

# Focal Atrial Tachycardia From the Ostium of the Coronary Sinus

## Electrocardiographic and Electrophysiological Characterization and Radiofrequency Ablation

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| <b>OBJECTIVES</b>  | The goal of this study was to characterize the electrocardiographic and electrophysiologic features and frequency of focal atrial tachycardia (AT) originating from the coronary sinus ostium (CS).   |
| <b>BACKGROUND</b>  | The ostium of the coronary sinus has been described as a site of origin of AT, but detailed characterization of these tachycardias is limited.  |
| <b>METHODS</b>     | Thirteen patients (6.7%) of 193 undergoing radiofrequency ablation (RFA) for focal AT are reported. Endocardial activation maps (EAM) were recorded from catheters at the CS (10 pole), crista terminalis (20 pole), and His positions. The P waves were classified negative, positive, isoelectric, or biphasic.   |
| <b>RESULTS</b>     | The mean age was $41 \pm 6$ years, seven female patients, with symptoms for $8 \pm 3$ years. Tachycardia was induced by programmed extra-stimuli in eight patients, was spontaneous in three patients, and in response to isoproterenol in two patients. These foci had a characteristic P-wave morphology. At the CS ostium, the P-wave was deeply negative in all inferior leads, negative or isoelectric becoming positive in lead $V_1$ , then progressively negative across the precordium. Lead aVL was positive in all patients. Earliest EAM activity occurred at the proximal CS at $20 \pm 3$ ms ahead of P-wave. Mean activation time at the successful RFA site = $-36 \pm 8$ ms; RFA was acutely successful in 11 of 13 patients. Long-term success was achieved in 11 of 11 over a median follow-up of $25 \pm 4$ months. |
| <b>CONCLUSIONS</b> | The CS ostium is an uncommon site of origin for focal AT (6.7%). It can be suspected as a potential anatomic site of AT origin from the characteristic P-wave and activation timing. Long-term success was achieved with focal ablation in the majority of patients. (J Am Coll Cardiol 2005;45:1488-93) © 2005 by the American College of Cardiology Foundation  |

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With the advent of radiofrequency ablation (RFA), it has become well-recognized that there is a characteristic anatomic distribution for focal atrial tachycardia (AT). In the right atrium (RA), foci tend to occur along the long axis of the crista terminalis (CT) (1), in the para-hisian region, and around the tricuspid annulus (2). In the left atrium these foci particularly tend to cluster around the pulmonary veins (3) and, more recently, have been described at the mitral annulus (4). Although the ostium of the coronary sinus (CS) is recognized as a site of origin for AT, the frequency and characteristics of AT arising from this site are incompletely described (1,5,6). In the present study, we characterize the electrocardiographic and electrophysiological features of focal AT originating from the CS ostium and its frequency in a consecutive series of patients undergoing RFA.

## METHODS

**Study population.** The study population included 13 patients of a consecutive series of 193 patients undergoing RFA for focal origin of 203 ATs. All patients had clinically documented paroxysmal or incessant AT for which they were having RFA.

All patients underwent electrophysiological study after the provision of informed written consent. The study was approved by the Melbourne Health Research Ethics Committee. Patients were studied in the fasted awake state with minimal use of sedation. All antiarrhythmic drugs were ceased a minimum of five half-lives before the procedure. Two patients had been taking amiodarone.

**Catheter positioning.** Catheter positioning and the approach used in our laboratory for ablation of AT have been previously published (3). In brief, catheters were positioned in the following manner: 1) CS catheter (10-pole, 2-5-2 mm interelectrode spacing) positioned with the proximal bipole at the ostium of the CS by best septal left anterior oblique projection; 2) CT catheter (20-pole, 1-3-1 mm interelectrode spacing) positioned along the CT. Intracardiac echocardiography (9 MHz) was used to aid in posi-

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#### Abbreviations and Acronyms

|     |                           |
|-----|---------------------------|
| AT  | = atrial tachycardia      |
| AV  | = atrioventricular        |
| CS  | = coronary sinus          |
| CT  | = crista terminalis       |
| RA  | = right atrium            |
| RFA | = radiofrequency ablation |

tioning of the catheter when necessary; 3) His bundle electrogram catheter; and 4) mapping and ablation catheter.

