Global Right Atrial Mapping of Human Atrial Flutter: The Presence of Posteromedial (Sinus Venosa Region) Functional Block and Double Potentials

A Study in Biplane Fluoroscopy and Intracardiac Echocardiography

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Background—Previous studies of atrial flutter have found linear block at the crista terminalis; this was thought to predispose the patient to the arrhythmia. More recent observations, however, have demonstrated crista conduction. We sought to characterize the posterior boundary of atrial flutter.

Methods and Results—Patients with counterclockwise flutter (n = 20), clockwise flutter (n = 3), or both (n = 5) were studied using two 20-pole catheters. Biplane fluoroscopy determined catheter positions. During counterclockwise flutter, craniocaudal activation occurred along the entire lateral and posterior right atrial walls. Septal activation proceeded caudocranially. In all patients, a line of block was seen in the posteromedial (sinus venosa) right atrium; this was manifested by the presence of double potentials where the upward and downward activations collided. Anatomic location was confirmed by intracardiac echocardiography in 9 patients. In patients with clockwise flutter, the line of block and double potentials were seen in the same location during counterclockwise flutter, but the activation sequence around the line of block was reversed. Pacing near the site of double potentials during sinus rhythm excluded a fixed line of block, and premature atrial complexes demonstrated functional block with manifest double potentials. In 2 patients, posterior ectopy organized to subsequently initiate isthmus-dependent atrial flutter.

Conclusions—(1) A functional line of block is seen at the posteromedial (sinus venosa region) right atrium during counterclockwise and clockwise atrial flutter. (2) All lateral wall right atrial activation can be uniform during flutter, without linear block or double potentials in the region of the crista terminalis. (3) Activation at the site of posteromedial right atrial functional block can organize to subsequently initiate isthmus-dependent atrial flutter. (Circulation. 2000;101:1568-1577.)

Key Words: atrial flutter • catheter ablation • intracardiac echocardiography • crista terminalis

Atrial flutter results from a right atrial macroreentrant circuit bounded anteriorly by the tricuspid valve annulus. In typical human counterclockwise flutter, the wavefront proceeds in a lateral to septal direction along the isthmus of tissue between the inferior vena cava and tricuspid valve, passes between the tricuspid annulus and Eustachian ridge, and then propagates up the septum in a caudocranial direction. A linear ablation lesion between the tricuspid annulus and the inferior vena cava or between the tricuspid annulus and the Eustachian ridge transects the critical isthmus of tissue, terminates atrial flutter, and predicts long-term freedom from recurrence. Additionally, using intracardiac echocardiography (ICE), some authors have found that the postero-lateral boundary of the atrial flutter circuit is the crista terminalis and that double potentials are seen at the crista terminalis during atrial flutter as the craniocaudal impulses anteriorly collide with the caudocranial wavefront posteriorly at this nonconductive barrier (Figure 1A). It has been proposed that conduction block at the crista terminalis is critical for the predisposition to and maintenance of atrial flutter as it prevents the flutter circuit from “short circuiting” itself in the posterolateral right atrium.

