Modes of Initiation of Two Types of Atrial Reentry in a Patient with Typical Atrial Flutter: Isthmus-dependent Micro-reentry versus Macro-reentry

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We studied the modes of initiation of two types of atrial reentrant tachycardias (i.e., micro-reentry isthmus tachycardia and counterclockwise atrial flutter) in a 39-year-old male with typical atrial flutter. Rapid atrial pacing from proximal coronary sinus at a cycle length of 220 msec initiated micro-reentry isthmus tachycardia (non-sustained), while rapid atrial pacing at a cycle length of 210 msec initiated sustained atrial flutter circulating counterclockwise around the tricuspid annulus. It was suggested that initiation of the counterclockwise atrial flutter was associated with a pacing-induced conduction block in the entire width of the isthmus, whereas initiation of the micro-reentry isthmus tachycardia was associated with a pacing-induced conduction block in a limited segment of the isthmus (i.e., partial isthmus block).

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Introduction

Several patterns of isthmus-dependent reentrant tachycardia have been described in the past literature, including typical atrial flutter circulating around the tricuspid valve (TV) annulus,1,2) double-wave reentry3) and lower-loop reentry around the inferior vena cava (IVC).4) We herein describe a new pattern of isthmus-dependent reentrant tachycardia, i.e., micro-reentry isthmus tachycardia within the isthmus area.5) This micro-reentry isthmus tachycardia, although non-sustained, was inducible with rapid atrial pacing from the proximal coronary sinus (CS) in a patient with typical atrial flutter.

Case Report

A 39-year-old man was admitted to our hospital because of palpitations. The electrocardiogram during an episode of palpitation showed typical atrial flutter with saw-toothed flutter waves. No underlying heart disease was noted from various laboratory examinations.

Electrophysiologic study and radiofrequency catheter ablation

Electrophysiologic study was performed using...
standard catheterization techniques after informed written consent was obtained. A 6-French decapolar electrode catheter with an inter-electrode distance of 2-8-2 mm was introduced via the right internal jugular vein and placed within the CS. An 8-French 20-polar electrode catheter with an inter-electrode distances of 5 mm was introduced percutaneously via the femoral vein and placed around the tricuspid annulus, with the most distal electrode pair (A1–2 electrodes) positioned at the ostium of CS (Figure 1). Electrodes A3 through A8 covered the atrial electrograms of the right atrial isthmus. A 6-French quadripolar electrode catheter was placed across the TV for recording His bundle electrogram (HBE). Stimuli were 2 msec in duration and approximately 3 times the diastolic threshold in intensity, delivered by a digital programmable stimulator.

The patient was in regular sinus rhythm at the beginning of the electrophysiologic study, and attempts were made to induce atrial flutter with rapid atrial pacing from the ostium of CS at cycle lengths of 300–200 msec in 10-msec steps. Rapid CS pacing at a cycle length of 210 msec induced typical counterclockwise atrial flutter; furthermore, another type of reentrant tachycardia (i.e., micro-reentry isthmus tachycardia), although non-sustained, was also inducible with rapid CS pacing at a cycle length of 220 msec (this cycle length was tested twice).

Figure 2 shows initiation of counterclockwise atrial flutter. In Figure 2, the clockwise wavefront from the S8 impulse was blocked between the A3–4 and A5–6 recording sites in a Wenckebach fashion, whereas the counterclockwise wavefront from the S8 impulse traveled all the way around the TV annulus and returned to the isthmus from an opposite direction. Counterclockwise atrial flutter then started because the isthmus conductivity was restored, so that the counterclockwise wavefront could propagate through the isthmus and circulate around the TV annulus.

Initiation of micro-reentry isthmus tachycardia at a paced cycle length of 220 msec is shown in Figure 3. Diagnosis of micro-reentry isthmus tachycardia was made based on the following observations. First, the earliest atrial activation during atrial tachycardia was registered at the A3–4 recording site (indicated by an arrowhead) within the isthmus, while collision of clockwise and counterclockwise wavefronts occurred at the A15–16 recording site along the TV annulus, excluding a macro-reentry. Second, atrial activation proceeded from proximal to distal CS (a potential → A1–2 site → CS3–4 site). Third, tachycardia induction was dependent on the critical conduction delay at the A3–4 recording site, consistent with reentry regarding its mechanism. In Figure 4A, atrial tachycardia was non-inducible when “a–b” interval was 85 msec, whereas the tachycardia was induced when “a–b” interval was sufficiently prolonged to 118 msec (Figure 4B). Micro-reentry isthmus tachycardia (indicated by straight and curved arrows) was initiated after the S10 stimulus, and terminated between the A3–4 and the A1–2 recording sites in the fourth beat.

