Electrophysiological Characteristics of Idiopathic Ventricular Tachycardia in Children

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**Background:** Idiopathic ventricular tachycardia (VT) has been reported to have a good prognosis, but there still might be the potential risk of sudden death.

**Methods and Results:** The 46 consecutive children (mean age 11.7±3.4 years) with idiopathic VT were enrolled in this study. Monomorphic VT was detected in 39 patients and polymorphic VT in 7 patients. The VT originated from the right ventricle (RV) in 22 patients, and left ventricle (LV) in 17 patients. The VT was induced by exercise in 68% of the RVVT, 41% of the LVVT, and 100% of the polymorphic VT. The VT was induced by programmed ventricular stimulation in 41% of the RVVT, 35% of the LVVT, and none of the polymorphic VT. Adenosine triphosphate terminated the VT in 9 of 15 patients (60%). The mechanism of the VT was suspected to be triggered by activity in 36.4%, automaticity in 40.9%, and re-entry in 22.7% of the RVVT, whereas it was 52.9%, 5.9%, and 41.2% of the LVVT, respectively.

**Conclusions:** The exercise inducibility was higher in polymorphic VT than the RVVT and LVVT, but no difference in the programmed stimulation. The sensitivity to adenosine tri-phosphate was not different between the RVVT and LVVT. In some patients with idiopathic VT, a non-verapamil sensitive re-entry was documented, which was more common in patients with ischemic heart disease or cardiomyopathy. (Circ J 2011; 75: 672–676)

**Key Words:** Idiopathic ventricular tachycardia; Monomorphic ventricular tachycardia; Polymorphic ventricular tachycardia; Ventricular fibrillation
catheters were introduced via the femoral, cervical or subclavian veins, and positioned in the right atrium, right ventricle (RV), septal leaflet of the tricuspid valve, and coronary sinus. Programmed ventricular stimulation was performed by applying burst ventricular pacing and up to triple extra stimuli from the right ventricular apex and outflow tract in the basal state following 8 beats of ventricular pacing during 2 basic cycle lengths. If the tachycardia was not induced, programmed stimulation was repeated under an infusion of 0.01–0.03 μg/kg/min of isoproterenol. The mechanism of the VT was confirmed by the inducibility and terminability of the VT by programmed stimulation, entrainment pacing during VT, and the effects of several medications. If VT were reproducibly induced and terminated by programmed stimulation, and an entrainment phenomenon was documented, it was defined as re-entry. The VT were defined as triggered activity when the VT were induced and terminated by programmed stimulation but not reproducibly, or the VT were terminated by an injection of adenosine tri-phosphate (ATP). The VT were defined as automaticity if the VT were not induced nor terminated by programmed ventricular stimulation. The origin of the VT was suspected from the surface electrocardiograms of the VT, earliest ventricular activation, pace mapping, activation mapping using 3-dimensional electroanatomical mapping (CARTO, Biosense-Webster), and the termination point of the VT by radiofrequency ablation (RFA).

A statistical analysis was made using a Fisher’s exact probability test and Pearson’s chi-square test by employing JMP v 5.1 software (SAS Institute Inc, NC, USA), and a P value of <0.05 was considered statistically significant.

Results

Monomorphic VT was detected in 39 patients. Six patients had catecholaminergic polymorphic VT and 1 patient had Andersen-Tawil syndrome.

Origin of the VT
The origin of the VT was from the RV in 22 patients, and left ventricle (LV) in 17. In 21 of 22 patients with a right ventricular VT, the origin was from the right ventricular outflow tract (RVOT), the VT in the other patient was from the right ventricular apex. Left ventricular origins of the VT were observed in the left ventricular outflow tract (LVOT) in 4 patients, left ventricular apex in 7, intraventricular septum in 5, and lateral wall of the LV in one.

VT was induced by exercise tests in 68% of the right ventricular VT, 41% of the left ventricular VT, and 100% of the polymorphic VT (Figure 1). The exercise inducibility was higher in polymorphic VT than the RVVT and LVVT (P=0.02).

VT was induced by programmed ventricular stimulation in 41% of the patients with right ventricular VT, 35% of those with left ventricular VT, and in none of those with polymorphic VT. There was no difference in the programmed stimulation inducibility between the RVVT, LVVT, and polymorphic VT (P=0.13).

ATP was administered during VT in 15 patients, and 9 (60%) VT were terminated by an ATP injection (Figure 2). The sensitivity to ATP was not different between the RVVT and LVVT (P=0.23). Verapamil was injected in 11 patients and the VT terminated in all of those patients.

