Paroxysmal atrial fibrillation originating from the cavotricuspid isthmus: Utility of self-reference mapping with a high-density grid mapping catheter for identification of non–pulmonary vein triggers

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Introduction
Pulmonary vein isolation (PVI) is an effective method for the treatment of paroxysmal atrial fibrillation (AF). Thus, correct identification of non–pulmonary vein (PV) triggers could lead to a higher success rate of catheter ablation (CA). However, the mapping techniques to identify non-PV triggers originate from various sites, and it is challenging to identify the site of AF initiation with pinpoint accuracy. Linear ablation between the tricuspid valve and the inferior vena cava orifice, ie, the cavotricuspid isthmus (CTI), is routinely performed for CTI-dependent atrial flutter (AFL) and has a high success rate of bidirectional conduction block creation. In the present case, the CTI was the precise origin of an under-recognized non-PV trigger identified by self-reference mapping using a high-density grid (HDG) mapping catheter (Advisor HD Grid Mapping Catheter, Sensor Enabled; Abbott, St. Paul, MN).

Case report
A 34-year-old man with symptomatic paroxysmal AF was referred to our institution for CA. He experienced AF attacks approximately once every 2 weeks, and he had never been diagnosed with AFL prior to the CA. His medical history was noncontributory for any cardiac disease or cardiothoracic surgery. Echocardiography revealed a normal ejection fraction and a left atrial diameter of 33 mm. The patient underwent preoperative prone-positional computed tomography to rule out intracardiac thrombi. Anatomical variants such as a prominent eustachian ridge or diverticulum were not observed in the 3-dimensional computed tomography angiography. After informed consent was obtained, PVI was performed during sinus rhythm using a 28 mm fourth-generation cryoballoon catheter (Arctic Front Advance; Medtronic, Inc, Minneapolis, MN). Spontaneous AF initiation was reproducibly observed with a single non-PV trigger and without isoproterenol infusion. Moreover, AF reinitiated a few seconds after electrical cardioversion repeatedly.

The earliest activation site of the non-PV trigger was in the region of the distal electrode pair of the duodecapolar Halo catheter (H1-2), which was placed along the tricuspid annulus. Therefore, the HDG mapping catheter using a 3-dimensional mapping system (EnSite X; Abbott) was positioned in the lateral right atrium at a lower level than the distal tip of the Halo catheter. After intracardiac cardioversion, the earliest activation site of the non-PV trigger was at D-spline 2-3 and 3-4 of the HDG mapping catheter (Figures 1A and 2A). Therefore, the HDG mapping catheter was moved to a more inferior site from the lateral site. After intracardiac cardioversion, the earliest activation site of the non-PV trigger was at D-spline 2-3 and 3-4 of the HDG mapping catheter (Figures 1A and 2A). Therefore, the HDG mapping catheter was moved to a more inferior site from the lateral site. After intracardiac cardioversion, the earliest activation site of the non-PV trigger was at D-spline 2-3 and 3-4 of the HDG mapping catheter (Figures 1A and 2A). Therefore, the HDG mapping catheter was moved to a more inferior site from the lateral site.
The (activation site of the non–PV trigger at D-spline 2-3 and 3-4 of the HDG mapping catheter (green tags) was maintained in almost the same position, shows the earliest activation site of the non–PV trigger at C-spline 2-3 and 3-4 of the HDG mapping catheter (HDG) mapping catheter (Figure 1C). The HDG mapping catheter was moved further inferiorly, shows the earliest activation site of the non–PV trigger at D-spline 2-3 and 3-4 of the HDG mapping catheter (Figure 1B). The HDG mapping catheter was further moved to a more inferior site, shows the earliest activation site of the non–PV trigger at D-spline 2-3 and 3-4 of the HDG mapping catheter (dark green tags). D: The fourth self-reference map, after the HDG mapping catheter was moved further inferiorly and posteriorly, shows the earliest activation site of the non–PV trigger at C-spline 2-3 and 3-4 of the HDG mapping catheter (yellow tags). E: The fifth self-reference map, while the HDG mapping catheter was maintained in almost the same position, shows the earliest activation site of the non–PV trigger at C-spline 2-3 and 3-4 of the HDG mapping catheter (bright green tags). The earliest activation site of the non–PV trigger exists in the HDG mapping catheter. F: Earliest activation sites of the non–PV trigger of the HDG mapping catheter. The red circle shows the earliest activation site. IVC = inferior vena cava; LA = left atrium; TA = tricuspid annulus.

also remained at D-spline 2-3 and 3-4 of the HDG mapping catheter (Figure 1B). The HDG mapping catheter was further moved inferiorly. After intracardiac cardioversion, the earliest activation site of the non–PV trigger still remained at D-spline 2-3 and 3-4 of the HDG mapping catheter (Figure 1C). The HDG mapping catheter was moved further inferiorly and posteriorly and was placed at the mid portion of the CTI. After intracardiac cardioversion, the earliest activation site of the non–PV trigger was observed at B-spline 3-4 and C-spline 3-4 (Figure 1D) of the HDG mapping catheter. Notably, the local electrograms of D-spline, which was only 3 mm apart from C-spline, were markedly delayed compared with those of C-spline. Finally, intracardiac cardioversion was repeated while maintaining the HDG mapping catheter at almost the same position. The earliest activation sites were C-spline 2-3 and 3-4 (Figures 1E and 2B), again. The local electrograms of the eustachian ridge were delayed compared with those of the central portion of the CTI. The earliest activation site of the non–PV trigger at the mid portion of the CTI was successfully identified with reproducibility (Figure 1F).

