Electro-Anatomical Characteristics of Typical Atrial Flutter

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Type 1 atrial flutter (AFL) is a macroreentrant tachycardia in the right atrium; the anterior barrier of the common AFL circuit is located at the tricuspid annulus (TA) and the posterior border is functional line of block at the posteromedial (sinus venosa region) right atrium. The upper turnover site of the wave front is mainly located at the anterior to the superior vena cava. Conduction property across the posteromedial (sinus venosa region) right atrium in patients with and without AFL is different. i.e., functional conduction block occurs at the lower pacing rate in patients with AFL, and the majority of patients with chronic AFL demonstrate conduction block across the posteromedial (sinus venosa region) right atrium even during sinus rhythm. Catheter ablation therapy for AFL is creation of linear lesion between tricuspid annulus and inferior vena cava. Changes in activation sequence around the tricuspid annulus are used to confirm bidirectional block. However, it is difficult to demonstrate the bidirectional block in the presence of transverse conduction around the inferior vena cava. In such a case, bidirectional block should be confirmed by differential pacing or 3-dimensional mapping system.

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on AFL and thus provided a new technique for rapid identification of ablation success. They showed 1) that the EVR forms a line of fixed conduction block between the IVC and the coronary sinus, 2) that the TA provides boundaries for the AFL reentrant circuit, and 3) that verification of a complete line of block between the TA and the EVR is a fairly reliable criterion for long-term ablation success (Figure 1). We also studied the role of EVR as a conduction barrier of common type AFL by recording electrograms on the EVR during pacing below and above the EVR before and after IVC-TV linear ablation (Figure 2a–c; data presented at the 61st Annual Scientific Meeting of the Japanese Circulation Society, 1997). We also changed the strategy for radiofrequency ablation of common AFL from an electrogram-guided method12) to an anatomic approach using a halo catheter (Figure 3a, b; data presented at the 60th Annual Scientific Meeting of the Japanese Circulation Society, 1996, and the 13th Annual Meeting of Japanese Society of Electrocardiology, 1996).

Anterior and posterior borders of type 1 AFL

Nakagawa et al.14) and Kalman et al.15) demonstrated that the anterior barrier of the common AFL circuit is located at the TA. The posterior border was plotted by Olgin et al.16,17) by activation and entrainment mapping guided by intracardiac echocardiography, which identified the crista terminalis and EVR as posterior barriers (Figure 4). However, Friedman et al.18) reported 1) a functional line of block at the posteromedial (sinus venosa region) right atrium during counterclockwise (CCW) and clockwise (CW) AFL, 2) uniformity of lateral wall right atrial activation during flutter, and 3) activation at the site of posteromedial right atrial functional block resulting in subsequent initiation of isthmus-dependent AFL (Figure 5). We studied the relation between the anatomic location of the crista terminalis and double potentials recorded during type 1 AFL by detailed electrophysiologic and two-dimensional and three-dimensional intracardiac echocardiographic analysis and discovered that the functional line of block in CCW and CW AFL is located not at the crista terminalis but at its septal edge or at the posteromedial right atrium (sinus venosa region) (Figure 6a, b).19) Previous in-vitro studies revealed that the propagation velocity of the crista terminalis is approximately 10 times greater in the longitudinal direction than in the transverse direction.20) Thus, the crista terminalis might serve as an “express pathway” rather than a conduction barrier during type 1 AFL.

The upper turnover site in the reentry circuit of type 1 AFL

Tsuchiya et al.21) and Arribas et al.22) reported, on the basis of entrainment studies, that the upper turnover site of the reentry circuit of type 1 AFL is anterior to the orifice of the superior vena cava (SVC). However, recent studies have shown that the

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**Figure 1**

Schematic of the right atrium, as viewed in the right anterior oblique projection, illustrates the reentrant circuit in type 1 atrial flutter (arrows) and the role of the eustachian valve/ridge in forming a line of conduction block between the inferior vena cava (IVC) and the coronary sinus ostium (CS).

SVC: superior vena cava, TA: tricuspid annulus

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upper turnover portion of the reentry circuit for type 1 AFL is posterior to the SVC in one-quarter to one-third\textsuperscript{23} or even a majority\textsuperscript{24} of patients with type 1 AFL. We studied the upper turnover site in the reentry circuit of type 1 AFL by means of three-dimensional mapping and entrainment pacing around the SVC (Okumura et al., data to be presented at the Heart Rhythm Society’s 31\textsuperscript{st} Annual Scientific Sessions, 2010) and showed that the upper turnover site of the AFL circuit is located at the cranial and anterior portion of the SVC–right atrial junction in the majority of patients with type 1 AFL (Figure 7).

