 160 consecutive patients referred for ablation of counterclockwise and/or clockwise typical atrial flutter, an additional atypical atrial flutter was mapped to the right atrial free wall. Five patients had no prior cardiac surgery. Incisional atrial tachycardia was excluded in the remaining patient. High-density electroanatomic maps of the reentrant circuit were obtained in 3 patients. Radiofrequency energy application from a discrete midlateral right atrial central line of conduction block to the inferior vena cava terminated and prevented the reinduction of atypical atrial flutter in each patient. Atrial flutter has not recurred in any patient (follow-up, 18 ± 17 months; range, 3 to 40 months).

Conclusions—Atrial flutter can arise in the right atrial free wall. This form of atypical atrial flutter could account for spontaneous or inducible atrial flutter observed in patients referred for ablation and is eliminated with linear ablation directed at the inferolateral right atrium. (Circulation. 2000;101:270-279.)

Key Words: atrial flutter • reentry • mapping • catheter ablation

Data obtained from investigations of atrial flutter in experimental models and humans indicate that macro-reentrant circuits may be confined by anatomic, surgical, and/or functional barriers remote from the tricuspid annulus–eustachian ridge (TA-ER) isthmus.1–5 These macro-reentrant tachycardias that do not use the TA-ER isthmus (ie, are not a form of typical atrial flutter) and do not exhibit reentry around atrial incisions can be classified as “true” atypical atrial flutters.1 Data characterizing the various forms of atypical atrial flutter in humans are limited. Treatment has been restricted to pharmacological management or AV junctional ablation to control ventricular rate.

In this study, we describe a specific type of atypical atrial flutter in which macro-reentry was confined to the right atrial free wall (RAFW). The study patients were identified from a group of patients referred for ablation of atrial flutter. The electrophysiological characteristics and ablation of this tachycardia are described.

Methods

Definitions

Typical (type 1) atrial flutter was defined as a macro-reentrant atrial tachycardia using the TA-ER isthmus.6 Atypical atrial flutter was defined as a macro-reentrant atrial tachycardia not using the TA-ER isthmus and not produced by prior atriotomy (ie, not an “incisional atrial tachycardia”).1 For the purposes of this study, tachycardia was considered to be sustained if persistent for ≥5 minutes in the absence of pace termination.

Patient Selection

During electrophysiological evaluation in 160 consecutive patients referred for ablation of typical atrial flutter, burst pacing at cycle lengths decreased by 10 ms per burst from 300 ms to 2:1 atrial capture from ≥2 right atrial sites resulted in the induction of an additional sustained atypical atrial flutter in 36 patients. Atypical atrial flutters were mapped to the left atrium in 22 patients and to the lateral RAFW in 6; localization was indeterminate in 8 patients. This study presents data on the 6 patients in whom the atrial flutter circuit was mapped to the RAFW.

Patient characteristics are presented in the Table. Diagnoses included coronary artery disease (3 patients) and mitral valve disease, idiopathic dilated cardiomyopathy, and no structural heart disease (1 patient each). Operative records of the patient with previous mitral valve replacement for mitral incompetency (11 years before the development of atrial flutter) indicated no RAFW atriotomy. No other patient had prior cardiac surgery. In the 5 patients with structural heart disease, echocardiographic examination demonstrated mild biatrial enlargement, trivial tricuspid insufficiency, and normal right ventricular systolic pressure. In 1 patient, echocardiographic examination was normal. Five patients had previously required direct-current cardioversion for atrial flutter. No patient had atrial fibrillation documented before study. One patient was receiving amiodarone at the time of study.

