Focal Atrial Tachycardia Originating From the Non-Coronary Aortic Sinus

Electrophysiological Characteristics and Catheter Ablation

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OBJECTIVES	We sought to investigate electrophysiological characteristics and catheter ablation in patients with focal atrial tachycardia (AT) originating from the non-coronary aortic sinus (AS).
BACKGROUND	In patients with failed ablation of focal AT near the His bundle (HB) region, an origin from the non-coronary AS should be considered because of the close anatomical relationship.
METHODS	This study included 9 patients with focal AT, in 6 of whom attempted radiofrequency (RF) ablation had previously failed. Activation mapping was performed during tachycardia to identify an earliest activation in the atria and the AS. The aortic root angiography was performed to identify the origin in the AS before RF ablation.
RESULTS	Focal AT was reproducibly induced by atrial pacing. Mapping in atria demonstrated that the earliest atrial activation was located at the HB region, whereas mapping in the non-coronary AS demonstrated that an earliest atrial activation preceded the atrial activation at the HB by 12.2 ± 6.9 ms and was anatomically located superoposterior to the HB in all 9 patients. Also, His potentials were not found at the successful site in the non-coronary AS in all 9 patients. The focal AT was terminated in <8 s in all 9 patients. Junctional beats and PR prolongation did not occur during RF application in all 9 patients. No complications occurred in any of the nine patients. All 9 patients were free of arrhythmias without antiarrhythmic drugs during a follow-up of 9 ± 3 months.
CONCLUSIONS	In patients with focal AT near the HB region, mapping in the non-coronary AS can improve clinical outcome. (J Am Coll Cardiol 2006;48:122–31) © 2006 by the American College of Cardiology Foundation

Catheter ablation has become the treatment of choice in patients with focal atrial tachycardia (AT) (1–12). Focal AT is commonly located in the crista terminalis (1,2), near the tricuspid and mitral annulus (3–6), within the pulmonary veins (4,7,8), at the ostium of the coronary sinus (CS) (9), and at the para-hisian region (10–12). In patients with failed ablation of a focal AT, an unusual location of an arrhythmogenic origin must be considered (5,6,13). A case report has described that a focal AT in one patient could be successfully ablated from the non-coronary aortic sinus (AS) (13). However, the arrhythmic characteristics of these patients have not been described in detail. In this study, we report on the arrhythmic characteristics and the ablation procedure in nine patients with a focal AT within the non-coronary AS, and the anatomical relationship between the non-coronary AS and the atria.

METHODS

Patient population. Nine patients (6 female; ages 54 ± 12 years, range 32 to 66 years) with frequent paroxysmal supraventricular tachycardia (PSVT) were referred to our centers (5 patients from AK St. Georg in Hamburg and 4 patients from Fuwai Hospital in Beijing). The clinical tachycardia presented with abrupt onset and offset in all nine patients and could be terminated by vagal stimulation in three patients. Patient symptoms included disabling palpitations and dizziness in 9 patients, presyncope in 1 patient, and syncope in 1 patient. The tachycardia was first diagnosed 8.6 ± 2.3 years (range 2 to 20 years) before referral and had been ineffectively treated by a mean of 3.0 ± 0.8 oral antiarrhythmic drugs. An electrophysiological study had previously been performed in 6 of the 9 patients. The clinical tachycardia had been diagnosed as a focal AT originating at the His bundle (HB) area in 5, and as possible atrioventricular node re-entrant tachycardia (AVNRT) in 1 patient. Radiofrequency (RF) ablation with a 4-mm thermocouple catheter had been attempted near the HB area in 5 patients and at a slow pathway area in 1 patient. Ablation was unsuccessful with one procedure in 5 patients and two procedures in 1 patient. No previous cardiac surgery was performed in any patient. No evidence of structural heart disease was found in any patient.

Electrophysiological study. After giving informed consent and withdrawing from antiarrhythmic drugs for at least five half-lives, all patients underwent an electrophysiological evaluation under intravenous sedation. Three catheters were

Abbreviations and Acronyms

AS	=	aortic sinus
AT	=	atrial tachycardia
AV	=	atrioventricular
AVNRT	=	atrioventricular nodal re-entrant tachycardia
CL	=	cycle length
CS	=	coronary sinus
ECG	=	electrocardiogram
HB	=	His bundle
LA	=	left atrium
PSVT	=	paroxysmal supraventricular tachycardia
RA	=	right atrium
RF	=	radiofrequency
RVA	=	right ventricular apex

introduced to the right atrium (RA), the right ventricular apex (RVA), and at the HB region via the femoral veins. Also, a 7-F multipolar catheter was advanced within the coronary sinus (CS) via the left subclavian vein. The stimulation protocol consisted of programmed stimulation at two basic cycle lengths (CLs) with up to two extrastimuli and burst pacing at the RA and the RVA. If the clinical tachycardia did not occur spontaneously and was not inducible during the baseline state, intravenous isoproterenol infusion (2 to 5 μg/min) was administered to provoke the clinical arrhythmia or facilitate the tachycardia induction. Programmed stimulation and burst pacing were used for tachycardia induction and termination, and they were repeated more than five times to ensure reproducibility of the response.

Focal AT is defined as: 1) atrial activation starting at a small area from which it radiates in all directions, and 2) range of activation times less than the tachycardia CL.

ECG analysis. P-wave morphology on the surface electrocardiogram (ECG) was assessed as previously described (4). The P-wave analysis was performed during periods of atrioventriclar (AV) block or after ventricular pacing. The P waves were described on the basis of 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 negative deviation; 3) biphasic if there were both positive and negative deflections from baseline; and 4) flat if there were no deflections from baseline.

Mapping and RF ablation. With induced tachycardia, activation mapping was performed via a 7-F 4-mm tip ablation catheter (Biosense-Webster, Inc., Diamond Bar, California). Activation time was measured from the onset of the local electrogram to a stable atrial electrogram recorded from a catheter within the CS. In all patients, initial mapping was performed in the RA and the left atrium (LA). Whenever mapping in both atria failed to identify a successful ablation site, mapping in the AS was performed retrogradely via the right femoral artery. The arrhythmia origin was defined by the site with the earliest atrial activation and successful ablation. Bipolar and unipolar

electrograms were filtered at 30 to 400 Hz and 0.05 to 400 Hz, respectively.