Standard electrophysiologic criteria were used to diagnose AT (7). Attempts at AT induction were made including atrial programmed extrastimulation and burst atrial pacing. If this was unsuccessful or when AT was not occurring spontaneously, isoproterenol was infused (1 to 6  $\mu\text{g}/\text{min}$ ). Mapping of the earliest site of endocardial activity relative to surface P-wave was performed with a 4-mm-tip mapping and ablation catheter.

Bipolar intracardiac electrograms were filtered between 30 and 500 Hz, recorded and stored digitally on a computerized system simultaneously with 12-lead surface electrocardiograms. Off-line analysis was performed using on screen digital callipers at 200 mm/s speed.

**Mapping of AT.** Anatomic localization of the atrial focus was performed during tachycardia or atrial ectopy by analysis of: 1) surface electrocardiogram P-wave morphology; 2) right atrial endocardial activation sequence during tachycardia (1,2,4); and 3) point mapping to locate site of earliest endocardial activation relative to surface P-wave onset with the mapping/ablation catheter. A fiducial point on a stable intracardiac electrode relative to P-wave onset was defined to perform point mapping. The average of three ectopic beats was used to calculate the activation time at each intracardiac site. Unipolar electrograms were not routinely recorded as this has not been the practice in our laboratory, although identification of a QS pattern in the unipolar electrogram to mark the site of origin is a useful technique for mapping.

In all patients, the RA was systematically mapped with the ablation electrode to include the following regions not covered by the standard multipolar catheters: high, mid-, and low lateral RA, tricuspid annulus, high, mid, and low septum, the superior vena cava, and the region of the triangle of Koch. Right atrial mapping was particularly focused around the region of earliest endocardial activation. If an early activation time could not be readily identified at the CS ostium (>20 ms before P-wave onset), then a transeptal puncture was performed to allow mapping of the left septum.

In all patients the ostium of the CS was identified by contrast venography, and the proximal pair of the decapolar CS catheter was positioned at this site. Tachycardias were considered to arise from the ostium of the CS when earliest activation was recorded and successful ablation achieved within 1 cm of the ostium of the CS (2).

**P-wave morphology.** Surface 12-lead electrocardiographic P-wave morphology was assessed as previously described (5). The P-wave was analyzed during periods of atrioventricular (AV) block or after ventricular pacing. P waves were described based on the deviation from baseline during the T-P interval as being: 1) positive (+): if there was a positive deviation from the isoelectric baseline; 2) negative (-): if there was a negative deviation; 3) isoelectric: arbitrarily defined when there was no P-wave deviation from baseline of  $\geq 0.05$  mV(4); and 4) biphasic: if there were both positive and negative (+/- or -/+) deflections from baseline. The P-wave amplitudes were measured from the peak to nadir.

**RA endocardial activation sequence.** The consistent deployment of a 20-pole catheter on the CT, a decapolar catheter in the CS, and a catheter in the His position allowed characterization of the right atrial endocardial activation sequence maps. Activation timing was measured from the onset of the P-wave in lead II of the surface electrocardiogram (ECG) (arbitrarily assigned a time of 0 ms) to each of the intracardiac bipoles of these catheters. Activation times were measured in a standardized fashion to onset of the first rapid deflection from the baseline.

**RFA and outcome.** Radiofrequency ablation was performed with continuous temperature feedback control of power output to achieve a target temperature of 50°C to 60°C for a maximum power of 40 to 50 W. Acute procedural success was defined by the absence of tachycardia or ectopy 30 min after ablation despite infusion of isoproterenol (up to 6  $\mu\text{g}/\text{min}$ ) and burst atrial pacing.

