Ventricular Tachycardias Arising From the Aortic Sinus of Valsalva: An Under-recognized Variant of Left Outflow Tract Ventricular Tachycardia

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OBJECTIVES   To describe a normal heart left bundle branch block, inferior axis ventricular tachycardia (VT), that could not be ablated from the right or left ventricular outflow tracts.

BACKGROUND   Whether these VTs are epicardial and can be identified by a specific electrocardiographic pattern is unclear.

METHODS   Twelve patients with normal heart left bundle branch block, inferior axis VT and previously failed ablation were included in this study. Together with mapping in the right and left ventricular outflow tracts, we obtained percutaneous epicardial mapping in the first five patients and performed aortic sinus of Valsalva mapping in all patients.

RESULTS   No adequate pace mapping was observed in the right and left ventricular outflow tracts. Earliest ventricular activation was noted in the epicardium and the aortic cusps. All patients were successfully ablated from the aortic sinuses of Valsalva (95% CI 0% to 18%). The electrocardiographic pattern associated with this VT was left bundle branch block, inferior axis and early precordial transition with Rs or R in V_2 or V_3. Ventricular tachycardia from the left sinus had rS pattern in lead I, and VT from the noncoronary sinus had a notched R wave in lead I. None of the patients had complications and all remained arrhythmia-free at a mean follow-up of 8 ± 2.6 months.

CONCLUSIONS   Normal heart VT with left bundle branch block, inferior axis and early precordial transition can be ablated in the majority of patients from either the left or the noncoronary aortic sinus of Valsalva. (J Am Coll Cardiol 2001;37:1408–14) © 2001 by the American College of Cardiology

Ventricular tachycardia (VT) in patients with no structural heart disease commonly arises from the right or left ventricles, preferentially from the outflow tracts or in relation to the fascicles of the conduction system (1–4). Radiofrequency ablation has provided a curative approach for patients with outflow tract VT, resulting in symptom alleviation and, in rare cases, reversal of tachycardia-induced cardiomyopathy (5). Despite an overall high success rate of catheter ablation, there remain arrhythmias that cannot be ablated by conventional approach. One cause could be an inaccessible site of origin such as an epicardial focus. Recent case reports have recognized the existence of ventricular outflow tract tachycardia that was successfully ablated from the aortic root in the sinus of Valsalva (6,7) or via the epicardial veins (8,9).

We report our experience in 12 patients with ventricular tachycardia who shared an under-recognized origin in the absence of any structural heart disease. In our series, mapping from both endocardial and epicardial aspects gave some insight into the VT site of origin.

METHODS

Patients. The study included 12 out of 66 patients (18%) who did not have any structural heart disease or coronary artery disease and were referred for radiofrequency ablation of ventricular tachycardia with outflow tract origin morphology (left bundle branch block [LBBB], inferior axis). All patients had undergone at least one failed attempt at radiofrequency ablation using conventional endocardial mapping techniques. They had been tried on multiple antiarrhythmic medications including beta-blocker, calcium antagonist, sotalol and flecainide without adequate control of their ventricular tachycardia. Symptoms were palpitations (seven patients) and syncope (five patients). All patients underwent complete history, physical examination, electrocardiogram (ECG), Holter monitoring, echocardiography and magnetic resonance imaging of the heart prior to the procedure.

Electrophysiologic study. All patients gave written informed consent for the procedure. The electrophysiologic study was performed in the postabsorptive state after withdrawal of antiarrhythmic medications and under sedation. Both groins and epigastric region were initially cleansed with betadine and draped in a sterile fashion. Catheters were placed via the right femoral vein. Ventricular tachycardia induction was initially attempted by programmed stimulation (up to three extrastimuli at two different drive

Abbreviations and Acronyms

ECG = electrocardiogram
LBBB = left bundle branch block
LVOT = left ventricular outflow tract
RVOT = right ventricular outflow tract
VT = ventricular tachycardia

trains and at two pacing sites) and burst pacing. If induction with programmed stimulation failed, intravenous isoproterenol infusion (1 to 6 μg/min) was initiated. If VT remained noninducible, phenylephrine infusion began.