When the earliest atrial activation was identified in the ASs, a 5-F pigtail catheter was inserted into the aortic root via the left femoral artery. The three ASs were visualized by aortic root angiography. Radiofrequency energy with a thermocouple 4-mm tip was delivered as previously described using a target temperature of 55 °C (14). Radiofrequency energy was started at 20 W and increased up to 40 W in the AS under continuous fluoroscopy to reach the target temperature. Radiofrequency energy was maintained for 90 s if tachycardia termination occurred with 10 s and catheter dislodgement was not observed.

Follow-up. After the procedure, patients were treated with oral aspirin for 3 months. Each patient returned for evaluation in the outpatient clinics at 1, 3, 6, and 12 months. Transthoracic echocardiography and 24-h Holter recordings were performed the day after the procedure and at the 3-month follow-up visit.

Statistical analysis. Data are expressed as mean ± SD.

RESULTS

Electrophysiological characteristics. In these nine patients, clinical tachycardia did not occur spontaneously. Sustained clinical tachycardia with a CL of 357.6 ± 62.5 ms (270 to 435 ms) was easily and reproducibly induced by both programmed atrial stimulation and atrial burst pacing in 8 patients, and by only burst atrial pacing in 1 patient. Also, all inducible tachycardias were easily terminated by burst atrial pacing. No evidence of accessory pathway was found during atrial and ventricular stimulation in any of the 9 patients. No tachycardia was inducible by ventricular stimulation in any patients. A critical delay from the atrial-to-His activation interval was not required for tachycardia induction in these 9 patients. The inducible tachycardia activated the ventricle with one-to-one AV conduction in 6 patients, and two-to-one or one-to-one AV conduction in the other 3 patients, including the 1 patient with misdiagnosed AVNRT in the previously failed ablation procedure. Intravenous infusion of isoproterenol was not required for tachycardia induction in all 9 patients before ablation. On the basis of these findings, clinical tachycardia was diagnosed as a focal AT.

Also, intravenous administration of 12 mg adenosine during tachycardia resulted in a change in the tachycardia CL, which led to a change of activation interval from the atrial-to-His activation in the following beat. Finally, intravenous adenosine terminated the tachycardia before AV block occurred during sinus rhythm (Fig. 1).

ECG characteristics. The P waves in lead I and aVL during tachycardia were positive in these nine patients. Also, the P waves in leads V$_1$ and V$_2$ during tachycardia were negative/positive in all nine patients. The P waves in II, III, and aVF during tachycardia were negative/positive
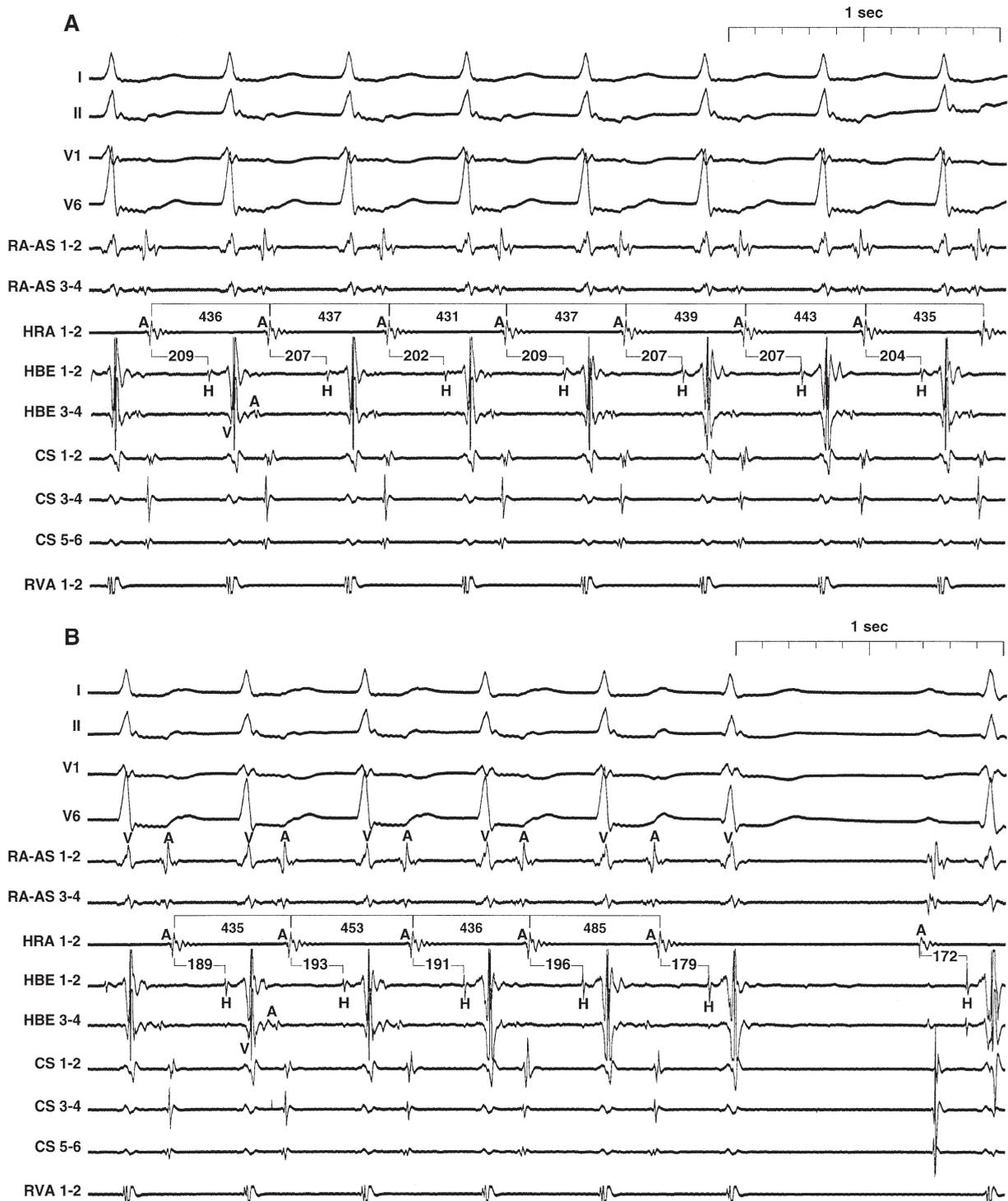
Figure 1. Continuous recordings from tracings **A** to **B** show electrocardiogram (ECG) leads I, II, V₁, V₆, and intracardiac electrogram recorded from a mapping catheter at the His bundle region (RA-AS 1 to 2, RA-AS 3 to 4, RA-AS uni), a catheter in the right atrium, a catheter at the His bundle region, a catheter within the coronary sinus, and a catheter at the right ventricular apex during intravenous administration of 12 mg adenosine in a patient with induced sustained focal atrial tachycardia originating from the non-coronary aortic sinus. Note that: 1) there is the change in the tachycardia cycle length (TCL), from 431 to 485 ms, before termination during intravenous administration of 12 mg adenosine; 2) the interval from atrium to His activation (AH interval) during the tachycardia depends on the previous TCL, which results in short AH with previous long TCL or long AH interval with previous short TCL; and 3) the tachycardia termination occurs without sequential atrioventricular block during sinus rhythm. AS = aortic sinus; CS = coronary sinus; HBE = His bundle region; RA = right atrium; RVA = right ventricular apex.