Two patients (patients 5 and 6, the Table) had spontaneous atypical atrial flutter at the onset of study as confirmed by subsequent
Electrophysiological Testing

Informed consent was obtained before electrophysiological study and ablation. Baseline electrophysiological evaluation was performed as previously described. If spontaneous atrial flutter was present at the onset of study, pacing was performed at multiple sites along the inferior TA to determine the participation of the TA-ER isthmus before pace termination. All patients underwent burst pacing from the medial and lateral TA-ER isthmus during sinus rhythm. Pacing was performed at twice the diastolic current threshold at an initial cycle length of 300 ms. Pacing cycle length was then decreased by 10 ms per burst until either atrial flutter induction or 2:1 atrial capture. For each spontaneous or induced sustained atypical atrial flutter, flutter cycle length (FCL), 12-lead ECG, atrial activation pattern, and response to pacing at multiple right atrial sites were examined.

In 3 patients, 1 or 2 custom, deflectable 18-electrode 7F catheters (1.5-mm intra-electrode and 10-mm interelectrode pair spacing) Cordis Webster) were positioned systematically at multiple right atrial locations during atypical atrial flutter to assess activation sequence and to identify double or fractionated potentials indicating the possible presence of conduction delay or block. In the remaining 3 patients, electroanatomic mapping (Biosense Webster) was used to characterize the tachycardia circuit. This system uses magnetic catheter tracking to construct spatially precise 3-dimensional endocardial activation maps. Distances and conduction velocities within the reentrant circuit were determined from high-density electroanatomic maps. Local activation was determined by electrogram onset.

Entrainment pace mapping was used to localize the tachycardia circuit in each patient. During entrainment mapping, bipolar pacing (2-mm interelectrode spacing) was performed at diastolic current threshold at cycle lengths 10 to 30 ms less than the FCL. During entrainment, any change in atrial activation sequence compared with baseline tachycardia as determined by analysis of all available surface and intracardiac ECG recordings was considered to represent manifest fusion. Concealed entrainment was considered to be present if pacing resulted in no change in atrial endocardial activation or surface F-wave morphology. To identify sites within the reentrant circuit, the postpacing interval (PPI) was analyzed.

The PPI was defined as the interval from the stimulus artifact to the beginning of the next atrial activity on the ECG. The PPI was measured during pacing from the medial and lateral TA-ER isthmus during sinus rhythm. Only 1 type of sustained atypical atrial flutter was inducible in each patient. In the absence of earlier pace termination, episodes of sustained atypical atrial flutter persisted for >1 hour in each patient. Spontaneous transitions between atypical and typical atrial flutter were observed in 2 patients.

Atypical Atrial Flutter Ablation and Follow-Up

In the initial 3 patients, ablation of atypical atrial flutter was performed with radiofrequency (RF) energy (Radionics RFG-3D or Medtronic lesion generators) delivered from an 8-mm tip electrode of an ablation catheter (EP Technologies or custom Medtronic) to 2 cutaneous patch electrodes. Guiding sheaths (Daig) were used to optimize ablation electrode contact. In the remaining 3 patients, electroanatomic map-guided ablation was performed with the use of a 4-mm electrode-tipped electroanatomic mapping/ablation catheter and 1 cutaneous patch electrode. RF energy was applied during sustained atypical atrial flutter. Energy applications (20 to 40 W, 30 to 60 seconds per application) were performed sequentially to produce a linear lesion. After ablation, burst pacing was performed from ≥2 right atrial sites at a cycle length decreased by 10 ms per burst from 300 ms to 2:1 atrial capture. Successful ablation was defined as termination of atypical atrial flutter during RF energy application and inability to reinduce atrial flutter.

Patients were discharged off antiarrhythmic drugs. Arrhythmia recurrence was excluded through serial examinations and ambulatory ECG monitoring.

Results

Atypical Atrial Flutter Induction, FCL, and Surface ECG

In all 6 patients, sustained atypical atrial flutter could be induced reliably with burst pacing at cycle lengths of 200 to 240 ms from the coronary sinus (CS) or at baseline. In each case, atrial fibrillation was not observed as an intermediate rhythm. Only 1 type of sustained atypical atrial flutter was induced in each patient. In the absence of earlier pace termination, episodes of sustained atypical atrial flutter persisted for >1 hour in each patient. Spontaneous transitions between atypical and typical atrial flutter were observed in 2 patients.

