Different Patterns of Interatrial Conduction in Clockwise and Counterclockwise Atrial Flutter

Joseph E. Marine, MD; Victoria J. Korley, MD; Ogundu Obioha-Ngwu, MD; Jane Chen, MD; Peter Zimetbaum, MD; Panos Papageorgiou, MD, PhD; Paul Milliez, MD; Mark E. Josephson, MD

Background—The terms counterclockwise (CC) and clockwise (C) atrial flutter (Afl) are used to describe right atrial activation around the tricuspid valve in the left anterior oblique view. The manner in which the left atrium is activated, as reflected by coronary sinus (CS) recordings, has not been systematically evaluated.

Methods and Results—Nine patients with both CC and C Afl underwent electrophysiological study with CS recordings during both rhythms with the use of a decapolar catheter with the tip placed in the distal CS. Patterns of CS activation during each type of Afl as well as during sinus rhythm were categorized into 1 of 3 patterns: sequential proximal-to-distal, sequential distal-to-proximal, and fused, indicating activation from different directions. In 7 of 9 patients, the pattern of CS activation in CC Afl and C Afl differed, with a proximal-to-distal pattern in CC Afl and a fused pattern in C Afl. In 2 patients, pacing the high right atrial septum near the presumed site of Bachmann’s bundle in sinus rhythm showed a similar fused pattern of CS activation.

Conclusions—These results demonstrate different patterns of CS activation in CC Afl and C Afl in the majority of patients and are consistent with a model in which the left atrium is activated predominantly over Bachmann’s bundle during C Afl and over the CS os in CC Afl. These findings may have implications for maintenance of Afl, interpretation of flutter wave morphology on surface ECG, and left atrial mechanical function in Afl. *(Circulation. 2001;104:1153-1157.)*

Key Words: atrial flutter ● conduction ● arrhythmia

Because of renewed interest in the mechanism and treatment of atrial arrhythmias, a better understanding of interatrial conduction is of paramount importance. Several patterns of interatrial conduction during sinus rhythm (SR) and atrial pacing have been identified by the use of Bachmann’s bundle (BB), the fossa ovalis, and the coronary sinus (CS) os as breakthrough points. Patterns of interatrial conduction during typical isthmus-dependent atrial flutter (Afl) are not well understood.

Typical Afl has been shown to involve a reentrant circuit in the right atrium revolving around the tricuspid annulus, which forms the anterior border. The flutter circuit is bounded posteriorly by the Eustachian ridge and crista terminalis. The left atrium is passively activated and is not necessary for maintenance of the tachycardia. The terms counterclockwise (CC) and clockwise (C) are used to describe right atrial activation around the tricuspid valve in the left anterior oblique (LAO) view. We evaluated patterns of left atrial activation, as reflected by CS recordings, in patients with both C and CC Afl.

Methods

The study population consisted of 9 patients undergoing electrophysiological study for typical isthmus-dependent Afl. All patients gave written informed consent; the study was approved by our institutional review committee, and procedures followed were in accordance with institutional guidelines. The 9 patients underwent electrophysiological study with CS recordings during both C and CC Afl with the use of a decapolar catheter with 5-mm interelectrode spacing with the distal tip placed at the furthest site at which discrete atrial activity could be recorded. Catheters were withdrawn as needed to evaluate the entire CS. For purposes of comparing CS activation, the CS catheter was placed in a stable position in the distal CS in each patient. The tip of the catheter recorded a large ventricular deflection and was typically at the 12 to 1 o’clock position in the 60° LAO projection, with the proximal pair at approximately the 4 o’clock position. In 2 patients (patients 1 and 5), the CS catheter could not be passed beyond the 3 o’clock position. A 10- to 20-pole catheter was positioned anterior to the crista terminalis along the anterolateral right atrium, with tip in the lateral cavoatricuspid isthmus. This catheter was also anterior to the CS catheter in the right anterior oblique (RAO) view. Quadrupolar catheters were placed in the His bundle region and in the right ventricular (RV) apex. C and CC Afl were defined according to standard criteria, based on right atrial activation pattern in the LAO view. Isthmus
dependence of atrial flutters was proven by (1) demonstrating concealed entrainment of flutter by stimulation in the isthmus; (2) observing the postpacing interval within 10 to 15 ms of flutter cycle length after isthmus pacing; and (3) terminating and preventing initiation of flutter by the production of bidirectional block in the isthmus by radiofrequency ablation. Two patients underwent electroanatomic mapping of the right atrium in both CC and C Afl with the use of the Biosense CARTO system (Cordis-Webster). This provided detailed analysis of the right atrial septal activation in C and CC flutter. Both patients also had CS recordings made during pacing of the high right atrial septum, near Bachmann’s bundle, during sinus rhythm.