Mechanism of the VT
From the results of the programmed stimulation and response to the drugs, the mechanism of the VT was suspected to be triggered activity in 36.4%, automaticity in 40.9%, and re-entry in 22.7% of the right VT. The mechanism was suspected to be re-entry in 52.9%, triggered activity in 5.9%, and automaticity in 41.2% of the left VT (Figure 3).

Radiofrequency Ablation
RFA was performed in 32 patients and 22 patients (69%) were successfully ablated. RFA was not performed in 5 patients because we did not start doing RFA at EPS. RFA was not performed in 9 patients after starting RFA, because no VT was induced during the EPS in 1 patient, the VT focus was very close to the His potential recording site in 1 patient,
polymorphic VT was noted in 7 patients. In 12 of the automatic VT patients, 8 (67%) were successfully ablated by RFA, 1 patient was controlled with anti-arrhythmic agents, and 3 were followed without any medications; in 9 of the VT patients with triggered activity, 7 (78%) were successfully ablated by RFA, 1 patient was controlled with anti-arrhythmic agents, and 1 was followed without any medications; in 11 of the re-entrant VT patients, 7 (64%) were successfully ablated with RFA, and 4 were controlled with anti-arrhythmic agents. RFA was not performed in 7 patients with polymorphic VT, however those patients were controlled with anti-arrhythmic agents, and 2 additionally underwent implantable cardioverter defibrillator (ICD) implantation. In 7 of the 10 unsuccessful patients, RFA was performed without using a CARTO system. The reason for the failure of the ablation before using the CARTO system was as follows; the VT focus was very close to the His potential recording site (para-Hisian VT) in 2 patients, failure to initiate the VT because the mechanism of the VT was automaticity in 2 patients, and it was technically impossible to reach the ventricular focus in 3 patients. After using the CARTO system, the ablation in 2 patients with left ventricular epicardial VT, and in 1 patient with a para-Hisian VT was unsuccessful. However, the other 81% of the VT were successfully ablated.

**Discussion**

Most of the data from the electrophysiological findings of idiopathic VT have been obtained from adult patients.\(^2\)\(^-\)\(^5\) However, relatively few data on the electrophysiological findings have been reported in pediatric patients.\(^1\)\(^6\)\(^-\)\(^8\) The largest VT study in pediatric patients was reported by Pfammatter et al.\(^1\) They concluded that most of the VT in children originated from the RVOT (70%), and the mechanisms of the VT were

![Figure 2. Effect of adenosine triphosphate (ATP) and verapamil on the ventricular tachycardia. This panel shows the effects of ATP and verapamil on the origin of the ventricular tachycardia. RVOT, right ventricular outflow tract; LVOT, left ventricular outflow tract; LV, left ventricle.](#)

![Figure 3. Suspected mechanism of the ventricular tachycardia. This panel shows that from the results of the programmed stimulation and response to the drugs, the mechanism of the ventricular tachycardia could be presumed. RV, right ventricle; LV, left ventricle; ISP, isoproterenol.](#)
enhanced automaticity or triggered activity. Throughout the past decade, several studies have been undertaken to define the clinical course and outcome in pediatric patients with idiopathic VT, but the electrophysiological mechanisms have not been evaluated in detail. This study examined the detailed electrophysiological mechanisms in pediatric patients with idiopathic VT.

In another report in adult patients, up to 90% of ventricular outflow tract VT originate from the RV, mainly from the RVOT, but also from other regions of the RV, including sites above the pulmonary valve. LVOT tachycardia can arise from endocardial sites, epicardial sites, the area of the aorto-mitral continuity, as well as from foci accessible from the aortic sinuses of Valsalva. In our study, 56% of the idiopathic VT originated from the RV, and 95% of those originated from the RVOT. In 2 of 4 patients with a LVOT origin, the VT originated from epicardial sites.

Most RVOT VT is sensitive to adenosine and verapamil. These findings are consistent with VT due to cyclic adenosine monophosphate-mediated triggered activity dependent on delayed after depolarizations. However, it is unclear whether LVOT VT shares a similar clinical phenotype, mechanism, and electrophysiological properties as RVOT VT.