Endocardial mapping. Pace mapping and activation mapping of the VT was done initially in the right ventricular outflow tract (RVOT) with 7 F deflectable 4 mm tip catheter (Navistar, Cordis-Webster, Baldwin Park, California) connected to the CARTO mapping system. Mapping with CARTO system was performed as previously described (10). If suitable ablation sites were not found in the RVOT, the femoral artery was cannulated and the left ventricular outflow tract (LVOT) was mapped to obtain adequate pace mapping with early local activation times. In each patient, radiofrequency ablation at the best possible site was attempted in the LVOT. Radiofrequency energy was delivered by impedance monitoring starting at 15 W and then adjusted to achieve an impedance drop of about 10 ohms while avoiding impedance rise. When ablation at the LVOT was not successful, epicardial mapping was performed in the first five patients following percutaneous instrumentation of the pericardial space.

Percutaneous epicardial mapping. Under 15° left anterior oblique fluoroscopic view an 8.9 cm 17 gauge epidural needle (Arrow International, Reading, Pennsylvania) was gently advanced in the left subxyphoid region with intermittent injection of a small amount of contrast material. Entry into the epicardial space was verified by injection of contrast showing characteristic "layering" in the epicardial space. A floppy guide wire was advanced through the needle and the position of the guide wire in the epicardial space was confirmed. Then an 8 F vascular sheath was advanced over the wire. A 7 F 4 mm tip deflectable Catheter (Navistar, Cordis Webster, Baldwin Park, California) was placed in the epicardial space via this sheath and mapping of VT was carried out using the CARTO system.

The ablation catheter was advanced in the aorta after inability to achieve successful ablation was demonstrated by mapping and ablation as described in the preceding text. The catheter was carefully manipulated in the aortic root above the cusps of the aortic valve and in the region of the sinuses of Valsalva until an early ventricular electrogram was recorded and pace mapping showed a QRS identical to the clinical VT morphology. Pace mapping at these sites often required high output pacing (between 5 and 40 mA). Radiofrequency lesions in the aorta were delivered only at the site with identical pace mapping. Before ablation, these sites were tagged with the CARTO system and catheter

stability was monitored. Energy delivery in the aorta was started at a power setting of 15 W, which was increased to no more than 25 W. Special care was taken to avoid ostia of the coronary arteries. Energy delivery was discontinued in case of even minimal dislodgment from the site showing identical pace mapping.

RESULTS

The patients' mean age was 27 $\pm$ 10 years. Seven were female and five male. Ventricular tachycardia was paroxysmal in four patients and repetitive in eight. None of the patients had any evidence of structural heart disease.

ECG description. The VT in all these patients showed a QRS morphology, which shared similar characteristics. All patients had a LBBB, inferior axis with pointed QRS in the inferior leads and early transition in the precordial leads, mostly in V_2 or V_3. The patients whose VTs were successfully ablated from the left coronary cusp region showed rS pattern in lead I (Fig. 1). The three patients who had successful ablation from the noncoronary cusp region had a notched R wave in lead I (Fig. 2). Table 1 summarizes the ventricular tachycardia morphology, mapping and ablation sites.

Provocation tests and response to acute drug administration. In the drug-free state, VT was never initiated in any of the patients by programmed stimulation or burst pacing. Isoproterenol infusion resulted in spontaneous initiation of sustained, incessant or repetitive tachycardia in eight patients and allowed initiation of VT by programmed stimulation in one patient. In the remaining patients, phenylephrine infusion was given after the termination of isoproterenol and this initiated repetitive tachycardia.

Mapping and ablation. In each patient pace maps in the RVOT region failed to achieve the early transition, and the local activation in VT was never earlier than -25 ms. Mapping of the LVOT also failed to show perfect pace mapping, being unable to reproduce the VT QRS morphology in L1. In each patient, radiofrequency lesions were delivered unsuccessfully at the earliest activation site in the LVOT. In five of the 12 patients who underwent the procedure early in this series, epicardial mapping was carried out and very early local activation times were recorded at epicardial sites adjacent to the LVOT in all patients with left-cusp VT (four out of five patients). The local ventricular activation preceded the surface QRS by 50 to 60 ms. But these sites also demonstrated large atrial electrograms, and pacing in these areas resulted in left atrial pacing (Fig. 3).

