Case Report

Identification and Radiofrequency Catheter Ablation of a Nonsustained Atrial Tachycardia at the Septal Mitral Annulus with the Use of a Noncontact Mapping System: A Case Report

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Here we report a case of a 16-year old female with symptomatic nonsustained atrial tachycardia (NSAT) originating from the septal mitral annulus. NSAT was induced by atrial burst pacing after an intravenous isoproterenol (ISP) injection. The array mode of the noncontact mapping system (NCM) allowed us to quickly identify the tachycardia focus at the septal mitral annulus, where the contact bipolar voltage map revealed no low voltage area (<0.5 mV). The NSAT was eliminated by a radiofrequency energy application to the identified tachycardia focus during sinus rhythm, and the patient has been free from any symptoms during 10 months of follow-up.
(J Arrhythmia 2008; 24: 214–218)

Key words: Ablation, Atrial tachycardia, Noncontact Mapping

Introduction

Idiopathic left atrial tachycardias (LA-AT) have been reported to originate from the pulmonary vein(s), LA appendage, posterior LA, mitral annulus and LA septum,[1–5] and the mechanisms based on the activation patterns and responses to atrial pacing include macroreentry, microreentry and focal discharges, depending on the case. However, there has been a limited number of reports which have described a focal AT originating from the left atrial septum,[4] especially of the septal mitral annular region.[5] In the case we report here, we found an NSAT originating from the septal mitral annulus with the use of NCM, which was eliminated by a radiofrequency energy application to the NSAT focus.

Case Report

A 16-year old female was referred for a second catheter ablation session for frequent symptomatic

Received 26, August, 2008: accepted 11, December, 2008.
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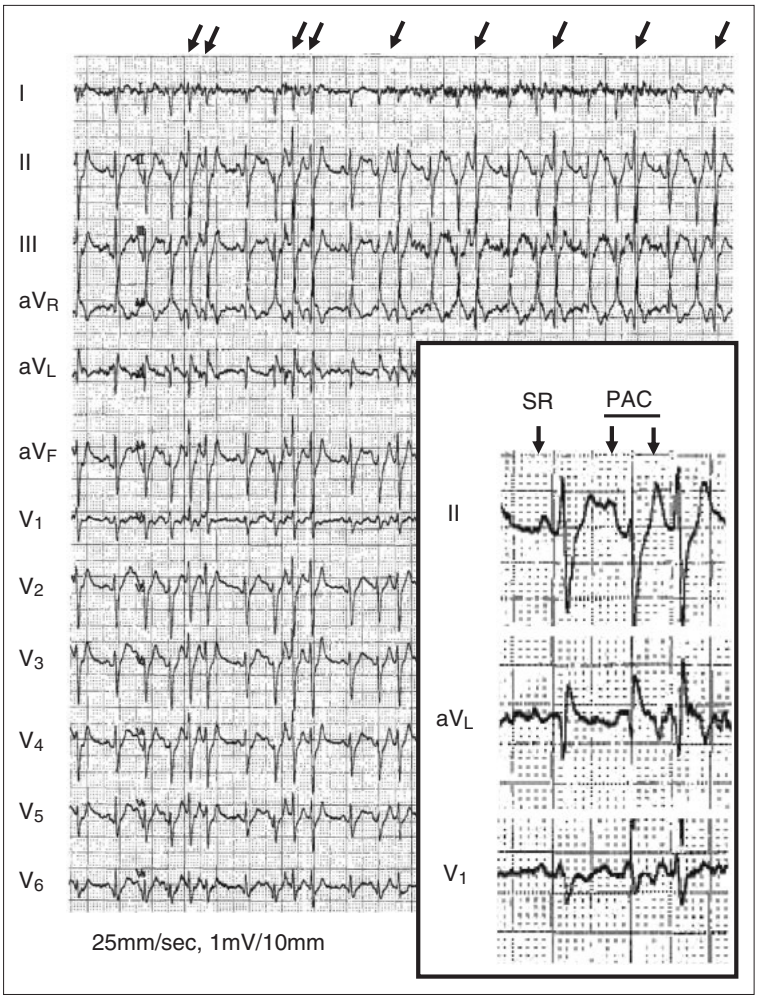

Figure 1 Twelve lead electrocardiogram during a treadmill exercise test.
Frequent premature atrial contractions (PAC) spontaneously occurred (arrows) around the peak exercise, during which the patient complained of palpitations. The P wave during the PACs was negative in lead aVL and positive in the inferior leads and V1, suggesting that the PACs originated from the left atrium.

premature atrial contractions (PAC) and NSAT. The first session was performed in other hospital, and failed to eliminate the arrhythmia. The frequent and successive PACs occurred only during the treadmill exercise test, and the P wave in the electrocardiogram during the PACs had a negative deflection in lead aVL, and was positive in the inferior leads and V1, suggesting that it originated from the anterior or superior LA[1] (**Figure 1**). No organic heart disease was documented by electrocardiography during sinus rhythm, chest radiography or echocardiography.

