

EXERCISE – INDUCED VENTRICULAR TACHYCARDIA IN CHILDREN*

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Two children with exercise-induced tachycardia, one with idiopathic long-QT syndrome, are presented. The patients were evaluated by exercise testing and electrophysiologic study. From the onset of treatment with the beta-blocking agent, pindolol, the patients have been symptom-free. These findings emphasize that children with syncope must be evaluated by ECG, exercise testing, 24-h Holter-monitoring, and finally, electrophysiological study. *Key words:* tachycardia, exercise-induced, children.

Exercise-related ventricular tachycardia is an uncommon finding in children. Current knowledge is based on reports of series comprising small numbers of patients. In a relatively large series, Bricher et al¹ found exercise-related tachycardia to have a frequency of approximately 0.8 percent among a referral population of children with cardiac disease. Although tachycardia, which occurs in children during exercise, is generally well tolerated, it is well known that in some cases it may cause syncope and even result in sudden death². In this report we describe two children with unexplained syncope associated with exercise-induced ventricular tachycardia.

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Case Reports

Case 1

A 14-year-old boy with the diagnosis of unexplained syncope who was followed up for four years had a VVI (ventricular pacing sensing inhibiting) permanent pacemaker implanted at the age of 13. Despite the presence of pacemaker back-up, he continued to have fainting episodes and was reevaluated. Physical examination revealed that there was only an apical late systolic murmur grade I/VI. ECG was normal, apart from a sinus bradycardia. Echocardiography revealed no structural abnormality. Neurological evaluation showed no disorder. The permanent pacemaker was removed and an electrophysiologic study was performed which revealed no abnormality. An exercise test was performed because of a probable association between exertion and syncope, and it revealed unifocal ventricular premature beats and short runs of ventricular tachycardia (Fig. 1a.b). Pindolol (Visken^R) therapy was instituted and the dosage was gradually increased to 45 mg bid. The medication was well tolerated and the syncopal attacks completely disappeared. The patient started to play basketball on the school-team, and was a successful student. During the four-year follow-up period he showed normal physical and psychological growth.

Case 2

A 9-year-old boy with the diagnosis of unexplained syncope was followed up for three years. Despite antiepileptic treatment, there was no improvement in his fainting episodes. A careful examination of the history revealed that there was a possible relationship between the fainting and exertion. According to the family history, the parents of the child were close relatives. In addition, two of the patient's male cousins, both siblings, had died of unexplained syncope, one at 15 years of age and the other at 12 years of age. Moreover, the patient's father had had palpitations and attacks of dizziness upon exertion, but had never used any medication for these conditions. Physical examination revealed normal physical findings. Neurologic examination was also unremarkable. Electrocardiographically, the QTc interval was 420 milliseconds. Two-dimensional echocardiography was normal. An electrophysiologic study was performed and revealed no abnormality except for a prolonged QTc interval (Fig. 2). A simple controlled exercise test, consisting of jumping, precipitated a bout of ventricular tachycardia and syncope. Pindolol (Visken^R) in a dose of 5 mg b.i.d., was started and 24-h Holter-monitoring showed the patient to be free of ventricular tachyarrhythmias. The patient, diagnosed as having idiopathic long QT syndrome, has been syncope-free for two years.