Subsequent mapping of the region of the aortic sinus of Valsalva showed local activation times in the left aortic sinus that were earlier than the LVOT sites. At those sites identical pace mapping was obtained. All four patients were successfully ablated from the left sinus of Valsalva. The fifth patient undergoing epicardial instrumentation was ablated from the noncoronary sinus. In this patient there was no earlier activation recorded from
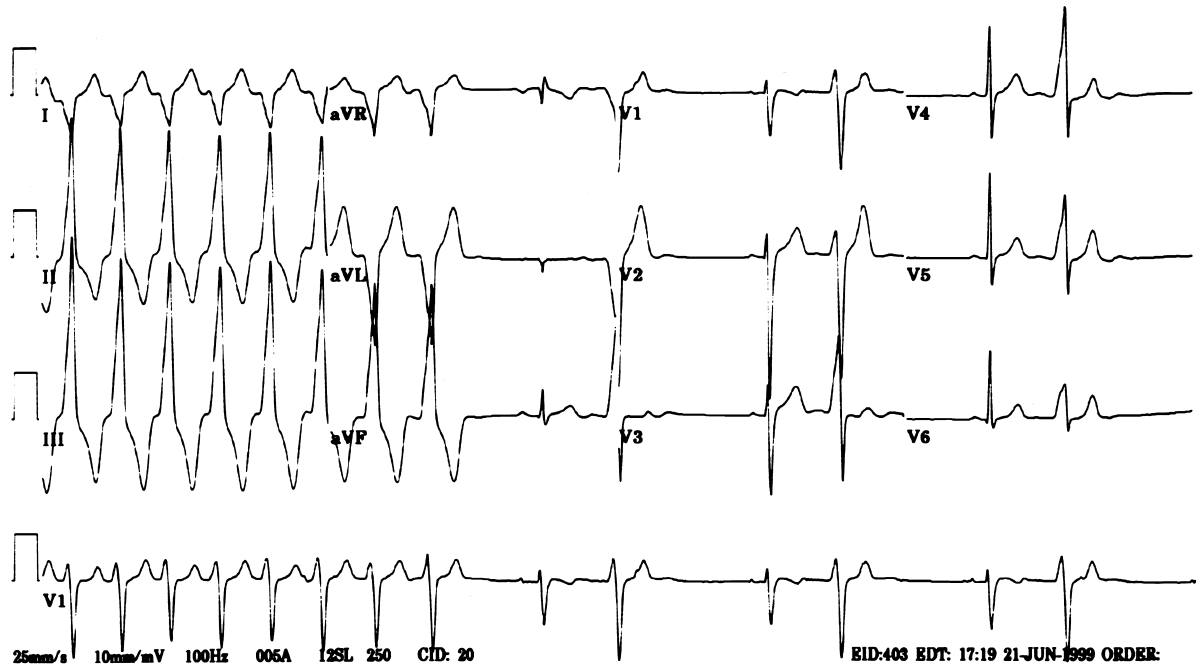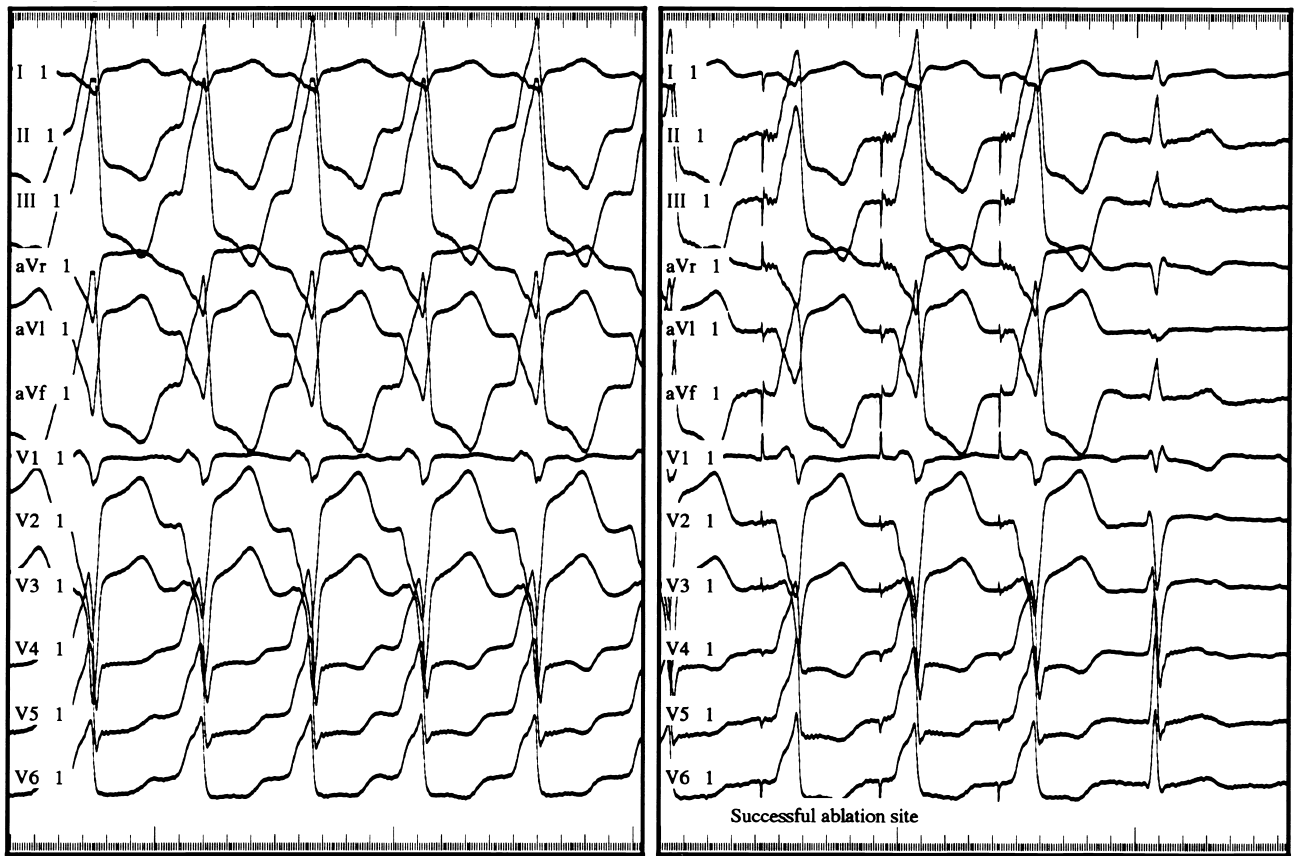