Several recent observations, however, have suggested that the crista terminalis may not be a barrier to conduction in isthmus-dependent atrial flutter, and thus may not be critical in arrhythmia mechanism. First, a variant of atrial flutter, lower loop reentry, has been described by Cheng et al. Lower loop reentry uses the sub-Eustachian isthmus, but the circuit encircles the inferior vena cava, penetrates the crista terminalis, and subsequently “splits” to both return across the
Figure 1. A, Global right atrial activation as defined in Reference 6. Note that the crista terminalis serves as a posterolateral line of block and that double potentials are seen at the crista as the craniocaudal wavefront strikes the crista anteriorly and a caudocranial wavefront strikes it posteriorly. B, Wavefront propagation during lower loop reentry.10 The isthmus is activated in a lateral-to-septal direction, as with typical atrial flutter. Activation then spreads posteriorly around inferior vena cava and penetrates the crista terminalis, demonstrating transverse crista permeability. The wavefront then splits, with one branch returning across the isthmus to close the dominant loop and the other ascending the anterolateral right atrium, where it collides with counterclockwise activation in the superior right atrium. ROV indicates roving catheter; numbers are as explained in C. C, Intracardiac electrograms from a patient with spontaneous lower loop reentry. Catheter positions are shown in B; electrode pairs are labeled consecutively beginning with I1,2 at distal end of catheter. Throughout tracing, isthmus activation proceeds from lateral (I19,20) to septal (I1,2). In the first half of the tracing, lateral wall activation on roving (ROV) catheter is caudocranial, with simultaneous activation of the distal leads, suggesting fusion (at ROV 15,16) between lateral low-to-high activation and a counterclockwise wavefront exiting the sub-Eustachian isthmus. This is consistent with lower loop reentry around the inferior vena cava. Asterisks at ROV 1,2 show point of earliest lateral wall activation, indicating a wavefront breaking through the crista terminalis at that point. At the point at which the cycle length increases to 305 ms and asterisks move to ROV 19,20, conduction no longer penetrates through the low crista terminalis, lateral wall activation becomes craniocaudal, and the cycle length increases as circuit length increases beyond that of the inferior vena cava circumference to that of the typical flutter circuit around the tricuspid annulus. In subsequent cycles, early lateral activation is seen at both ROV 19,20 and ROV 7,8, suggesting that wavefronts are now penetrating the crista at ROV 7,8 but with enough delay for a wavefront to reach ROV 19,20 first. Nonetheless, because of “shortcut” at ROV 7,8, cycle lengths (measured at the lateral isthmus) are shortened, although they are still generally longer than when earliest activation was lower at ROV 1,2. Lower loop reentry is described in greater detail elsewhere.10 RV indicates right ventricle; HIS, His bundle; CS, coronary sinus; PROX, proximal; and DIST, distal.
isthmus and ascend the anterolateral right atrium (Figure 1, B and C). This is a stable rhythm, occurs spontaneously, is isthmus dependent, and has crista penetration at various atrial sites within the same patient.10 The occurrence of lower loop reentry demonstrates the absence of crista terminalis conduction block during atrial flutter, indicating that conduction block at the crista terminalis is not necessary for arrhythmia maintenance.

Additional evidence has also suggested that the crista terminalis may not always function as the posterior boundary during human atrial flutter. In preliminary mapping studies of atrial flutter using multipolar catheters, biplane fluoroscopy, and ICE, we found double potentials (a sign of linear block)1,2 in the posteromedial (sinus venosa) right atrium, the smooth-walled region posterior to the crista terminalis. The purpose of the present study, therefore, was to define the posterior boundary of typical isthmus-dependent atrial flutter and to determine whether this posterior boundary is associated with specific anatomical barriers. Additionally, this study sought to determine whether the boundary is fixed or functional.

Methods

Patient Characteristics

A total of 28 consecutive patients (22 men and 6 women) with atrial flutter who underwent global right atrial mapping were included in the study. Patients had a mean age of 61 ± 13 years (range, 25 to 81 years). Nine patients had coronary artery disease, and 6 of these patients had previous bypass surgery. Four patients had previous atrial septal defect repair; 5 patients had hypertension; 5 patients had an ejection fraction ≤ 40% (range, 35% to 40%); and 7 had structurally normal hearts. The mean ejection fraction of all patients was 54 ± 11% (range, 35% to 79%).

Electrophysiological Study and Global Right Atrial Mapping

Patients were brought to the electrophysiology laboratory in the postabsorptive state and were lightly sedated with fentanyl, diaze-
pam, and midazolam. All antiarrhythmic drugs had been discontinued for ≥5 half-lives, except in 2 patients who discontinued amiodarone 5 and 14 days before the procedure. Oxygen saturation and blood pressure were continuously monitored. A 7-French orthogonal catheter was advanced into the coronary sinus via the right internal jugular vein. Via the femoral veins, a 5-French quadripolar catheter was placed in the right ventricle, a 6-French octapolar catheter was placed near the His bundle, and two 7-French 20-pole deflectable mapping catheters were positioned in the right atrium. These 20-pole catheters had 10 electrode pairs with 1 mm of distance within pairs and 4 mm of spacing between pairs. All intracardiac electrograms were displayed at 5 to 20 mV/cm after filtering from 30 to 500 Hz.

**Global Right Atrial Mapping**

One of the 20-pole mapping catheters (the isthmus catheter) was positioned via a long guiding sheath (SR0, Daig, Inc) across the sub-Eustachian isthmus; it was kept there throughout the study. The second 20-pole catheter (the roving catheter) was initially placed at the anterolateral tricuspid valve annulus, and subsequently repositioned around the atrium to record 5-mm lengths of right atrial activation, resulting in a global map (Figure 2). Standardized recordings were obtained at the anterolateral, lateral, posterolateral (in the region of the crista terminalis), posterior, posteromedial (sinus venosa region), septal (vicinity of the fossa ovalis), and anteroseptal right atrium. Catheter positions were recorded via biplane cine fluoroscopy (right and left anterior oblique positions), and electrograms were stored on an electrophysiology workstation (Prucka, Inc).