Studies were recorded on optical disk by standard digital recording systems (Prucka Engineering and Bard Electrophysiology). Patterns of CS activation during each type of Afl, as well as during sinus rhythm (SR), were categorized into 1 of 3 theoretically possible patterns: sequential proximal-to-distal (PD), sequential distal-to-proximal (DP), and fused (F), indicating activation from different directions. A schematic diagram of these 3 patterns is shown in Figure 1. Timing of activation along the CS catheter and atrial cycle lengths were measured in each patient during each rhythm, taking a mean of 10 consecutive intervals.

Results
The patients’ clinical characteristics are shown in Table 1. There were 7 men and 2 women studied, with a mean age of 69±12 years. Five patients had structural heart disease and 4 were taking class I or class III antiarrhythmic drugs for treatment of atrial fibrillation. The mean cycle length (CL) of CC Afl in all patients was 250±34 ms, and the mean cycle length of C Afl was 255±36 ms.

Results of assessment of CS activation are shown in Table 2. In 7 of the 9 patients studied, the pattern of CS activation differed between CC and C Afl. During CC Afl, CS activation was PD all 9 patients. During C Afl, CS activation was fused in 7 patients and PD in 2 patients. CS activation pattern during C Afl was similar to that in normal sinus rhythm (NSR) in all patients. Timing of CS activation was similar (<10 ms difference in proximal coronary sinus bipole and distal coronary sinus bipole activation) between C Afl and NSR in 7 of 9 patients. Comparing morphology and polarity of CS bipoles between C Afl and NSR showed 5 of 5 match in 4 patients and 4 of 5 match in an additional 4 patients. Comparison of CS recordings of 2 typical patients (patients 8 and 9) during CC Afl, C Afl, and NSR are shown in Figure 2.

Two patients (patients 6 and 7) underwent pacing (at 300 ms) of the high right atrial septum, near the presumed site of BB during sinus rhythm. The results, illustrated in Figure 3, show that CS activation during BB pacing is similar to that during C Afl (showing a fused pattern in each case) and different from that during CC Afl (showing a PD pattern).

The same two patients underwent electroanatomic mapping of the right atrium during both CC and C Afl with the

![Figure 1. Schematic diagram of patterns of CS activation.](image)

<table>
<thead>
<tr>
<th>Patient</th>
<th>CC CL, ms</th>
<th>C CL, ms</th>
<th>CC CS Pattern</th>
<th>C CS Pattern</th>
<th>NSR CS Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>286</td>
<td>301</td>
<td>PD 40</td>
<td>PD 37</td>
<td>F 9 -10</td>
</tr>
<tr>
<td>2</td>
<td>233</td>
<td>288</td>
<td>PD 32</td>
<td>F 9</td>
<td>F 10 -10</td>
</tr>
<tr>
<td>3</td>
<td>226</td>
<td>225</td>
<td>PD 67</td>
<td>F 19</td>
<td>F 20 -10</td>
</tr>
<tr>
<td>4</td>
<td>302</td>
<td>302</td>
<td>PD 71</td>
<td>F 13</td>
<td>F 15 -10</td>
</tr>
<tr>
<td>5</td>
<td>202</td>
<td>197</td>
<td>PD 17</td>
<td>PD 19</td>
<td>PD 41 -10</td>
</tr>
<tr>
<td>6</td>
<td>275</td>
<td>245</td>
<td>PD 30</td>
<td>F 16</td>
<td>F 10 -10</td>
</tr>
<tr>
<td>7</td>
<td>272</td>
<td>262</td>
<td>PD 86</td>
<td>F 16</td>
<td>F 19 -10</td>
</tr>
<tr>
<td>8</td>
<td>230</td>
<td>245</td>
<td>PD 47</td>
<td>F 16</td>
<td>F 19 -10</td>
</tr>
<tr>
<td>9</td>
<td>222</td>
<td>230</td>
<td>PD 40</td>
<td>F 10</td>
<td>F 10 -10</td>
</tr>
</tbody>
</table>