Iwai et al reported that in 122 patients with outflow tract arrhythmias in adult patients, the VT in 100 (82%) patients originated from the RVOT, and from the LVOT in 22 (18%) patients. In this study, from 41% of the RVOT foci and 50% of the LVOT foci, sustained VT was inducible, and the induction of VT was catecholamine dependent in 66% of the RVOT foci and 73% of the LVOT foci. The VT was sensitive to adenosine (88% and 78% of the RVOT and LVOT VT, respectively) as well as to the blockade of the slow inward calcium currents (70% of the RVOT and 80% of the LVOT VT) in both groups. The above data suggest that the most common arrhythmogenic mechanism was consistent with cyclic adenosine monophosphate-mediated triggered activity for both the RVOT and LVOT arrhythmias. Based on these similarities, these arrhythmias should be considered as a single entity, and classified together as ‘outflow tract arrhythmias’.

In the present study, the mechanisms of the LVOT VT were automaticity in 75%, and triggered activity in 25%. Furthermore, in some patients with RVOT VT, the mechanisms were verapamil sensitive re-entry and verapamil non-sensitive re-entry.

VT at a very young age is reported mostly to be exercise-related VT. Rocchini et al reported that graded treadmill exercise testing increased the degree of the ventricular arrhythmias in 9 of 21 pediatric patients (43%). In those patients, the ambulatory monitoring and exercise testing revealed an 88% and 57% incidence of VT, respectively. Deal et al reported that exercise-related symptoms were present in 9 of 24 pediatric patients (38%). Of those 9 patients with a history of exercise-related VT, treadmill testing induced sustained tachycardias in 5 and non-sustained tachycardia in one. Among the 15 patients with no history of exercise-related VT, non-sustained tachycardia was induced by exercise in 1 patient. Up to 50% of the idiopathic VT in our study was induced by exercise. The mechanism of the exercise-related VT is known to be triggered activity. However, to the best of our knowledge there have been no detailed reports on the mechanism of exercise-related VT. Exercise-related VT was present in 6 of 11 patients with triggered activity associated VT (55%), 10 of 15 with automaticity associated VT (67%), and 6 of 13 with re-entry associated VT (46%) in our study. There was no difference in the mechanism of the idiopathic VT induced by exercise.

As in the adult patients, where RFA of idiopathic VT was proven to be associated with high success rates, several reports on the RFA of VT in children have also shown a similar safety and efficacy of the procedure in pediatric patients. The success rate for the ablation of either right or left VT has ranged from 83 to 88%. In this study, the radiofrequency catheter ablation was successful in 69% of the patients. Two patients with an epicardial origin were incapable of being ablated from the ventricular endocardium or the aortic sinus of Valsalva. Tanner et al reported that in 5 of 33 patients with idiopathic outflow tract VT, it was not possible to ablate the VT from the left or right ventricular endocardium or the aortic sinus of Valsalva. Three patients underwent a successful ablation via a coronary venous approach and 2 by a transpericardial approach. In those cases, a coronary venous approach or transpericardial approach can be successful for the catheter ablation of the VT focus. However, reports of the transpericardial approach being used in the pediatric age group are limited, and the manipulation involved in this catheter ablation might have potential risks of coronary sinus rupture and cardiac tamponade. Irrigation catheters might increase the success rate for some of the epicardium origin VT.

Three of the patients with a para-Hisian origin were unable to be ablated in this study. In those cases, cryoablation might have been useful for ablating the VT focus. However, it is still difficult to ablate the VT in patients with automatic mechanisms of the VT because of the difficulty in inducing the VT. EnSite Array mapping system was reported effective for non-sustained VT or premature ventricular contractions. In the patients with RVOT VT and LVVT, the success rate of the radiofrequency catheter ablation was fairly successful even in children, and we recommend radiofrequency catheter ablation in these patients. However, in the patients with polymorphic VT or autonomic VT, anti-arrhythmic drugs or an ICD implantation might be the first choice of treatment in these patients. The development of a smaller sized mapping system with a simultaneous recording of the onset of the VT would make it easier to ablate the autonomic or polymorphic VT in children. The development of a small sized ICD system could be of benefit for children whose VT was difficult to control either by RFA or anti-arrhythmic drugs.

Conclusions
The exercise inducibility was higher in polymorphic VT than the RVVT and LVVT. There was no difference in the programmed stimulation inducibility between the RVVT, LVVT, and polymorphic VT. The sensitivity to ATP was not different between the RVVT and LVVT. In some patients with idiopathic VT, non-verapamil sensitive re-entry was documented, and was more common in patients with ischemic heart disease or cardiomyopathy.

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References