**Electroanatomic characteristics of the right atrial posterior wall and IVC-TV isthmus in patients with or without AFL with two- and three-dimensional intracardiac echocardiography**

We compared the electrophysiologic and anatomic characteristics of the right atrial posterior wall and IVC-TV isthmus in patients with or without type 1 AFL.\textsuperscript{25} A functional block line was located on the septal side of the crista terminalis in all patients, whether with or without AFL, but limited transverse conduction capability (Table 1) and age-related enlargement of the crista terminalis (measured from the short axis area; 16.4 ± 6.5 mm\textsuperscript{2}/m\textsuperscript{2} vs. 11.3 ± 6.4 mm\textsuperscript{2}/m\textsuperscript{2}) were related to the AFL.\textsuperscript{25} Matsuyama et al.\textsuperscript{26} examined human autopsy hearts and reported that the sinus venosa region showed age-related fibrofatty replacement of the musculature. Gonzalez et al.\textsuperscript{27} examined pig hearts and reported that the sinus venosa and not the crista terminalis results in a rate-dependent line of block during transverse right atrial activation and that the morphologic characteristics of the sinus venosa, i.e., thin muscle bundles with variable fiber orientation embedded in abundant strands of collagenous tissue, appear to facilitate block in this region. We then conducted a study comparing the anatomy of the IVC-TV isthmus

\begin{figure}
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\caption{Changes in atrial activation time across the eustachian valve/ridge (EVR) during pacing above and below the EVR before and after IVC-TA isthmus ablation.}
\end{figure}
between patients with and without type 1 AFL and characterizing the anatomy of the IVC-TV isthmus in the patients with ablation-resistant AFL. The
IVC-TV isthmus was shown to be significantly longer in patients with AFL than in those without (median length 24.6 mm; range 17.0–39.1 mm vs. median length 20.6 mm; range 12.5–28.0 mm), respectively (p < 0.05). In 5 patients with ablation-resistant AFL, a deep recess and prominent EVR were observed.

**Electroanatomic differences between chronic and paroxysmal AFL**

We compared intracardiac echocardiographic images of the crista terminalis and transverse conduction properties of the crista terminalis between patients with chronic (lasting >1 month) type 1 AFL and patients with paroxysmal type 1 AFL. In all patients with chronic AFL, the crista terminalis was thick and continuous from the SVC to the IVC, and conduction across the crista terminalis was blocked at a pacing rate just above sinus rhythm. In contrast, three-dimensional images from paroxysmal AFL showed the CT to be thin and discontinuous, and conduction across the crista terminalis during mid-septal pacing was observed in five of the eight patients. Thus, the nature of AFL is determined, at least in part, by anatomic and electrophysiologic characteristics of the crista terminalis (Figure 8a, b).²⁹

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**Figure 6**

a) Three-dimensional intracardiac echocardiogram (3D-ICE) showing crista terminalis (CT) and 20-pole electrode catheter (A 20) (left panels) and intracardiac electrograms recorded from the A 20 catheter (right panel). Note that intracardiac electrograms show double potentials during counterclockwise atrial flutter. The A 20 catheter was located at the posterior right atrium adjacent to the CT.

b) Three-dimensional intracardiac echocardiogram (3D-ICE) showing crista terminalis (CT) and 20-pole electrode catheter (A 20) (left panels) and intracardiac electrograms recorded from the A 20 catheter (right panel). Note that intracardiac electrograms show double potentials during counterclockwise atrial flutter. The A 20 catheter was located at the posteroseptal right atrium.
Increased inducibility of AFL in patients with atrioventricular nodal reentrant tachycardia

Previous reports documented association between atrioventricular nodal reentrant tachycardia and inducible AFL and a shared pathway between atrioventricular nodal reentrant tachycardia and AFL.\textsuperscript{30,31} We compared three-dimensional morphology of the coronary sinus ostium and inducibility of type 1 AFL in patients with and without atrioventricular nodal reentrant tachycardia.\textsuperscript{32,33} The area of the coronary sinus lumen measured at 15 mm into the coronary sinus did not differ significantly between patients with and without atrioventricular nodal reentrant tachycardia. However, the area of the