Figure 1. **(A)** Twelve-lead ECG in a 17-year-old male with syncope and palpitations who had successful ablation from the left sinus of Valsalva. Note the nonsustained ventricular tachycardia (VT) had left bundle branch block, inferior axis morphology with transition in V_3 and rS pattern in lead I. **(B)** Identical pace mapping from the successful site above left coronary cusp is shown. The **left side of the panel** shows spontaneous nonsustained VT and the **right-side panel** shows the pace mapping at the successful site. The recordings V_2 and V_3 were obtained from sites lateral to the standard position because of placement of a defibrillator patch in the lower chest region.
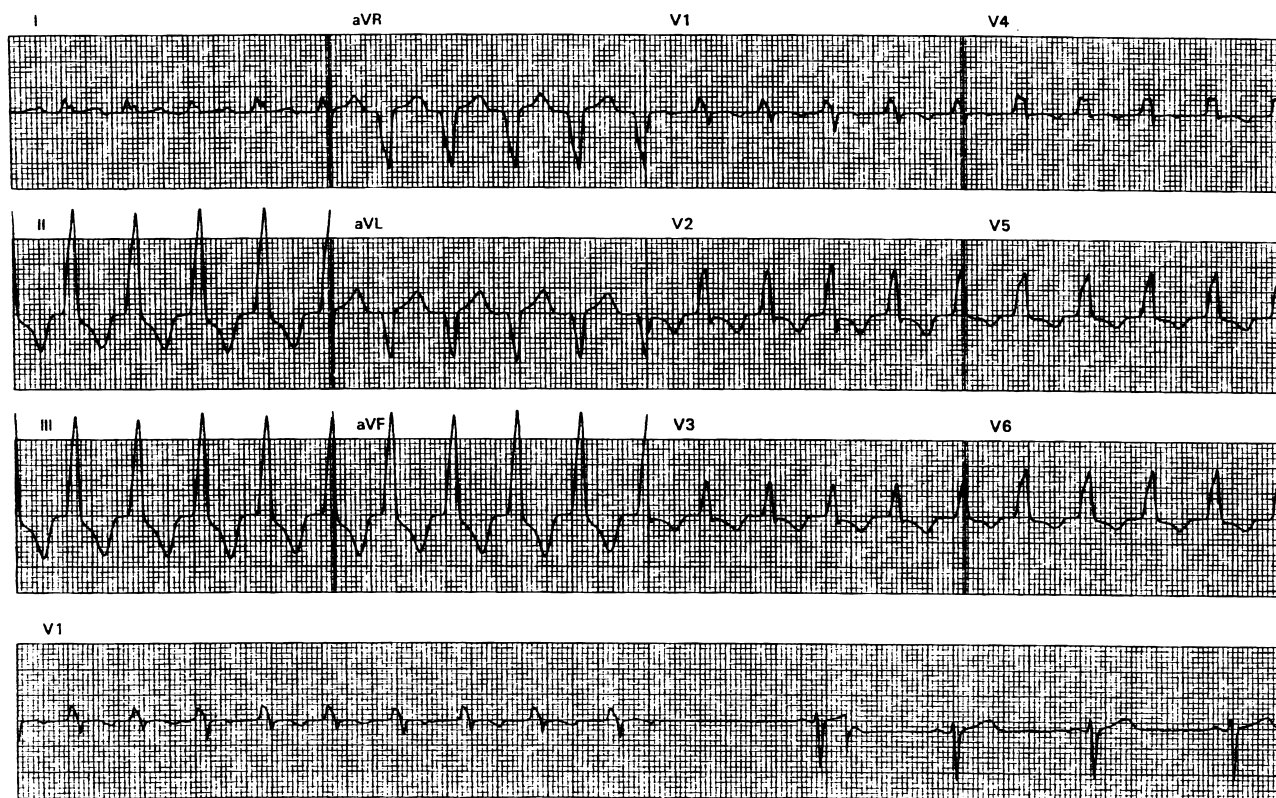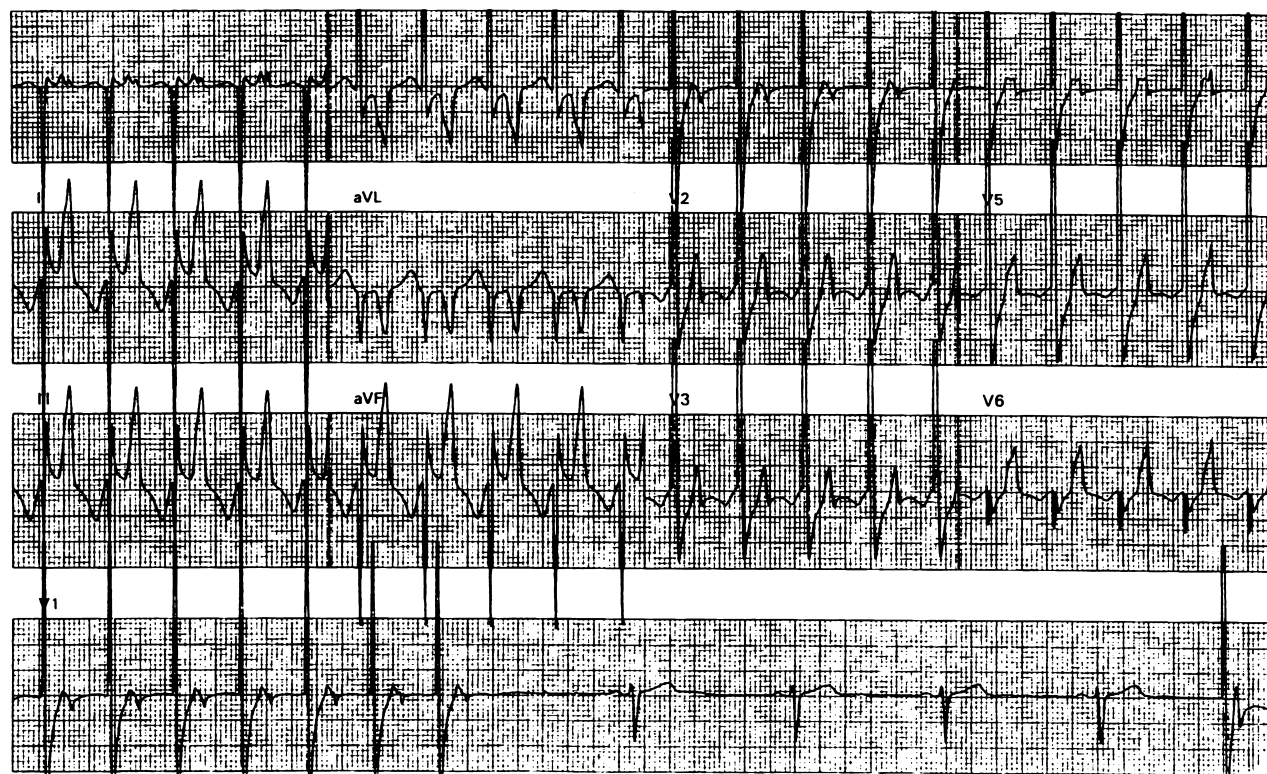
Figure 2. (A) 12-lead configuration of spontaneous ventricular tachycardia in a patient successfully ablated from the noncoronary cusp region. (B) Pace map at the successful site. Note left bundle branch block inferior axis morphology, precordial transition in V_2 and a notched R-wave lead I.