A linear radiofrequency catheter ablation was performed targeting the A3–4 recording site at the right septal isthmus. Atrial flutter was no longer inducible after complete isthmus block was obtained in a clockwise and counterclockwise direction.

Discussion
We believe that this is the first case report in which the modes of initiation of macro-reentry atrial flutter and micro-reentry isthmus tachycardia was fully analyzed in the same patient. By analyzing double atrial potentials (potentials a and b) in a small area of the isthmus (A3–4 recording site), we were able to demonstrate that initiation of counterclockwise atrial flutter was associated with a pacing-induced unidirectional conduction block in the entire width of the isthmus. In Figure 2, complete isthmus block over the entire isthmus is evident, since both c and b potentials disappeared in the S8 beat (note “a–c–b” sequence in the S7 beat), and there was no clockwise impulse propagation from the A5–6 to A15–16 recording sites along the TV annulus. In contrast, in Figure 3, S10 impulse propagated from the A1–2 to A15–16 recording site in a clockwise

Figure 1 Fluoroscopic view showing 20-polar electrode catheter positioned around the tricuspid annulus. The most distal electrode pair (A1–2 electrodes) was positioned at the ostium of CS. A3 through A8 electrodes covered the atrial electrograms of the right atrial isthmus.
Induction of atrial flutter by rapid coronary sinus (CS) pacing at 210 msec.

Typical counterclockwise atrial flutter was directly induced by the S8 impulse. Induced atrial flutter was being entrained by the subsequent S9 and S10 impulses and therefore, native atrial flutter in fact started after the S10 impulse. A3–4 through A7–8 electrodes covered the atrial electrograms of the right atrial isthmus.

Figure 2

Induction of micro-reentry isthmus tachycardia by rapid CS pacing at 220 msec.

Figure 3
direction, and there was no obvious block during initiation of micro-reentry isthmus tachycardia. Initiation of micro-reentry isthmus tachycardia may be explained schematically as in Figure 5. It is hypothesized in Figure 5 that right atrial isthmus consists of two (α and β pathways) or more conducting pathways with bridging bundles. This schema derives from the observation by Waki et al. that the structure of right atrial isthmus is composed of non-uniform muscular trabeculations with interlacing bundles and a multitude of crossovers. In Figure 5A, a paced impulse with a clockwise direction is blocked in the β pathway (this means “partial isthmus block” since the impulse is not blocked in the α pathway), then the impulse from the α pathway enters the β pathway via a bridging bundle and propagates antidromically within the β pathway (U-turn activation). Because of insufficient conduction delay (85 msec in Figure 4A), upper stream of β pathway is still refractory and does not allow the U-turn impulse (dotted line) to pass through the blocked area. In Figure 4B, because of sufficient conduction delay (118 msec in Figure 4B), upper

Figure 5 Diagrammatic explanation of initiation of micro-reentry isthmus tachycardia using a dual pathway model. For simplicity and illustrative purpose, right atrial isthmus is depicted as 2 muscular bundles (α and β pathways) and a single bridging bundle. It is hypothesized that refractory period of α pathway is shorter than that of β pathway. To complete the micro-reentry, another bridging bundle located at the upper stream of dual pathways is required but is excluded from the schema.
stream of $\beta$ pathway restores excitability and allows the U-turn impulse (dotted line) to pass through the initially blocked area, thus leading to initiation of micro-reentry isthmus tachycardia.

We have shown that partial isthmus block occurred at a cycle length of 220 msec, whereas entire isthmus block occurred at a cycle length of 210 msec. Dual pathway model (see Figure 5) will readily explain this phenomenon. At a cycle length of 220 msec, pacing stimulus encountered refractoriness of $\beta$ pathway, and not $\alpha$ pathway (partial isthmus block), since the refractory period of $\alpha$ pathway was shorter than that of $\beta$ pathway. At a shorter cycle length of 210 msec, pacing stimulus encountered refractoriness of both $\alpha$ and $\beta$ pathways, resulting in entire isthmus block.

**Conclusion**

We have documented a new pattern of isthmus-dependent reentrant tachycardia (micro-reentry isthmus tachycardia) in a patient with typical atrial flutter. Rapid CS pacing at a cycle length of 220 msec initiated micro-reentry isthmus tachycardia, whereas rapid CS pacing at a cycle length of 210 msec initiated counterclockwise atrial flutter. It was suggested that initiation of counterclockwise atrial flutter was associated with a pacing-induced conduction block over the entire width of the isthmus, whereas initiation of micro-reentry isthmus tachycardia was associated with a pacing-induced conduction block in a limited segment of the isthmus (i.e., partial isthmus block).

**References**


