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Eccentric activation of the coronary sinus during typical atrial flutter: What is the mechanism?

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1. Case presentation

A 79-year-old man with a history of ventricular septal defect underwent catheter ablation of drug refractory paroxysmal atrial flutter (AFL). The 12-lead electrocardiogram during tachycardia showed a biphasic, predominantly positive flutter wave with an initial negative component in leads II, III, and aVF, and positive flutter waves in leads V_1 through V_6 (Fig. 1, A), consistent with typical counterclockwise (CCW) AFL [1]. A decapolar catheter was advanced with its ♯17 and ♯18 poles in the proximal coronary sinus (CS), and a deflectable duodecapolar Halo catheter was placed parallel to the tricuspid annulus (TA), across the inferior vena cava (IVC)-TA isthmus, with its tip at the CS ostium (Fig. 1, B and C). Although we did not perform contrast CS venography to confirm the location of the CS ostium, we believe that the CS ostium is located near the 5 o'clock position of the mitral annulus as estimated by the shape of the CS catheter projected in the left anterior oblique fluoroscopic view, consistent with the location of the ♯17 and ♯18 poles of the CS catheter. The atrial activation sequence along the TA during ongoing tachycardia, combined with entrainment pacing at the IVC-TA isthmus, and 10 o'clock and 2 o'clock positions of the TA with a pacing cycle length of 220 ms confirmed the diagnosis of typical CCW AFL. We did not perform entrainment pacing from the CS. During entrainment pacing from any site along the TA, all atrial deflections in CS recordings immediately after the last pacing stimulus were captured with the activation sequence similar to that during the AFL and a spike-atrial interval shorter than AFL cycle length, consistent with an orthodromic capture. A line of radiofrequency energy delivery blocked conduction across the IVC-TA isthmus, terminated AFL, and restored sinus rhythm. Differential pacing from the low septal and lateral right atrial (RA) region confirmed the successful creation of bidirectional isthmus block. It is noteworthy that, during ongoing AFL before ablation, (1) a latency of low RA conduction, consistent with conduction across the septal isthmus, was observed along the distal Halo catheter (Fig. 1, D), and (2) recording of atrial activation along the CS, instead of being proximal to distal, was centrifugal from CS poles 9 to 10 (Fig. 1, D). What is the underlying electrophysiological mechanism of atypical atrial activation during typical CCW AFL?

2. Commentary

Recordings of the activation sequence from the CS during tachycardia are expected to help discriminate among atrial tachyarrhythmias [2]. During ongoing typical CCW AFL, most of the CS is activated from proximal to distal, because the CS

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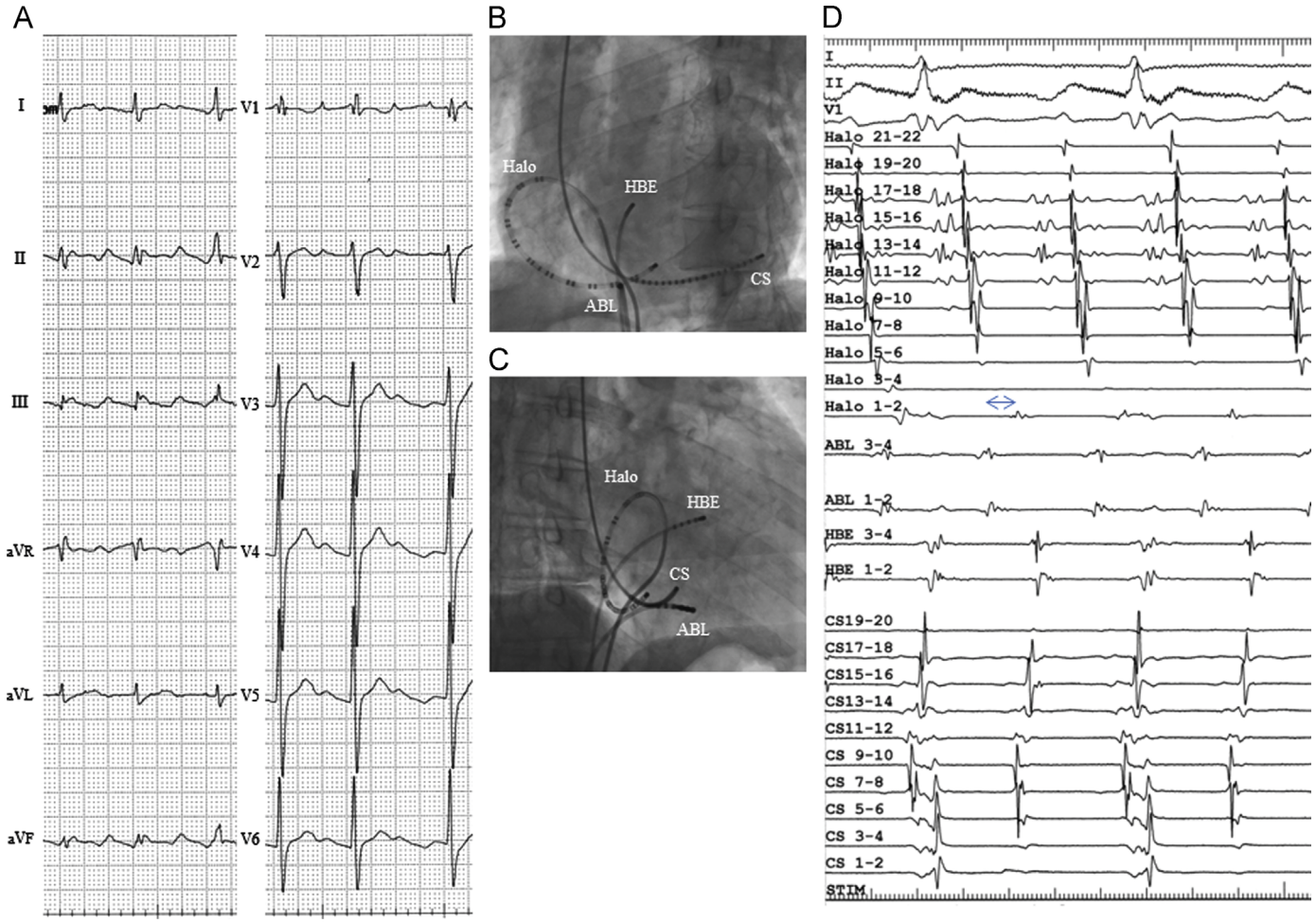