Table 1. Ventricular Tachycardia Morphology, Mapping and Ablation Sites

| | Tachycardia QRS Morphology | | | | Local |
	Lead I	Transition	Mapping Site/s	Successful Sites	Activation Time
Patient 1	rS	V$_3$	Endo/Epicardial	Left sinus	−52 ms
Patient 2	rS	V$_2$	Endo/Epicardial	Left sinus	−35 ms
Patient 3	rS	V$_3$	Endo/Epicardial	Left sinus	−30 ms
Patient 4	rS	V$_2$	Endo/Epicardial	Left sinus	−45 ms
Patient 5	Bifid R	V$_2$	Endo/Epicardial	Noncoronary sinus	−37 ms
Patient 6	Bifid R	V$_3$	Endocardial	Noncoronary sinus	−40 ms
Patient 7	Bifid R	V$_3$	Endocardial	Noncoronary sinus	−38 ms
Patient 8	rS	V$_2$	Endocardial	Left sinus	−40 ms
Patient 9	rS	V$_2$	Endocardial	Left sinus	−60 ms
Patient 10	rS	V$_2$	Endocardial	Left sinus	−35 ms
Patient 11	rS	V$_3$	Endocardial	Left sinus	−38 ms
Patient 12	rS	V$_3$	Endocardial	Left sinus	−40 ms

epicardial sites. In each patient identical pace mapping was observed only at a single site, which was tagged with the CARTO system. High output pacing was necessary in all patients to obtain capture. Older patients required both high output pacing and long pulse width (≥40 ms) to achieve capture. In all patients tachycardia termination was obtained with a single radiofrequency lesion at these sites (95% confidence interval for failure rate 0% to 18%). Two of these patients also had a different LBBB VT arising from the right ventricle. Both VTs were also successfully ablated. After the ablation, ventricular tachycardia or ventricular ectopic beats were no longer observed despite isoproterenol or phenylephrine infusions.
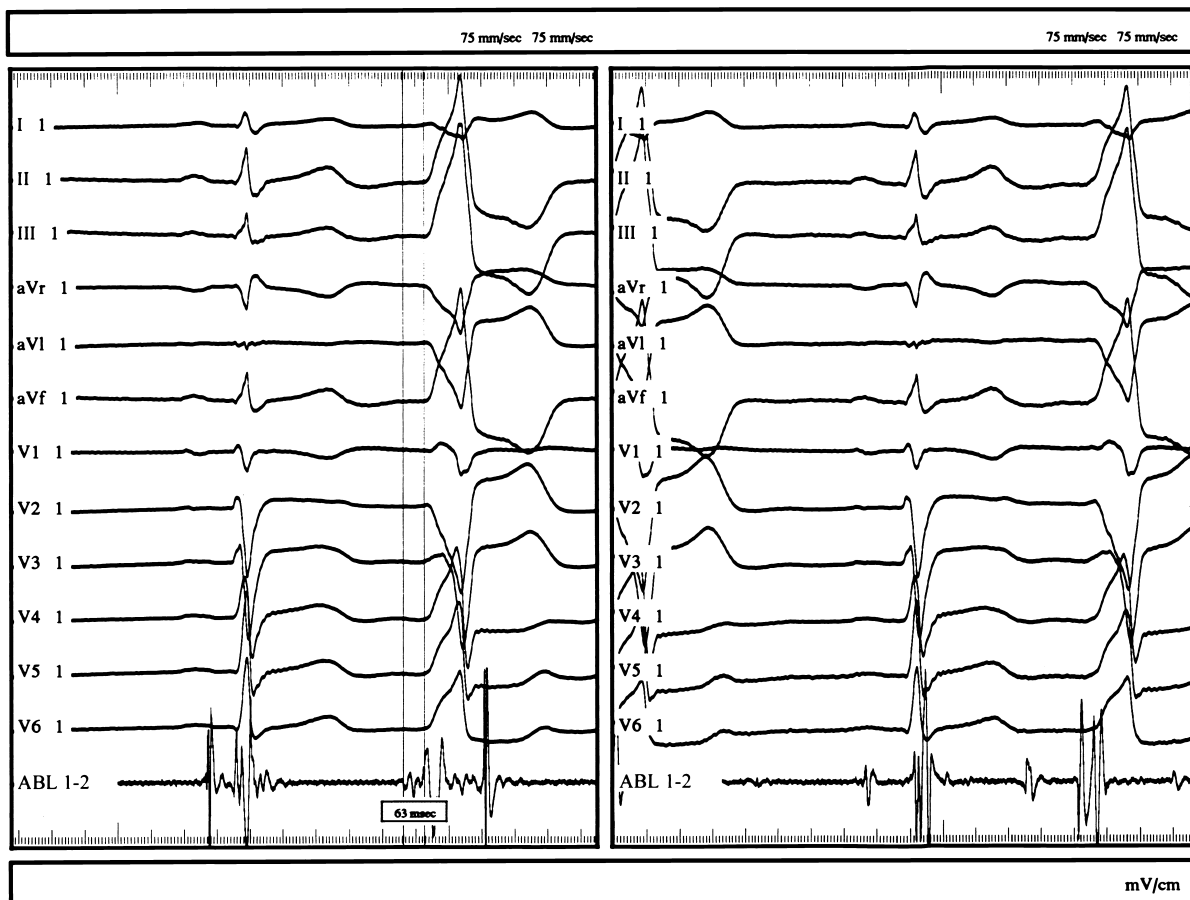

