Case Reports

Electroanatomical Mapping of a Right Atrial Tachycardia Originating within the Inferior Vena Cava

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The venae cavae have recently been implicated as sites of ectopic activity that can initiate and perpetuate atrial fibrillation. In the present report a case of a woman with paroxysmal atrial tachycardia is presented. The atrial arrhythmia was mapped with a non-contact electroanatomical mapping system and the exit site of the tachycardia was found within the inferior vena cava. The tachycardia was successfully ablated at a site where the mapping catheter did not record any electrical activity.

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Ectopic atrial foci giving rise to atrial tachycardias may be located at any site within the three-dimensional body of the right atrium as well as on the atrial septum.[1] Additionally, during the last few years a significant body of literature has implicated the pulmonary veins in the genesis of ectopic atrial activity that commonly initiates atrial fibrillation.[2,3,4] An important relation between the atrial anatomy and the distribution of ectopic atrial foci has been elaborated using conventional fluoroscopic mapping.[5,6] The right atrial sites that are usually involved in the genesis of atrial tachycardias are the region of the crista terminalis, the right atrial appendage and the region below the coronary sinus ostium.[7] Recently, the superior venae cavae have been implicated as sites of atrial tachycardias initiating paroxysmal atrial fibrillation.

Electroanatomical mapping with the non-contact system EnSite 3000 (Endocardial Solutions Inc.) has been developed as an efficient method for simultaneous multi-site mapping. Using a complex mathematical concept, the system allows the anatomical reconstruction of the evaluated cavity along with the calculation of the unipolar potentials from 3600 points of this cavity. The information obtained is displayed as isopotential maps of the propagation wavefront and as "virtual" unipolar electrograms from the corresponding sites.

The case of a focal atrial tachycardia that originated within the inferior vena cava (IVC) is presented. This case raises the question of the possible electrophysiological properties of the tissue below the right atrium-IVC junction.

Case presentation

A 49-year-old woman with drug refractory symptomatic atrial tachycardia was referred for electrophysiologic study and radiofrequency catheter ablation. The woman had been symptomatic for the last 10 years with frequent paroxysms of the tachycardia. The rest 12-lead surface ECG revealed salvos and short runs of long RP supraventricular tachycardia with a P wave morphology that was negative in the inferior leads, positive in leads I, aVL, aVR and biphasic in lead V1 (Figure 1). A trans-

Figure 1. Twelve lead ECG during the atrial tachycardia demonstrating the negative P waves in the inferior leads.

thoracic echocardiogram revealed the absence of structural heart disease.

During the electrophysiologic study, conventional catheters were introduced via the femoral veins and positioned in the high right atrium, His region and right ventricle. The non-contact system EnSite 3000 was used for the electroanatomical mapping of the tachycardia. Specifically, the multi-electrode basket catheter of the system was introduced into the right atrium via the left femoral vein and positioned in the low right atrium. The geometric reconstruction of the right atrium was achieved by dragging a mapping catheter along the walls of the cavity and specific anatomic landmarks such as the tricuspid annulus, the coronary sinus os, the right atrial appendage and the junctions of the superior and inferior vena cava with the atrial body. These junctions were characterized by the complete loss of the atrial electrograms.

Subsequently, a run of the clinical atrial tachycardia was recorded by the system and analyzed offline. After appropriate filtering of the signals in order to exclude repolarization potentials, the ectopic activation sequence of the right atrium was tracked backwards toward the exit site of the tachycardia. Tracking was accomplished via the isopotential maps provided by the mapping system. The exit site of the tachycardia was mapped in the posteromedial aspect of the IVC about 2 cm below the IVC-right atrium junction (Figure 2). The virtual electrograms at this