in seven of these 9 patients (the left panel in Fig. 2). In the remaining 2 patients, the P waves in the II, III, and aVF leads were negative in 1 patient (the middle panel in Fig. 2), and the P waves were positive in the II and aVF leads and flat in the III lead in 1 patient (the right panel in Fig. 2).
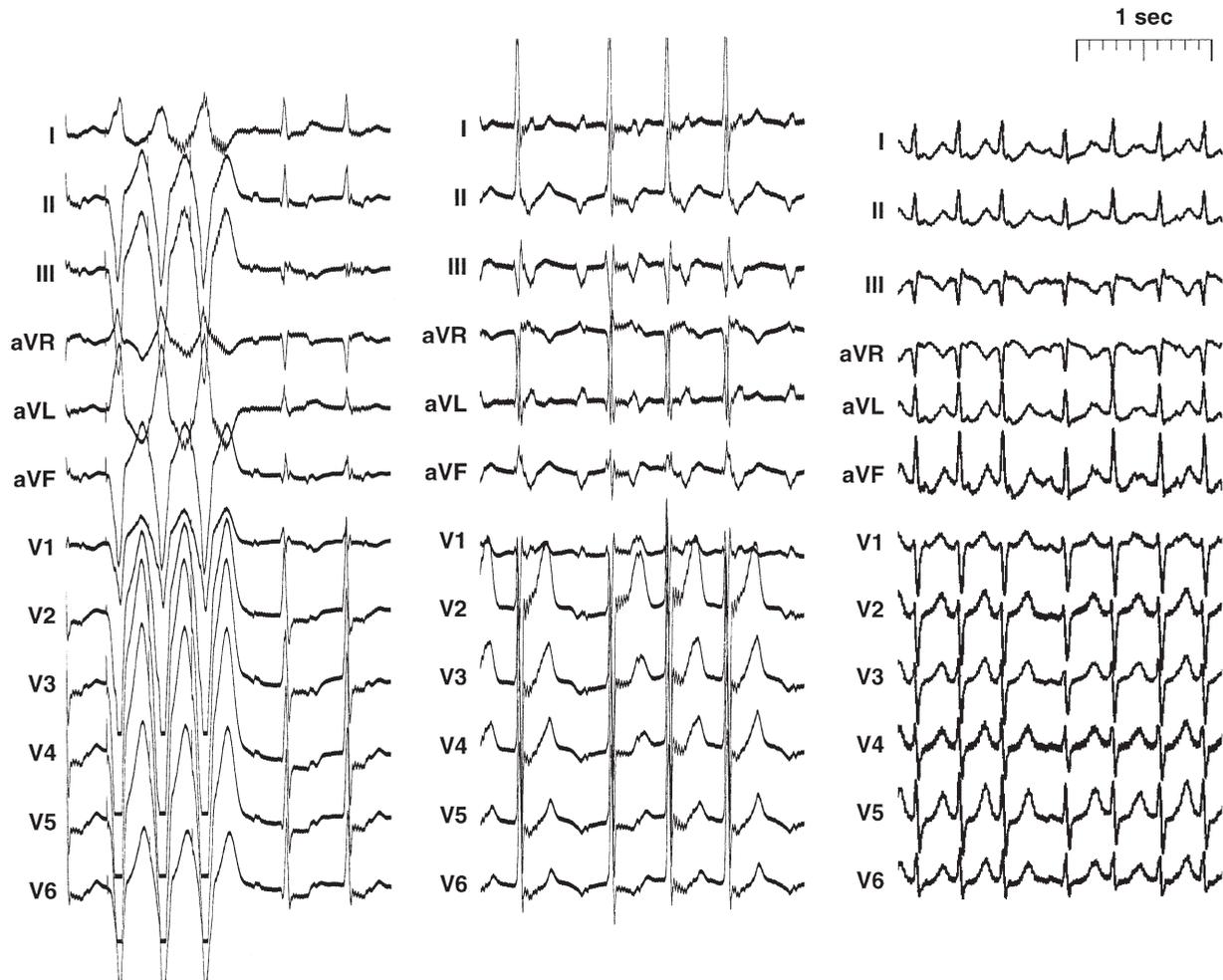
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Figure 2. P-wave morphology on 12-lead ECG in three patients with focal atrial tachycardia (FAT) originating from the non-coronary aortic sinus.

Mapping and ablation in the RA and LA. In these nine patients, mapping in the RA demonstrated that the earliest atrial activation was located near the HB area, and preceded the reference CS electrogram by 52.9 ± 8.7 ms (41 to 67 ms). The local electrograms at the earliest RA site presented with a distinct His potential in all 9 patients (Fig. 3A). In five of these 9 patients, a mean of 6.2 ± 1.0 RF applications (range 5 to 7) was delivered at the RA site with the earliest activation, and resulted in transient termination of the tachycardia after a mean of 29 ± 3 s (range 25 to 35 s) in three patients and transient prolongation of the PR interval after tachycardia termination. No ablation was performed in four patients, including three patients with previously failed ablation at this region.