Figure 3. The local electrogram from the epicardial aspect in the patient in Figure 1 is shown on the left side of the panel. The ventricular electrogram precedes the surface QRS by 63 ms, but there is also a large atrial electrogram recorded at this site. The **right side of the panel** shows local activation recorded from the left aortic sinus of Valsalva where the ventricular tachycardia was successfully ablated. At this site the local ventricular electrogram preceded the surface QRS by 52 ms.

Follow-up. All patients have remained tachycardia-free after a mean follow up of 8 ± 2.6 months. Eight patients have had postprocedure magnetic resonance imaging and all have had transthoracic echocardiograms without evidence of aortic valvular dysfunction or pericardial abnormality. At follow-up, all patients had Holter monitoring and nine underwent an exercise stress test with no arrhythmia.

DISCUSSION

Background. Although outflow tract tachycardia in patients with no structural heart disease has a benign prognosis, it often results in significant symptoms such as palpitations, syncope and restriction in physical activity. Many of these patients are young adults, and successful radiofrequency ablation would eliminate the tachycardia and improve the quality of life. Identifying patients with tachycardia having possible unusual sites of origin based on QRS morphology would help to finalize treatment plans, shorten procedure times and radiation exposures and improve the rate of successful ablation.

Study findings. All patients in our series had similar characteristics. None had any structural heart disease. The VT morphologies suggested outflow tract origins. However, mapping of the RVOT and LVOT regions did not yield any identical pace map or early local activation.

In our 12 patients, all of whom were successfully ablated from the aortic root, two characteristic QRS morphologies were observed. A LBBB QRS morphology, tall monophasic R waves inferiorly, early precordial lead transition with rS or RS in lead V_1 and Rs pattern in V_2 or V_3 were seen in all patients. In addition, the patients who had ventricular tachycardia abolished from the region of the left aortic sinus had an rS pattern in lead I. On the other hand, the patients who were successfully ablated from the noncoronary sinus had a notched R wave in lead I. No ventricular tachycardia was reproduced by pacing from the right coronary cusp region, which resulted in atrial rather than ventricular capture.

Previous studies. Callans et al. (3) suggested that patients with failed ablation showing LBBB and inferior axis VT with an early transition might have VT arising from the left ventricular outflow in the proximity of the His bundle. Krebs et al. (11) have also described VTs with transition in lead V_3 or V_2 that could not be successfully ablated from the right side. Interestingly, the VT morphology of the patients who could not be ablated from the right or left ventricular outflow tracts was similar to our patients ablated from the left sinus of Valsalva. Even though the electrocardiographic pattern was remarkably consistent in all patients in our series, one has to remember that surface ECG morphology of ventricular tachycardia depends not only on the site of origin, but also to some extent on the orientation of the heart in the chest cavity and on the position of the surface electrocardiographic leads. Therefore, although surface electrocardiograms may be highly suggestive of the site of origin of the VT, they may not be accurate in every patient.