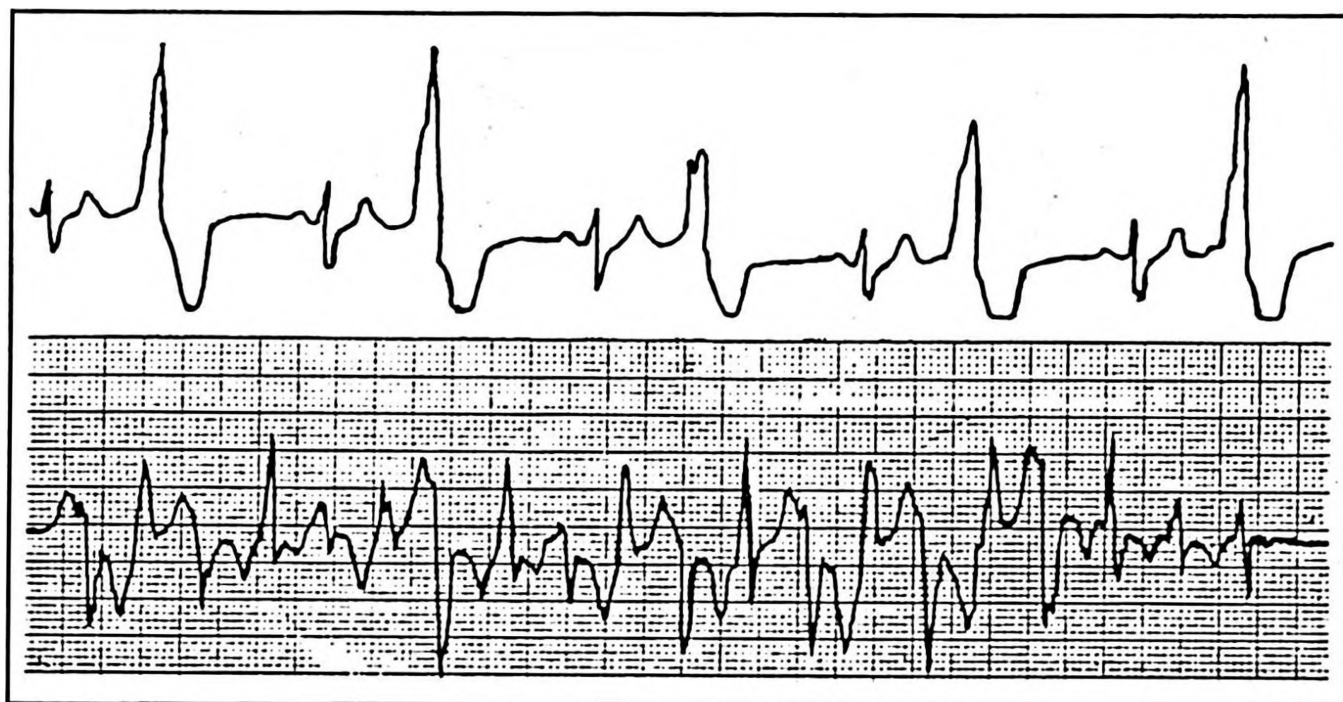
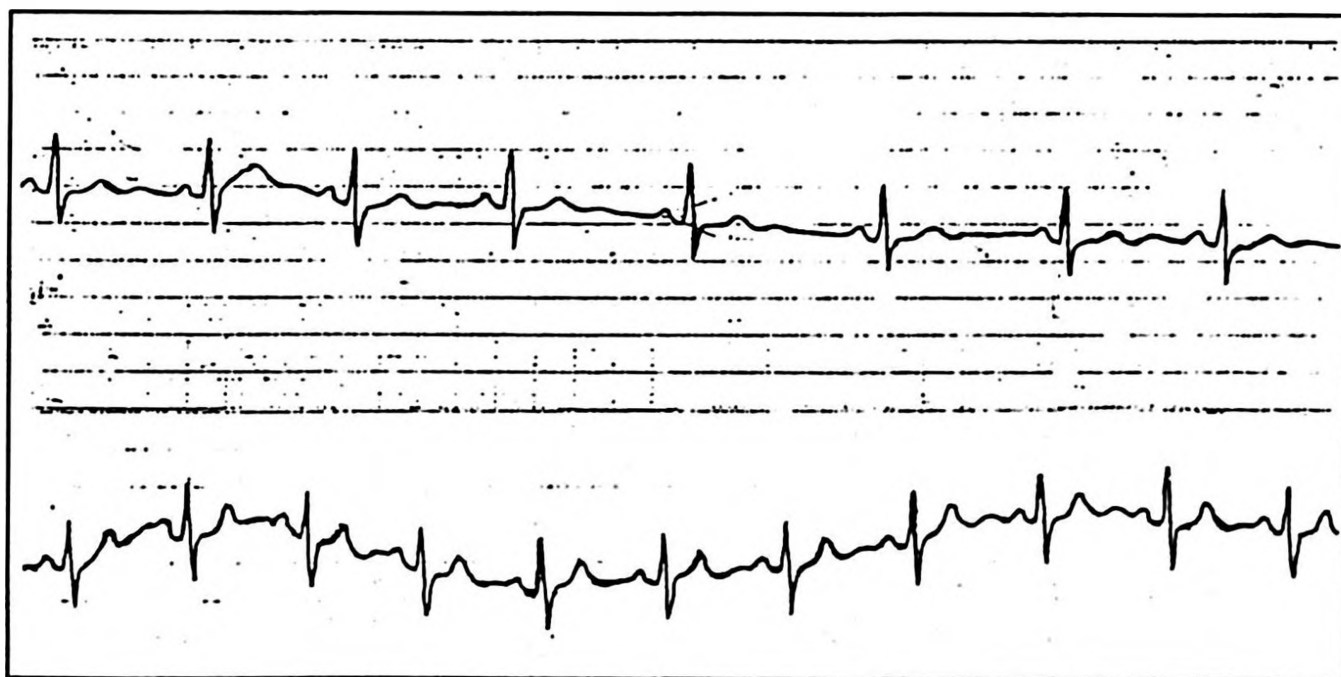
**a****b**