Mapping in the LA was performed in all nine patients. The earliest LA activation was located in the anteroseptal region immediately opposite to the HB area (Fig. 3B), and preceded the reference CS electrogram by 50.2 ± 8.3 ms (range 38 to 62 ms). The earliest LA activation was consistently later than that in the RA by 2 to 5 ms in all patients. No His potential was found at this site in any patients. A mean of 5.8 ± 0.8 RF applications (range 5 to 7) was delivered at the earliest LA activation without

tachycardia termination or the PR prolongation in eight patients.

Mapping and ablation in the non-coronary AS. In the nine patients with focal AT, mapping in the ASs during tachycardia demonstrated that an earliest atrial activation preceded the reference CS electrogram by 66.2 ± 3.6 ms (60 to 71 ms) (Fig. 3C) and the onset of the P-wave by 32.4 ± 4.6 ms (range 27 to 37 ms). Aortic root angiography demonstrated the mapping catheter located in the non-coronary AS via the left femoral artery in all 9 patients (Fig. 4). The earliest atrial activation in the non-coronary AS was earlier than that in the RA by 13.8 ± 5.5 ms (range 7 to 21 ms) in 8 patients (Fig. 4C). In 1 patient, the earliest atrial activation in the non-coronary AS and in the RA was the same (Figs. 5A and 5B), but the amplitude of the atrial activation in the AS was greater than that in the RA (Fig. 5B). In these 9 patients, no His potentials were found at the site with the earliest atrial activation in the non-coronary AS (Figs. 3C and 5B). Fluoroscopically, the successfully ablated site in the non-coronary AS was slightly supero-posterior to the site of HB recording in the RA (Fig. 4). The electrograms at the successful ablation sites were fractionated in 4 of 9 patients, and presented with a small atrial activation and a large
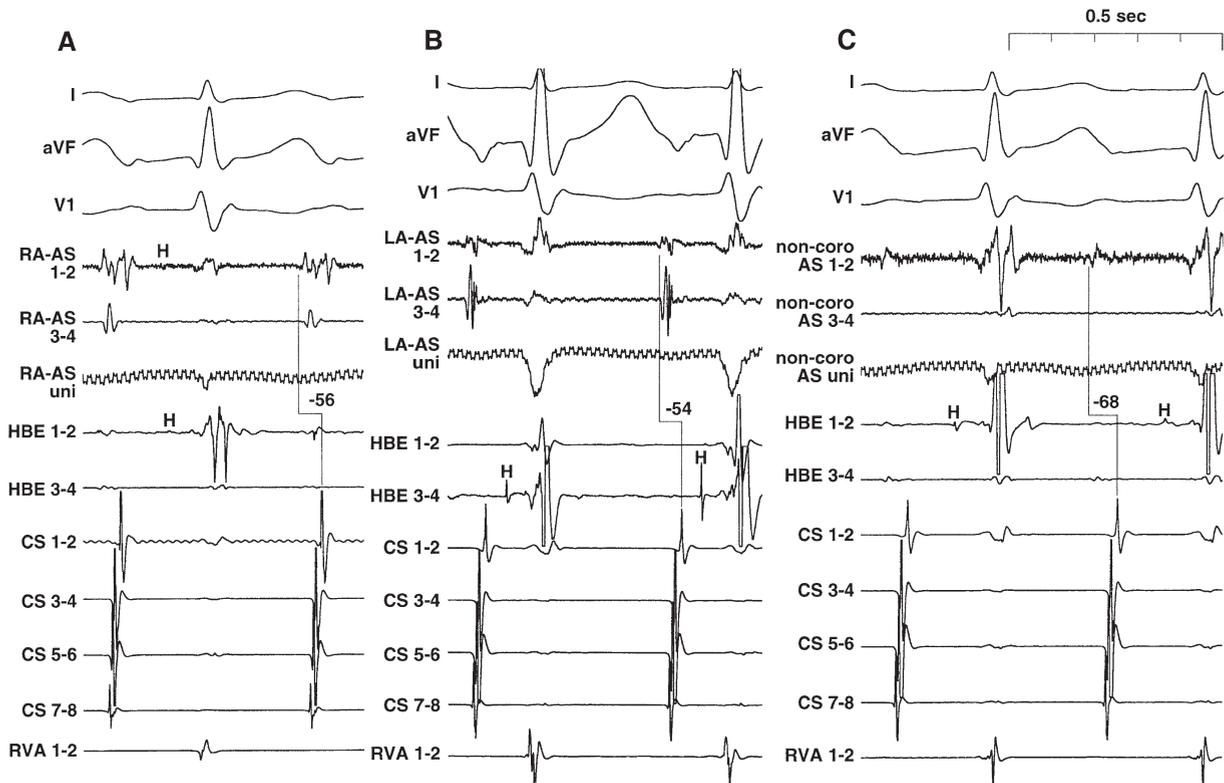
Figure 3. Tracings are ECG leads I, aVF, V$_1$, and intracardiac electrogram recorded from a mapping catheter, a catheter at the HBE, a catheter within the CS, and a catheter at the right ventricular apex (RVA) in a patient with focal atrial tachycardia originating from the non-coronary AS. **(A)** The earliest activation in the right atrium is shown at the His bundle region (RA-AS 1 to 2, RA-AS 3 to 4, RA-AS uni). Note that the earliest activation preceding the fixed atrial electrogram recorded from the CS 1 to 2 by 56 ms and a tiny His potential at this site. The tachycardia was unsuccessfully terminated by radiofrequency (RF) delivery at this site. **(B)** The earliest activation in the left atrium is located at the left anteroseptal region immediately opposite the His bundle in the RA (LA-AS 1 to 2, LA-AS 3 to 4, LA-AS uni). Note that the earliest activation preceded the fixed atrial electrogram recorded from the CS 1 to 2 by 54 ms and there was no His potential at this site. The tachycardia was also unsuccessfully terminated by RF delivery at this site. **(C)** The successful site with an earliest RA activation is shown at the non-coronary AS (non-coro AS 1 to 2, non-coro AS 3 to 4, non-coro AS uni). Note that the earliest activation preceded the fixed atrial electrogram recorded from the CS 1 to 2 by 68 ms and there was no His potential recording at this site. LA = left atrium; other abbreviations as in Figure 1.

ventricular potential in 6 patients, equal atrial and ventricular activation in 2 patients, and a large atrial activation and a small ventricular activation in 1 patient.