Inability to achieve successful ablation with conventional endocardial approaches has raised the possibility of an epicardial origin of this idiopathic LVOT tachycardia. Similarly, it has been suggested that certain QRS morphologies may reflect an arrhythmogenic focus of epicardial origin (12,13). Preliminary reports of mapping and ablation through the coronary venous system have indeed supported this hypothesis, showing an early epicardial activation and successful ablation (9). However, this approach is limited by the anatomy of the coronary venous tree and the ability to consistently cannulate those branches for mapping and ablation purposes.

Recently, mapping and ablation of VT has been successfully and safely carried out by percutaneous epicardial instrumentation (14–16).

Epicardial mapping. In five of our patients, percutaneous epicardial access was obtained. Of interest, simultaneous endoepicardial mapping in the patients with left aortic cusp VT showed an earlier activation on the epicardial surface. However, at the site of the early ventricular electrogram there was also a large atrial electrogram, and pacing resulted in atrial capture.

Only mapping of the aortic root in the sinus of Valsalva yielded early local activation times and identical pace mapping. On the other hand, in the noncoronary cusp VT, no early activation was recorded from the epicardium.

Therefore, even though it appeared that the origin of most of these VTs may have been closer to the epicardial surface, it was not possible to reach those sites via the epicardial approach because of the left atrial appendage interposition around the aorta and the LVOT region (Fig. 4). Successful radiofrequency ablation was then achieved from the aortic root in the sinus of Valsalva. It is conceivable that in patients with sclerotic or calcified aortic wall, radiofrequency ablation from the aortic sinus would not be able to reach the tachycardia focus.

Limitations and possible complications. Radiofrequency ablation in the region of the aortic root has been reported in a limited number of patients. Aortic valve damage and occlusion or stenosis of the coronary arteries are potential complications. However, aortic valve scarring and thermal damage might have been prevented by limiting the power settings. In our 12 patients there was no clinical evidence of ischemia or ischemic ECG changes during or after the procedure. In addition, we did not observe evidence of aortic valve dysfunction or morphological changes at follow-up.

We did not make an effort to correlate the successful ablation site with the ostium of the coronary artery by coronary angiogram. This could have resulted in unintentional ablation in the left main coronary artery. However, targeting sites suitable for ablation on the basis of ability to obtain identical pace mapping appeared safe in our experience. It is also possible that the real time nonfluoroscopic electroanatomical monitoring of the ablation catheter may
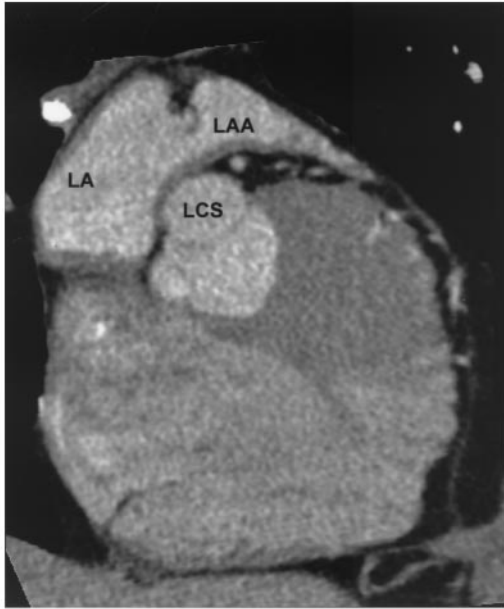
Figure 4. High resolution computerized tomographic images of the heart, showing an oblique view. The aortic root and left coronary cusp are partly covered by the left atrial appendage. LA = left atrium; LAA = left atrial appendage; LCS = left coronary sinus.

have prevented inadvertent energy delivery at different locations. Intravascular ultrasound imaging may provide an alternative approach to visualize the relationship between the ablation catheter and the coronary artery ostium.

Conclusion. Not all VTs with LBBB inferior axis can be ablated from the RVOT. We describe a ventricular tachycardia with LBBB inferior axis morphology in patients with normal heart, which was successfully ablated from the aortic sinuses of Valsalva. All these patients had an early transition in the precordial leads and either an rS pattern or a notched R wave in lead I. Recognition of this VT will increase our ability to successfully treat patients with ablation procedures. On the other hand, the vicinity of the coronary arteries and the potential for serious complications should be weighed when considering ablation.

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