Overall, there was no significant difference in the FCLs of typical and atypical atrial flutter, although the difference in FCL was >80 ms in 1 patient (the Table). In the 5 patients in whom typical atrial flutter ablation was performed before atypical atrial flutter ablation, TA-ER isthmus ablation did not affect the inducibility or FCL of atypical atrial flutter.

In each patient, a negative F wave in the inferior ECG leads was displayed during atypical atrial flutter (Figure 1A). The negative F-wave morphology was similar to that of counterclockwise typical atrial flutter (Figure 1B). In 2 patients, spontaneous transitions in F-wave polarity between negative and positive in the inferior leads during atypical atrial flutter were observed (see Activation Mapping).
Activation and Entrainment Mapping

In all patients, comprehensive analysis of mapping data obtained before TA-ER isthmus ablation suggested macroreentry confined to the RAFW. Right atrial activation preceded left atrial activation in each patient. Detailed right atrial mapping during atypical atrial flutter identified widely split double potentials in the midlateral RAFW distinct from the crista terminalis (Figure 2). Only discrete potentials were

Figure 1. A, Representative ECG patterns of RAFW atypical atrial flutter. Atypical atrial flutter with negative F-wave polarity in inferior leads was observed in each patient. Tachycardias in 2 left panels displayed counterclockwise RAFW activation (direction of rotation described in right lateral view). Tachycardia in far right panel displayed clockwise RAFW activation. B, F-wave morphology of counterclockwise typical atrial flutter (corresponding to patients in A). Note similarity with that of negative F-wave RAFW flutter.
recorded from this same area during normal sinus rhythm. During sinus rhythm, pacing at progressively shorter cycle lengths provoked the development of double potentials recorded from the midlateral RAFW before the induction of atrial flutter. This finding suggested the presence of rate-related conduction delay or block within the RAFW.

Mapping of the inferolateral RAFW identified fractionated electrograms of \( \approx 90 \) ms duration (representing \( \approx 34\% \) of the respective FCL; Figure 2). Although fractionated electrograms were recorded at several inferior RAFW sites (eg, sites 4, 5, and 8, Figure 3), only fractionated electrograms recorded from sites adjacent to the central line of double potentials (eg, sites 4 and 5, Figure 3) were within the tachycardia circuit (see below).

Pacing at sites immediately adjacent to the central line of double potentials (sites A through D, Figure 3) resulted in PPI\(_2\)FCL intervals \( >30 \) ms, indicating that these lateral RAFW sites were within the flutter circuit (Figure 4). Pacing from the CS os, mid–TA-ER isthmus, or sites posterior to the crista terminalis (site E, Figure 3) resulted in manifest fusion and PPI\(_2\)FCL intervals \( <30 \) ms.

Detailed mapping of the RAFW suggested clockwise (right lateral view) activation in 3 patients and counterclockwise activation in 3. Because most episodes of atypical atrial flutter were induced with CS os pacing, no correlation between pacing site and direction of activation could be determined. In 2 patients, the interval separating the lateral and medial TA-ER isthmus electrograms during atypical atrial flutter was less than the corresponding interval observed during either counterclockwise or clockwise typical atrial flutter or during inferolateral TA or proximal CS pacing. These findings immediately excluded the presence of typical atrial flutter. In 2 patients, activation of the proximal CS and the inferoseptal and superolateral right atrium was nearly simultaneous (Figure 5). This finding also suggested coincident rather than sequential (as with typical atrial flutter) activation of these sites. In 3 patients, brief episodes of spontaneous transient block from the lateral RAFW to the superolateral, inferior anterolateral, and/or inferoseptal right atrium was observed and thus excluded these sites from active participation in the flutter circuit (Figure 5).