**Statistical analysis.** All variables are expressed as mean  $\pm$  SD. Statistical comparisons within the group were made using a paired *t* test. A *p* value <0.05 was considered statistically significant.

## RESULTS

**Patient characteristics.** Of 193 patients who underwent RFA of 203 ATs, 13 (6.7%) were determined to have an AT originating from the CS. Seven of 13 patients were women (mean age  $41 \pm 6$  years; range 15 to 72 years). Symptoms attributable to tachycardia had been present for  $8.3 \pm 2.7$  years, and patients had failed a mean of  $1.3 \pm 0.2$  antiarrhythmic medications. Two patients had ischemic heart disease, and one patient had mild mitral valve disease. Previous RFA had been performed for AVNRT in two patients and AT arising from the superior mitral annulus in one patient.

**Tachycardia characteristics.** In seven patients tachycardia was induced by atrial programmed extra-stimuli alone, and in one with programmed extra-stimuli during isoproterenol infusion. In three patients AT was spontaneous, and, in another two patients, spontaneous tachycardia developed during isoproterenol infusion. Sustained tachycardia was present in 12 of 13 patients with a mean tachycardia cycle length of  $360 \pm 21$  ms. In all patients, AT could be differentiated from atypical AVNRT or a slowly conducting

**Table 1.** P-Wave Morphology

|           |        | ECG Lead |        |        |                |                |                |  |
|-----------|--------|----------|--------|--------|----------------|----------------|----------------|--|
| I         | II     | III      | aVL    | aVR    | V <sub>1</sub> | V <sub>3</sub> | V <sub>6</sub> |  |
| iso 11    | neg 13 | neg 13   | pos 13 | pos 13 | neg/pos 7      | neg 8          | neg 10         |  |
| low pos 2 |        |          |        |        | iso/pos 5      | biphasic 3     | iso 2          |  |
|           |        |          |        |        | neg 1          | iso 2          | biphasic 1     |  |

ECG = electrocardiographic; iso = isoelectric; neg = negative; pos = positive.

accessory pathway by the presence of persistent or transient AV dissociation.

**P-wave morphology.** These foci had a characteristic P-wave morphology (Table 1). The P-wave was deeply negative in leads II, III, and aVF in 13 of 13 patients. The negative amplitude was significantly greater in leads II and III than in aVF (mean P-wave amplitude in lead II  $0.19 \pm 0.02$  mV, lead III  $0.20 \pm 0.02$  mV, and in lead aVF  $0.12 \pm 0.02$  mV) ( $p = 0.02$  to lead III and  $p = 0.046$  to lead II). The inferior leads had a secondary upright component in 4 of 13 patients, which is demonstrated in Figures 1B and 1C.

Lead I was isoelectric defined by an amplitude  $<0.05$  mV in 11 of 13 patients (mean P-wave amplitude in lead I =  $0.03 \pm 0.01$  mV). Leads aVL and aVR were positive in 13 of 13 patients. For AT originating from the CS ostium, the P-wave in the precordial leads was characteristic. Lead V<sub>1</sub> showed an isoelectric component followed by an upright component in 5 of 13 patients or a mildly inverted component followed by an upright component in 7 of 13 patients. The P-wave in the remaining patient was negative in V<sub>1</sub>. As these two components were followed across the precordial leads, the initial deflection became more negative, and the second deflection became isoelectric. The extent of this precordial evolution was variable (Fig. 1).

The described P-wave morphology is very similar to that of typical atrial flutter, which has an atrial exit zone at the CS ostium (8).

