Atypical Right Atrial Flutter Patterns

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Background—The purpose of our study was to define the incidence and mechanisms of atypical right atrial flutter.

Methods and Results—A total of 28 (8%) of 372 consecutive patients with atrial flutter (AFL) had 36 episodes of sustained atypical right AFL. Among 24 (67%) of 36 episodes of lower loop reentry (LLR), 13 (54%) of 24 episodes had early breakthrough at the lower lateral tricuspid annulus, whereas 11 (46%) of 24 episodes had early breakthrough at the high lateral tricuspid annulus, and 9 (38%) of 24 episodes showed multiple annular breaks. Bidirectional isthmus block resulted in elimination of LLR. A pattern of posterior breakthrough from the eustachian ridge to the septum was observed in 4 (14%) of 28 patients. Upper loop reentry was observed in 8 (22%) of 36 episodes and was defined as showing a clockwise orientation with early annular break and wave-front collision over the isthmus. Two patients had atypical right AFL around low voltage areas (“scars”) in the posterolateral right atrium.

Conclusions—Atypical right AFL is most commonly associated with an isthmus-dependent mechanism (ie, LLR or subeustachian isthmus breaks). Non–isthmus-dependent circuits include upper loop reentry or scar-related circuits. (Circulation. 2001;103:3092-3098.)

Key Words: electrophysiology ■ atrial flutter ■ catheter ablation ■ mapping ■ tachycardia

Spontaneous clockwise (CW) and counterclockwise (CCW) isthmus-dependent atrial flutter (AFL) has been well described. More recently, other types of isthmus-dependent and non–isthmus-dependent flutter patterns have been described.1–10 The purpose of the present study was to define the incidence and mechanisms of right AFL in a large cohort of patients referred for catheter ablation of AFL.

Methods

A total of 372 consecutive patients with AFL were referred to our institutions for ablation from January 1996 to August 2000. None of the patients in the present study had been previously reported. Patients with surgically corrected congenital heart disease were excluded from the study. We excluded 328 patients with sole typical isthmus-dependent flutter and 16 with left AFL, leaving 28 (8%) patients (aged 59±13 years) with atypical right AFL for study. Among the 28 patients, 16 (57%) had documented atrial fibrillation and AFL, whereas 12 had AFL alone. Six patients had structural heart disease, but none had an atriotomy. Almost all antiarrhythmic drugs had been withdrawn for at least 5 half-lives before the study; however, 6 patients were being treated with amiodarone at the time of the study.

Informed written consent was obtained for all patients. Recordings were obtained from a 20-pole electrode catheter along the tricuspid annulus (TA), the coronary sinus (CS), and the anteroseptal region (His bundle region), as previously described.7 Entrainment mapping was attempted in all patients, and concealed entrainment was diagnosed when the difference between tachycardia cycle length (TCL) and postpacing interval was ≤30 ms, with identical intracardiac and surface flutter wave morphology. Electroanatomic mapping using the CARTO Biosense system (Biosense Webster Inc) was available in 4 of the 28 patients.

Definitions

Sustained tachycardia was defined as that lasting ≥30 seconds. Early breakthrough during AFL was described as a wave front traversing the subeustachian isthmus (SI) and activating the posterior right atrium (RA) with early annular activation (break), producing 2 wave fronts that result in collision along the TA.7

Statistical Analysis

The difference between flutter TCL for each patient with both typical and atypical flutter was analyzed by paired t test. A value of \( P<0.05 \) was considered statistically significant.

Results

We identified 36 episodes of atypical right AFL in 28 (8%) of 372 patients referred for ablation of AFL. Twenty patients had both typical and atypical flutter, whereas 8 patients had only atypical flutter. A total of 32 episodes (89%) of atypical flutter were sustained, and 26 (81%) of 32 were sufficiently prolonged to allow for mapping, entrainment pacing, and/or
ablation. During the studies, 22 (61%) of 36 episodes occurred spontaneously and were usually preceded by episodes of typical flutter, whereas 14 episodes were induced by pacing. Twenty (91%) of 22 spontaneous episodes and 12 (86%) of 14 pacing-induced episodes were sustained.

