Adenosine-Sensitive Ventricular Tachycardia From the Anterobasal Left Ventricle

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Objectives. This study demonstrates that exercise-provocable tachycardia resembling right ventricular outflow tract tachycardia may originate from the anterobasal left ventricle.

Background. Reentry is the operative mechanism of idiopathic left ventricular tachycardia, with a QRS complex of right bundle branch block and superior axis that is responsive to verapamil but not adenosine. Whether some mechanism other than reentry is operative in some idiopathic left ventricular tachycardias is unclear.

Methods. In 4 of 53 consecutive patients with idiopathic left ventricular tachycardia, the tachycardia was sensitive to adenosine. These four patients were women 63, 61, 61 and 31 years old and were the subjects of the present study.

Results. In all four patients, spontaneous tachycardia was related to exercise or emotional stress. The tachycardia displayed atypical left (one patient) or right (three patients) bundle branch block with an inferior axis and marked variation in cycle length. An intravenous bolus of adenosine triphosphate (10 to 20 mg) terminated tachycardia in all four patients. Tachycardia was terminated or prevented in three patients given intravenous or oral verapamil. Atrial or ventricular incremental or extrastimulus testing induced tachycardia in all four patients (three with, one without isoproterenol infusion). Electrically induced tachycardia also demonstrated marked variation in cycle length, which ranged from 230 to 390 ms. Entrainment was not demonstrable with overdrive pacing from multiple sites. Endocardial mapping during tachycardia revealed that the earliest activations were registered 25, 40, 35 and 50 ms before onset of the QRS complex, respectively, from the anterior aspect of the left ventricle just below the mitral annulus, adjacent to the left ventricular outflow tract. High frequency Purkinje spikes were not recorded at this site. Radiofrequency current delivered to this site successfully ablated the tachycardia in three of the four patients.

Conclusions. Exercise-provocable, catecholamine-mediated, verapamil-responsive, adenosine-sensitive ventricular tachycardia may arise from the anterobasal left ventricle adjacent to the outflow tract.

(J Am Coll Cardiol 1997;30:1339–45) ©1997 by the American College of Cardiology

Idiopathic left ventricular tachycardia is characterized by a QRS configuration of right bundle branch block and a superior axis (1–5). It is responsive to verapamil but not adenosine. Reentry is thought to be the operative mechanism of the tachycardia. There are sporadic case reports (6,7) showing that some idiopathic left ventricular tachycardias may be responsive to adenosine and that the operative mechanism may not be reentry. In the present study, we describe four patients with idiopathic left ventricular tachycardia originating from the anterobasal left ventricle adjacent to the outflow tract in which the clinical and electrophysiologic characteristics resemble those of exercise-provocable, catecholamine-mediated, verapamil-responsive, adenosine-sensitive ventricular tachycardia from the right ventricular outflow tract.

Methods

Patients. Between July 1991 and April 1997, a total of 53 consecutive patients with idiopathic left ventricular tachycardia underwent radiofrequency ablation at Chang Gung Memorial Hospital. The procedure was reviewed and approved by the hospital review board and was in accordance with local ethical standards. In 4 of these 53 patients, the tachycardia was demonstrated to be provokable by treadmill exercise and responsive to adenosine. These four patients were the subjects of the present study.

Electrophysiologic study. Electrophysiologic study was performed after discontinuance of cardioactive drugs for at least 5 half-lives and after written informed consent was obtained. Two 6F quadripolar electrode catheters with 10-mm
interelectrode spacing were respectively positioned at the high right atrium and right ventricular apex for pacing and recording of local electrograms. A 7F quadripolar steerable electrode catheter with a 4-mm tip and 5-mm interelectrode spacing was placed across the tricuspid valve for recordings of the His bundle electrogram. Another 7F steerable electrode catheter with a 5-mm tip and 2-mm interelectrode spacing between the distal two electrodes was retrogradely introduced into the left ventricle through the aorta for recordings of the local electrogram, pacing and ablation. Electrocardiographic leads I, aVF and V1 as well as intracardiac electrograms were simultaneously displayed and recorded on a multichannel oscilloscopic recorder (Midas-2500, PPG Industries Inc.).