Only patients with isthmus-dependent atrial flutter, as determined by surface ECG tracings, isthmus conduction during arrhythmia, and arrhythmia termination during isthmus ablation, were included. In 11 patients, entrainment studies were performed to confirm the arrhythmia mechanism; in another 17 patients, a global right atrial map consistent with isthmus-dependent flutter was thought to be sufficient with confirmation by arrhythmia termination during isthmus ablation. Patients with previous surgery who had scar reentry were excluded from this study.

**Localizing Posterior Block in Atrial Flutter**

While continuously recording electrograms, the roving catheter was swept from the anterolateral to posterolateral and then posteromedial right atrium. The site at which double potentials were recorded and a change in activation sequence from craniocaudal to caudocranial occurred (during CCW flutter) was taken as the posterior line of block. In CW flutter, the transition occurred in the opposite direction. Biplane fluoroscopy determined catheter position in all patients. In the left anterior oblique view, catheter positions medial to the SVC (as determined by the coronary sinus catheter in the SVC) were defined as septal; those lateral to the SVC were defined as lateral (Figure 2). Anterior and true posterior (ie, trabeculated versus sinus venosa region) positions were defined by the right anterior oblique image.

**ICE**

In 9 patients, ICE was used to determine catheter position relative to intracardiac structures (5 patients had coronary artery disease and 2 had an atrial septal defect). In the first 4 patients, a 6.2-French 12.5-MHz catheter (Boston Scientific) was used; in all subsequent
patients, a 9-French 9-MHz catheter was employed (Boston Scientific). With the roving catheter positioned at the posterior line of block during atrial flutter, the ICE catheter was advanced to the SVC and gradually withdrawn. The position of the roving catheter (continuously recording double potentials to confirm catheter stability at line of block) relative to intracardiac structures and fluoroscopic location were noted. The position of the crista terminalis (clearly seen at the junction of the smooth and pectinated right atrium) and the relationship of the intracardiac catheters to the crista terminalis were noted throughout the ICE study.

**Results**

During the course of the electrophysiological study, 20 patients had only counterclockwise (CCW) flutter, 3 patients had only clockwise (CW) flutter, and 5 patients had both. The mean atrial cycle length during CCW flutter was $263\pm57$ ms; during CW flutter, it was $260\pm53$ ms. In 23 patients with CCW atrial flutter, the posterior line of block occurred in a true posteroseptal (sinus venosa region) location, with double potentials seen medial to the superior vena cava (SVC) and posterior to the region of the fossa ovalis (Figure 2). In 2 patients, double potentials were seen at a true posterior position, with the roving catheter in line with the SVC. In all 6 patients in whom the global map was completed during the occurrence of CW atrial flutter, the posterior line of block was posteroseptal. In each of the 3 patients with both CCW and CW atrial flutter, the line of block occurred in the same (posteroseptal) location during CCW and CW flutter. In all patients, linear block and double potentials were also seen along the Eustachian ridge.

Figure 2 is an image-based table of the global right atrial map in a patient who had both CCW and CW atrial flutter during the course of the electrophysiological study. In that patient, activation propagated in a cranio-caudal direction along the entire lateral wall and the true posterior wall; caudocranial activation was not apparent until the roving catheter was placed in a posteromedia position, indicating that propagation occurred caudocranially in parallel along both sides of the crista terminalis during typical atrial flutter. During CW flutter, double potentials were seen at the same location, and activation spread caudocranially along the entire lateral wall and craniocaudally along the septum (Figure 3). Additionally, the double potentials formed an upside-down “V” configuration (Figure 2, C4 and C5), demonstrating a superior boundary to the line of block, around which wavefront propagation occurred.

ICE demonstrated that the posterior line of block occurred in the posteromedia right atrium in the sinus venosa region. Figure 4 shows the fluoroscopic, ICE, and electrographic data in a patient during typical CCW flutter. As seen in Figure 4, the double potentials occurred approximately midway between the fossa ovalis and the crista terminalis. Quantitative measurements were available in 7 of 9 patients who had ICE. With measurements taken at the midfossa level, the distance from the crista terminalis to the site of double potentials was $2.7\pm0.7$ cm; the distance from the double potentials to the posterior rim of the fossa ovalis was $1.8\pm0.6$ cm.