Written informed consent was obtained and the session was performed with mild conscious sedation. An NSAT with the same P wave morphology as the clinical AT in the 12 lead electrocardiogram occurred during atrial burst pacing after a bolus injection of ISP (4 μg) (**Figure 2**).

In order to identify the PAC/NSAT focus and elucidate the subsequent LA activation, a multielectrode array (MEA) catheter used with the NCM system (St. Jude Medical, Minnetonka, MN, USA) was introduced into the LA through a 10Fr. long transseptal sheath (Mullins, C.R. Bard, Inc.), which was advanced into the LA by a standard Brockenbrough technique. The tip of the MEA was deployed into the LA appendage. After the long sheath was introduced into the LA, heparin was injected into the LA to maintain an activated clotting time (ACT) between 300 and 400 seconds throughout the study. Every 30 minutes the ACT was monitored and, if it was <300 seconds, additional heparin was injected. The geometry of the LA was depicted with the 20-pole circular catheter (Spiral HP, St. Jude Medical, Minnetonka, MN, USA) and deflectable 8 mm-tip quadripolar electrode catheter (Fantasista, Japan Life Line, Co Ltd., Japan). The recording and analysis by the NCM was performed according to an established method reported by other researchers, the details of which have been described elsewhere.[6–10] During the review of the recorded data, a high-pass filter

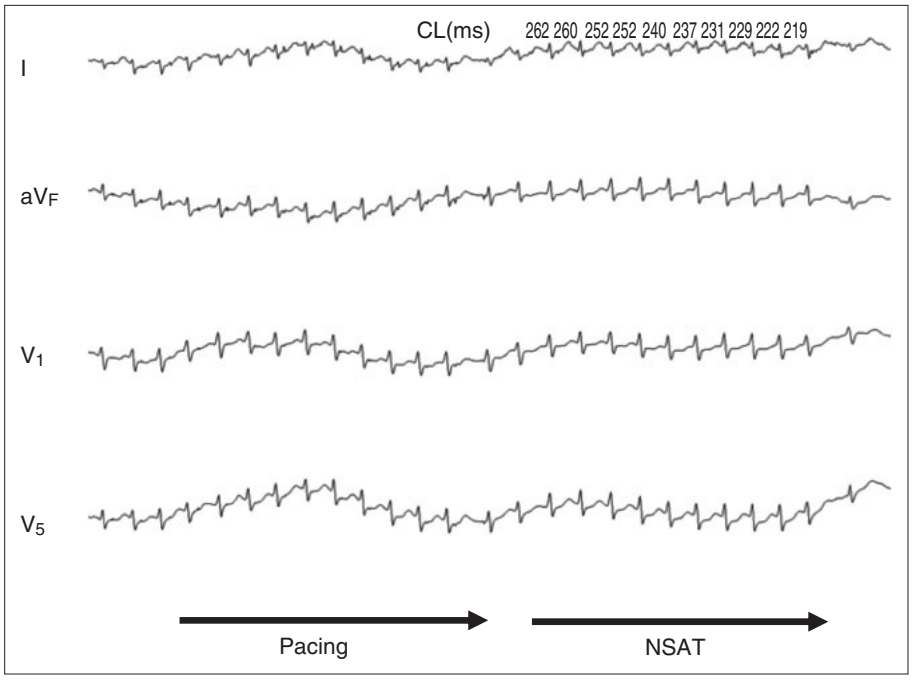

Figure 2 Electrocardiographic recording of nonsustained atrial tachycardia (AT) induced by atrial burst pacing after an intravenous isoproterenol injection (4 μg). The figures on the lead I indicate AT cycle length, suggesting a warm-up phenomenon.

setting of 2.0 Hz was used for the isopotential map created by the NCM.