Mean±SD 250±34 255±36

CC Afl indicates counterclockwise atrial flutter; C Afl, clockwise atrial flutter; CS, coronary sinus; NSR, normal sinus rhythm; PD, proximal-to-distal activation pattern; F, fused activation pattern. Numbers next to patterns represent timing, in milliseconds, from proximal to distal bipole of the CS catheter.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Sex</th>
<th>Structural Heart Disease</th>
<th>LVEF, %</th>
<th>LA Size, cm</th>
<th>AADs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>77</td>
<td>F</td>
<td>CAD/CABG, AVR</td>
<td>NA</td>
<td>NA</td>
<td>BB</td>
</tr>
<tr>
<td>2</td>
<td>66</td>
<td>F</td>
<td>None</td>
<td>NA</td>
<td>NA</td>
<td>BB, verapamil</td>
</tr>
<tr>
<td>3</td>
<td>73</td>
<td>M</td>
<td>CAD/CABG, AVR</td>
<td>40</td>
<td>4.6</td>
<td>Amiodarone, BB</td>
</tr>
<tr>
<td>4</td>
<td>83</td>
<td>M</td>
<td>CAD/CABG, HCM</td>
<td>35</td>
<td>3.9</td>
<td>Dig, diltiazem</td>
</tr>
<tr>
<td>5</td>
<td>61</td>
<td>M</td>
<td>None</td>
<td>60</td>
<td>4.3</td>
<td>BB</td>
</tr>
<tr>
<td>6</td>
<td>68</td>
<td>M</td>
<td>None</td>
<td>50</td>
<td>4.2</td>
<td>Sotalol, diltiazem</td>
</tr>
<tr>
<td>7</td>
<td>46</td>
<td>M</td>
<td>DCM</td>
<td>30</td>
<td>5.8</td>
<td>Flecainide, BB</td>
</tr>
<tr>
<td>8</td>
<td>65</td>
<td>M</td>
<td>None</td>
<td>60</td>
<td>4.0</td>
<td>None</td>
</tr>
<tr>
<td>9</td>
<td>82</td>
<td>M</td>
<td>CAD</td>
<td>45</td>
<td>4.5</td>
<td>None</td>
</tr>
</tbody>
</table>

Mean±SD 69±12 46±12 4.5±0.6

LVEF indicates left ventricular ejection fraction; LA, left atrium; AAD, antiarrhythmic drugs; CAD, coronary artery disease; CABG, coronary artery bypass grafting; AVR, aortic valve replacement; HCM, hypertrophic cardiomyopathy; DCM, dilated cardiomyopathy; BB, β-blocker; Dig, digoxin; and NA, not available.
use of the Biosense CARTO system. In patient 6, the CS os was activated 44 ms before the BB region during CC Afl (CL 275 ms) and 52 ms after the BB region during C Afl (CL 245 ms). In patient 7, the CS os was activated 79 ms before the BB region during CC Afl (CL 272 ms) and 56 ms after the BB region during C Afl (CL 262 ms).

