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High density mapping of inappropriate sinus tachycardia further looks into potential mechanisms

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ABSTRACT

Inappropriate sinus tachycardia (IST) is an incompletely understood condition associating unexpectedly fast sinus rates and debilitating symptoms whose management by sinus node modification/ablation demonstrated limited long-term success. We report about a case of IST who underwent two RF procedures using high density mapping system, highlighting some possibly specific features and discussing potential mechanisms.

Inappropriate sinus tachycardia (IST) is an incompletely understood condition associating unexpectedly fast sinus rates and a spectrum of debilitating symptoms including palpitations, weakness, fatigue, dizziness or near syncope [1]. Because of the relative incapacity of drug therapy in alleviating symptoms and reducing heart rate [1], sinus node modification/ablation by percutaneous RF (radio-frequency) ablation is sometimes proposed in disabled refractory patients, although often with limited long-term success [1–4]. This is probably due to a very incomplete knowledge about the mechanisms involved, but also possibly because of the limited precision in conventional electro-anatomical mapping.

Mechanisms involved in IST are still uncertain and probably multifactorial. Main hypothesis rely on mild dysautonomia, intrinsic elevated sinus node rate, hypersensitivity to catecholamines or M2 receptor blockade, still poorly known circulating mediators or neuropeptides, acquired structural alterations of the sinus node or psychiatric causes (see review in 1).

Detailed 3-dimensional mapping helps to identify earliest endocardial activation sites during IST, which are located all along the high crista terminalis area [1,3], which can be interpreted as the endocardial activation breakthroughs from the underlying anatomically subepicardial sinus node. Extended ablation in these areas usually lead to more and more inferior shifts of the activation source, together with changes in P wave morphology and progressive reduction in heart rate [1–3] evoking diffuse electrophysiological alterations. In other cases however, discrete localized lesions lead to an abrupt reduction in heart rate [1,2,5] with similar P wave changes [4], evoking more focally localized abnormal automaticity.

The Rhythmia system ™ (Boston Scientific, Inc.) is a useful tool for achieving very detailed atrial activation [6]. If high-density mapping is helpful in increasing knowledge about the mechanisms of IST is unknown. We report about a case of IST who underwent two RF procedures using the Rhythmia system ™, highlighting some possibly specific features and discussing potential mechanisms.

A 45 years-old woman with IST was referred because of intractable symptoms (palpitations and shortness of breath for minor exertion without any other cause). Symptoms lasted for several years despite repeated trials of single or associated drug therapy (nadolol 120 mg, verapamil, ivabradine 15 mg, diltiazem 200 mg daily). Mean heart rate was 108 bpm (diurnal 117, nocturnal 96, max 177, min 71) on ambulatory recording. Diagnosis of IST was made according to the consensus criteria [1,7] with exclusion of other causes. The patient had no other relevant medication and did not present with other comorbidities or mental health disease

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outside some mental distress due to the long lasting debilitating symptoms linked to IST (not present before). While sinus node modification is not recommended as a part of routine care for patients with IST [7], we choose to perform sinus node ablation/modification because of intractable symptoms despite optimal medical therapy.

A first ablation procedure was performed. Electro-anatomical map of the right atrium was created using the Rhythmia system ™ with the Orion catheter, which is a mini-basket catheter made of eight splines with eight 0.4 mm electrodes each (64 electrodes, 2.5 mm spacing). Activation map showed multiple areas of earliest activation along the sinus node area, crista terminalis and anterior parts of the superior vena cava opening (Fig. 1) unchanged after autonomic blockade. Ablation was delivered using irrigated 4 mm