Although the NSAT was induced by atrial burst pacing after the ISP injection in the beginning of the electrophysiologic study as shown in **Figure 2**, the NSAT was no longer induced other than for at most 2 successive PACs after the introduction of the MEA into the LA in spite of the best efforts to induce the AT by aggressive atrial pacing and/or an ISP injection. Because the P wave morphology of the PACs was identical to that of the NSAT, which suggested that both the tachycardia and PACs originated from the same site, an analysis of the activation was performed for the PACs using the NCM. A dynamic wavefront map of the PACs revealed that the PACs originated from the septal mitral annulus, from which the activation spread out to the entire LA (**Figure 3a**). The virtual unipolar electrogram at the PAC focus, demonstrated by dynamic wavefront map, exhibited a q-S pattern confirming the PAC focus.[11] The onset of the virtual electrogram preceded the P wave by 40 ms (**Figure 3b**). Contact bipolar recording at the AT focus was not obtained during the PAC because of the infrequent nature of the PAC occurrence.

A voltage map was also constructed with the contact bipolar electrograms during sinus rhythm using an 8 mm-tip quadripolar electrode catheter (Fantasista, Japan Life Line, Co Ltd., Japan), in which the low voltage zone (LVZ) was defined as an area with an electrogram amplitude of <0.5 mV. There was no LVZ at the septal mitral annulus corresponding to the PAC/NSAT focus but only a mild LVZ in the posterior LA. The mean voltage of the LVZ in the posterior LA was 0.3 ± 0.1 mV.

After applying a total of 4 radiofrequency energy applications with an energy of 30 W to the AT focus (**Figure 3c**), no further AT/ectopy was observed or induced by atrial pacing and/or an intravenous ISP injection. Thus the ablation procedure was finished at this point. There were no procedure-related complications. During a follow-up period of 10 months, the patient has been free from any symptoms and no PAC/NSAT has been observed at rest, during treadmill exercise testing or during 24-hour Holter electrocardiographic recordings.

Discussion

LA-AT is a well known entity, and is usually observed after radiofrequency catheter ablation (RFCA)[6,7] or open heart surgery for mitral valvular disease.[8] Among such arrhythmias, idiopathic focal LA-AT is uncommon, and several likely sites of origin were described in previous papers, which included the pulmonary veins, LA appendage, mitral annulus, and LA septum.[1–5] In the present patient, the AT focus was identified at the septal mitral annulus, which a few reports described as a site for an idiopathic focal AT focus.[4,5]