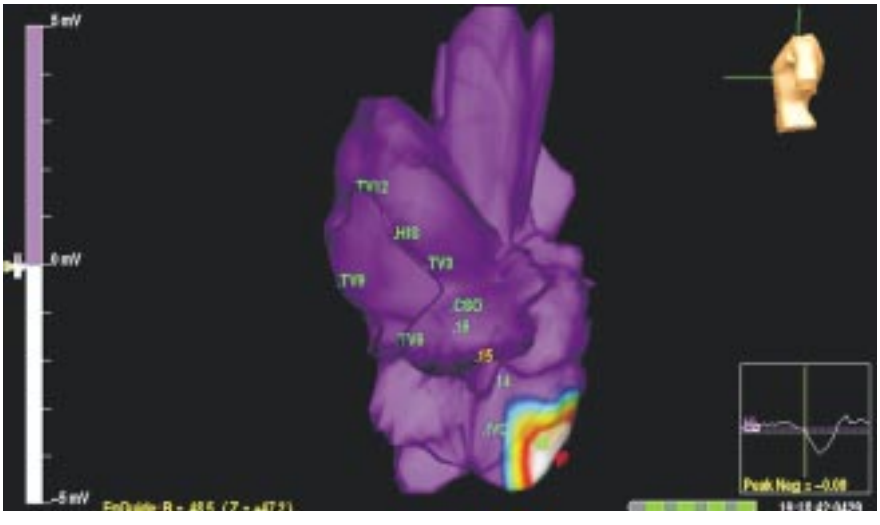

Figure 2. The initial exit site of the tachycardia within the inferior vena cava as shown on the anatomical reconstruction of the right atrium. The white color represents the most negative area at the initiation of the propagation sequence.
CSO: coronary sinus ostium, TV3, 6,9,12: tricuspid valve perimeter, His: His bundle, IVC: inferior vena cava.

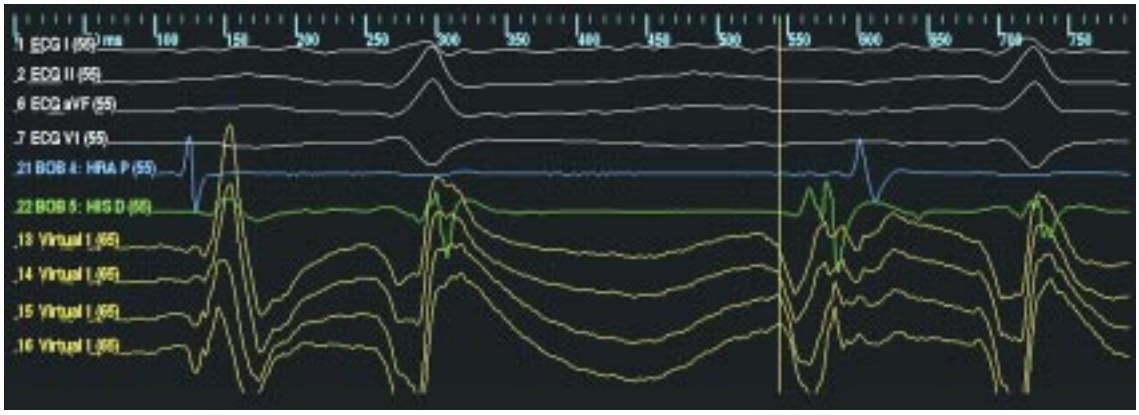

Figure 3. The "virtual" electrograms at the exit site. The QS morphology is characteristic in the unipolar recording 13 while the morphology becomes rS at sites 14, 15, 16, located as shown in the anatomic reconstruction.
CSO: coronary sinus ostium, TV3,6,9,12: tricuspid valve perimeter, His: His bundle, IVC: inferior vena cava.

site exhibited the characteristic QS morphology of an exit site, while electrograms located towards the coronary sinus os and superiorly to the exit site recorded an rS morphology (Figure 3). Advancement of the mapping catheter to the exit site of the tachycardia proved that this site was fluoroscopically below the hemidiaphragm and within the IVC (Figures 4,5). Radiofrequency energy was delivered in the area of the exit site that completely eliminated any ectopic activity. However, a few minutes later a new tachycardia arose and was reanalyzed as previously. The new exit site was located about 1 cm above the initial one at the IVC-right atrium junction (Figure 6). Paradoxically, the bipolar mapping catheter at both exit sites did not record any electrical activity. Entrainment attempts from multiple sites within the IVC failed to demonstrate reentry as a mechanism of arrhythmia.

Radiofrequency energy was again delivered in this area, resulting in the complete elimination of the tachycardia. The patient has remained asymptomatic during a 6-month follow-up period.