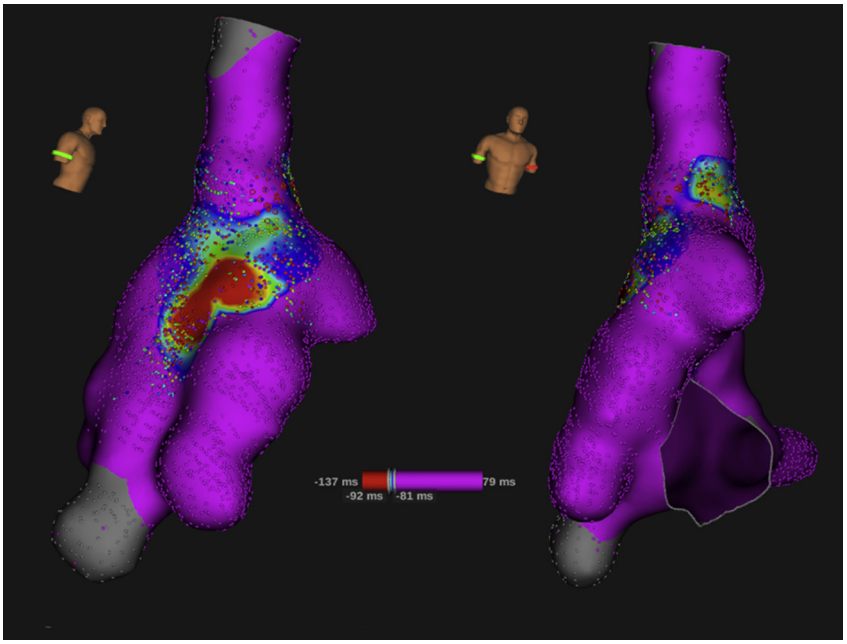

Fig. 1. Activation map during the first ablation procedure. Areas of earliest activation (in red) were rather diffuse, located along the sinus node area, crista terminalis and anterior parts of the superior vena cava (the window of activation scale is restricted to around 10 ms for better delineation of the earliest sources).

