Surface Electrocardiographic Characteristics of Right and Left Atrial Flutter

Andres Bochoeyer, MD*; Yanfei Yang, MD*; Jie Cheng, MD, PhD; Randall J. Lee, MD, PhD; Edmund C. Keung, MD; Nassir F. Marrouche, MD; Andrea Natale, MD; Melvin M. Scheinman, MD

Background—There is little information about the surface expression of non–cavotricuspid isthmus (CTI)–dependent right atrial (RA) or left atrial (LA) flutter circuits.

Methods and Results—We retrospectively evaluated 32 episodes (in 26 patients) of atypical RA and 22 episodes (in 21 patients) of LA flutter. The surface ECG of 13 patients with lower-loop reentry was similar to that of their pattern during counterclockwise (CCW) CTI atrial flutter (AFL), except for decreased amplitude of the terminal forces in the inferior leads. In 11 of 24 episodes characterized by high or multiple breaks over the crista, the ECG showed changes that depended on the initial activation sequence of the LA. In 7 of 8 episodes of upper-loop reentry, the ECG pattern completely mimicked that for clockwise (CW) CTI AFL. All 11 patients with an LA septal circuit showed a typical ECG pattern characterized by prominent forces in lead V1 with flat deflections in the other surface leads. Eleven patients with other LA circuits had a more variable pattern but showed decreased voltage in the inferior leads compared with that of a group with CCW-CTI AFL (1.6±1 vs 2.68±0.7 mV, respectively; P<0.05).

Conclusions—The RA surface-ECG patterns different from those of CCW or CW-CTI could still be CTI dependent. In contrast, a typical CW-CTI surface pattern was always seen in patients with upper-loop reentry, which was non–CTI dependent. LA AFL circuits had either flat or low-amplitude forces in the inferior leads. *(Circulation. 2003;108:60-66.)*

Key Words: electrophysiology ▪ atrial flutter ▪ mapping ▪ catheter ablation

Although a large variety of atrial flutter (AFL) circuits have been defined, there are few data that correlate known AFL circuits with surface ECG patterns. The purpose of this study was to define the ECG patterns of flutter circuits in an effort to help localize the flutter pathway.

Methods

Study Population

Right Atrial AFL

This study retrospectively analyzed consecutive patients referred for catheter ablation of AFL. The patients (n=372) were recruited from the University of California at San Francisco from January 1996 to August 2000.

Patients with clockwise (CW) or counterclockwise (CCW) cavotricuspid isthmus (CTI)–dependent flutter (CTI-AFL; n=328 patients) and 18 patients with prior cardiac surgery were excluded from study. We studied 26 patients who had experienced 32 episodes of atypical right atrial (RA)-AFL. In 20 of 26 cases, the patients also had CCW or CW CTI-AFL. Among these 32 episodes of atypical flutter, 20 (63%) were spontaneous and 12 were pacing induced. The flutters were generally sustained (>30 seconds) and allowed for mapping and ablation.

Left Atrial AFL

We included only those patients with left atrial (LA)-AFL circuits without atriotomy in whom a complete electroanatomic map (CARTO) was available. Eight were excluded because of an incomplete map. A total of 21 consecutive patients with 22 episodes of LA macroreentrant circuit were analyzed; 17 of 21 were studied at the Cleveland Clinic. All patients with LA-AFL were studied during their sustained, clinically manifest arrhythmias.

Analysis of the ECG

We compared simultaneous surface and intracardiac recordings and/or CARTO maps with the surface recordings. We also compared surface recordings from our study patients with 38 consecutive patients with CCW CTI-AFL who underwent successful CTI ablation. Surface flutter waves were usually analyzed during higher (>2:1) grades of atrioventricular block to avoid superimposition of the flutter on T waves. In 3 instances, recordings of 2:1 block were used because the P or flutter waves that preceded the QRS complexes were free of T waves.
Electrophysiology Study

