Sudden Change From Counterclockwise to Clockwise Flutter During Cavotricuspid Isthmus Ablation

What Is the Mechanism?

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Catheter ablation of cavotricuspid isthmus dependent flutter has become a commonly performed procedure with high success rate and is a prototype of macro-re-entrant arrhythmia. Although catheter ablation of isthmus-dependent flutter has become a routine procedure, not infrequently, its behavior in the electrophysiology laboratory is unusual. We present one such case highlighting some of the principles of electrophysiology of re-entrant arrhythmia and atrial flutter.

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Case Presentation

A 69-year-old woman with history of prior hospital admission for congestive heart failure with preserved left ventricular systolic function in the setting of atrial tachyarrhythmia presented for atrial flutter ablation. Atrial flutter appeared to be typical on surface ECG and intracardiac electrogram recordings through 20-electrode halo catheter placed along the tricuspid annulus. Recordings through the halo catheter showed counterclockwise activation sequence, and entrainment from the cavotricuspid isthmus confirmed isthmus-dependent flutter. Catheter ablation was done with 8 mm nonirrigated radiofrequency ablation catheter to create a line of block in the cavotricuspid isthmus from tricuspid annulus to the inferior vena cava. During the ablation, the activation pattern changed from counterclockwise to clockwise in direction (Figure 1). What is the mechanism of this change in activation?

Discussion

Classic atrial flutter involves a macro-re-entrant circuit in the right atrium around the tricuspid annulus. The cavotricuspid isthmus, a narrow strip of right atrial tissue between the anatomic boundaries formed by the tricuspid annulus and the inferior vena cava, is a part of the circuit. Catheter-based treatment of classic atrial flutter involves ablation of cavotricuspid isthmus, creating a line of block in the macro-re-entrant circuit. Documentation of bidirectional conduction block across the cavotricuspid isthmus is the end point of the ablation procedure and leads to freedom from recurrence in >90% of patients. Catheter ablation of cavotricuspid isthmus often leads to delay in conduction across the cavotricuspid isthmus without a block, which may lead to slowing or termination of atrial flutter. Sometimes ablation may lead to unidirectional conduction block. Unidirectional conduction block may allow atrial flutter only in 1 direction, either counterclockwise or clockwise.

In this case, ablation of cavotricuspid isthmus led to change from counterclockwise flutter to clockwise flutter. A closer look at Figure 1 shows that the counterclockwise flutter breaks, and there is one transition beat before the clockwise flutter starts. The last beat of the counterclockwise flutter does not conduct to the first 2 bipoles of the halo catheter (bipoles 1,2 and 3,4), which were located medial to the line of ablation lesions being created (see the disturbances in the signals on bipoles 5,6 of the halo catheter along which ablation was being done). This suggests conduction block in the cavotricuspid isthmus in lateral to septal direction, hence terminating the counterclockwise flutter circuit. Signal on the bipoles 3,4 of the cavotricuspid isthmus in lateral to septal direction, hence terminating the counterclockwise flutter circuit. Signal on the bipoles 3,4 of the cavotricuspid isthmus suggests reversal of direction of activation from septal to lateral. This activation wavefront appears to cross the isthmus with some delay (wavy arrow from bipoles 1,2 and 3,4 toward bipoles 5,6 and 7,8 on the halo catheter recording). Signal on the bipole 3,4 on the halo catheter during the intervening beat has initial deflection opposite to that during previously ongoing counterclockwise flutter, suggesting reversal of direction of activation from septal to lateral. This activation wavefront appears to cross the isthmus with some delay (wavy arrow from bipoles 1,2 and 3,4 toward bipoles 5,6 and 7,8 on the halo catheter recording). Figure 2) and activates the lateral wall of the right atrium in clockwise direction. The delay in conduction is likely because of the blocked activation wavefront from the transition beat coming down the lateral right atrial wall toward the cavotricuspid ablation line from the lateral side of the isthmus. In the subsequent flutter, the reversal of polarity of signal in the bipoles 3,4 on the halo catheter is maintained, which is consistent with clockwise activation of the atrium in that area.

Continuation of lesion placement along the isthmus led to termination of this flutter (Figure 3). Termination of clockwise flutter is clearly because of the development of block in the septal to lateral direction (clockwise) in the isthmus suggested by loss of atrial signals in the last beat of the flutter in bipoles beyond 5,6 on the halo catheter after activation of bipoles 1,2 and 3,4, which also proves that this second atrial tachycardia was indeed isthmus dependent.

The origin of the transition beat, however, is not clear and has few possible mechanisms. Electrogram recordings...
from the halo catheter suggest activation wavefront traveling down the lateral wall of right atrium toward the cavo-tricuspid isthmus ablation line. On the septal side of the ablation, the wavefront appears to be traveling in the opposite direction suggested by the reversed deflection pattern on the bipole right atrium (RA) 3,4 as discussed earlier. This is possible with an ectopic beat in the high right atrium or a sinus beat. However, a sinus beat coming in immediately after the flutter stops is unlikely, which is reaffirmed more strongly by the delay in initiation of sinus activity seen after the clockwise flutter finally breaks. The morphology of P wave, although similar to the sinus P wave (Figure 3), has subtle differences with longer P-wave duration. Moreover, the coupling interval of this atrial beat with the last counterclockwise flutter beat is very close to the flutter cycle length, suggesting a re-entrant mechanism and a link of this beat to the last counterclockwise flutter beat. The likely mechanism in this case appears to be conduction of the last counterclockwise flutter beat across the crista terminalis breaking into the midseptal area and activating the septum, upward and downward, a clockwise lower loop re-entry.3,4 Manifest conduction of the last beat of counterclockwise flutter across the crista terminalis with minimal delay (255 versus 245 ms measured at the bipole RA 19,20) likely becomes possible because of the absence of a competing wavefront of activation after the development of unidirectional block in the cavo-tricuspid isthmus. The upward wavefront subsequently travels up to the high right atrium and then down the lateral wall of the right atrium. The downward wavefront in turn activates the lower septum and the cavo-tricuspid isthmus from the
septal side toward the ablation line. The lateral wavefront of the transition beat reaches the ablation line from the lateral side slightly earlier and does not conduct because of unidirectional block. However, this concealed activation of the cavotricuspid isthmus ablation line leads to delay in conduction of the septal to lateral wavefront initiating clockwise flutter (Figure 2).

This case shows mechanism of termination of atrial flutter during catheter ablation and shows the importance of bidirectional conduction block in the cavotricuspid isthmus during the procedure.

Disclosures

None.

References


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