Exercise-induced Ventricular Tachycardia/ Ventricular Fibrillation in the Normal Heart

Risk Stratification and Management

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KEYWORDS

- Ventricular tachycardia • Ventricular fibrillation • Exercise
- Arrhythmogenic right ventricular cardiomyopathy • Mitral valve prolapse
- Catecholaminergic polymorphic ventricular tachycardia

KEY POINTS

- Ventricular tachycardia (VT) occurs only rarely in patients without organic heart disease, and most of these arrhythmic events are not related to exercise; consequently, the literature on exercise-induced VT/ventricular fibrillation in normal hearts is limited.
- The most common form of idiopathic monomorphic VT is the one originating from the outflow tract.
- The main point in risk stratification of exercise-induced monomorphic VT is distinguishing idiopathic monomorphic VT from VT related to subtle heart disease, particularly right ventricular dysplasia.
- Exercise-induced polymorphic VT is potentially lethal, particularly when caused by genetic disorders.

INTRODUCTION

Ventricular tachycardia (VT) and ventricular fibrillation (VF) occur only rarely in patients without organic heart disease, and most of these arrhythmic events are not related to exercise. Consequently, the literature on exercise-induced VT/VF in normal hearts is limited.[1–3] Idiopathic exercise-induced VT is rare even in the young population, in which organic heart disease is less common. For example, in a recent analysis of consecutive young (<18 years of age) patients treated for VT during a 25-year period, roughly half were free of organic heart disease and only half of these had exercise-induced VT.[4] Furthermore, an arrhythmogenic genetic disorder was ultimately diagnosed in one-third of the patients with exercise-induced VT with apparently normal hearts.[4] Consequently, only 10 patients with exercise-induced idiopathic VT were identified during a 15-year period in a specialized referral center.[4] Analogously, among consecutive athletes undergoing evaluation for ventricular arrhythmias discovered during preparticipation screening, only 23% had persistence (9%) or worsening (14%) of arrhythmias during exercise; most had transient suppression of their ventricular arrhythmias during exercise, which reappeared during the recovery phase of the exercise test.[5] In addition, idiopathic VF is definitively rare and

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is not exercise related.[6,7] This article focuses on exercise-induced monomorphic (Fig. 1A) and polymorphic (Fig. 1B, C) VT in the absence of evident heart disease, with an emphasis on the differential diagnosis and its implications for risk stratification.

ARRHYTHMOGENIC EFFECTS OF EXERCISE

The mechanisms of ventricular arrhythmias in the normal heart are discussed (see Luebbert J, Auberson D, Marchlinski F: PVCs in Apparently Normal Hearts, in this issue). Exercise is arrhythmogenic, albeit causing mainly extrasystoles and nonsustained VT. The anticipation of dynamic exercise is associated with inhibition of vagal tone, increasing the sinus rate even before exercise begins.[8] Once exercise begins, the interplay between sympathetic activation and vagal withdrawal leads to increased heart rate and augmented ventricular contraction. In the postexercise phase, vagal reactivation decelerates the sinus rate, which may return to baseline within a few minutes while sympathetic tone is still increased. Sudden and unexpected exercise is associated with a different vagal withdrawn to sympathetic activation balance caused by earlier and more dramatic sympathetic activation.[8] Arrhythmogenic effects of exercise include shortening of the ventricular refractory periods, increased velocity of impulse conduction, and enhanced amplitude of afterpotentials. All known mechanisms of cardiac arrhythmias (ie, reentry, triggered activity, and enhanced automaticity) play a role in the genesis of exercise-related arrhythmias.[8]

IDIOPATHIC MONOMORPHIC VENTRICULAR TACHYCARDIA

In its strictest form, the diagnosis of idiopathic VT should be reserved for patients with normal cardiac structure and function as assessed by noninvasive and invasive studies, including cardiac MRI, coronary angiography, ventriculography, and cardiac biopsy. However, in clinical practice, invasive evaluation is often voided when the clinical history and noninvasive studies suggest benign disease and the electrocardiogram (ECG) of the VT is consistent with well-described variants of idiopathic VT. Specific forms of VT often presenting in patients without apparent organic heart disease are presented in Fig. 2.[9] The most common form of idiopathic monomorphic VT (involving about 70% of idiopathic VT cases) originates from either the right ventricular outflow tract (RVOT) or the left ventricular outflow tract (LVOT). This form of VT is the ventricular arrhythmia most often encountered in otherwise healthy athletes.[10] The QRS morphology during VT is of left bundle branch block (LBBB) pattern with tall R waves in the

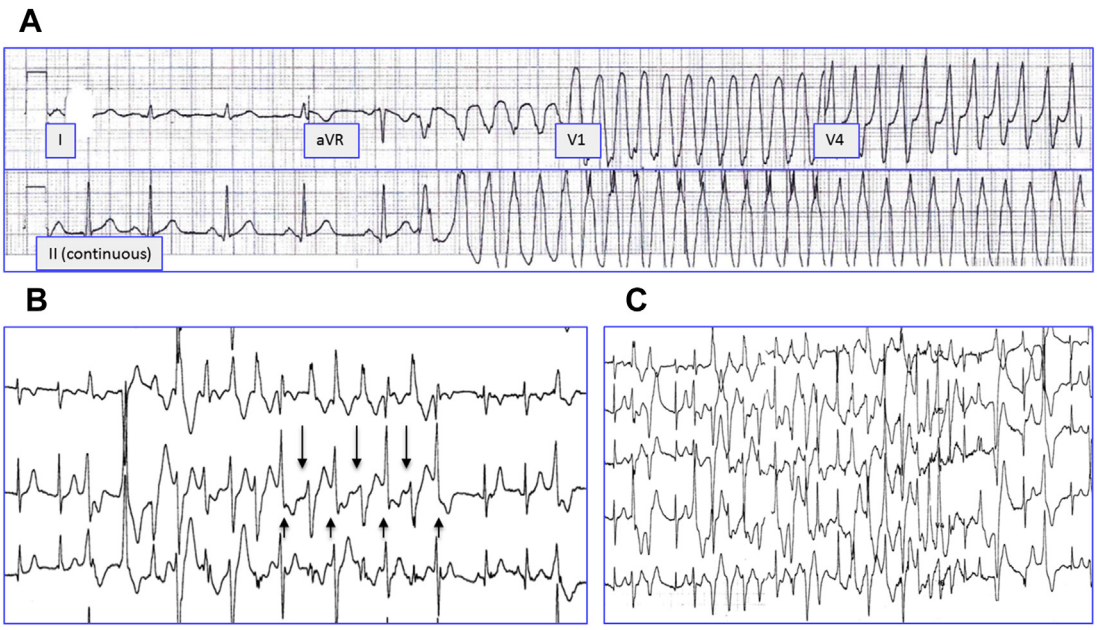

Fig. 1. Exercise-induced monomorphic and polymorphic VT. (*A*) A typical exercise-induced monomorphic VT. The arrhythmia is well tolerated despite its rapid rate. (*B*) The typical sequence of events recorded in patients with catecholamine-sensitive polymorphic VT. As the intensity of exercise increases, patients begin to develop more and more frequent ventricular ectopy and (if the exercise test continues) repetitive bursts of VT that may be bidirectional (*arrows in B*) and frankly polymorphic (*C*).