The study was approved by the University of California at San Francisco and Cleveland Clinic institutional review boards. Antiarrhythmic drugs were discontinued for at least 5 half-lives before the study, except for 11 patients who were treated with amiodarone. The study was performed as previously described by using recordings from a 20-pole halo, His bundle, and coronary sinus (CS) catheters. The distal pole of the halo catheter was positioned at 7 o'clock on the tricuspid annulus (TA) in the left anterior oblique projection. Twelve-lead ECGs and intracardiac recordings were obtained and displayed simultaneously. Entrainment mapping was attempted in all patients at a cycle length (CL) of 10 to 30 ms less than the flutter CL. Concealed entrainment was present when the difference between the tachycardia CL and postpacing interval was \( \pm 25 \) ms with identical intracardiac activation sequence and surface flutter wave morphology (when possible). Electroanatomic mapping with the CARTO system (Biosense, Cordis-Webster Inc) was available for 2 patients with atypical RA-AFL and for 21 patients with LA-AFL. Low voltage was defined as a voltage amplitude <0.1 mV and scar that showed no recordable activity. Entrainment mapping was attempted from various sites in those with lower-loop reentry (LLR) and upper-loop reentry (ULR). In all patients with LLR, concealed entrainment was present in the CTI but was absent from the CTI in those with ULR. CTI ablation was successful in arrhythmia termination and resulted in a lack of arrhythmia inducibility for all patients with LLR.

Statistics

All numeric variables are expressed as mean±SD. A value of \( P<0.05 \) was considered statistically significant. Mean voltage comparisons were made with use of a t-test for a 2-tailed distribution with 2 samples of unequal variance. Sensitivity and specificity were calculated by using Fisher’s exact probability test.

Results

RA Flutter

This group consisted of 24 men and 2 women with an average age of 59±13 years. The demographic data for patients with RA-AFL is detailed in Table 1. In both patients who changed from CCW to LLR or from CW to ULR, the CL decreased from 266 to 253 ms (\( P<0.02 \)) and from 259 to 250 ms (\( P<0.03 \)), respectively.

Counterclockwise LLR

A total of 19 patients with 24 episodes showed a pattern of LLR, which involves a breakthrough over the crista with the circuit around the inferior vena cava; hence, LLR is a CTI-AFL. We found 3 different ECG patterns for patients with LLR. The first pattern was previously described by Cheng et al and involved a wavefront break over the low lateral RA (Figure 1). This circuit was found in 13 of 24 episodes and was usually associated with a slight decrease in the amplitude of the late positive deflection in the inferior leads. It was thought that the decrease in the late inferior forces was caused by an impulse collision over the lateral portion of the RA, which served to cancel the forces caused by late craniocaudal lateral RA activation. Because activation of the septum and LA remains the same as with CCW CTI-AFL, the AFL wave patterns remain very similar.

The second ECG pattern involved a total of 11 of 24 episodes that showed wavefront breaks over more superior portions of the TA. An illustrative example of a “high break” is shown in Figure 2A and is characterized by advancement of the atrial electrogram from the His bundle region compared with the CS recordings during CCW-AFL. Of note was the finding that the surface recordings were identical during CCW CTI-AFL when compared with the LLR pattern. We interpreted this finding as being due to capture of the LA from the CS connection. The wavefront collision occurred high over the septum, and hence, most of lateral wall was still activated in a craniocaudal direction.

Figure 2B shows another example of LLR with a high break over the TA. In this patient, the change to a higher break site (TA) was associated with a marked change in the surface ECG morphology. Note that the surface ECG during LLR (with the break at TA) showed a pattern that mimicked CCW-AFL. With conversion to the higher break (TA), the surface ECG pattern was now more consistent with that seen in CW-AFL. We explain this finding by assuming earlier...
activation of the septum and anterior LA over Bachmann’s bundle, with the wavefront akin to that observed in CW-AFL.

The third type of flutter circuit was observed in 9 patients and was characterized by LLR, with multiple, early breaks over the lateral TA. A very unusual ECG pattern evolved as the flutter circuit changed from CCW to LLR with multiple breaks (Figure 3). The flutter wave in V1 changed from positive to negative and was associated with diminished late forces in the inferior leads. The latter is explained by a collision of wavefronts over the lateral wall, with cancellation of the late craniocaudal forces. The change in polarity of V1 might result from slightly earlier activation of the LA from Bachmann’s bundle (Figure 3).

Clockwise ULR
Another flutter pattern was characterized by a circuit confined to the upper portion of the RA that was non-CTI dependent. There were 8 episodes of CW-ULR in 7 patients: 5 were spontaneous and 3 were induced by pacing. Seven of the 8 episodes showed breakthrough over the low crista terminalis, with wavefront collision over the midlateral TA. An example is shown in Figure 4. Note that there is no change in the surface ECG pattern despite clear-cut evidence of change from a typical CW circuit to 1 with a wavefront collision in the CTI, which was associated with an early break over the low lateral TA. This pattern is explained by the identical craniocaudal activation sequence of the septum and LA for both flutter circuits. One patient had a persistent 2:1 atrioventricular conduction, which impaired our ability to compare the surface pattern with CW CTI-AFL.