Induction of tachycardia was conducted by incremental atrial and ventricular pacing as well as atrial and ventricular extrastimulus testing, as previously described (8). Programmed ventricular stimulation was done by a maximum of three ventricular extrastimuli at two different driven cycle lengths from the right ventricular apex and outflow tract. If ventricular tachycardia was not induced, the stimulation was repeated after isoproterenol infusion (1 to 4 \( \mu \)g/min to achieve a 20% increase in sinus rate). If sustained tachycardia was induced, entrainment study was conducted by overdrive ventricular pacing from the right ventricular apex and outflow tract. The pacing stimuli were twice the diastolic threshold and 2 ms in duration and were provided by a digital programmable stimulator (Bloom and Associates, DTU-200).

Radiofrequency ablation. The potential ablation site was selected at a site where the endocardial activation was the earliest and where pace mapping displayed a QRS complex resembling that of the tachycardia (8). A test current of 20 to 35 W was then applied during an episode of tachycardia under continuous digital monitoring of power strength and impedance. If the tachycardia was terminated within 10 s, additional current was applied for another 60 to 120 s. The output current of a radiofrequency generator (Radionics RFG-3C) was delivered to the distal electrode of the large-tipped ablation catheter and a posteriorly positioned cutaneous patch. Programmed stimulation was performed after ablation during isoproterenol infusion to ensure successful ablation. The successful ablation site was then recorded by cineradiograph at the right and left anterior oblique and lateral projections. An initial bolus of 3,000 U of heparin was administered immediately before application of the radiofrequency current. Additional heparin (1,000 U) was given every hour until completion of the procedure.

Results

Patient 1. A 63-year old woman had paroxysmal palpitations for 5 years. The palpitations were precipitated by exertion or postural change and lasted for 10 to 20 min. The rest ECG was normal. The ECG recorded during palpitation showed a regular, wide QRS tachycardia at a rate of 240 beats/min and a pattern of atypical left bundle branch block with an inferior axis (Fig. 1A). Physical examination, chest roentgenogram and echocardiogram were normal. Treadmill exercise tests reproducibly provoked sustained ventricular tachycardia identical to the spontaneous tachycardia during the recovery phase. Heart rate varied from 230 to 260 beats/min before termination of the tachycardia. An intravenous bolus of 10 mg of adenosine triphosphate (ATP) terminated the tachycardia in 10 s (Fig. 1A). However, a few bursts of tachycardia were observed after termination. Prolongation of tachycardia cycle length was observed after ATP. An intravenous injection of 8 mg of verapamil also terminated the tachycardia during a separate episode of tachycardia, but intravenous xylocaine failed to terminate the tachycardia. Control programmed stimulation with rapid atrial or ventricular pacing as well as delivery of atrial or ventricular extrastimuli failed to induce tachycardia. After isoproterenol infusion (3 \( \mu \)g/min), both sustained and nonsustained tachycardias occurred spontaneously; rapid ven-
tricular pacing at a cycle length of 260 ms reproducibly induced tachycardia. The cycle length of tachycardia varied from 230 to 260 ms. Entrainment of the tachycardia was not demonstrated by overdrive pacing. Activation mapping from both the right and left ventricles revealed that the earliest endocardial activation was registered 25 ms before the onset of the QRS complex at the superior aspect of the left ventricle adjacent to the left ventricular outflow tract (Fig. 2A). The endocardial activation at this site was 20 ms earlier than the earliest recorded activation site from the right ventricular outflow tract. Pace mapping at this site revealed a QRS configuration that matched that of the spontaneous tachycardia (11 of 12 leads) (Fig. 3), whereas pace mapping from the right ventricular outflow tract, including the earliest activation site in this area, displayed a QRS configuration different from that of the spontaneous tachycardia. Radiofrequency ablation was unsuccessful in ablating the tachycardia. The patient subsequently received nadolol 80 mg/day; multiple follow-up treadmill exercise tests failed to provoke the tachycardia, and this patient has been free of symptoms for 12 months.