In these 9 patients with focal AT, the focal AT was terminated by a single RF application in 7 patients, and by two RF applications in 2 patients. The time to termination at the successful site was 3.2 ± 2.1 s (range 0.5 to 8.0 s). It occurred within 2 s in 4 patients (Fig. 6); within 4 s in 8 patients, including the patient with simultaneous atrial activation in the non-coronary AS and at the HB region; and within 8 s in all 9 patients. No prolongation of the PR interval or a junctional rhythm was observed in any of the nine patients during RF delivery. The ablation catheter in the non-coronary AS was very stable during continuous fluoroscopic monitoring. No dislodgement of the ablation catheter was observed in any of the patients. After ablation, focal AT was not inducible by atrial stimulation with and without isoproterenol infusion.

Follow-up. No procedure-related complications occurred in these nine patients immediately after ablation and during follow-up. No evidence of aortic valve damage was demonstrated during follow-up. All 9 patients were free of the

arrhythmias without antiarrhythmic drugs during a follow-up of 9 ± 3 months.

DISCUSSION

Prevalence of non-coronary cusp location of focal AT. Focal AT is commonly located in the crista terminalis (1,2), near the tricuspid and mitral annulus (3–6), around the pulmonary veins (4,7,8), and at the septal regions (9–12). Previous studies have demonstrated that focal ATs originating from the septal regions can be reproducibly induced and terminated by programmed atrial stimulation, and are sensitive to intravenous adenosine in most patients (9–12). Most patients with these tachycardias are amenable to RF ablation with a high success rate. In a single center experience from Hamburg in the last two years, the non-coronary AS origin was found only in 5 of 123 patients (4.1%) with focal AT. However, the true incidence may be overestimated, because the majority of our patients had complex arrhythmias in this center.

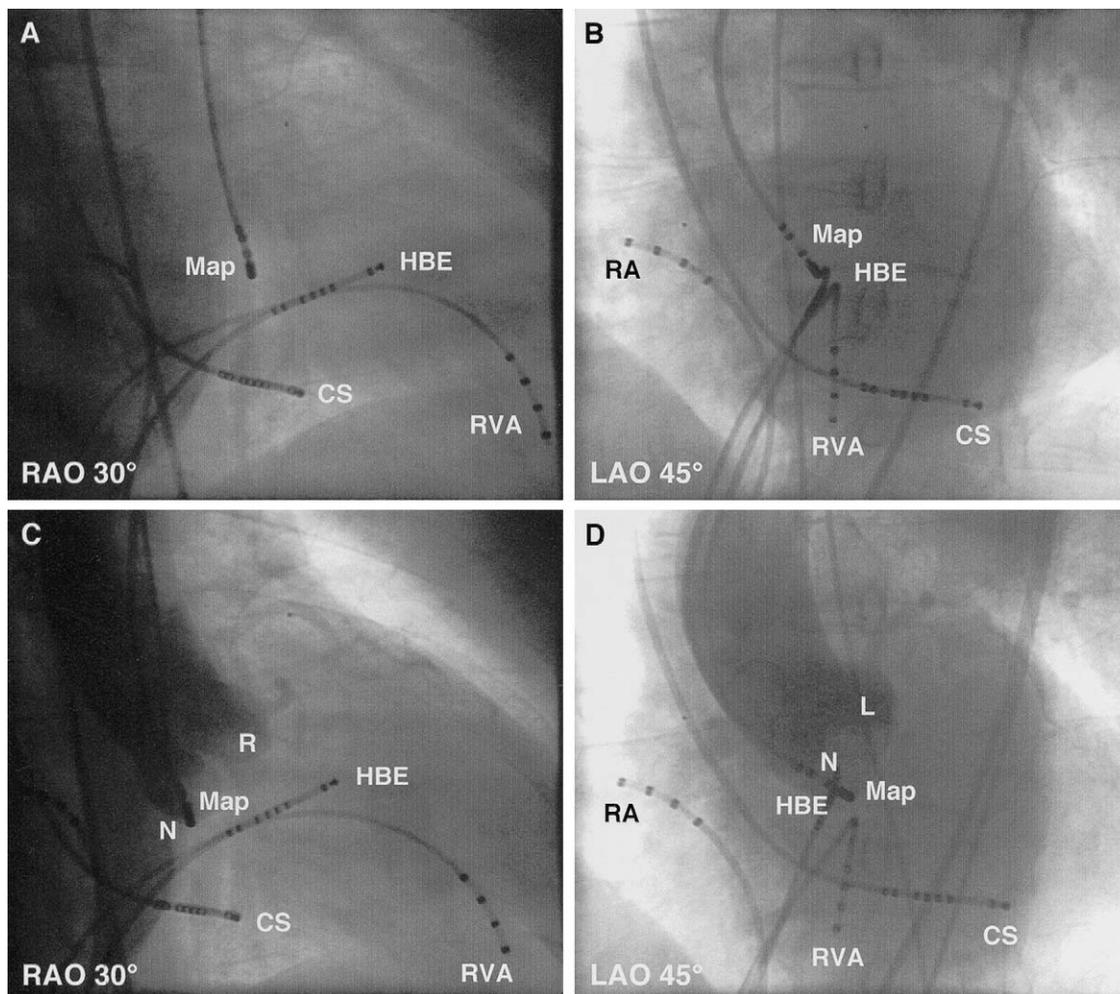**Focal AT originating in the non-coronary AS.** In the present study, all patients with focal AT had clinical

Figure 4. Right (30°) and left (45°) oblique radiographic view show the mapping catheter (Map) at the successful ablation site in the AS **(A,B)**, a multipolar catheter inside the CS, a multipolar catheter at the HBE, a catheter in the RA, and a catheter in the RV. Aortic root angiography in right (30°) and left (45°) oblique radiographic view **(C,D)** shows a mapping catheter in the non-coronary aortic sinus supero-posterior to sites with His bundle potential recording at distal one to four electrodes. L = left aortic sinus; N = non-coronary aortic sinus; R = right aortic sinus; other abbreviations as in Figure 1.

symptoms with abrupt onset and offset, which are similar to those in patients with PSVT. During electrophysiological study, the clinical tachycardias were reproducibly induced and terminated only by atrial stimulation in all nine patients. The tachycardias were sensitive to intravenous administration of adenosine. On the basis of these findings, the tachycardia mechanism was due to either micro–re-entry or trigger activity. Also, electrophysiological findings and the P-wave morphology on the surface ECG during tachycardia were very similar to the focal AT originating near the septal region (9–12), especially near the para-hisian area (10–12), but several features differentiate them. In the present study, mapping demonstrated the earliest atrial activation in the vicinity of the non-coronary AS and an attempted RF ablation at the HB area failed in 8 of the 9 patients (in five patients during the procedure and in three patients during a previous procedure).