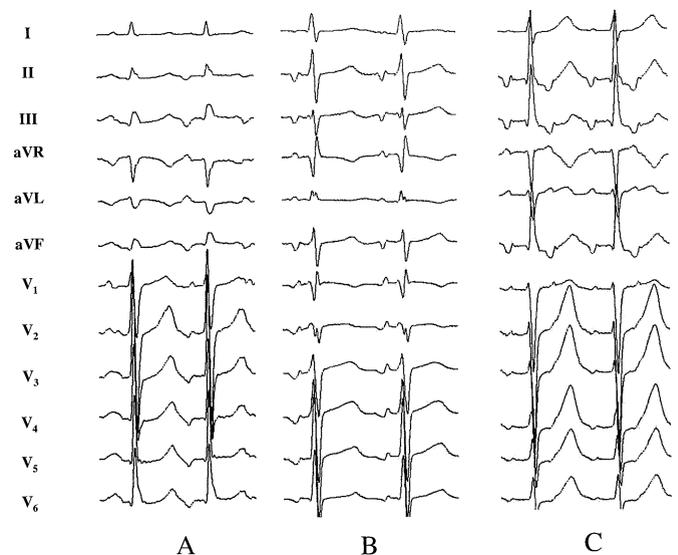
**Atrial endocardial activation sequence mapping.** Earliest endocardial activation on the standard catheters (His bundle, CT, and CS) occurred at the CS ostium in 13 of 13 patients. The mean activation times to P-wave onset were as follows: His bundle  $4 \pm 6$  ms; high CT  $25 \pm 7$  ms, mid-CT  $11 \pm 19$  ms, and low CT  $3 \pm 17$  ms. At the CS the mean activation times to P-wave onset were: proximal CS  $-20 \pm 3$  ms; mid-CS  $-16 \pm 4$  ms, and distal CS  $4 \pm 5$  ms ( $p < 0.01$  for proximal CS to His, all cristal locations, and distal CS). The characteristic atrial endocardial activation sequence is demonstrated in Figure 2. In general, for CS tachycardias in this location, proximal CS activation was earliest, then His with the CT activated from low to high.

**RFA.** Mapping for the earliest endocardial activity was performed during tachycardia (12 patients) or with atrial ectopy (1 patient) (Fig. 3). Coronary sinus venography did not reveal abnormal anatomy or diverticula in any patient.

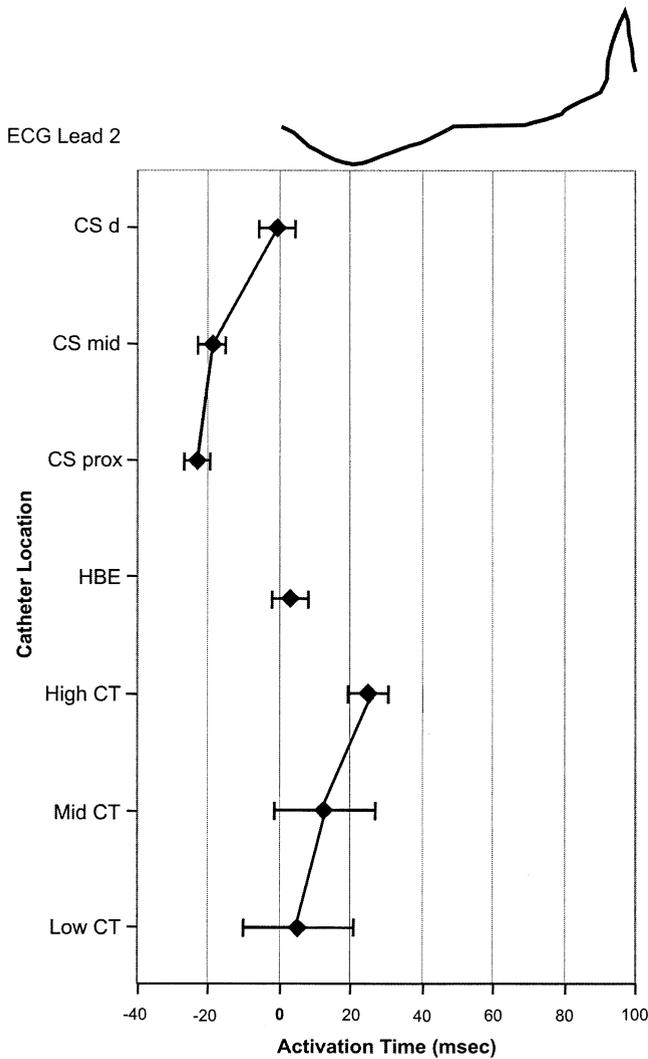
At the CS ostium, endocardial atrial activation preceded the onset of the P-wave by a mean of  $36 \pm 8$  ms (Fig. 3);