**Patient 2.** A 61-year old woman experienced recurrent palpitations for 3 years. Attacks of palpitation lasted for ~30 min and were related to exertion and emotional stress. The rest ECG was normal. The ECG recorded during palpitation displayed a regular, wide QRS tachycardia at a rate of 150 beats/min and a configuration of atypical right bundle branch block with an inferior axis (Fig. 1B). Physical examination, chest roentgenogram and echocardiogram were normal. Treadmill exercise tests provoked sustained ventricular tachycardia identical to the spontaneous tachycardia. Heart rate varied from 170 to 220 beats/min. An intravenous bolus of 20 mg of ATP terminated the tachycardia in 18 s (Fig. 1B). A progressive slowing of the tachycardia was noted before termination. Carotid massage was ineffective in terminating the tachycardia. Control programmed stimulation was unable to induce tachycardia. After isoproterenol infusion (2.5 μg/min), ventricular tachycardia identical to the clinical tachycardia occurred spontaneously and was reproducibly induced by programmed stimulation with rapid atrial or ventricular pacing as well as delivery of atrial or ventricular extrastimulus (Fig. 4A). The cycle length of the induced tachycardia varied from 280 to 300 ms. Entrainment of the tachycardia was not demonstrated by overdrive pacing. Activation mapping of the tachycardia revealed that the earliest endocardial activation was registered 40 ms before the onset of the QRS complex at the high anterior superior aspect of the left ventricle, below the mitral annulus and adjacent to the left ventricular outflow tract (Fig. 2B and 4B). Delivery of radiofrequency current (23 W) at this site terminated the tachycardia in 2.2 s (Fig. 4C). Repeat

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**Figure 2.** Radiographic recordings (right [top] and left anterior oblique [bottom] projections) showing the earliest activation site (ablation site) during ventricular tachycardia in Patients 1 (A) and 2 (B). Arrow indicates the tip of the mapping (ablation) catheter at the earliest activation site.

**Figure 3.** Simultaneous 12-lead ECGs in Patient 1, showing QRS configuration during induced ventricular tachycardia (A) and pace mapping (B) from a site at the superior aspect of the left ventricle adjacent to the left ventricular outflow tract. Note that the QRS complex during pacing displayed a near-perfect match to that during tachycardia, although a subtle difference was noted in lead V1.
treadmill exercise tests 2 days after ablation and thereafter revealed no provocation of the tachycardia. The patient has been free of symptoms for 12 months without medication.

Patient 3. A 61-year old woman was referred to this institution because of daily attacks of palpitation for 10 years. The attacks were precipitated by exertion and emotional stress. An electrophysiologic study was performed at another laboratory 8 months before the referral and revealed both atrioven-tricular reentry tachycardia incorporating a concealed left lateral accessory pathway and ventricular tachycardia. The concealed left lateral accessory pathway was successfully ablated; however, attempted ablation of ventricular tachycardia was unsuccessful. Physical examination revealed a grade 2/6 diastolic rumbling murmur at the apex. The chest roentgenogram and rest ECG were normal. The echocardiogram demonstrated mild mitral stenosis. Treadmill exercise tests provoked sustained ventricular tachycardia. Heart rate varied from 150 to 230 beats/min, and the QRS configuration displayed a pattern of atypical right bundle branch block with an inferior axis (Fig. 5A). An intravenous bolus of 18 mg of ATP terminated the tachycardia in 20 s, whereas an intravenous injection of 7.5 mg of verapamil terminated the tachycardia in 34 s. Oral propranolol (40 mg/day) in four divided doses effectively prevented provocation of ventricular tachycardia by exercise. Baseline programmed stimulation with rapid atrial or ventricular pacing as well as delivery of atrial or ventricular extrastimulus induced sustained tachycardia. The cycle length of the tachycardia was 390 ms. Entrainment of the tachycardia was not demonstrated by overdrive stimulation; however, the tachycardia also occurred spontaneously during isoproterenol infusion. The cycle length of the tachycardia during isoproterenol infusion ranged from 270 to 300 ms. Activation mapping of the tachycardia showed that the earliest endocardial activation was registered 35 ms before the onset of the QRS complex at the superior aspect of the left ventricle, below the mitral valve and close to the left ventricular outflow tract. Delivery of radiofrequency current (25 W) at this site terminated the tachycardia in 10 s. A treadmill exercise test performed 3 days after the ablation showed no induction of the

Figure 4. Recordings from Patient 2 showing induction, mapping and radiofrequency ablation of the ventricular tachycardia. A, Induction of ventricular tachycardia during isoproterenol infusion with delivery of two atrial extrastimuli at an atrial-driven cycle length of 400 ms and the first and second coupling interval of 260 and 230 ms, respectively. B, Endocardial activation mapping during tachycardia showing that the earliest activation was registered 40 ms before the onset of the QRS complex at the high anterosuperior aspect of the left ventricle, below the mitral annulus close to the left ventricular outflow tract. C, Termination and successful ablation of the ventricular tachycardia after delivery of radiofrequency current (arrow). A = atrial response; HRA, HBE, RV = bipolar electrograms recorded from the high right atrium, His bundle area and right ventricle, respectively; LVd, LVP = bipolar electrograms recorded from the distal and proximal two electrodes, respectively, of the mapping (ablation) catheter located in the left ventricle; RVOTd, RVOT2, RVOT3, RVOT4 = unipolar electrograms recorded from the distal, second, third and fourth electrodes of the quadripolar catheter, respectively, positioned at the right ventricular outflow tract; S1 = basic driven stimulus; S2 = first extrastimulus; S3 = second extrastimulus; V = ventricular response.