Mapping during tachycardia demonstrated that the earliest atrial activation in the non-coronary AS preceded the RA activation at the HB region by 12.2 ± 6.9 ms in these 9 patients. Also, an interesting finding in the study was that the earliest LA activation was consistently later than the earliest RA activation by 2 to 5 ms in these 9 patients. This finding may be explained by anatomical relationship. The non-coronary AS is anatomically closer to the HB region in the RA compared to the anteroseptal region in the LA (15). In our patients, the clinical and electrophysiological characteristics of these focal AT were very similar to those described by Tada et al. (13) in a patient with focal AT originating from the non-coronary AS. These data strongly suggest that mapping of focal AT in the non-coronary AS may avoid aggressive RF delivery near the HB, with the potential consequence of AV block when tachycardia mapping demonstrated the earliest atrial activation at the HB region, especially in the patients with previously failed ablation at the HB region.

RF ablation of focal AT in the non-coronary AS. Previous studies have shown that RF ablation in the AS can be
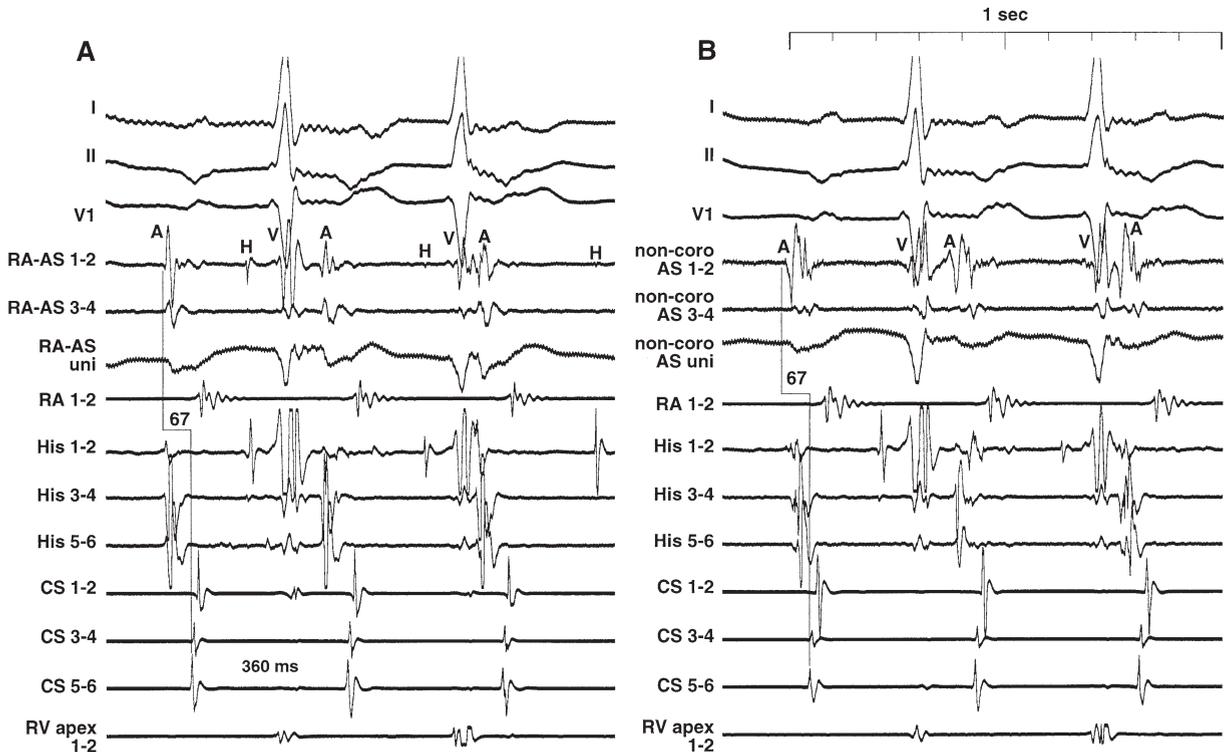
Figure 5. Tracings are ECG leads I, II, V$_1$, and intracardiac electrogram recorded from a mapping catheter, a catheter in the RA, a catheter at the HBE, a catheter within the CS, and a catheter at the RVA in a patient with focal atrial tachycardia originating from the non-coronary aortic sinus. **(A)** Mapping in the RA during tachycardia demonstrates that the earliest right atrial activation is found at the HBE (RA-AS 1 to 2, RA-AS 3 to 4, RA-AS uni). Note that: 1) the earliest activation preceded the fixed atrial electrogram recorded from the coronary sinus by 67 ms and a discrete His potential at this site; and 2) the amplitude change of His activation during the tachycardia indicates an unstable catheter position at the HBE. **(B)** Mapping in the AS demonstrates the earliest atrial activation in the non-coronary AS (non-coro AS 1 to 2, non-coro AS 3 to 4, non-coro AS uni) with the same activation time compared to the earliest right atrial activation. Note that: 1) the earliest activation preceded the fixed atrial electrogram recorded from the coronary sinus by 67 ms; and 2) no His activation is found at the successful site in the non-coronary AS. Abbreviations as in preceding figures.

successfully performed with a 4-mm thermocouple ablation catheter in patients with repetitive ventricular tachycardia (14,16,17). Recently, Tada et al. (13) also demonstrated that conventional RF energy could be successfully performed in the non-coronary AS in one patient with focal AT. In the present study, all focal ATs were successfully terminated with one RF application in 7 patients and with two RF applications in 2 patients. Additionally, the focal AT was also successfully terminated within 4 s in the patient with simultaneous activation in the non-coronary AS and at the RA site with the HB recording, although no attempted RF energy was performed at this site. More importantly, no recording of His potentials was found at the successful AS site. This information may explain why prolongation of the PR interval and junctional beats did not occur during RF delivery in our 9 patients. These findings were different from the previous study finding that focal AT could be induced by ventricular pacing and that infrequent junctional beats occurred during RF ablation (13). On the basis of our study, we strongly suggest that careful mapping in the non-coronary AS may be necessary to minimize a potential risk of injuring the AV node or the HB in focal AT with earliest activation at the HB region, especially with previous failed ablation at this region.