Fig. 1: The top of the panel shows ventricular bigeminy and exercise-induced multiform ventricular tachycardia. (a) after treatment with pindolol ventricular bigeminy disappeared, and exercise could not provoke ventricular tachycardia (b).

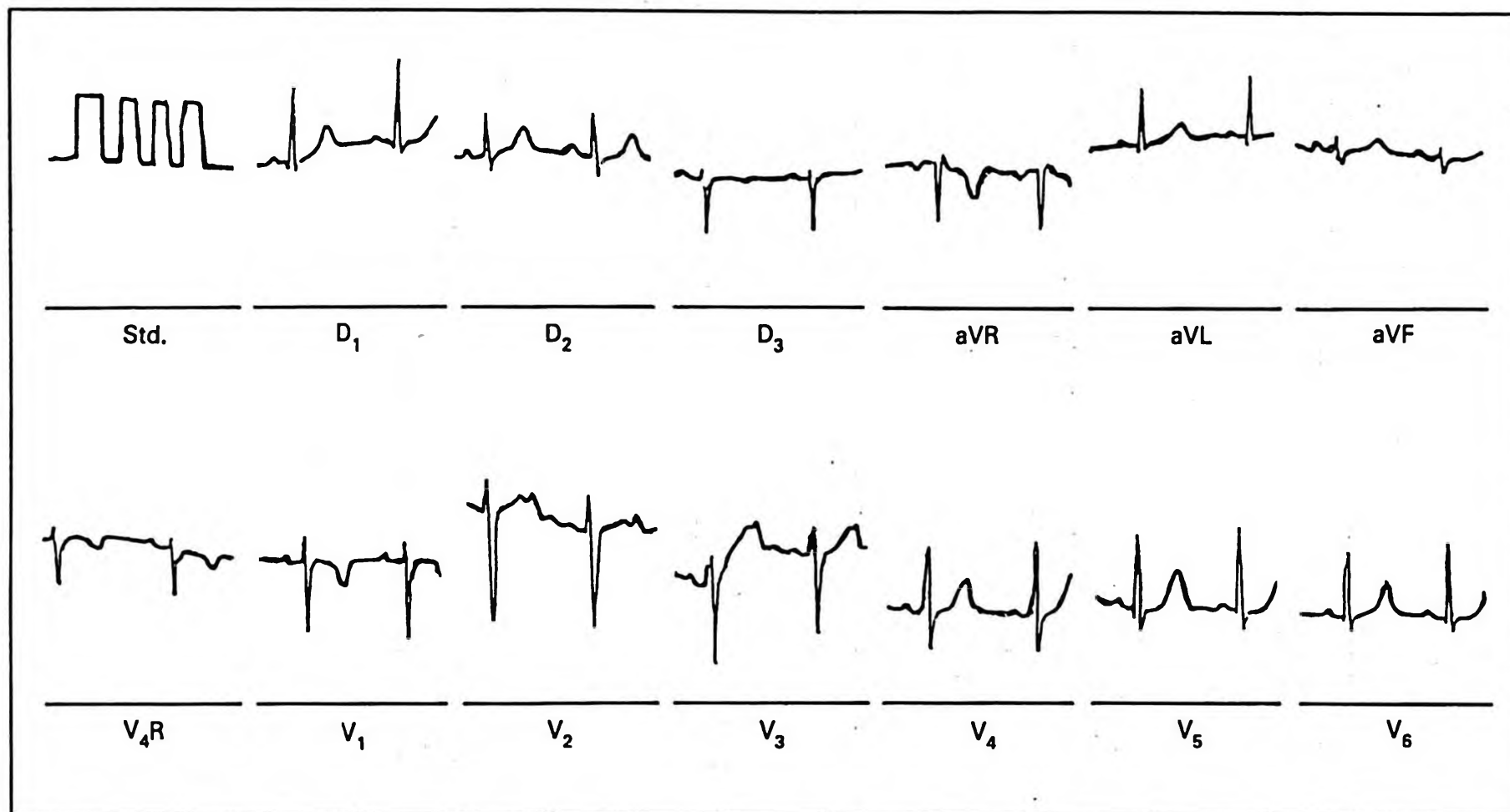


Fig. 2: The rhythm strip revealed a normal sinus rhythm and a corrected QT interval of 420 milliseconds.

Discussion

Ventricular arrhythmias in children are uncommon, especially in the absence of congenital structural heart disease or other predisposing factors³⁻⁵. The diagnosis of ventricular tachycardia in children is commonly an incidental finding in asymptomatic patients⁶. Idiopathic ventricular tachycardia is generally believed to carry a favorable prognosis, though the natural history and the need for treatment still remains controversial⁷⁻⁹. However, patients with exercise-related ventricular tachycardia may be at an especially high risk of sudden death, and antiarrhythmic therapy has been recommended, even in asymptomatic patients^{1,10}.

Exercise increases sympathetic tone and promotes secretion of catecholamines. Consequently, exercise enhances diastolic (phase 4) depolarization, improves membrane conductivity, shortens refractory periods of all cardiac tissues, and activates the adenylate cyclase-cyclic AMP system, thereby increasing transmembrane calcium influx. Therefore, exercise can theoretically provoke tachyarrhythmias via all three major mechanisms: re-entry, automaticity and triggered rhythmic activity. Patients with exercise-induced tachycardia generally do not have excessive secretion of catecholamines. These findings have led investigators to suggest the presence of a low sympathetic threshold¹¹. Sung et al¹² have demonstrated that beta-adrenoreceptor blockade is especially effective in suppressing ventricular tachycardia. From a clinical standpoint, it is thus quite reasonable to initiate beta-blocker therapy in patients with exercise-induced ventricular tachycardia unless clinical findings indicate otherwise¹³. In