In 6 patients (3 with only CCW flutter, 3 with CCW and CW flutter), the arrhythmia terminated at a time when the roving catheter was positioned at the line of block. In all 6 patients, a separate pacing catheter was placed adjacent to the roving catheter, and extrastimuli were delivered to assess...
local conduction. In all patients, no double potentials were recorded on the roving catheter during normal sinus rhythm or with pacing at long cycle lengths; thus, conduction proceeded from the pacing catheter across the sinus venosa region of the right atrium at which the roving catheter was located without interruption (ie, no line of block). With the placement of premature atrial complexes and with spontaneous premature atrial complexes, double potentials appeared, demonstrating functional block (Figure 5). In 2 patients, sinus venosa atrial extrastimuli initiated atrial flutter. Interestingly, a reentrant arrhythmia with double potentials appeared in the sinus venosa region before the occurrence of organized reentry at the isthmus, demonstrating that at times, sinus venosa functional block with premature complexes may lead to local reentry, which is subsequently followed by stable isthmus-dependent atrial flutter (Figure 6).

**Discussion**

**Key Findings**

With the integrated use of multipolar catheters, biplane fluoroscopy, and ICE, we found double potentials (a sign of linear block)\(^1\,^2\) in the posteromedial sinus venosa right atrium, removed from the crista terminalis, with uniform cranio-caudal conduction on either side of the crista terminalis during typical flutter. With the mapping catheter in the posteromedial right atrium, minor lateral deflection (to the true posterior smooth right atrium) demonstrated cranio-caudal activation during typical flutter, and medial deflection toward the septum demonstrated caudo-cranial activation (Figure 3). Posteromedial double potentials, 2 distinct atrial

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**Figure 3.** Composite of global right atrial activation during CCW atrial flutter, taken from mapping depicted in Figure 2.
Figure 4. Integrated biplane fluoroscopy, multipolar catheters, and intracardiac echocardiography identify anatomic region of block. In A, right anterior oblique fluoroscopic view at site of double potentials is shown. ICE indicates intracardiac echo catheter, and I sheath, guiding sheath through which isthmus catheter passes. Roving and isthmus catheters are labeled as in Figure 2, with proximal poles 19,20 and distal poles 1,2 identified. B is a computer model made from fluoroscopic and ICE images showing catheter positions in the right anterior oblique view. C and D represent left anterior oblique fluoroscopic and computer model views, respectively. Note that roving catheter is in the posteromedial right atrium in the sinus venosa region. E shows ICE image obtained with catheters in positions shown. Distal roving catheter is in the sinus venosa region, equidistant between crista terminalis and fossa ovalis. F shows electrograms recorded by the roving catheter at position shown—double potentials are present as ascending and descending wavefronts collide at a line of block. Surface leads and isthmus catheter show ongoing typical CCW atrial flutter. At Rov 5,6, double potentials fuse, indicating that this is the terminus or end of the line of block. At more cranial electrodes (such as Rov 9,10), no double potentials exist, indicating that the electrodes are above the end of the line of block. PRES I indicates blood pressure (tracing not shown); other abbreviations as in Figure 2.
potentials separated by an isoelectric interval, were seen as electrograms caused by caudocranial propagation; when these waves collided with the line of block from the septal side, the wavefront propagated over the top of the line of block and then proceeded from a craniocaudal direction to collide with the line of block from the lateral side (Figure 3). The observation of a line of block removed from the crista, and identical craniocaudal propagation on either side of the crista, makes it unlikely that crista terminalis impermeability is causative in the pathophysiology of atrial flutter in these patients. Additionally, the line of block was functional, as demonstrated by the absence of double potentials with pacing adjacent to the roving catheter at the line during sinus rhythm and the appearance of double potentials with premature atrial complexes.

Although many investigators have noted double potentials during atrial flutter and their association with a line of block,1-3 ICE permits the correlation of the electrical phenomenon with intracardiac structures.6,7 In a series of elegant studies using ICE, Olgin et al6-9 and Kalman et al11 found the crista terminalis was the posterolateral boundary in human flutter, in contrast to the present finding of sinus venosa block. It is unclear why our results differed from theirs. There is likely heterogeneity in right atrial activation outside of the sub-Eustachian isthmus in patients with atrial flutter.12 This might explain why lower loop reentry, which clearly proves crista terminalis permeability to transverse wavefront propagation, was seen in some (6 of 20), but not all, patients in the study by Cheng et al10 Similarly, in a study assessing crista conduction in patients with and without atrial flutter, nearly half of flutter patients demonstrated crista terminalis permeability.13 This again suggests variability in crista terminalis conduction in patients with atrial flutter. Because all of our patients had sinus venosa linear block, we were unable to determine whether any structural or patient characteristics predicted which patients might have crista impermeability.