In the present study, focal ATs were terminated in <8 s with RF energy. Therefore, we suggest that RF application should be stopped if focal AT is not terminated after 10 s. In addition, continuously fluoroscopic visualization of the mapping catheter may decrease a potential risk of complications during RF delivery in the non-coronary AS, although the mapping catheter during RF application was very stable.

Anatomical considerations. For a better understanding of the arrhythmia origins and the approaches for catheter ablation, a note on the anatomical arrangement between the non-coronary AS and the atrial anteroseptal annulus is relevant. Spatially, the aortic root occupies a central location within the heart, with the non-coronary AS anterior and superior to the paraseptal region of the left and right atria close to the superior atrioventricular junctions. In all normally structured human hearts, the non-coronary AS is adjacent to atrial myocardium on the epicardial aspect (Fig. 7a). The rightward (anterior) margin of the non-coronary AS is related to the paraseptal region of the right atrial wall (Figs. 7b and 7c), whereas the leftward margin is related to the left atrial wall (Figs. 7d and 7e). The part of the right atrial wall that can be targeted from the non-coronary AS lies superior to the central fibrous
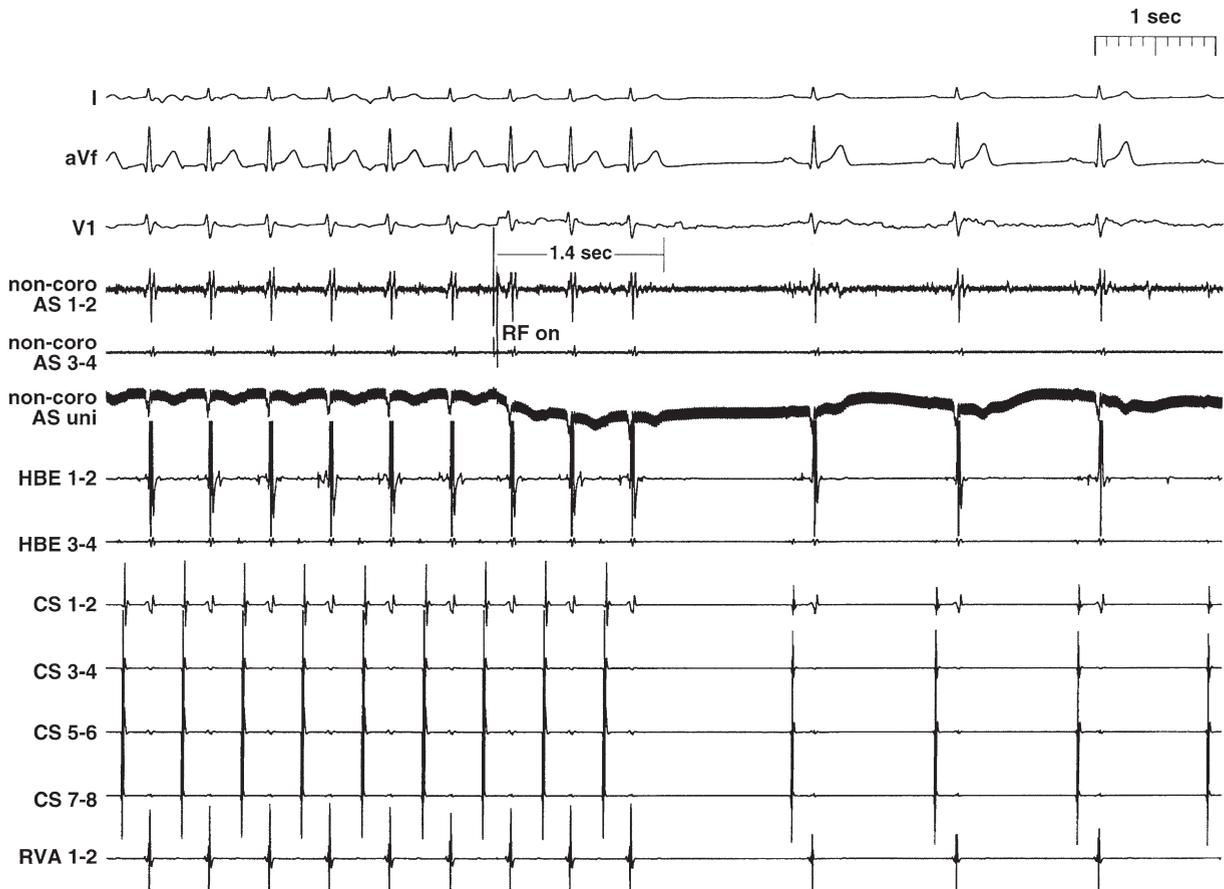
Figure 6. Tracings are ECG leads I, aVF, V₁, and intracardiac electrogram recorded from a mapping catheter in the non-coronary AS (non-coro AS 1 to 2, non-coro AS 3 to 4, non-coro AS uni), a catheter at the HBE, a catheter within the CS, and a catheter at the RVA during RF delivery in a patient with FAT. Note that FAT is successfully terminated after 1.4 s during the first RF application. RF = radiofrequency; other abbreviations as in preceding figures.

body (Fig. 7c). The HB penetrates through the central fibrous body and continues as the atrioventricular conduction bundle that then passes to the crest of the muscular ventricular septum, immediately beneath the membranous septum (Fig. 7c).

In humans, most hearts do not have ventricular myocardium interposing between the aortic and mitral valves; hence there is fibrous continuity between the two valves (13,14). In approximately 15% of horses, however, there is fibrous discontinuity, and all three coronary ASs contain some ventricular myocardium (18). This arrangement occasionally occurs in humans, and when it does, there is then the potential for accessory atrioventricular pathway or arrhythmogenic foci within the AS. In humans, the so-called aortic-mitral separation (19) is the region of perceived discontinuity owing to the overlap of atrial wall over the atrial surface of the mitral leaflet (Fig. 7d).