Figure 5. ECGs showing termination of ventricular tachycardia after intravenous bolus of ATP in Patients 3 (A) and 4 (B). See text for discussion.
tachycardia. The patient has been free of symptoms without medication for 6 months.

**Patient 4.** A 31-year old woman presented with episodic palpitations for 2 years. The attacks were related to exercise and emotional stress and lasted ~5 min. The ECG recorded during sinus rhythm showed frequent ventricular extrasystoles. The ECG recorded during palpitations displayed a monomorphic wide QRS tachycardia at a rate ranging from 160 to 260 beats/min and a configuration of atypical right bundle branch block and an inferior axis (Fig. 5B). Physical examination, chest roentgenogram and echocardiogram were normal. Treadmill exercise tests reproducibly provoked sustained ventricular tachycardia identical to the clinical tachycardia. An intravenous bolus of 18 mg of ATP terminated the tachycardia in 7 s; termination of tachycardia occurred abruptly without slowing of the rate (Fig. 5B). Subsequent exercise tests on separate days demonstrated that intravenous xylocaine (100 mg bolus), oral propranolol (80 mg/day in four divided doses) or oral verapamil (160 mg/day in four divided doses) effectively prevented exercise provocation of the tachycardia. Programmed stimulation during control electrophysiologic study was unable to induce tachycardia. After isoproterenol infusion (3 μg/min), both sustained and nonsustained tachycardia identical to the clinical tachycardia occurred spontaneously. The tachycardia was also inducible by rapid atrial or ventricular pacing. The induced tachycardia had a cycle length that varied from 230 to 320 ms. Entrainment of the tachycardia was not demonstrated by overdrive pacing. Activation mapping revealed that the earliest endocardial activation was registered 50 ms before the onset of the QRS at the superior aspect of the left ventricle, beneath the anteromedial aspect of the mitral annulus adjacent to the left ventricular outflow tract (Fig. 6B). Pace mapping at this site displayed a QRS configuration matching that of clinical tachycardia. Delivery of 22 W of radiofrequency current to this site terminated the tachycardia in 17 s. This site was also documented by transthoracic two-dimensional echocardiography in addition to left ventricular angiography. Repeat treadmill exercise tests 5 days after ablation and thereafter showed no provocation of tachycardia. The patient has been free of symptoms for 1 month, with no medication.

**Discussion**

Exercise-provocable right ventricular outflow tract tachycardia versus idiopathic left ventricular tachycardia. Idiopathic ventricular tachycardia may arise from the right or left ventricle. The tachycardia from the right ventricle is characterized by a QRS configuration of left bundle branch block and an inferior or normal axis and originates from the right ventricular outflow tract (9–12). Some tachycardias are frequently provoked by stress, exercise or isoproterenol infusion and are responsive to vagal maneuvers, edrophonium, ATP, dipyridamole, verapamil and beta-adrenergic blocking agents (11–14). Programmed stimulation may induce tachycardia, especially during isoproterenol infusion, but entrainment is not seen with overdrive pacing. Wu et al. (11) and Lerman et al. (13) have suggested triggered afterdepolarization as the operative mechanism of the tachycardia. Idiopathic tachycardia from the left ventricle is characterized by a QRS configuration of right bundle branch block with a superior or indeterminate axis. This tachycardia, first reported by Zipes et al. (15), was shown to be verapamil responsive by Belhassen et al. (16) and was subsequently designated as a specific clinical entity by Lin et al. (1). The tachycardia is inducible and terminable by programmed stimulation and can be entrained by overdrive ventricular pacing (1–4,8,17,18). It is responsive to calcium blocking agents but not ATP or beta-blockers. A sharp Purkinje potential is recorded before the onset of the QRS complex during tachycardia. This tachycardia can be ablated successfully from the inferoapical or midseptum of the left ventricle (8). Reentry is the operative mechanism of the tachycardia.