Table 1 Anatomic characteristics of the posterior right atrium on three-dimensional ICE images of control and AFL patients

<table>
<thead>
<tr>
<th></th>
<th>Control (n = 16)</th>
<th>AFL (n = 15)</th>
<th>p value</th>
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<tr>
<td>ERP/SW (ms)</td>
<td>220 (200–253)</td>
<td>600* (270–725)</td>
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<td>4</td>
<td>3</td>
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<tr>
<td>Long</td>
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<td>9</td>
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<tr>
<td>ERP/FW (ms)</td>
<td>215 (188–260)</td>
<td>280 (230–675)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Short</td>
<td>7</td>
<td>2</td>
<td></td>
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<tr>
<td>Medium</td>
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<td>8</td>
<td>0.07</td>
</tr>
<tr>
<td>Long</td>
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<td>5</td>
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ERP: effective refractory period of conduction, SW: mid-septal wall pacing, FW: mid-fee wall pacing.

The Mann-Whitney’s U-test or chi-square test was used for comparisons between the control and AFL patients.

\*p < 0.05 vs ERP/FW using Student’s t-test.

The ERP shown is the median value and interquartile range.

Short: <RA ERP, Medium: 600 ms>, >RA ERP, Long: ≥600 ms

Figure 7
Non-contact activation map of the right atrium during counterclockwise atrial flutter. Note that caudal to cranial activation of the posteroseptal right atrium was blocked at the sinus venosa region (panel 3), and the wavefront was directed from the posteroseptal right atrium to the anterolateral right atrium by turning around the anterior side of the superior vena cava (panel 4).
coronary sinus ostium was significantly larger in patients with atrioventricular nodal reentrant tachycardia than in those without. The coronary sinus ostium was flared in patients with atrioventricular nodal reentrant tachycardia, giving it a “windsock” appearance (Figure 9a, b). Type 1 AFL was induced in a greater percentage of patients with atrioventricular nodal reentrant tachycardia (53%) than in

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**Figure 8**
Comparison of the morphology of the crista terminalis (CT) in representative patients with paroxysmal and chronic atrial flutter (AFL). The CT on the three-dimensional intracardiac echocardiogram (3D ICE) of patients with chronic atrial flutter is thick and continuous from the superior vena cava to the inferior vena cava (a) and that of patients with paroxysmal AFL is thin and discontinuous (b).

RAA: right atrial appendage, FO: fossa ovalis

**Figure 9**
Comparison of the morphology of the coronary sinus by three-dimensional intravascular ultrasound (3D IVUS) in patients with and without atrioventricular nodal reentrant tachycardia (AVNRT). Note that the area of the coronary sinus ostium in the patient with AVNRT (b) is larger than that of the patient with concealed Wolff-Parkinson-White (WPW) syndrome (a), giving it a “windsock appearance.”

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patients without (27%). In patients with atrioventricular nodal reentrant tachycardia and inducible AFL before slow pathway ablation, AFL was also inducible after slow pathway ablation. There was no difference in the cycle length of induced AFL before and after ablation. Thus, there may be a common posteroseptal perinodal atrium area participating in the two tachycardia circuits; however, the width of the AFL circuit at the septal cavotricuspid isthmus should be larger than the circuit at the slow pathway location.

Relation between polarity of the flutter wave in the surface ECG and the endocardial atrial activation sequence in patients with type 1 AFL

There is general consensus on the distinct morphology of the 12-lead ECG during CCW type 1 AFL, i.e., a distinct “sawtooth pattern” with predominantly negative AFL waves in the inferior leads and V6 combined with a positive AFL wave in V1. Electrocardiographic features found to be most specific for CW type 1 AFL are more variable and include a predominantly positive AFL wave in the inferior leads and V6 as well as a short plateau phase with a wide negative component in the inferior leads and an overall negative component in V1. We compared polarity of the flutter wave in the surface ECG and the endocardial activation-atrial activation sequence in patients with type 1 AFL. CCW AFL with negative flutter wave in the inferior leads was characterized by proximal to distal coronary sinus activation and inferior to superior esophageal lead activation, and Bachmann’s bundle activation occurred later than the coronary sinus and esophageal electrograms. CCW AFL with a positive flutter wave in the inferior leads was characterized by Bachmann’s bundle activation that occurred earlier than the coronary sinus and esophageal electrograms, and the esophageal electrogram activation occurred in the superior to inferior direction. CW AFL with a positive flutter wave in the inferior leads was characterized by Bachmann’s bundle activation preceding activation in the esophageal leads and coronary sinus, esophageal electrogram activation occurring in the superior to inferior direction, and proximal to distal, middle to proximal and distal, proximal, or distal to middle coronary sinus activation. CW AFL with a negative flutter wave in the inferior leads was characterized by coronary sinus activation from the proximal to distal direction, and coronary sinus activation was either earlier than or simultaneous to Bachmann’s bundle activation. Esophageal electrograms were activated from the inferior to superior direction. Furthermore, we have reported two cases of type 1 CCW AFL in which changes in the left atrial activation sequence produced significant changes in flutter wave polarity without a change in the activation sequence within the right atrium. These findings highlight the possible role of alteration of the interatrial connections in the genesis of atypical manifestations of type 1 AFL (Figure 10).