Figure 2. A, Left-hand panels show 12-lead ECGs of spontaneous episodes of CCW and LLR. Right-hand panels show intracardiac recordings during CCW and LLR. Note that during LLR, patient developed “high” cristal break over TA9 (see schema). High break explains early activation over RA roof as well as advancement of atrial deflection on His bundle electrogram relative to CS recordings. TA8–TA9 electrograms are probably out of plane relative to His atrial recordings, because they show small changes relative to that from His. Direction of both LLR and CCW wavefronts are identical (caudocranial) and explain why 12-lead ECGs (left-hand panels) are identical for both circuits. Isth indicates cavotricuspid isthmus. B, Serial recordings from patient with CCW-AFL (not shown) who developed 2 patterns of LLR. Left-hand panel shows surface ECGs of both, and right-hand panel shows corresponding intracardiac recordings. In 1 pattern (LLR-1), 12-lead ECG shows flat flutter waves in inferior leads and positive flutter waves in V1, (see arrows). In endocardial recordings shown on right, LLR-1 is characterized by cristal break over TA8 (marked by asterisk), whereas LLR-2 shows higher break (TA9) as shown in schema at bottom. Note that higher cristal break in LLR-2 again results in earlier activation of His bundle atrial electrogram relative to CS (compare LLR-2 with LLR-1). Larger circuit (LLR-2) is associated with shorter transisthmus conduction time, suggesting that circuit size determines isthmus conduction time. Surface ECG leads for LLR-2 (left) shows positive flutter waves in inferior leads and negative flutter waves in V1, and are explained by craniocaudal activation of septum and LA. CSos indicates the ostium of CS.
LA Flutter

The demographic pattern for these patients is detailed in Table 1. The LA-AFL patients were separated into 3 groups.

LA Septal Circuit

We observed 11 episodes of LA-AFL around the left fossa ovalis. All patients showed an ECG pattern characterized by a dominant positive or multiphasic flutter wave in V1 with near-isoelectric flutter waves in the others leads (Figure 5A). CARTO mapping in these patients showed either CCW or CW revolution around the left side of the septum primum (Figure 5B). Concealed entrainment was found in the isthmus between the right pulmonary veins and the left septum primum. We failed to terminate the flutter circuit by radio-frequency ablation in 1 patient.

Mitral Annulus AFL

Three patients had a reentrant CCW circuit around the mitral annulus. All had concealed entrainment from both the CS and mitral annulus. Two had a surface ECG pattern that showed prominent, positive P waves in V1/V2, with inferior leads that showed 1-mV positive or multiphasic waves (Figure 6). The third case had an ECG pattern that mimicked a typical CCW pattern, except for a negative P wave of 1 mV in lead I. In these patients, the mitral annulus was the anterior boundary, and low-voltage areas in the posterior wall served as the posterior boundary; successful ablation was achieved in 2 of the 3 patients.

Posterior Wall Scar Circuits

There were 8 patients who had low-voltage or scarred areas over the LA posterior wall and posterior scar circuit. The surface ECG of 4 of them mimicked typical CCW CTI-AFL patterns in terms of P-wave orientation, except for 1 patient, whose ECG showed a negative deflection in V1 without the classic “sawtooth” pattern in the inferior leads. One patient had a surface ECG with a low-voltage CW pattern, and another 2 cases showed the characteristic LA septal circuit pattern previously described (Figure 7). One patient had a fixed 2:1 atrioventricular block, which precluded definition of the surface wave morphology. Ablation was successful in terminating these circuits in all 8 patients.

Surface ECG Comparison

Comparison of the 12-lead ECG between those with LA-AFL and those with CCW CTI-AFL is detailed in Table 2. Inferior leads in all patients with the LA septal circuit pattern had almost flat-line recordings. The maximal amplitude in any of the 3 inferior leads (II, III, aVF) was significantly smaller for those with mitral annulus or posterior wall scar circuits compared with those with CCW CTI-AFL (1.6 ± 1 vs 2.68 ± 0.7 mV, P < 0.05).
Discussion

Major Findings
We found that surface flutter recording for those with LA-AFL might often be an inadequate indication of CTI-AFL, whereas LA-AFL shows even more inhomogeneous patterns. Previous elegant animal studies showed that flutter wave polarity was dependent on the activation sequence of the LA,\textsuperscript{10–13} and this principle has been confirmed in human studies.\textsuperscript{14–16} More recently, studies with 62-lead body-surface mapping in humans with CCW-AFL showed evidence that the terminal positive deflections in the inferior leads could be caused by late craniocaudal activation of the lateral RA wall.\textsuperscript{17} We used these principles to explain the relation between surface and endocardial recordings.

