A 60-year-old male patient had a history of paroxysmal atrial fibrillation and typical atrial flutter. Radiofrequency ablation for pulmonary vein isolation and cavotricuspid isthmus (CTI) had been performed 2 years previously. This time, he presented with symptoms of palpitation. Electrocardiogram (ECG) showed regular narrow QRS complex tachycardia with sawtooth pattern (Figure 1A). CTI re-connection with recurrent typical atrial flutter was assumed and electrophysiological study was performed. Intracardiac ECG showed typical counterclockwise atrial flutter (Figure 1B). Bi-directional block of the CTI, however, was confirmed on differential pacing. What is the mechanism of this tachycardia and ECG pattern?

The posterior barrier of typical atrial flutter is formed by the crista terminalis and the anterior barrier is formed by the tricuspid annulus. The impulse in the right atrium travels around the large macroreentry circuit. In counterclockwise cases, the impulse ascends anteriorly through the lower septum and descends along the lateral free wall. The activation pattern of coronary sinus is from coronary sinus opening to the distal part (Figure 2A). The sawtooth pattern on surface ECG and the sequential activation pattern on intracardiac ECG reflect the aforementioned impulse travel. CTI was involved in such a macroreentry circuit. Therefore, typical counterclockwise atrial flutter is impossible with a bi-directionally blocked CTI. In the present case, entrainment was performed first. Tachycardia, however, was terminated after pacing and was reinitiated spontaneously. Therefore, we constructed a right atrial activation map using 3-D electroanatomical mapping (Figures 2B, C). The tachycardia mapping resembled counterclockwise activation pattern.
conduction and the ECG pattern. An artificial conduction barrier may alter the nature of impulse that a typical electrophysiological pattern could be mimicked ing other types of arrhythmia. AT may also cause an atypical intracardiac ECG pattern mimicking atrial flutter not possible. In the usual case, the impulse from the focus of AT would spread out all over the atrium. Due to the origin being located in the lower septum, the impulse would ascend, regardless of whether it traveled along the septum or along the lateral free wall. CTI block, however, had previously been achieved by radiofrequency ablation. The impulse from the coronary sinus could not travel through the CTI; therefore, the free wall could be activated only in the downward direction, rather than the upward direction. Origin at the proximal coronary sinus roof also made the coronary sinus activation pattern exactly the same as that for typical atrial flutter (Figure 2A). Because of the pre-existing bi-directionally blocked CTI, the ECG of the AT with origin at the proximal coronary sinus roof mimicked typical counterclockwise atrial flutter. The double potential at the middle CTI further confirmed complete block, making typical atrial flutter (tachycardia cycle length, 257 ms; Figure 2D). With radiofrequency ablation at this area and the proximal roof of coronary sinus, the tachycardia was successfully terminated. Therefore, atrial tachycardia (AT) originating in the proximal coronary sinus roof was the final diagnosis.

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Entrainment pacing at CTI can be performed to further differentiate AT and typical atrial flutter. In the present case, however, tachycardia was terminated by entrainment pacing, making differential diagnosis using this method not possible. Also, the nature of the tachycardia termination by entrainment pacing made reentry or triggered activity possible as the mechanism of AT. In previous studies, reentry or triggered activity were the mechanism of AT from the coronary sinus ostium or proximal part and is compatible with the present case. With the advance of radiofrequency ablation for arrhythmia, artificial conduction barrier may alter the nature of impulse conduction and the ECG pattern. Anatomical abnormality may also cause an atypical intracardiac ECG pattern mimicking other types of arrhythmia. This presentation reminds us that a typical electrophysiological pattern could be mimicked by another arrhythmia. When interpreting electrophysiological tracings, previous procedures and subsequent electrophysiological changes should always be kept in mind.

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