Isthmus-Dependent AFL

Lower Loop Reentry Tachycardia

A total of 24 of 36 episodes of atypical AFL fit the diagnostic criteria of lower loop reentry tachycardia (LLR), suggested by Cheng et al.7 Twenty of the 24 episodes were sustained, and 4 were nonsustained. Fifteen episodes occurred spontaneously in patients with or without typical flutter, whereas 9 occurred after atrial pacing.

In 4 patients, episodes of LLR alternated with typical CCW flutter; Figure 1 (left panel) shows beat-to-beat changes in activation sequence and TCL. In 13 of the 24 episodes, the early breakthrough occurred at the lower lateral RA (TA1 to TA3) with wave-front collision over the lateral wall similar to that described.7 In contrast, we found that 11 of 24 episodes had an early breakthrough at TA5 to TA8 and collision of wave fronts over the high RA or high septum. This sequence was confirmed by both CARTO Biosense mapping and basket catheter mapping during flutter in 1 patient. Figure 2 shows a patient with typical CCW flutter who spontaneously developed AFL with a CCW orientation and early breakthrough at TA5. Concealed entrainment was found at the isthmus. Radiofrequency (RF) ablation applied to the isthmus showed tachycardia termination at the isthmus.

Another new finding for those with LLR was the presence of multiple early breakthroughs with multiple collision sites along the TA (Figure 3, right panel). The pattern of multiple early breaks occurred in 9 (38%) of the 24 episodes of LLR.

We noted only minor changes in flutter-wave morphology, with changes from typical CCW flutter to LLR when the breakthrough occurred over the low lateral RA.7 More pronounced changes were sometimes seen when the collision occurred over the high lateral or septal RA areas. As shown in Figure 4, LLR that was associated with early break at T6 and collision of wave front at high RA had flatter flutter waves in the inferior leads (Figure 4A) compared with the pattern during CCW flutter (not shown), whereas LLR with higher early break (TA8) and collision at the septum had positive flutter waves in the inferior leads and negative flutter waves in lead V1 (Figure 4B). The latter was more compatible with a CW flutter pattern and is explained by activation of the septum and left atrium by a cranial-caudal sequence, owing to reversal of activation of these structures by superior breakthrough on the annulus.

Figure 1. Right panel illustrates catheter sites in left anterior oblique projection. A 20-pole catheter was placed along TA with its distal pair of electrodes (TA1) at 7 o’clock and proximal electrodes (TA10) at high atrial septum. Left panel shows simultaneous recordings of surface leads II, aVF, and V1, distal (HBEp) and proximal (HBEp) His bundle recordings, and distal (CS3) and proximal (CS2) CS recordings, together with recordings from the low lateral (TA1) to the high septal (TA10) RA. Recordings show evidence of early breakthrough over midlateral wall (TA6) (stars) with collision at TA8 alternating on beat-to-beat basis with typical CCW flutter. Note variation of electrogram morphology in TA5 and TA6, as well as oscillation of TCL. Schematic diagram depicts proposed circuit. CT indicates crista terminalis; CSOS, CS ostium; and \( \_\_\_\_\_\_ \), wave front collision.

Figure 2. Collision of wave front over high/lateral RA in patient who originally showed CCW flutter. Early breakthrough is depicted by stars at TA5. During RF application to the SI, gradual prolongation of the TCL was recorded before block in isthmus. Note that area of annular collision was maintained until flutter was terminated. CS indicates middle of coronary sinus; other abbreviations and symbols as in Figure 1.
For those with LLR, proof that the isthmus was part of the circuit was confirmed by concealed entrainment during isthmus pacing in 12 patients and by flutter termination in the isthmus during RF ablation in all patients. After achievement of bidirectional block, the flutter was no longer inducible; however, the same early breakthrough(s) could also be demonstrated by pacing at the CS ostium in 7 patients; early breakthrough was also confirmed in 1 of these patients by CARTO mapping.