Spontaneous transitions in F-wave polarity during atypical atrial flutter were observed in 2 patients (Figure 5). Transitions

![Figure 2. Activation mapping of atypical atrial flutter. Right anterior oblique (RAO) fluoroscopic view illustrates positions of multipolar mapping and mapping/pacing catheters in lateral RAFW. Additional catheters were positioned in His bundle region and CS. Numerical designations signify RAFW recording sites (locations depicted in fluoroscopic view and in Figure 3) corresponding with electrograms in composite activation map on right. Double potentials were recorded from sites 3 and 4 and fractionated electrograms from sites 5 and 8. Analysis indicated that sites 2 through 5 were within and site 8 was outside the atrial flutter circuit (see Activation and Entrainment Mapping section). Arrows (activation map) indicate activation sequence.](http://circ.ahajournals.org/)

![Figure 3. Right atrium indicating reentrant circuit (right anterior oblique [RAO] view). Numerical designations signify RAFW recording sites (locations depicted in Figure 2). Designations A through E indicate pacing site locations. Entrainment mapping indicated that sites A through D were within reentrant circuit, and sites posterior to crista terminals (CT) were outside reentrant circuit (site E). During ablation, RF energy applied to sites 4 through 7 resulted in elimination of tachycardia. Both clockwise and counterclockwise activation sequences were observed in separate patients. SVC indicates superior vena cava; IVC, inferior vena cava; and TA, tricuspid valve annulus.](http://circ.ahajournals.org/)
Figure 4. Entrainment mapping during counterclockwise atypical atrial flutter. Displayed (from top to bottom) are 3 surface ECG leads, electrograms from superolateral (high) right atrium (HRA), distal and proximal electrode pairs of mapping/pacing (MAP/PACE) catheter, proximal (RA 16–15) to distal (RA 2–1) electrode pairs of multipolar mapping catheter (catheter placement as in Figure 2), proximal (CS 8–7) and distal CS, and stimulation markers (STIM). Pacing cycle length is 200 ms. PPI is represented by interval from last stimulus artifact (S1) to first electrogram recorded from mapping/pacing catheter (arrow, A). A, Pacing from mid-anterolateral RAFW (B, Figure 3); B, pacing from mid-posterolateral RAFW (D, Figure 3). PPI–FCL was \( \leq 20 \) ms at each site. Mid-anterolateral RAFW pacing resulted in concealed fusion (no change in intracardiac electrograms or surface ECG), reflecting pacing from "protected" site between central line of block and tricuspid annulus in which antidromic capture of any site outside of reentrant circuit is prevented. Posterolateral RAFW pacing resulted in manifest fusion, as evidenced by change in electrograms recorded from most proximal electrode pair of multipolar mapping catheter (RA 16–15 or site 1 in Figure 2; solid arrow denotes paced complex; open arrow, baseline activation) consistent with antidromic capture of site near reentrant circuit. Surface ECG remains unchanged. This response indicates degree of local fusion during pacing from relatively "unprotected" site, permitting antidromic capture of site (RA 16–15) near reentrant circuit. Pacing from adjacent RAFW pacing sites on opposite sides of central line of block results in markedly different stimulus to RA 8–7 electrogram intervals (asterisks).
in F-wave polarity were not rate dependent. Reversal of F-wave polarity from negative to positive was associated with a change in early activation from the inferior to the superior septum, suggesting that the interatrial septum and left atrium were activated passively during atypical atrial flutter (Figure 5).13

Magnetic Electroanatomic Mapping
High-density electroanatomic maps (160 to 410 recording sites per map) demonstrated clockwise (2 patients) or counterclockwise (1 patient) activation within the RAFW (Figure 6). Vertical central lines of block (identified by the isolated line of double potentials) were identified 1.5 to 2.0 cm anterior to the crista terminalis. The lengths of the central lines of block ranged from 2.0 to 2.4 cm. Within each circuit, multiple areas of slow conduction (conduction velocity <0.4 m/s) were identified (Figure 7).