RFA was acutely successful in 11 of 13 patients. Electrogram timing at successful sites was significantly earlier than at unsuccessful sites ( $15 \pm 6$  ms ahead of P-wave onset,  $p < 0.001$ ). The electrogram at successful sites was complex and fractionated (9) in 3 of 11 patients. The mean number of radiofrequency applications was  $7 \pm 5$ . When RFA was performed during tachycardia, the time to termination at the successful site was  $8 \pm 5$  s. At the site of successful ablation, “speeding” occurred in six patients and deceleration of tachycardia before termination in one.

For the last 10 cases, the CS ostium was divided into four sectors: anterior, posterior, superior, and inferior. The site of successful ablation was at the superior aspect in four patients (Fig. 4), posterior lip in three patients, and at the floor or inferior border of the CS ostium in one patient. In another patient earliest activity was recorded in the superior lip of the CS ostium where a large His potential was recorded. In view of the risk of AV block, and prior discussion with the patient, no ablation was performed. In one other patient, activation timing was earliest at the posterior lip of the CS ostium (30 ms before P-wave). Ablation at this site produced speeding and tachycardia termination, but infrequent ectopy continued to be observed. The clinical outcome was



**Figure 1.** The P-wave morphology from three patients is presented. The characteristic findings were: a deeply inverted P-wave in the inferior leads with 4 of 13 patients having a secondary upright component (B and C). Lead V<sub>1</sub> was inverted (B and C) or isoelectric (A) then upright. Leads aVL and aVR were positive in 13 of 13 patients.



**Figure 2.** Graphic representation of the mean activation times at each of the recorded endocardial sites for the 13 patients. Earliest endocardial activation on these standard catheters occurred at the proximal coronary sinus (CS) in all 13 patients. CT = crista terminalis; HBE = His bundle. Numbers refer to bipolar pair.

significant symptomatic benefit, but with a requirement for ongoing medical therapy.

The mean procedure time was  $112 \pm 29$  min, and the fluoroscopy time was  $28 \pm 5$  min. An irrigated tip ablation catheter was not required in any patient. There were no procedural complications.

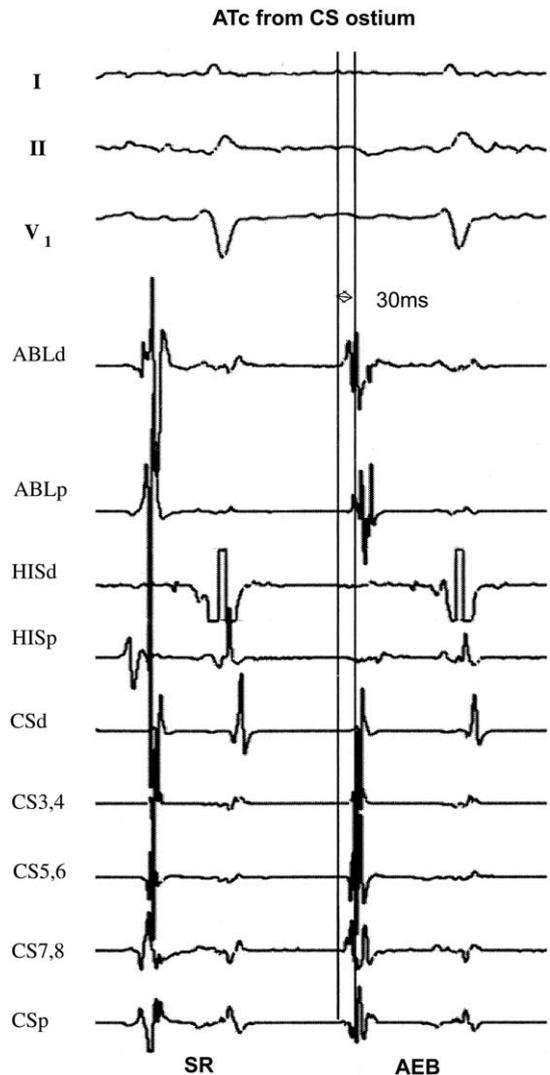
Three patients had a prior ablation for a different arrhythmia (two AV nodal reentrant tachycardia and one AT from the superior mitral annulus).