Partial Isthmus-Dependent SI Short Circuit

In 4 (14%) of the 28 patients with atypical right AFL, a short circuit of the eustachian ridge barrier was suspected by recording early activation of the atrial septum (Figure 5). The patient had spontaneous CCW AFL with premature activation of the CS ostium and impulse collision at the isthmus of both the orthodromic CCW wave front and another front emerging from the CS ostial region (Figure 5, left panel). The TCL of this flutter was 223 ms. Overdrive pacing at the TA margin of the medial portion of the isthmus and CS ostium showed that it was not part of the tachycardia circuit. Concealed entrainment was demonstrated by pacing from the high RA septal area between the superior vena cava (SVC) and the fossa ovalis (FO). Application of RF energy to this region slowed the tachycardia without termination in 1 of the patients. In the other patient, the 20-pole catheter showed a CW flutter pattern with an early breakthrough over the 7 o’clock position.

Non–Isthmus-Dependent AFL

Upper Loop Reentry Tachycardia

Another pattern observed was a non–isthmus-dependent reentrant circuit involving the upper portion of the RA, which we designated upper loop reentry tachycardia (ULR). A total of 8 episodes were recorded in 7 patients. ULR was characterized as a CW flutter circuit with early breakthrough over the lateral annulus and the collision of wave fronts over the isthmus or low lateral RA wall. Overdrive pacing of the isthmus showed that it was not part of the tachycardia circuit. Four episodes of ULR either occurred spontaneously or emerged spontaneously from typical CW flutter or LLR and were pacing-induced in 3 (38%) episodes; 2 patients had spontaneous ULR alone. Seven (88%) of the 8 episodes of ULR were sustained.

During the episodes of ULR, attempts to ablate the circuit were performed in 2 patients. In both, concealed entrainment was demonstrated by pacing from the high RA septal area between the superior vena cava (SVC) and the fossa ovalis (FO). Application of RF energy to this region slowed the tachycardia without termination in 1 of the patients. In the other patient, the 20-pole catheter showed a CW flutter pattern with an early breakthrough over the 7 o’clock posi-
tion. Concealed entrainment was also documented in the area between the inferior vena cava (IVC) and FO (Figure 6A). In the same patient, double potentials were shown as the 20-pole catheter was moved to the septum (Figure 6B). Ablative lesions applied from the FO to the orifice of IVC resulted in tachycardia slowing and then termination. We suspect that the tachycardia circuit for ULR, at least in some patients, involves a reentrant wave around the region of the FO (Figure 6C). Electroanatomic mapping was not available for these patients.

In 3 of 20 patients with CCW-LLR, spontaneous isthmus block occurred and resulted in a pattern of CW flutter with ULR (Figure 7). This arrhythmia was associated with impulse collision over the isthmus.

**Right Atrial Scar Reentry**

Two patients without prior atriotomy had evidence of reentry around large low-voltage regions of the posterolateral RA, as determined by CARTO Biosense mapping. Both patients had spontaneous episodes of typical CW or CCW as well as atypical flutter. Ablation at the isthmus abolished typical flutter, but the atypical flutter persisted. Entrainment mapping at multiple sites showed evidence of manifest entrainment at the midseptum, CS ostium, and SI. Concealed entrainment was present only over the superior-posterolateral and inferior-posterolateral sections of the RA. Voltage mapping confirmed large areas of low voltage (<0.25 mV) over the posterolateral RA. Propagation and activation mapping showed a macroreentrant circuit between the low-voltage areas and the vena cava (Figure 8). Linear ablation from the “scar” to the IVC terminated the flutter.

**Changes of Flutter TCL**

In the present study, 14 patients had both typical CCW flutter and LLR; 4 patients had both typical CW flutter and ULR; 1 patient had typical CCW flutter and ULR; and 1
patient had typical CCW and CW flutter, as well as LLR and ULR. For the patients with both CCW and LLR, there was a statistically significant decrease in TCL comparing LLR (241±46 ms) with CCW (253±45 ms) (P=0.005). Similarly, compared with those with CW flutter (259±39 ms), those with ULR had a shorter TCL (250±36 ms) (P=0.029). These results are best explained by the shorter circuits of the atypical flutter patterns compared with the typical forms.

Discussion

Main Findings
In an unselected group of patients referred for ablation of AFL, we found that 8% had atypical right AFL patterns. These episodes generally (61%) occurred spontaneously from preexisting typical flutter patterns and tended to be sustained (89%). Our results indicated that these atypical patterns were a reflection of independent stable circuits and not transient patterns induced by rapid atrial pacing.