Discussion

Our results demonstrate different patterns of CS activation in CC Afl and C Afl in the majority of patients. All 9 patients had a sequential PD CS activation pattern during CC Afl, whereas 7 of 9 had a fused activation pattern during C Afl, suggesting activation from at least 2 different directions. A pure DP pattern, which could suggest left atrial origin, was not seen in any patient. However, if the CS catheter could be manipulated further, for example, to the aortic valve, a DP pattern may have been seen. We have noted such patterns in sinus rhythm in other patients studied for different reasons. CS activation pattern during C Afl was similar to that in NSR in all patients, although some difference in timing of CS activation or electrogram morphology was seen in 6 of 9 patients.

The finding that CS activation was nearly identical during BB pacing and C Afl in 2 patients, coupled with the pattern of right atrial septal activation seen during electroanatomic mapping, suggests that BB serves as the dominant interatrial breakthrough site during C Afl. The CS os appears to serve as
the dominant breakthrough site during CC Afl. The 2 patients who did not fit the predominant pattern had less distal placement of CS catheter but also may have had delayed conduction through the BB. This latter explanation is less likely because flutter wave morphology, which is influenced by left atrial activation, was similar in these 2 patients and the others.

Our results differ from those of other investigators who have commented on CS activation pattern during C Afl. In one study, ECG findings and ablation results were correlated with intracardiac electrograms in 18 patients with C Afl, of whom 12 had CS recordings. All 12 patients showed PD CS activation similar to what was seen in CC Afl. Our results may differ because of the more distal placement of the CS catheter. Other investigators who have studied clockwise Afl have focused on right atrial activation and obtained little information on CS or left atrial activation pattern.

Since Bachmann first described the function of the canine interauricular band (since known as Bachmann’s bundle), a number of reports have advanced understanding of interatrial conduction. Two groups have described specialized conduction properties in BB in the dog, reinforcing its importance in interatrial conduction. Dissimilar atrial rhythms, though uncommon, support the idea that interatrial conduction occurs through discrete pathways, which can develop varying degrees of conduction delay and block. Antz et al, working with a canine Langendorff preparation, have shown that the CS os forms a discrete muscular connection between the right and left atria in dogs. Sun et al, working in the canine intact beating heart, mapped both sides of the interatrial septum by using multi-electrode basket catheters and demonstrated discordant activation of its right and left aspects of the septum. They also showed that there appeared to be discrete functional electrical connections in the superior septum, near the BB, and the inferior septum, near the CS os.

Roithinger et al studied left-to-right interatrial conduction in 18 patients by using electroanatomic mapping of the right atrium while pacing the CS or left atrium during sinus rhythm. This group showed that there appeared to be 3 preferential sites of conduction using the BB, the CS os, and the fossa ovalis region. Most patients had a single dominant breakthrough site, which varied by the patient and by site of pacing. Data from our laboratory suggest a marked influence of site of pacing on left-to-right atrial activation. Bachmann’s bundle plays a prominent role with superior left atrial or CS stimulation, whereas the os of the CS plays the dominant role when inferior sites are stimulated. The fossa ovalis plays a lesser role with CS stimulation than direct posterior left atrial stimulation (V.J. Koro and M.E. Josephson, manuscript in preparation).

Okumura et al, working with a canine sterile pericarditis model of atrial flutter, obtained biatrial maps by using a 95-pole grid. They showed that the pattern of left atrial activation depended on the pattern of rotation of Afl in the right atrium. In the dogs with tachycardia analogous to CC Afl in humans, left atrial activation proceeded inferior-to-superior beginning in the region of the CS os. In the dogs with tachycardia corresponding to C Afl, left atrial activation proceeded superior-to-inferior, beginning in the region of the BB. Although we did not perform detailed left atrial mapping in our patients, our results are consistent with this model. A proposed schema of left atrial activation in human CC and C Afl is outlined in Figure 4.