Catheter Ablation and Follow-Up
Ablation of RAFW flutter was performed at the time of primary electrophysiological study and typical atrial flutter ablation in 5 patients. RF energy applications delivered sequentially from the inferior vena cava os superiorly to the midlateral RAFW double potentials terminated atrial flutter in each patient (Figure 8). The mean number of RF energy applications was 8 with the use of 8-mm ablation electrodes and 14 with the use of 4-mm electrodes with electroanatomic guidance. Corresponding ablation parameters with 8-mm ablation electrodes (3 patients) were power output of 30 to 55 W, current of 703 to 754 mA, impedance of 70 to 85 Ω, and ablation electrode temperature of 49°C to 54°C; with 4-mm ablation electrodes (3 patients), power output was 20 to 30 W, current was 452 to 582 mA, and impedance was 92 to 108 Ω. In 5 patients, termination of atrial flutter during energy application occurred without a change in FCL. In 1 patient, progressive cycle length prolongation without oscillation was observed during the 2 energy applications before flutter termination (Figure 9). After ablation, burst pacing protocols from the CS os and lateral right atrium did not induce any sustained atrial flutter. There were no complications.

One patient in whom TA-ER isthmus ablation was not performed at the time of ablation of spontaneous RAFW atypical flutter developed typical atrial flutter 8 months later. During subsequent electrophysiological evaluation, no atypical atrial flutter was induced. There has been no recurrent arrhythmia in this patient for an additional 8 months of follow-up after TA-ER isthmus ablation. In the remaining 5 patients, there has been no recurrence of any atrial flutter at 3

Figure 5. A, Additional positive F-wave morphology of RAFW atypical atrial flutter observed in 2 patients. Spontaneous transitions in F-wave polarity between positive (left) and negative (right) inferior leads were observed. B, Right atrial activation during same atypical atrial flutter with positive (left) and negative (right) F waves in surface lead II. Cycle lengths are identical. Note nearly simultaneous activation of high right atrial (HRA), low septal right atrium (LSRA), and proximal CS. With transition in F-wave polarity from positive to negative, activation of proximal CS now precedes HRA (arrows), indicating some degree of inferosuperior left atrial activation. Note the 2:1 intra-atrial block from the anterolateral (ANTLAT) to LSRA. This finding excludes LSRA from active participation in the reentrant circuit (see text).
to 40 months of follow-up. One patient developed atrial fibrillation at 1 month and was treated with amiodarone (subsequent follow-up of 8 months without recurrent arrhythmia). Collectively, there has been no recurrent atypical atrial flutter during a follow-up of 18 ± 17 months.

**Discussion**

This study describes a specific type of atrial flutter originating in the RAFW. This form of atypical atrial flutter constituted a spontaneous clinical tachycardia in at least 3 of the study patients. In the remaining patients, RAFW flutter was induced during the evaluation of typical atrial flutter. No patient had a previous right atriotomy scar. Conduction delay within the midlateral RAFW was demonstrated during rapid pacing in sinus rhythm. The precise nature of the conduction delay or block within the RAFW, ie, the presence of fixed versus purely functional block, was not definitively established. Although similar findings in experimental models have been interpreted as indicating the presence of functional block, exclusion of a fixed line of block can be difficult.

The surface ECG does not readily distinguish RAFW atypical atrial flutter from typical atrial flutter. Fortunately, they can be differentiated by entrainment (or electroanatomic) mapping. These findings emphasize the importance of determining the precise mechanism of a “negative F-wave” atrial flutter before TA-ER isthmus ablation.