Discussion

The presence of excitable myocardial tissue within the wall of the venae cavae has long been reported in the literature.[8,9] Potentials that represent depolarization of cardiac myocytes have been recorded within the superior vena cava and have been implicated in the initiation of atrial fibrillation in certain patients.[10] A histologic study in humans demonstrated the uninterrupted presence of smooth muscle and cardiac myocytes within the superior and inferior vena cava at a distance of 45 mm and 18 mm from the right

Figure 4. Left anterior oblique fluoroscopic view with the ablation catheter at the exit site of the tachycardia within the inferior vena cava. Also shown are the catheters in the high right atrium, the His region and the right ventricle. The "balloon" catheter is located within the right atrial cavity.

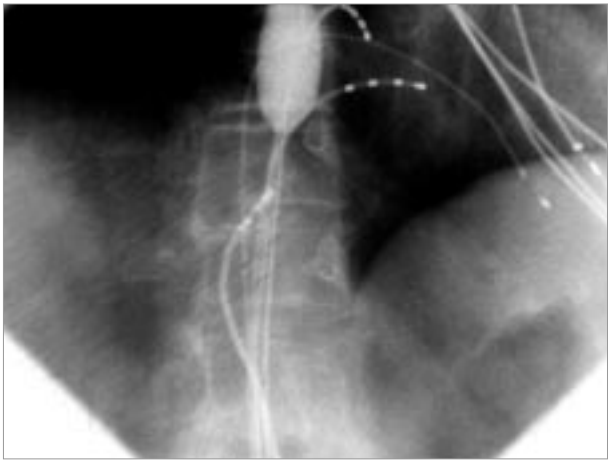

Figure 5. Right anterior oblique fluoroscopic view with the ablation catheter at the exit site of the tachycardia within the inferior vena cava.

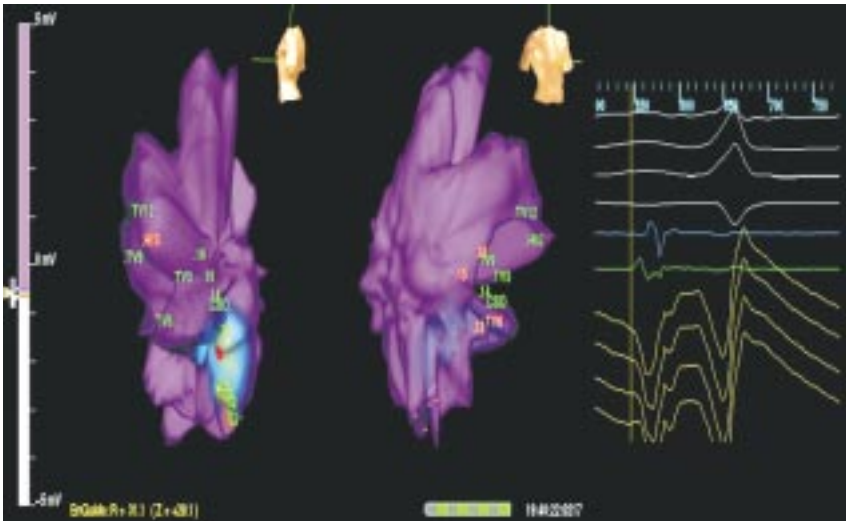

Figure 6. The exit site during the second tachycardia at the right atrium-inferior vena cava junction, characterized by a QS morphology of the "virtual" electrograms.

atrial junction respectively.[11] In the IVC the cardiac muscle fibers extended continuously from the atrium to a level just under the diaphragm. Their fibers were bundled, running circularly or obliquely, and being more abundant in the anterior wall than in the posterior. From these findings, the investigators concluded that the venae cavae close to the atrium are histologically regarded as an extension of the atrium. In a recent report the IVC was the site of atrial ectopy associated with the initiation of atrial fibrillation.[12]

In the present case, an atrial tachycardia originating from within the IVC is reported. The tachycardia was mapped with a non-contact electroanatomical mapping system and the initial exit site of the tachycardia was found at a distance of approximately 2 cm from the IVC-right atrial junction. However, after the initial successful ablation, the recurrence of the tachycardia from a site higher than the initial one probably suggests the presence of a larger triggering area along the musculature of the IVC. The absence of a discrete electrogram on the bipolar mapping catheter at the initial exit site of the tachycardia could be explained by a deeper origin of the tachycardia within the wall of the inferior vena cava that was only revealed by the unipolar "virtual" electrogram.

In conclusion, this case demonstrates that, in rare cases, arrhythmogenic muscular sleeves can be found in the IVC and that the IVC ectopy can be safely ablated using radiofrequency energy.

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