**Follow-up.** Of the 11 tachycardias successfully ablated, one patient had a recurrence at 24 h after the initial procedure. A repeat procedure was performed with successful ablation at the original site at the ostium of the CS. One patient who had been in persistent tachycardia had infrequent ectopy after ablation, which was controlled on medical therapy. Long-term success was achieved in 11 of 11 patients off antiarrhythmic medication over a median follow-up of  $25 \pm 4$  months.

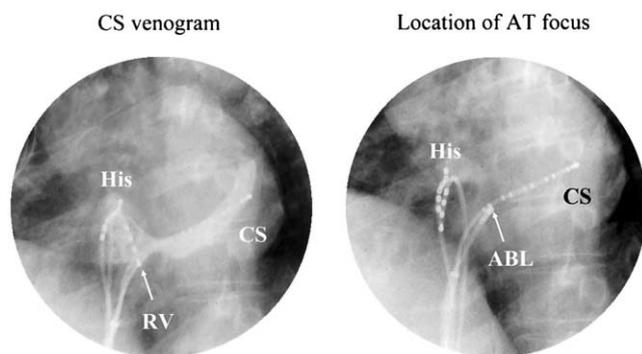
## DISCUSSION

Although most case series include the ostium of the CS as a recognized anatomic site of origin for focal AT (1,5,6), the nature of these tachycardias has not been well-described. In the present series, the ostium of the CS was the site of origin in 13 (6.7%) of 193 patients undergoing RFA for focal AT from all sites and represented 9% of ATs arising in the RA. These tachycardias have a characteristic P-wave morphology and can be ablated with a high success rate using standard mapping tools. In this series, the superior and posterior lip of the CS ostium were the most common sites of origin.

**Prior studies.** Previous ablation series have reported on small numbers of patients undergoing ablation for focal AT arising from the CS ostium (1,5,6,10,11) making it difficult to reach conclusions regarding its frequency and electrocardiographic and electrophysiologic features. Chen et al. (12)



**Figure 3.** Endocardial electrograms demonstrating the ablation signal (ABLd) at the successful site 30 ms ahead of P-wave onset. The coronary sinus (CS) is activated from proximal to distal ahead of His activation. AEB = atrial ectopic beat; SR = sinus rhythm.



**Figure 4.** Left anterior oblique projections demonstrating a coronary sinus (CS) venogram on the **left panel** and the successful location for ablation of focal atrial tachycardia at the superior lip of the CS ostium on the **right panel**. ABL = ablation catheter; RV = right ventricle.

reported 49 (34.7%) patients with AT originating from the atrial septum including 22 from the posteroseptal region. It was not clear what proportion of the total AT series these represented or their relation to the CS ostium; ECG data presented for inferior leads and  $V_1$  were similar to those in the current study.