Murine embryological studies have recently traced the specialized conduction system around the aortic root and the AV canal in early stages and found marked regression in later developmental stages (6,20). These investigators hypothesized that arrhythmogenic substrates may be due

to persistence of the developing conduction system in this region. In the present study, on the basis of the immediate tachycardia termination, we hypothesize that the arrhythmogenic foci are located in the subepicardium of the atrial wall immediately posterior to the non-coronary AS, or the atrial wall overlapping the mitral leaflet. Placing the catheter in the AS provides more stability and precision than atrial approaches.

Conclusions. This study includes 9 patients with focal AT, in 6 of whom previous attempted ablation had failed. In these 9 patients with focal AT, mapping in the atria demonstrated the earliest atrial activation at HB region, whereas mapping in the non-coronary AS demonstrated that that earliest atrial activation preceded the earliest RA activation by 12.2 ± 6.9 ms. All focal ATs were successfully ablated in <8 s in the non-coronary AS with one application in seven patients, and with two RF applications in two patients. These data suggest that some myocardium may be located superficially within the non-coronary AS. We have shown that ablation can be carried out in the AS without any deleterious effects on the aortic wall or any complications during follow-up.
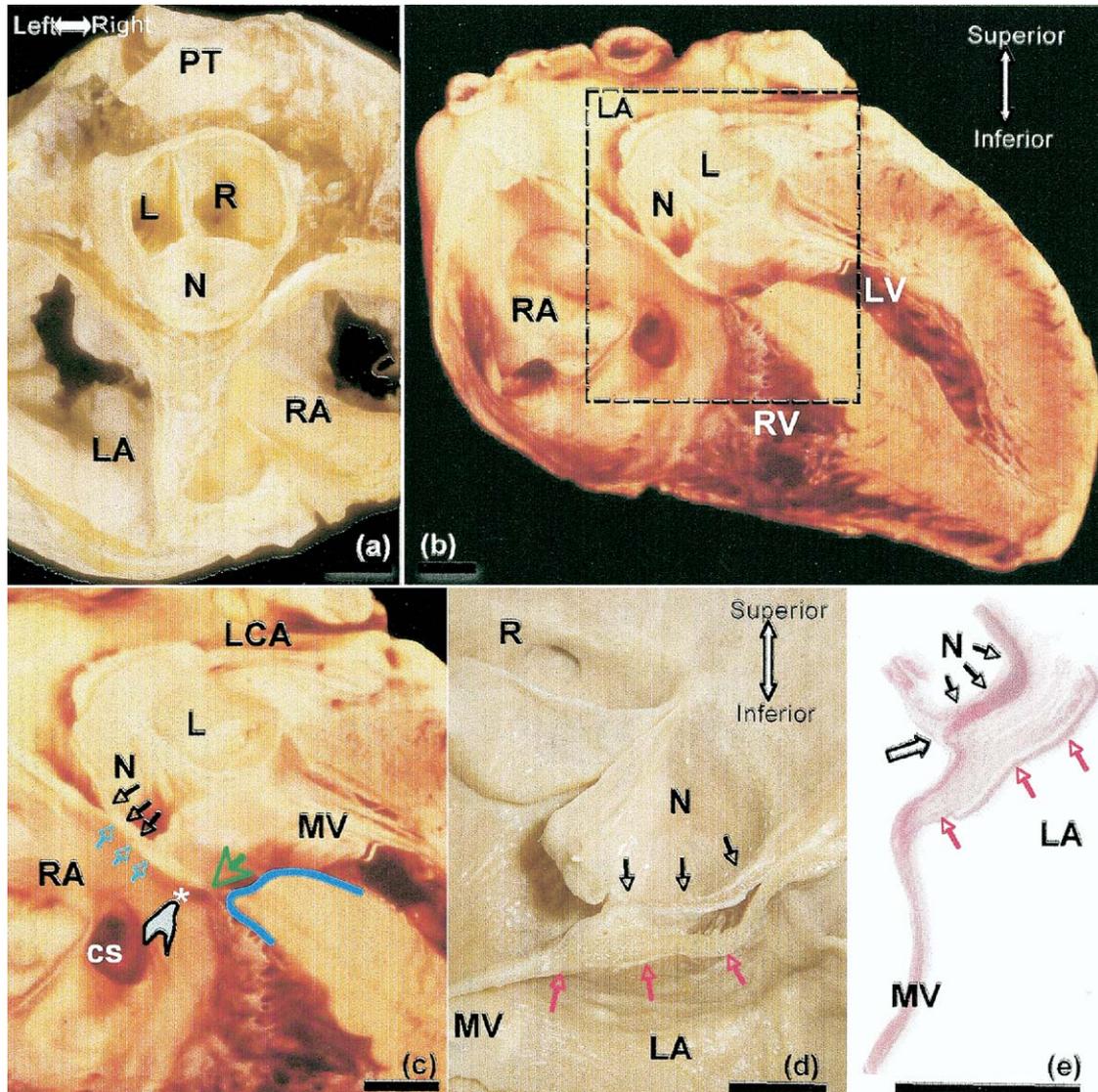
Figure 7. (a) The cardiac base is displayed to show the central location of the aortic valve and proximity of the non-coronary AS to the paraseptal region between right and left atria. **(b and c)** A heart specimen is sectioned in the paracoronal plane to show the rightward extent of the non-coronary AS **(black arrows)** and the right atrial wall **(blue arrows)**. The **green arrow** indicates the membranous septum. The **filled shape** marks the location of the atrioventricular node; the asterisk denotes the His bundle, and the branching atrioventricular conduction bundle is represented by the **blue line. (d)** This simulated parasternal long axis cut through a heart specimen shows the leftward extent of the non-coronary AS **(black arrows)** overlying the left atrial wall **(red arrows). (e)** This histologic section stained with van Gieson stain corresponds to the cut shown in panel D. On the ventricular aspect there is fibrous continuity **(open arrow)** between aortic and mitral valve. On the atrial aspect, left atrial myocardium overlaps the fibrous continuity. Scale bars are 1 cm. LCA = left coronary artery; LV = left ventricle; MV = mitral valve; R = right coronary AS; other abbreviations as in preceding figures.

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