Exercise-provocable right ventricular outflow tract tachycardia (3,11–14) and verapamil-sensitive idiopathic left ventricular tachycardia share (1–4,8,17,18) some similarities; however, differences between these two tachycardias can readily be identified. Provocation of idiopathic left ventricular tachycardia by exercise or isoproterenol infusion is noted only occasionally and appears to be related to achievement of a critical heart rate. Electrical induction of exercise-provocable right ventricular outflow tract tachycardia is less consistent and usually requires isoproterenol infusion. The cycle length of idiopathic left ventricular tachycardia is stable; however, cycle
length alternans is seen not infrequently. The cycle length of exercise-provocable right ventricular outflow tract tachycardia displays wide variation depending on the level of catecholamine or sympathetic tone, but cycle length alternans is only seen rarely (19,20). The first coupling interval of the induced idiopathic left ventricular tachycardia demonstrates an inverse relation to the paced cycle length or the coupling interval that induces the tachycardia, whereas the first coupling interval of the induced right ventricular outflow tract tachycardia demonstrates a concordant relation to the paced cycle length or the coupling interval that induces the tachycardia. The idiopathic left ventricular tachycardia demonstrates entrainment by overdrive ventricular pacing, but the exercise-provocable right ventricular outflow tract tachycardia does not. Both tachycardias are responsive to calcium blockers, but only the exercise-provocable right ventricular outflow tract tachycardia is responsive to beta-blockers or vagal maneuvers.

**Adenosine and idiopathic ventricular tachycardia.** Adenosine invariably interrupts exercise-provocable right ventricular outflow tract tachycardia (3,13,14) but is effective only in sporadic cases of idiopathic left ventricular tachycardia (6,7). DeLacey et al. (6) described a patient with nonsustained tachycardia showing a QRS complex of right bundle branch block, a left axis and a rate of 125 beats/min. The tachycardia was not inducible at baseline but occurred spontaneously during isoproterenol infusion. Both adenosine and verapamil terminated the tachycardia. The earliest activation during tachycardia was identified at the inferoapical region of the left ventricle and was not preceded by a high frequency Purkinje spike. Kobayashi et al. (7) described two patients with verapamil-sensitive idiopathic left ventricular tachycardia, in whom the tachycardias were responsive to adenosine, dipyridamole, acetylcholine and vagal maneuvers. The tachycardias were provokable by exercise and isoproterenol infusion, and displayed a QRS configuration of atypical right bundle branch block, inferior axis and variation in cycle length. The earliest activation site during tachycardia was located at the anterobasal or upper midseptal region of the left ventricle without a Purkinje spike preceding the QRS complex. Entrainment was not demonstrated by overdrive ventricular pacing. Lee et al. (20) noted that 6 of their 22 patients had verapamil-sensitive idiopathic left ventricular tachycardia responsive to adenosine; however, the electrophysiologic characteristics of these 6 patients were not available for analysis. The electrophysiologic characteristics of the four patients in the present study as well as the two patients of Kobayashi et al. (7) resemble those of the exercise-provocable right ventricular outflow tract tachycardia in that the tachycardia is exercise provocable, catecholamine mediated, verapamil responsive and adenosine sensitive. Delayed afterdepolarizations appear to be the operative mechanism of this arrhythmia, which tends to arise from the anterobasal left ventricle adjacent to the outflow tract. Triggered activity has been demonstrated in cardiac muscle fibers in the anterior mitral valve leaflet of the monkey heart (21). Regarding the patients of DeLacey et al. (6) and Lee et al. (20), the mechanism of adenosine responsiveness is less clear. Whether adenosine responsiveness in their patients was mediated through the same mechanism as that in our patients or was due to a nonspecific antagonistic effect of adenosine on isoproterenol is unclear.

Two reports of adenosine-sensitive idiopathic left ventricular tachycardia (Callans et al. [22] and Lerman et al. [23]) have recently appeared. The observations in the present study are concordant with those of Callans et al. (22) and Lerman et al. (23).

**Conclusions.** Exercise-provocable tachycardia resembling the exercise-provocable right ventricular outflow tract tachycardia may arise from the left ventricle. This tachycardia is sensitive to adenosine, appears to be mediated by catecholamines and tends to arise from the anterobasal left ventricle adjacent to the outflow tract. Delayed afterdepolarization is likely to be the operative mechanism of the tachycardia.

**References**