**P-wave morphology.** Although P-wave morphology can provide only a general guide to the localization of ATs, it was remarkably consistent in the current series. Indeed, the P-wave is similar in many respects to the flutter wave morphology in counterclockwise atrial flutter consistent with an atrial exit zone at the CS in the latter arrhythmia. A similar P-wave morphology has been reported for tachycardias in the midseptal and perinodal regions, although it is less usual to observe a deeply inverted P-wave in inferior leads at these locations (12). Similarly, tachycardias arising from the left side of the septum even in the inferoposterior region usually demonstrate isoelectric or biphasic P waves in inferior leads (13). Tachycardias at the inferior and septal region of the tricuspid annulus (near to the ostium of the CS) will generally have an inverted P-wave in  $V_1$  (and be inverted across precordial leads) (2). Finally, tachycardias arising from the body of the CS, several centimeters beyond the ostium, generally show a uniphasic upright P-wave in  $V_1$ , which remains upright across most precordial leads (14).

**Activation mapping and RFA.** Activation mapping demonstrated earliest activity to be at the proximal CS ahead of P-wave onset in all patients (mean  $20 \pm 3$  ms ahead of P-wave). With careful mapping around the rim of the CS, the mean earliest activation time at the successful ablation site was  $36 \pm 8$  ms. Prior studies have suggested that early sites can be recorded in the RA at the septal region (38 ms ahead of P-wave) even when the tachycardia originates from the left septum (13). In our experience, if a clear P-wave onset (unencumbered by the T-wave) is recorded and referenced to an intracardiac signal, then when sites are found in the RA 20 ms before P-wave it is probable that the tachycardia will be right atrial in origin. This was the case in the current series. Although we did not utilize a three-dimensional mapping system in the current series, this may

have resulted in shorter fluoroscopy times and improved success rates. We observed that CS ostial tachycardias occurred most frequently from the posterior and superior lip of the CS ostium but could arise from around the entire circumference. These observations emphasize the importance of careful circumferential mapping.

It is well-recognized that ablation within the CS ostium, particularly in the roof (superiorly), can interfere with fast pathway conduction. In one patient in the current series, earliest activity was recorded at the roof of the CS where a large His potential could be recorded. Ablation was deferred, and this patient has elected medical therapy for the present.

**ATs at the CS ostium.** While the mechanism of AT originating at the CS ostium cannot be determined from a clinical study, an appreciation of the particular anatomy of this region potentially provides some insights. The CS is an important site of interatrial connection (15). The embryological origin of the CS is the left horn of the primitive sinus venosus, and a sheath of striated myocardium persists as a remnant from the sinus venosus surrounding the proximal end of the CS (16). This cuff of striated muscle is continuous with the right atrial myocardium and extends a variable distance to terminate in the region of the Valve of Vieussens (17). The ostium is characterized by an abrupt change in right atrial fiber orientation in the region of the Thebesian valve (17). This region of abrupt change in fiber orientation may potentially provide the anisotropic conduction necessary for the initiation of re-entry. In this study 8 of 13 CS ostial tachycardias could be initiated and terminated by programmed stimulation suggesting re-entry as a possible mechanism. In the remaining five, tachycardia occurred spontaneously or in response to isoproterenol more in keeping with abnormal automaticity or triggered activity (11). In a series of septal ATs from Chen et al. (12) 44 of 49 were inducible with programmed stimulation, and 22 (45%) were responsive to catecholamines. Information regarding AT arising from the posteroseptal region was not presented separately.

**Conclusion.** The CS ostium is an uncommon site of origin for focal AT (6.7% of AT). It can be suspected as a potential anatomic site of AT origin from the characteristic P-wave and activation timing. Long-term success was achieved with focal ablation in the majority of patients.