The posterior line of block we found formed an inverted V pattern, suggesting an upper limit or terminus to the line of block. In the present study, we were unable to determine the exact relationship of this line of block to the SVC; however, in a study using electroanatomical mapping,12 the flutter circuit was constrained by a superior isthmus (between the tricuspid annulus and SVC) in some patients but not others, demonstrating that at least in a subpopulation of flutter patients, the posterior line of block does not extend superiorly to the SVC. Interestingly, in that report, double potentials
were seen in the posterior right atrium in a position corresponding to the location of double potentials in the present report (Figure 4 in Reference 12). That study, like ours, showed variability in the flutter circuit outside of the sub-Eustachian isthmus.

Given the known occurrence of lower loop reentry in some patients, it can be postulated that flutter macro reentry is composed of 2 broad and competing pathways with a figure-of-eight type anatomy—one circuit follows the tricuspid annulus (as shown in Figure 3), and the other flows around the inferior vena cava (the lower loop reentry circuit, Figure 1B). The degree of posterior functional block and crista terminalis permeability would determine which circuit dominates. The combination of crista permeability and a short line of block at the sinus venosa region may make available a shorter (and hence dominant) circuit around the inferior vena cava rather than the “usual” circum-tricuspid circuit. Because the sub-Eustachian isthmus is critical to both circuits, successful isthmus ablation would not distinguish between the 2 variants of isthmus-dependent flutter. Because the flutter wave is predominantly determined by left atrial activation and both variants have similar surface lead appearances, surface electrocardiography cannot readily distinguish the forms either. The concept of variability of the flutter circuit outside of the sub-Eustachian isthmus—and the inability to observe this variability by isthmus ablation or by surface electrocardiography—has been supported by electroanatomical 3D mapping.

There has been great interest in determining which anatomic boundaries may be critical for or predispose a patient to the development of intra-atrial arrhythmias. This study found that outside of the isthmus region, functional properties may be as important as anatomic boundaries in human atrial flutter. This is consistent with other reports in which a site-dependent atrial conduction delay in patients prone to intra-atrial arrhythmias has been described and in which an alteration of functional characteristics by dual-site atrial pacing may account for the possible benefit of that tech-

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**Figure 6.** Posterior initiation of atrial flutter during lateral right atrial pacing. Catheters are positioned as in Figure 3, with roving catheter adjacent to the line of posteroseptal block. The last organized activation across the isthmus from a lateral to septal direction is the last paced wavefront (this starts with the second electrogram on I 19,20 after vertical line). Subsequently, posteromedial double potentials appear (asterisks on Rov catheter electrograms), demonstrating development of functional block and suggesting posterior reentry. After 2 disorganized isthmus activations, CW isthmus-dependent atrial flutter begins (first organized isthmus activity is demarcated by +, and arrow demonstrates septal to lateral propagation). Note that posteroseptal double potentials develop before organization of isthmus conduction into isthmus-dependent flutter.
nique.15 Posteromedial functional block may occur because embryologically right and left atrial myocytes with differences in electrophysiological properties join in this region. Interestingly, in 2 patients, extrastimuli led to sinus venosa linear block and double potentials that were followed by organized isthmus-dependent flutter only after several posterior reentrant cycles (Figure 6). This suggests that posterior functional block may support rotors of reentry that initiate atrial flutter, in addition to its required presence for preventing the posterior “collapse” of the flutter circuit. Other investigators have observed that atrial flutter often begins with a brief episode of atrial fibrillation, the development of functional posterior block, and subsequent stable flutter.16 These observations are consistent with our finding of functional posterior block during atrial flutter, which at least in some cases precedes the establishment of stable flutter.

Conclusions
We found that a functional line of block is present at the posteromedial (sinus venosa) right atrium during counterclockwise and clockwise atrial flutter. The absence of double potentials in the region of the crista terminalis suggested that crista terminalis block was not required for the maintenance of atrial flutter in these patients. Outside of the sub-Eustachian isthmus, functional characteristics may be important for atrial flutter initiation and for the determination of whether flutter preferentially occurs around the tricuspid annulus or the inferior vena cava (lower loop reentry).

References
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