Electroanatomic mapping of the right atrium in 2 of our patients showed that the BB region was activated 52 ms and 56 ms before the CS os in C Afl, whereas the CS os was activated 44 ms and 79 ms before the BB region in CC Afl. The difference in timing of activation of the breakthrough points may largely explain the different patterns of CS activation that we found. In addition, anisotropic influences may play a role. For example, conduction velocity along the interatrial septum may differ between the clockwise and counterclockwise directions. The difference in timing of CS and BB activation during CC and C Afl in our 2 patients support this idea. In addition, conduction time over BB and CS os breakthrough sites might depend on the direction from which they are activated, although we have no direct evidence to support this hypothesis.

Although CC Afl is clinically much more common than C Afl, both types can be induced in most patients in the electrophysiological laboratory with the use of rigorous stimulation protocols. This finding raises the question of why C Afl is not more commonly seen clinically. One explanation is that atrial premature complexes from the left atrial conduct over the BB and at the same time block clockwise from the CS in the isthmus, leading to CC Afl. Olgin and colleagues have suggested this mechanism by inducing CC Afl from the CS and C Afl from the right atrium. Difference in the pattern of left atrial activation provides another possible explanation. Predominant right-to-left atrial activation over the BB, as occurs in C Afl, may be less stable, resulting in reentry in the left atrium with either degeneration to Afl or termination to sinus rhythm. Further research into the mechanism of spontaneous initiation and termination of C Afl, as well as a better understanding of where and how wave fronts collide in the left atrium during CC and C Afl, will be needed to assess these hypotheses.

Differences in left atrial activation between CC and C Afl should be reflected in the morphology of the flutter wave on surface ECG. During CC Afl, CS activation corresponds to the discrete negative deflection in the flutter wave in the
inferior leads, also consistent with inferior-to-superior activation of the left atrium. Superior-to-inferior activation of the left atrium in C Afl should be manifested as a positive deflection in the flutter wave in those leads. We have observed such discrete positive p waves coinciding with CS activation in some patients with C Afl, but this is not a universal finding. In contrast, the flutter wave morphology in CC Afl always begins with a negative deflection with a variable height of the terminal positive part of the flutter wave.

Another implication of the present study is that left atrial mechanical function may differ between CC and C Afl as the result of different sequence of activation. Although patients with Afl have been shown to be at risk for thromboembolism and for left atrial appendage (LAA) thrombus, it is unknown whether risk varies by type of flutter.16,17 Because the left atrium and left atrial appendage are activated in C Afl similarly to NSR, our results suggest the hypothesis that patients with C Afl might have more normal LAA contractile function and reduced risk of thromboembolism compared with patients with CC Afl.

The major limitation of this study is that CS mapping provides an incomplete picture of left atrial activation, and therefore breakthrough sites on the interatrial septum must be inferred. In addition, we did not induce both CC and C Afl in all patients that we studied for Afl, and therefore the patterns we found may not be representative of all patients with Afl. We were unable to reach the most distal CS superiorly in 2 patients. Finally, we studied a relatively small number of patients with heterogeneous clinical characteristics, 4 of whom were taking antiarrhythmic drugs, which may affect interatrial conduction.

Conclusions

Left atrial activation, as assessed by coronary sinus recordings, differs between CC and C atrial flutter in the majority of patients that we studied. Our results are consistent with a model in which the left atrium is activated predominantly over the BB during C Afl and over the CS os in CC Afl. The patterns probably reflect the differences in engagement of the CS os and BB and the time to cross the BB to distal CS versus conduction time from proximal to distal CS. Our results may have implications for the relative infrequency of C Afl, for the morphology of the flutter wave on surface ECG, and for mechanical properties of the left atrium in CC versus C Afl.

References

Different Patterns of Interatrial Conduction in Clockwise and Counterclockwise Atrial Flutter
Joseph E. Marine, Victoria J. Korley, Ogundu Obioha-Ngwu, Jane Chen, Peter Zimetbaum, Panos Papageorgiou, Paul Milliez and Mark E. Josephson

Circulation. 2001;104:1153-1157
doi: 10.1161/hc3501.095478

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2001 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/104/10/1153

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/