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## REFERENCES

1. Kalman JM, Olgin JE, Karch MR, Hamdan M, Lee RJ, Lesh MD. "Cristal tachycardias": origin of right atrial tachycardias from the crista terminalis identified by intracardiac echocardiography. *J Am Coll Cardiol* 1998;31:451–9.
2. Morton JB, Sanders P, Das A, Vohra JK, Sparks PB, Kalman JM. Focal atrial tachycardia arising from the tricuspid annulus: electro-

- physiologic and electrocardiographic characteristics. *J Cardiovasc Electrophysiol* 2001;12:653-9.
3. Kistler PM, Sanders P, Fynn SP, et al. Electrophysiological and electrocardiographic characteristics of focal atrial tachycardia originating from the pulmonary veins: acute and long-term outcomes of radiofrequency ablation. *Circulation* 2003;108:1968-75.
  4. Kistler PM, Sanders P, Hussin A, et al. Focal atrial tachycardia arising from the mitral annulus: electrocardiographic and electrophysiologic characterization. *J Am Coll Cardiol* 2003;41:2212-9.
  5. Tang CW, Scheinman MM, Van Hare GF, et al. Use of P-wave configuration during atrial tachycardia to predict site of origin. *J Am Coll Cardiol* 1995;26:1315-24.
  6. Natale A, Breeding L, Tomassoni G, et al. Ablation of right and left ectopic atrial tachycardias using a three-dimensional nonfluoroscopic mapping system. *Am J Cardiol* 1998;82:989-92.
  7. Saoudi N, Cosio F, Waldo A, et al. Classification of atrial flutter and regular atrial tachycardia according to electrophysiologic mechanism and anatomic bases: a statement from a joint expert group from the Working Group of Arrhythmias of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *J Cardiovasc Electrophysiol* 2001;12:852-66.
  8. Schwartzman D, Callans DJ, Gottlieb CD, Dillon SM, Movsowitz C, Marchlinski FE. Conduction block in the inferior vena caval-tricuspid valve isthmus: association with outcome of radiofrequency ablation of type I atrial flutter. *J Am Coll Cardiol* 1996;28:1519-31.
  9. Jais P, Shah DC, Haissaguerre M, et al. Mapping and ablation of left atrial flutters. *Circulation* 2000;101:2928-34.
  10. Kay GN, Chong F, Epstein AE, Dailey SM, Plumb VJ. Radiofrequency ablation for treatment of primary atrial tachycardias. *J Am Coll Cardiol* 1993;21:901-9.
  11. Higa S, Tai CT, Lin YJ, et al. Focal atrial tachycardia: new insight from noncontact mapping and catheter ablation. *Circulation* 2004;109:84-91.
  12. Chen CC, Tai CT, Chiang CE, et al. Atrial tachycardias originating from the atrial septum: electrophysiologic characteristics and radiofrequency ablation. *J Cardiovasc Electrophysiol* 2000;11:744-9.
  13. Marrouche NF, SippensGroenewegen A, Yang Y, Dibs S, Scheinman MM. Clinical and electrophysiologic characteristics of left septal atrial tachycardia. *J Am Coll Cardiol* 2002;40:1133-9.
  14. Badhwar N, Kalman JM, Sparks PB, Kistler PM, Randall JL, Scheinman MM. Atrial tachycardia arising from the coronary sinus musculature: electrophysiologic characteristics and long term follow up (abstr). *Heart Rhythm* 2004;1:57.
  15. Antz M, Otomo K, Arruda M, et al. Electrical conduction between the right atrium and the left atrium via the musculature of the coronary sinus. *Circulation* 1998;98:1790-5.
  16. Gerlis LM, Davies MJ, Boyle R, Williams G, Scott H. Pre-excitation due to accessory sinoventricular connexions associated with coronary sinus aneurysms. A report of two cases. *Br Heart J* 1985;53:314-22.
  17. Chauvin M, Shah DC, Haissaguerre M, Marcellin L, Brechenmacher C. The anatomic basis of connections between the coronary sinus musculature and the left atrium in humans. *Circulation* 2000;101:647-52.