**Ablation of RAFW Atypical Atrial Flutter**

In the present study, an area between an anatomic barrier (the inferior vena cava os or inferior crista terminalis) and the center of the reentrant circuit was targeted for ablation. This resulted in the elimination of atypical atrial flutter after a limited ablation procedure in each patient. This site was targeted primarily because of the close proximity of the center of the circuit to the inferior vena cava os. Additionally, the phrenic nerve and major components of the conduction system were avoided. It is possible that linear ablation between the center of the circuit and other barriers may also have been effective. Use of electroanatomic mapping simplified the characterization of the reentrant circuit and permitted the precise localization of pacing sites and ablation lines.

Lesions contiguous with anatomic barriers may generate or extend a line of conduction block, thus potentially creating
the substrate for other tachycardias and/or facilitating their occurrence. It is possible that the RAFW ablation line could facilitate the occurrence of typical atrial flutter in patients with intact TA-ER isthmus conduction. In the present study, typical atrial flutter subsequently developed in the single patient who had not undergone concomitant TA-ER isthmus ablation. This possibility should be considered when ablation is planned in patients presenting with only atypical atrial flutter.

Relation to Experimental Models of Atrial Flutter
Several experimental models of atrial flutter have been described, including atrial incisional (intercaval and right atrial crush injury, RAFW “Y” incision, atrial surgery), right (tricuspid insufficiency/pulmonary stenosis) and left (subclavian arterial/pulmonary venous shunt) atrial enlargement, and sterile pericarditis models. Macro-reentry with purely functional obstacles in the RAFW has been demonstrated in sterile pericarditis and atrial enlargement models. Shimizu et al demonstrated the presence of multiple areas of slow conduction within RAFW reentrant circuits in a sterile pericarditis model. The characteristics of atrial flutter in our study patients seem to reflect a similar tachycardia mechanism of single-loop macro-reentry incorporating multiple discrete areas of slow conduction within the RAFW.

Study Limitations
The study population represents a select group of patients referred for ablation of atrial flutter. The precise prevalence of RAFW flutter in an unselected patient population is unknown. In this study, we describe 1 type of atypical atrial flutter arising in the right atrium. It is likely that additional atypical macro-reentrant circuits will be identified in the future. Induction
protocols used in the present study were limited, and the precise mechanisms of tachycardia initiation and conduction block within the RAFW were not definitively established. It is anticipated that new high-resolution mapping systems will facilitate further characterization of these tachycardias.

**Clinical Implications**

Atrial flutter in humans may be due to macro-reentry confined to the RAFW. Although the surface ECG does not readily distinguish atrial flutter arising in the RAFW from typical atrial flutter, they can be differentiated through the use of high-resolution mapping systems.

![Figure 8](image1.png)

**Figure 8.** Catheter ablation (first 3 patients). Top and bottom panels depict left (LAO) and right anterior oblique (RAO) fluoroscopic views, respectively. RF energy was applied sequentially from inferior vena cava os (RF 1) superiorly toward central line of double potentials (solid white lines in far right panels indicate ablation line). Atrial flutter terminated during RF 5. Ablation was guided with electro-anatomic maps to generate similar ablation lines in final 3 patients.

![Figure 9](image2.png)

**Figure 9.** Termination of atypical atrial flutter during ablation. Displayed (from top to bottom) are 3 surface ECG leads, proximal (MAP 6–5) to distal (MAP 2–1) electrode pairs of multipolar mapping catheter positioned in lateral RAFW (see Figure 2), proximal and distal electrode pairs of ablation catheter (ABL), proximal CS electrogram, and RF generator output (RF/WATTS). Electrograms are included (A) from distal electrode pair of ablation catheter (ABL DIS) at site where RF energy application resulted in termination of tachycardia. Analysis of right atrial activation at time of termination (B) is consistent with block at ablation site. In this patient, FCL increased from 318 ms (285 ms at baseline) to 409 ms immediately before tachycardia termination.
of entrainment pace mapping or electroanatomic mapping techniques. This form of atrial flutter can be eliminated with linear ablation between the center of the reentrant circuit and the inferior vena cava.

References
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