A case of persistent atrial fibrillation recurrence related to multiple non-pulmonary vein triggers. - Fast and easy mapping of the use of a high-density grid-style mapping catheter -

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Introduction: The recent advances in the mapping systems and catheters for atrial fibrillation (AF) ablation are remarkable. We experienced a fast and easy mapping using a high-density grid-style mapping catheter.

Methods: N/A

Result: A 71-year-old man was referred to our hospital for treatment of an AF recurrence. His medical history included hypertension, dyslipidemia, and percutaneous coronary intervention for unstable angina. On admission, his laboratory findings revealed an elevated BNP level of 257.2 pg/mL. Transthoracic echocardiography revealed a dilated left atrium (LAD 48 mm) and normal systolic left ventricular function (LVEF 61%). He underwent an initial ablation procedure, including a pulmonary vein (PV) isolation with radiofrequency ablation, for persistent AF 6 months prior. Sinus rhythm was maintained with anti-arrhythmic drugs (AADs) after an early phase recurrence, however, the AF also recurred while taking the AAD. He underwent a second ablation procedure after hospitalization. Although there were no PV reconnections, it was impossible to maintain sinus rhythm because of the immediate recurrence of the AF even shortly after repeated intracardiac DC shocks. The intracardiac atrial electrograms of the repeated initiators exhibited the same sequence, suggesting that the firing originated from the same site. When a single-beat multi-point simultaneous mapping of the initiator was created using EnSite NavX and the Advisor HD Grid catheter (a high-density grid-style mapping catheter), it was diagnosed as a septal site in the lower right atrium (RA). Subsequently, another map of the fractionated electrograms across the RA was created during sustained AF before the ablation. The activation was organized throughout the entire RA, except for at the foregoing firing site with a significant area of fractionated electrograms closely associated with the area of the initiator. It was suggested that one of the mechanisms of the fractionated electrograms was associated with the initiator of the AF. The firing disappeared until an isoproterenol infusion after the ablation of that site. Furthermore, the firing appeared after frequent APCs from another site during a high-dose isoproterenol infusion. Similarly, the second initiator was diagnosed (by single-beat multi-point simultaneous mapping) as being located at a septal site in the lower left atrium. It was possible to maintain sinus rhythm throughout the session after ablating those two non-PV triggers. There have been no recurrences for 12 months without any AADs.

Conclusion: We were able to diagnose and treat multiple triggers by a systematic fast and easy
mapping using a high-density grid-style mapping catheter.