Patterns of LLR
An earlier study from our laboratory defined the characteristics of LLR.7 In that study, early breakthrough over the TA occurred over the low lateral RA. Our present study confirms and extends these observations. For example, we found that ≥1 annular break could occur at the lateral or anterolateral regions of the annulus. Proof that these circuits were isthmus dependent was shown by concealed entrainment and/or response to isthmus ablation. The observed patterns of atypical right AFL could not be artifacts due to spontaneous changes in catheter position because the surface ECG frequently showed changes in unison with changes in the endocardial recordings(Figures 3, 4B, and 7).

SI Short Circuit
Another novel finding was demonstration of circuits with early activation of the CS region. In this circuit, a typical CCW wave front negotiated the lateral portion of the isthmus and skirted posterior to the CS ostium and the septum. One possible explanation is the presence of a pectinate muscle band from the crista effectively separating the isthmus into anterior and posterior compartments. In all 4 patients, bidirectional isthmus block induced by RF lesion terminated the tachycardia.

Upper Loop Reentry Tachycardia
ULR is interpreted as the “converse” of LLR with a CW circuit and break over the lateral or anterolateral annulus with impulse collision in the isthmus. It should be emphasized that electroanatomic mapping studies were not available during ULR; hence, the precise confines of the circuit are not clear, although detailed entrainment mapping in 2 of these patients showed concealed entrainment at the posterior RA septal region between the FO and either the SVC or the IVC. Also, supportive of our schema for ULR is the finding of spontaneous conversion of either typical CW flutter or LLR to ULR (Figure 7). In 1 patient with LLR and multiple breakthroughs, conduction block over the isthmus was associated with the start of an ULR loop.

Previous Studies
Lower Loop Reentry
Our hypothesis explaining LLR has been verified by a number of other authors.3,5,11–15 The finding of collision sites along the lateral or superior TA as well as LLR with multiple breaks has actually been illustrated in a previous study (Figure 1 of Friedman et al15) Moreover, this important study showed that a posterior (intercaval) functional line of block formed the posterior barrier for typical flutter circuits while transverse conduction over the crista terminalis was present. They hypothesized that the flutter circuit was a result of competing wave fronts. Because the intercaval line was of variable length, activation of the posterior wall with penetration of the crista allowed for expression of different collision sites over the annulus. This formulation well explains our observations.

SI Short Circuit
Prior studies by Olgin et al16 and Nakagawa et al17 showed that breakthrough over the eustachian ridge posterior to the CS ostium may be observed in ≈25% to 50% of patients with typical forms of flutter. In these reports, there was almost simultaneous activation of the septum by wave fronts advancing both anterior and posterior to the CS. In the patients described in the present study, the only way to explain the proposed circuit is to postulate that the wave front advancing anterior to the CS is delayed sufficiently, allowing for collision with a return impulse from the CS that was previously conducted via the posteriorly directed wave front (Figure 5). It should be emphasized that bidirectional isthmus......
block in these patients always resulted in tachycardia termination.

**Upper Loop Reentry Tachycardia**

A prior report has described an atypical flutter circuit similar to our ULR.4 In addition, a very complete report by Shah et al,11 who used electroanatomic mapping, revealed a variable pattern of activation of the superior RA in patients with typical CCW flutter. They showed an apparent isthmus between the SVC and the superior portion of the TA. We hypothesize that ULR might use the channel between these structures.

**Scar Reentry**

We found 2 patients with atypical flutter circuits due to broad areas of low voltage found over the posterolateral portion of the RA. In both instances, tachycardia termination was accomplished by an RF lesion placed from the scar region to the IVC. Similar findings involving similar mechanisms in both the right and left atrium have been reported by others.8–10 On the basis of our limited observations, we cannot exclude the possibility that some of the patients with ULR may, in fact, have this pattern because of low-voltage or scarred areas.

**Limitations of the Study**

Our hypothetical circuits were derived largely from deductive reasoning based on typical flutter circuits. We appreciate that precise delineation of the tachycardia circuit(s) is not possible without advanced imaging techniques.

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