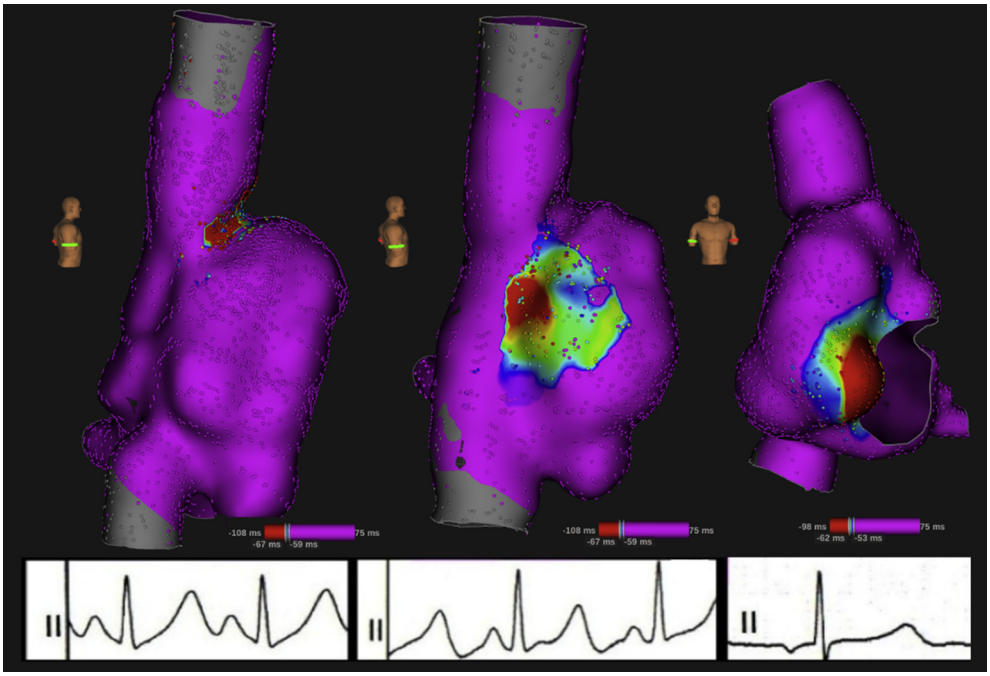

Fig. 2. Activation maps during the second ablation procedure. Areas of earliest activation (in red) were much more localized, initially located at the arcuate ridge (A), then slightly inferior (B) and then at the infero-lateral part for the tricuspid annulus (C), together with changing rates and P wave morphologies (see text) (the window of activation scale is similarly restricted to 10 ms). Note that automatic annotation with the Rhythmia system sometimes led to apparently inconsistent maps due to the recording of large number of points, some being incorrectly annotated (points of different colors in areas with the same activation time). But due to the high number of points, only the "dominant" local activation time is depicted as a background color on the map, avoiding the need to check and reannotate all points in case of clear mechanism.

RF catheter (Cordis Thermocool ™) with 30 W power setting. Despite repeated RF applications (total 30 minutes) all over these areas, only very limited decrease in heart rate was acutely achieved (105–95 bpm) together with changing areas of earliest activation but which were similar to what was already depicted in the first map. Mean heart rate was 96 bpm after the procedure (diurnal 99, nocturnal 92, max 128, min 78) then increasing to 112 bpm a few months later still while on ivabradin 15 mg daily, and a second procedure was planned six months after.

The first activation map during this second procedure (Fig. 2A) displayed a localized focus along the arcuate ridge [1] with a fixed rate of 120 bpm. Of note that the area of earliest activation appeared quite narrower than during the previous procedure, showing also areas of block, possible consequences of the previous procedure. RF application at this site was quickly followed by an abrupt decrease in heart rate (100 bpm) without change in P wave morphology. The second activation map then showed a slightly change in the focus location, which was located about one centimeter inferiorly to the first one, at the anterior aspect of the expected location of the crista terminalis (Fig. 2B). New RF application here also quickly led to a sudden decrease in heart rate (90 bpm) together with a clear change in P wave morphology, which became negative in the inferior leads. A third activation map then showed a displaced focus along the infero-lateral part of the tricuspid annulus (8 o'clock in left anterior view) (Fig. 2C), once again with a quite narrow area of earliest activation. RF application at this third site then led to an additional decrease in heart rate (85 bpm) with occurrence of stable junctional rhythm with constant 1/1 retrograde atrial activation included in the QRS. Over 30 minutes, no any spontaneous P wave was observed, neither at baseline, after bursts of atrial pacing nor during isoproterenol infusion (max heart rate 100 bpm) with a junctional stable rhythm at 80 bpm at the end of the procedure.

During the three following days, repeated ECG demonstrated the reappearance of low atrial rhythm (95 bpm) while ambulatory recording showed a mean heart rate of 86 bpm (diurnal 90 bpm, nocturnal 79 bpm, max 126 bpm, min 65 bpm). Sinus node rhythm only emerged during 6 minutes-walking test (max 120 bpm). The patient was discharged under ivabradin 15 mg daily.

Three months later, while still under ivabradin, the patient was in low atrial rhythm (Fig. 3), ambulatory recording reveal a constant ectopic atrial rhythm with a mean heart rate of 85 bpm (diurnal 93 and nocturnal 73, max 138, min 40) with nocturnal non significant atrial pauses without junctional escape. Low atrial rhythm was present throughout the treadmill test (max 113 bpm). Functional status improved especially for the palpitations, although with persisting dyspnea without apparent pulmonary, biological or cardiac cause and despite normalized heart rates.