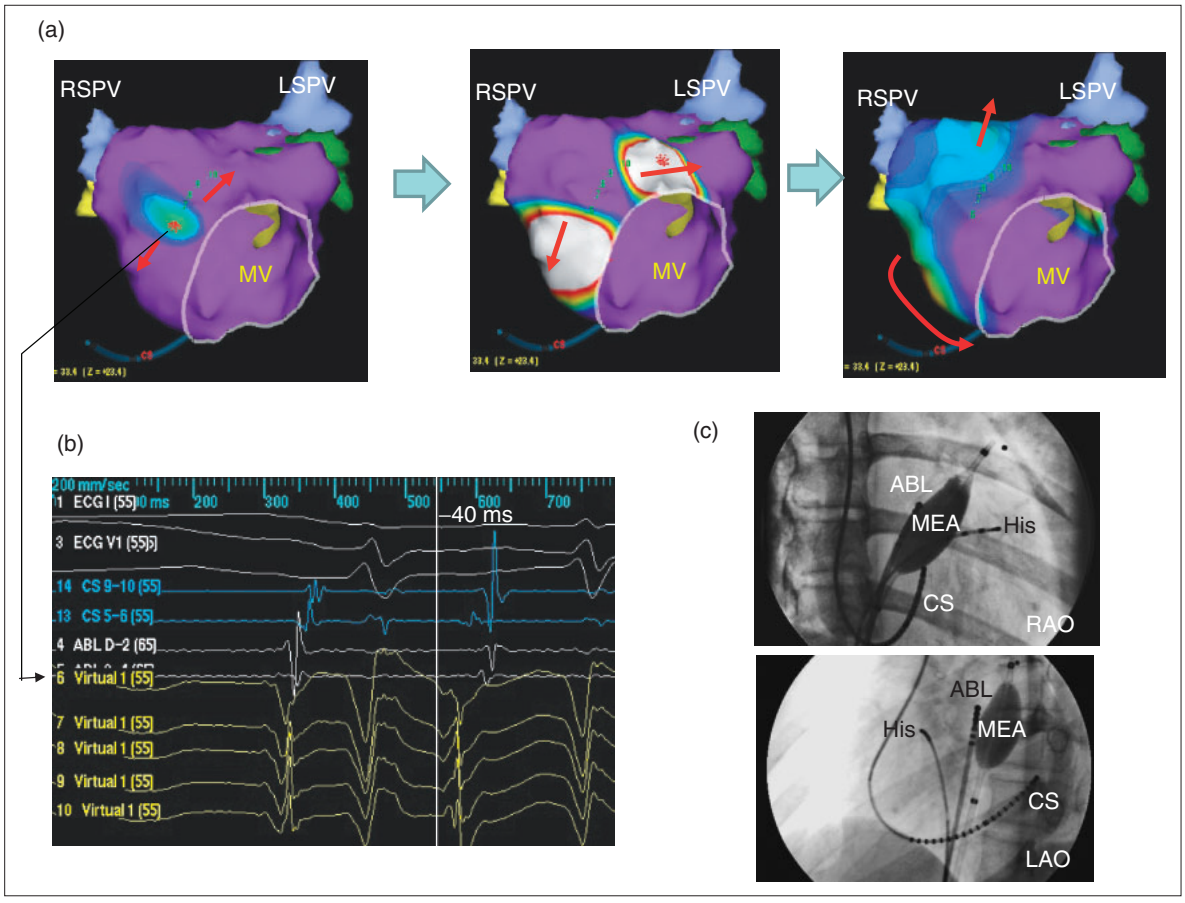

Figure 3

(**a**) Dynamic wavefront map created during a premature atrial contraction (PAC). The focus of the PAC was identified at the septal mitral annulus. The activation that originated from the focus propagated radially into two different directions, one to the anterior left atrium (LA), and the other toward the LA septum and subsequently toward the posterior LA. (**b**) The virtual unipolar electrograms at the PAC focus site, indicated by "6. Virtual 1" in the figure, exhibited a q-S pattern, the beginning of which preceded the P wave by 40 msec. The ablation catheter was not located at the earliest activation site. (**c**) Location of each electrode in the RAO and LAO fluoroscopic views.
ABL: Ablation catheter, MEA: Multielectrode array catheter.

Kistler et al. reported that focal AT aroused from the mitral annulus was observed in 7 out of 172 patients with AT, the precise location of which was close to the left fibrous trigone or mitral-aortic continuity.[5] They defined mitral annular AT as followings: 1) the successful ablation site was located at the annular lesion by the fluoroscopic view, 2) ratio of the atrial electrogram amplitude to ventricular one was <1.0 as well as the ventricular electrogram amplitude > 0.5 mV at the successful ablation site, 3) the AT and ectopy were both abolished by RFCA at this site,[5] and all these findings were also observed in the presented patient. AT originating from the left septal region was also reported by Marrouche et al.[4] and they identified an AT foci at the left inferoposterior or left midseptal region. In the former, the P wave morphology was negative in aVL and had a biphasic negative-positive deflection in lead V1, but in the latter, positive, biphasic negative-positive deflections, or isoelectric P waves were observed in lead V1 and a positive or biphasic P-wave morphology was observed in the inferior leads. In the present case, the P wave morphology was negative in lead aVL, and positive in the inferior leads and V1, and was consistent in both reports.

The dynamic regional activation map constructed with the virtual unipolar electrograms revealed that the PACs directly spread out from the AT focus to the entire LA without conducting through any preferential pathways, which is sometimes observed with focal ATs during an activation analysis using the array mode of the NCM system. Higa et al.[11] reported that many ATs that originated from the right atrium passed through preferential pathways and propagated to the entire right atrium. The reason

for the difference between their report and ours in terms of the conduction pattern might be due to the difference in the substrate. The anisotropic conduction is also a speculated mechanism that is expressed as preferential pathway, and the difference in the conduction property around the AT focus could describe the phenomenon. The patients reported by Higa et al., all had a LVZ covering the area of the AT focus,[11] whereas ours had no LVZ around the AT focus.

The mechanism of the present AT was unclear, but appeared to be due to a focal discharge or microreentry mechanism, rather than macroreentry because the AT was characterized by a radial spread of the activation, and the endocardial activation did not covered the entire AT cycle length. The contact bipolar voltage map revealed that those PACs and NSAT originated from an area with a normal electrogram amplitude, which suggested that there was no abnormal background underlying the focus. The detailed mechanism of the focal AT in the present patient is unclear, but there is a possibility that the AT was due to abnormal automaticity because warm-up phenomenon was observed in the beginning of the induced AT by rapid atrial pacing under ISP injection (4 μg).

Because the sustained AT did not spontaneously occur or was not induced in the present patient, we employed the array mode of the NCM system to quickly identify the AT focus. Tada et al.[12] also showed the efficacy of the NCM to identify the origin of swallowing-induced atrial tachyarrhythmias. We think that the array mode of the NCM system is very useful especially for some specific tachycardias which are characterized by hemodynamic instability, a nonsustained form, polymorphism or pleomorphism.

Acknowledgement

We thank Mr. Yuuho Iwanaga for his technical assistance.

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