Lower-Loop Reentry
The surface ECG pattern was very similar for those with LLR and CCW CTI-AFL. For some, the changes were manifested by a decreased amplitude of the late positive waves in the inferior leads, probably as a result of wavefront collision over the lateral RA wall. Of interest was the finding that high cristal breaks could be associated with a CW pattern (Figure 2B). Even more interesting surface patterns were recorded with LLR circuits and multiple cristal breaks (Figure 3). These surface patterns were typical for neither CCW nor CW-AFL and emphasize the need for careful entrainment mapping of the CTI to document its involvement.

Upper-Loop Reentry
ULR is a circuit with CW orientation in the upper RA with wavefront collision in the CTI. In our series of 7 patients with...
LA Atrial Flutter

Those with LA-AFL had a significantly higher incidence of LA enlargement, atrial fibrillation, and longer tachycardia CL compared with those with RA-AFL. Jais et al\(^8\) first reported LA-AFL maps and pointed out a rather variegated pattern of surface expression for the LA-AFL population.

LA Septal Circuit

Patients with LA septal circuit typically showed large, usually positive waves in V\(_1\) with almost flat waves in other leads. We suggest that this pattern might be caused by a septal circuit with anterior-posterior forces projecting on V\(_1\) and the cancellation of caudocranial forces. This pattern was 100% sensitive for an LA septal circuit, but the specificity of this pattern for any LA-AFL was only 64% (\(P<0.002\)).

Mitral Annulus AFL

Most of these patients showed prominent forces in V\(_1\)/V\(_2\) with diminished amplitude in the inferior leads. It is suggested that a posterior LA scar allows for domination by anterior LA forces. This constellation of findings might mimic CCW or CW CTI-AFL, but the decreased amplitude of frontal plane forces suggests an LA circuit (Table 2).

Posterior Wall Scar Circuits

This group showed the most variable surface ECG patterns. The only possible distinguishing feature was the relatively low-amplitude signals in the inferior leads. The voltage in lead I was significantly greater in this group compared with those with CCW CTI-AFL (0.9±1 vs. 0.005±0.4 mV; \(P<0.05\); Table 2). Hence, low-amplitude inferior forces and prominent, positive forces in lead I suggest a posterior wall scar circuit.

Limitations

For most of those with RA-AFL, a detailed electroanatomic map was unavailable; hence, our explanations require confirmation by more detailed studies. The data derived from this study might not be an accurate indication of the incidence of LA-AFL circuits. This is because some LA circuits could not be completely mapped and thus, were not included in this study.

Clinical Implications

Our studies for those with RA-AFL highlight the importance of entrainment mapping. We found instances of atypical surface patterns that were CTI dependent and vice versa. LA-AFL showed more inhomogeneous patterns. A large-amplitude flutter (or P) wave in V\(_1\) associated with flat, inferior P waves was pathognomonic of LA septal circuit but was observed in other LA circuits. In addition, those with LA-AFL had a tendency for a greater isoelectric interval, which might reflect wide areas of slow conduction.\(^20\)

### References


### Table 2. Surface ECG Features: Comparison of Mean Voltage (in mV)

<table>
<thead>
<tr>
<th></th>
<th>RA CCW (n=38)</th>
<th>LA Septal Circuit (n=11)</th>
<th>Mitral Annulus (n=3)</th>
<th>Posterior Wall Macroleentry (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior leads*</td>
<td>2.62±0.7</td>
<td>0.08±0.1†</td>
<td>1.2±0.35†</td>
<td>1.6±1†</td>
</tr>
<tr>
<td>Lead I</td>
<td>0.005±0.4</td>
<td>0</td>
<td>-0.3±0.57‡</td>
<td>0.9±1†</td>
</tr>
<tr>
<td>V(_1)</td>
<td>1.97±0.35</td>
<td>2.3±0.4§</td>
<td>1.6±0.35§</td>
<td>1.6±1.2§</td>
</tr>
</tbody>
</table>

*Mean±SD of maximal amplitude in lead II, III, or aVF.
†\(P<0.05\) by \(t\) test compared with CCW.
§Only 1 patient had negative flutter wave in lead I.
§\(P=\)NS compared with CCW.


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