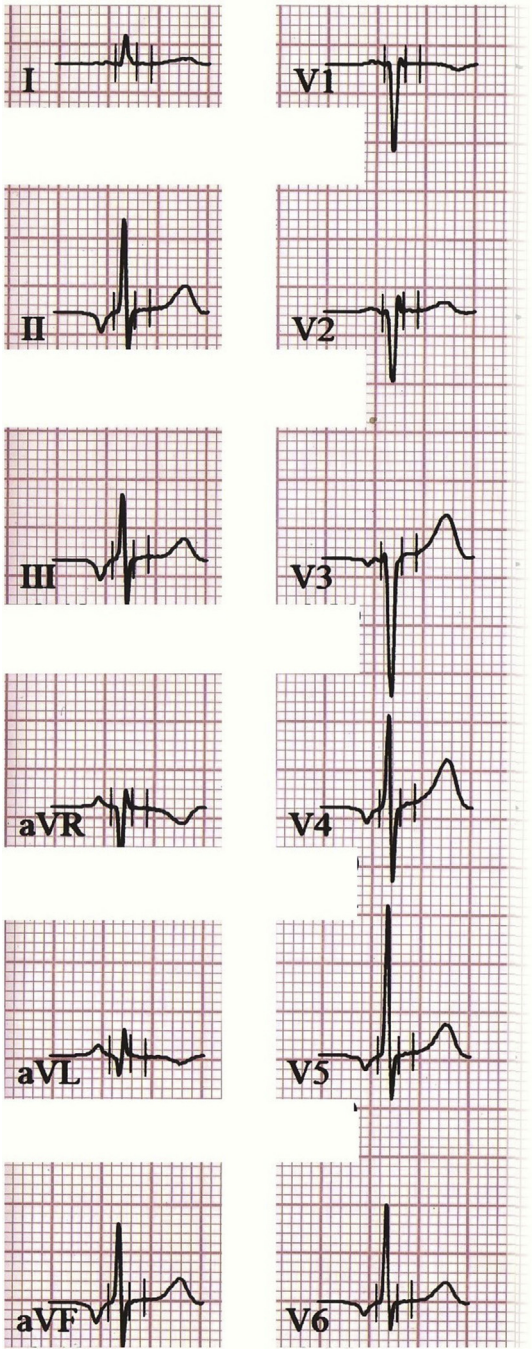

Fig. 3. Baseline 12-lead ECG 3 months after the second procedure, as recorded just before treadmill, showing low right atrial rhythm.

1. Discussion

Apart from a very recent case [5], this is the first report of IST where repeat high-density mapping was used. Endocardial activation in both procedures may add some arguments in favour of a multifocal extended - but not diffuse - area of enhanced abnormal automacity in this particular case, which may not reflect the whole spectrum of IST. The first activation map before ablation displayed a wide area of earliest endocardial activations located all around the sinus node, crista terminalis and neighbouring areas. Large amounts of RF applications at these sites probably let some sources untargeted, which were then able to be successfully eliminated during the second procedure because of easier-to-locate focal residual sources. Alternatively, it is quite possible that the time resolution of the mapping system was initially unable to select the driving foci or endocardial breakthoughs of subepicardial sources, because of very closed activation times, and that RF applications during the first procedure did not target the true culprit zones. The apparent failure of the first procedure may also be caused by the lack of anatomical correlations between true pacemaker sources and apparent earliest endocardial activations because of complex scheme of sino-atrial conduction [6], but this may have been simplified by the first ablation. This case also highlights the possible success of redo endocardial ablation for refractory IST instead of choosing an epicardial approach [1].

Whatever the true location of dominant pacemaker in IST, the areas of abnormal automaticity, if apparently widely extended - or representing the widely extended endocardial projection of subepicardial sources – were not diffuse. They did not extend in the

right and to the left atrium, since no more fast atrial spontaneous activation was observed after removal of culprit sources during the second procedure. This means that the underlying pathophysiological mechanism involved in IST is not diffuse, even if the rate of discharge of the residual atrial pacemaker in our case was rather high for a low atrial escape rhythm.

In conclusion, high-density mapping and repeated ablation of extended focal sources during IST may increase long-term success rate. Even if multiple or widely extended far below the expected sinus node area, sources of abnormal automaticity during IST remain focal and not diffuse throughout the atria.

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