Right atrial angiography revealed no unusual anatomical findings, such as a prominent eustachian ridge or a diverticulum. Figure 3A shows the HDG mapping catheter positioned at the mid portion of the CTI, and Figure 3B shows the first radiofrequency (RF) application at the mid portion of the CTI. Ablation of the non–PV trigger was performed using a 3.5 mm irrigated-tip contact force–sensing ablation catheter (TactiCath, Sensor Enabled; Abbott) at the mid portion of the CTI (Figure 3C). Multiple RF applications were delivered intensively at the mid portion of the CTI during AF, limited to 30 watts with the contact force maintained between 10 and 15 g and lesion size index (LSI) of 4.0–5.0 at each lesion. Pink (LSI 4.0–5.0) or red (LSI 5.0–6.0) dots indicated ablation tags. After focal RF ablation at the CTI, non–PV triggering AF was never provoked even by high-dose isoproterenol infusion and burst pacing from the high right atrium. Omnipolar mapping with an HDG catheter, which displays the wavefront vector map as green arrows, at this CTI region was then performed during coronary sinus (CS) pacing (Figure 3D). Finally, the 2 conduction gaps, both of which were at the distal and proximal portions of the CTI, were successfully ablated by additional RF applications. The bidirectional CTI block was confirmed using a differential pacing maneuver and omnipolar mapping during pacing from the proximal poles of the CS (Figure 3E). AF originating from the eustachian ridge has already been reported; thus, it is necessary to distinguish whether this non–PV trigger appears at the eustachian ridge or at the CTI. The voltage map and electrophysiological findings after non–PV trigger ablation support the evidence that the non–PV trigger originated from the middle of the CTI region, not from the eustachian ridge. During 12 months of follow-up, the patient remained asymptomatic with no recurrence of atrial arrhythmias.

Discussion

PVs are the major source of AF, and non–PV triggers can arise from the superior vena cava, left atrium posterior
wall, crista terminalis, CS, inferior vena cava, ligament of Marshall, both atrial septae, and both atrial appendages.2–6 Kato and colleagues observed non-PV triggers in 211 of 647 patients (32.6%), arising most frequently from the superior vena cava.7 Older age, female sex, and lower body mass index were significantly associated with non-PV triggers.

Figure 2  Earliest atrial activation site during atrial fibrillation initiation. A: The intracardiac electrogram shows the earliest activation (red arrowhead) at D-spline 3-4 of the high-density grid (HDG) mapping catheter during atrial fibrillation (AF) initiation, and the coupling interval is 220 ms. The activation sequence of the Halo catheter is distal to proximal (blue dotted arrow) and that of each spline on the HDG mapping catheter is proximal to distal (red dotted arrows). B: The intracardiac electrogram shows the earliest activation (red arrowhead) at C-spline 3-4 of the HDG mapping catheter during AF initiation at the mid portion of the cavotricuspid isthmus, and the coupling interval is 200 ms. The activation sequence of the Halo catheter is distal to proximal (blue dotted arrow) and that of each spline on the HDG mapping catheter is proximal to distal (red dotted arrows). CS = coronary sinus; Halo = halo duodecapolar catheter; HDG = high-density grid; LAO = left anterior oblique; RAO = right anterior oblique.
The presence of a non-PV trigger was an independent predictor of AF recurrence. Herein, the patient was a young man with a normal body mass index. The characteristics of this patient were different from those previously reported.

Non-PV triggers at the CTI were detected correctly by self-reference mapping with the HDG mapping catheter. Another study reported a method of self-reference mapping using a multipolar high-resolution mapping catheter.
The effectiveness of self-reference mapping using an HDG mapping catheter to identify a rare non-PV trigger originating from the prominent eustachian ridge has been previously reported by Yamauchi and colleagues. We used an HDG mapping catheter to identify the correct origin using its structure with both vertical and horizontal splines, which does not change the relative positions of the 16 electrodes in a 4 × 4-square lattice. Self-reference mapping was performed by tagging the earliest activation site of an HDG mapping catheter on a 3-dimensional mapping system and then moving the catheter upstream until the earliest excitatory site was identified for catheter reproducibility. It is necessary to distinguish whether this non-PV trigger appears at the eustachian ridge or at the CTI in this case. The voltage map and electrophysiological findings after non-PV trigger ablation give a boost to the evidence that the non-PV trigger originated from the middle of the CTI region, not the eustachian ridge. To the best of our knowledge, this is the first case of AF with a non-PV trigger that originated from the CTI.

RF ablation of the CTI is believed to be generally safe and has a high success rate for treating CTI-dependent AFL. A previous study reported that CTI ablation, in addition to PVI, significantly reduced both AF and atrial arrhythmia inducibility. In another study, the clinical recurrence rate after CA for AF was lower in the additional CTI group than in the circumferential PVI-alone group. The decrease in the induction of AF might be due to the treatment of non-PV trigger from the CTI, as seen in the present case.

**Conclusion**

Self-reference mapping using an HDG mapping catheter could facilitate accurate non-PV trigger identification, which increases the success rate of non-PV trigger ablation. More importantly, the CTI is a potential and rare source of non-PV triggers.

**References**

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