selective cases, electrophysiologic studies are needed to define the underlying mechanism of this abnormality and to select an effective pharmacological agent^{14,15}. Our first case had exercise-induced tachycardia, and a beneficial effect was achieved with beta-blockade, which is consistent with the above-mentioned data. Since we could not demonstrate any anatomic substrate either by echocardiographic or electrophysiologic studies, and because there was no clinical evidence for increased sympathetic activity, we propose a low sympathetic threshold as the cause of the ventricular arrhythmia.

Idiopathic long QT syndrome is a ventricular repolarization abnormality characterized by autosomal recessive or dominant inheritance. The diagnosis of this disease is made basically by measuring the corrected QT interval (QTc), which extends beyond the normal limits for age. Affected individuals have ventricular tachycardia and syncope induced by exercise and stress². Electrophysiological studies are not usually helpful, however, they may be valuable in doubtful cases to rule out other possible causes^{14,15}. Treatment is based on the administration of beta-blocker drugs. Some patients may experience syncope despite this medication and require high thoracic left sympathectomy². The present case has been on pindolol for two years and has remained symptom-free. If drug tolerance develops in this patient, a sympathectomy might be planned as a more definitive approach.

In conclusion, occult arrhythmias are important causes of syncope in some children with both structurally normal hearts and negative neurologic evaluation. Children with negative findings for non-cardiac causes of syncope should be evaluated by ECG, 24-h Holter-monitoring, maximal exercise testing, and a detailed cardiac electrophysiologic study. These recommendations are especially important in children with exercise-induced syncope because of the fatal nature of the outcome related to this arrhythmia¹⁶.

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