Fig. 1. (A) 12-lead electrocardiogram during ongoing atrial flutter (AFL) with variable atrioventricular conduction. (B and C) Right and left, respectively, oblique fluoroscopic views of the position of the catheters. In (B) the ablation catheter (ABL) is located on the ablation line of the isthmus. HBE is the His-bundle electrogram and CS is the coronary sinus. (D) Intracardiac electrograms during ongoing AFL. A regular atrial rhythm at a cycle length of 232 ms was recorded in a counterclockwise activation sequence around the tricuspid annulus. A conduction delay is present between the atrial electrograms recorded at the nearly adjacent Halo 5–6 and Halo 1–2 recording sites (bidirectional arrows). An abrupt decrease in the amplitude of the atrial deflections accompanied by an abrupt increase in the amplitude of the ventricular deflections in the distal CS recordings suggest that the distal tip of the CS catheter might be inserted into the lateral vein branching from the CS. Halo 21–22 is the proximal and 1–2 is the distal recording sites on the Halo catheter; HBE 3–4 is the proximal and 1–2 is the distal His-bundle electrograms; CS19–20 is the proximal and CS1–2 is the distal CS recordings.

musculature, by being continuous with the RA myocardium at the ostium and connected to the LA myocardium [3], is a major RA-to-LA connection [4–6]. However, several cases of typical CCW AFL with an atypical activation of the CS associated with changes in the morphology of FL waves have been described, probably because of changes in the interatrial connection caused by conduction block at the CS ostium [7–10], the mechanism of which remains unexplained.

We recorded pacing from the ostium of CS in search of interatrial conduction along the CS. During pacing at a cycle length slightly longer than the AFL cycle length, the wavefront propagated in a proximal-to-distal CS activation sequence (Fig. 2), suggesting the presence of conduction across an interatrial electrical connection at the CS ostium. Moreover, during pacing at a cycle length close to that of AFL, the activation sequence became centrifugal and earliest at the mid CS (CS11–12), similar to that during AFL. Additionally, the interval between the pacing spike and the atrial deflection recorded at the bundle of His prolonged with an atypical Wenckebach periodicity, consistent with a spike-atrial conduction delay in the RA. The electrophysiological observations shown in Fig. 3 are explained as follows: (1) the wavefront of the S2- and S3-paced cycles passed through the ostium of the CS; (2) the S4-paced cycle was blocked because of refractoriness at the ostium of the CS (CS19–20 or more proximal), whereas another wavefront traveling in a caudal-to-cranial direction along the right-sided interatrial septum, returned via the Bachmann bundle, the interatrial septum, or both, in a cranial-to-caudal direction along the left-sided interatrial septum, reached the mid-CS (CS11–12), and finally encountered residual refractoriness at the CS ostium caused by the third paced RA-to-LA wavefront; (3) the S5-paced cycle was blocked at the ostium of the CS because of residual refractoriness caused by LA-to-RA concealed penetration from the S4-paced cycle; and (4) each new impulse was similarly blocked at the ostium of CS because of residual refractoriness from concealed penetration of the previous paced cycle (linking phenomenon) [11,12]. During captured pacing, the spike-atrial conductive properties represented atypical Wenckebach periodic conduction delay, followed by S10-atrial conduction block;(5) because of the cancellation of the linking phenomenon by the noncaptured S10 stimulus, the wavefront of the S11-paced cycle passed through the ostium of the CS again, although S12-, S13-, and S14 were similarly blocked at the ostium of the CS (not shown in Fig. 3). This pacing study suggests, therefore, that linking between dual conductive pathways of CS musculature and left atrial muscle was the mechanism responsible for the perpetuation of conduction block at the ostium of the CS. This may have