Induction of type 1 AFL

We compared the inducibility of type 1 AFL by rapid atrial pacing from the coronary sinus ostium
and low lateral right atrium in 10 patients with AFL. CCW AFL was induced by pacing from the coronary sinus ostium in 6 of the 10 patients, and CW AFL was induced by pacing from the low lateral right atrium in 4 of the 10 patients. Conduction block at the IVC-TV isthmus was demonstrated in 6 of the 6 patients in whom CCW AFL was induced by pacing from the coronary sinus ostium, but 1 of the 4 patients in whom CW AFL was induced by pacing from the low lateral right atrium showed conduction block at the IVC-TV isthmus at the initiation of AFL (data presented at the 14th Annual Meeting of the Japanese Society of Electrocardiology, 1997).

Relation between atrial fibrillation and type 1 AFL

Roithinger et al. documented a remarkably stereotypical pattern of endocardial organization during spontaneous conversion from atrial fibrillation to AFL. Over 1313 seconds of fibrillation, there were 171 episodes of “organized atrial fibrillation.” Characteristics of the organized atrial fibrillation activation sequence may shed light on the mechanism underlying onset of clinical AFL. The more common appearance of CCW as opposed to CW AFL is explained by the predominant craniocaudal streaming of activation during organized AF.
activation of the right atrial free wall during organized atrial fibrillation in patients with the anatomic substrate for type 1 AFL (“streaming of activation,” Figure 11). We reported a case in whom conversion from atrial fibrillation to both CCW and CW AFL was observed.38)

Determination of IVC-TV isthmus block after type 1 AFL ablation

With intact IVC-TV isthmus conduction, ascending activation from pacing the low septal right atrium collides with descending activation on the anterior wall. With pacing from the low anterior right atrium, the opposite occurs—ascending and descending activation fronts collide on the right atrial septum. IVC-TV isthmus block after ablation prevents ascending activation on the right atrial wall opposite the pacing front (Figure 3a, b).39) Thus, we have reported a simple method for confirming IVC-TV isthmus block by changes in P wave polarity and the PR interval in the limb leads during low lateral right atrial pacing. During such pacing, P wave polarity in the limb leads shifted from negative to flat or positive or flat to positive after ablation. There was also an increase in P wave width and the stimulus-to-QRS wave interval, which was not observed during low right atrial septum pacing.40) However, ablation can cause long local conduction delays, indistinguishable from complete block,41) and transverse conduction across the crista terminalis during low septal or coronary sinus ostial pacing can produce a collision pattern on the anterior right atrial wall, suggesting conduction across the IVC-TV isthmus.21,42,43) Thus, demonstration of a complete line of double potentials along the ablation line is a fairly accurate modality for confirming complete linear block of the IVC-TV isthmus.44) However, on-site demonstration of clear-cut, widely spaced double atrial potentials is often difficult because of the ambiguity of electrogram interpretation along the ablation line.45) Shah et al.46) reported a simple method for distinguishing IVC-TV isthmus block from persistent conduction across an ablation line, i.e., the distal and proximal bipoles of a quadripolar catheter placed close to the ablation line were successively stimulated during recording from the ablation line. They hypothesized that because the initial and terminal components of local potentials reflected activation at the respective ipsilateral and contralateral borders of the ablation line, a change to a more proximal pacing site without moving the catheter would prolong the stimulus to the initial component timing, whereas the response of the terminal component would depend on the presence of block or persistent conduction. No change in the timing or a shortened terminal component would indicate block, whereas lengthening would indicate persistent gap conduction. Moreover, as shown previously, conduction block across the crista terminalis is associated with anisotropy, i.e., transverse conduction block across the crista terminalis occurs at a longer pacing cycle length or a longer coupling interval during pacing from the right atrial posterior

![Figure 12](image-url)

**Figure 12**
Assessment of inferior vena cava (IVC)–tricuspid annulus (TA) block by non-contact mapping system. Upper panel (a) shows changes in peak negative voltage of the virtual unipolar electrogram with wide-band filtering at three recording sites. Recording of a decrease in peak negative voltage at the mid-isthmus is the most specific technique to predict conduction block. Lower panel (b) shows that in the presence of transcristal conduction, an r’-wave pattern in the second component of unipolars at the ablation line indicates complete isthmus block.
wall than from the free wall. These differences may be a critical determinant of the CCW rotation of type 1 AFL. However, Scaglione et al. and Matsushita et al. described unidirectional CCW block at the isthmus, and Otomo et al. described CARTO® maps of transcristal conduction during low-rate proximal coronary sinus pacing in which activation sequence mapping by duodecapolar catheter showed an incomplete block pattern masquerading as persistent CW isthmus conduction in 9% of cases. Because Shah et al. paced only from the lateral side of the isthmus, we conducted “differential pacing” from posteroseptal and anterosetal right atrial pacing sites after linear ablation. Of 14 patients with type 1 AFL who exhibited incomplete conduction across the isthmus, 13 exhibited complete isthmus block in response to differential pacing, and 1 exhibited slow conduction across the isthmus. We also described a simple method for discriminating complete block from slow conduction across the isthmus. 

A duodecapolar halo catheter with 2-mm electrode spacing was placed at the isthmus, and bipolar electrograms were recorded with a band pass filter of 0.05–500 Hz. In 18 of 19 patients, the bipolar electrograms revealed CCW activation and negative polarity during proximal coronary sinus pacing only after complete IVC-TV isthmus block. In 1 of these 18 patients, additional applications of radiofrequency energy changed polarity of the bipolar electrograms from positive to negative although the conventional 2-8-2-mm halo electrogram activation sequence suggested complete isthmus block. In 7 patients who showed transverse conduction across the crista terminalis during proximal coronary sinus pacing, the conventional halo electrogram activation sequence suggested incomplete isthmus block; however, in 6 of these 7 patients, bipolar electrograms showed CCW activation with predominantly negative polarity. Thus, the polarity of wide range-filtered high-density bipolar electrograms recorded just lateral to the ablation line during proximal coronary sinus pacing may be used as a simple and an accurate indicator of complete isthmus block.

**Three-dimensional electroanatomic mapping systems**

Nakagawa et al. reported the utility of a three-dimensional nonfluoroscopic electroanatomic mapping system (CARTO) to examine the global right atrial activation pattern in patients during AFL, to localize the anatomic boundaries, and to create a complete line of conduction block by ablation across the subeustachian isthmus. Radiofrequency ablation across the subeustachian isthmus is performed during coronary sinus pacing. Beginning at the TA, the ablation electrode is moved toward the EVR in 2-3-mm increments. Each move is marked on the right atrial map to depict the ablation line. In the event of residual conduction across the ablation line, defects in the ablation line are located by mapping the previous ablation sites, guided by the CARTO system, to locate the transition from the double atrial potentials (indicating block) to a single atrial potential (indicating conduction). Radiofrequency ablation of the site of the single atrial potential along the ablation line produces complete conduction block across the subeustachian isthmus. Thus, the CARTO electroanatomic mapping system allows precise three-dimensional localization of the anatomic boundaries of the AFL reentrant circuit, and it facilitates ablation by accurately depicting defects in the ablation line.

Lin et al. reported both an optimal electrogram recording technique for detecting acute ablative tissue injury and the characteristics of virtual unipolar electrograms for detecting isthmus block during radiofrequency ablation of type 1 AFL with the use of a noncontact mapping system (Ensite 3000, Endocardial Solutions, St. Paul, MN). We tested their hypothesis and showed that peak negative voltage decrease with wide-band filtering (0.5-IC 150 Hz) was the most specific recording technique for predicting conduction block (Figure 12a, data presented at the 25th Annual Meeting of the Japanese Society of Electrocardiology, 2008), and a predominant R-wave pattern in the second component of unipolar potentials at the ablation line indicates complete isthmus block, even in the presence of transcristal conduction (Figure 12b, data presented at the Japanese Arrhythmia Society, 2008).

**References**


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