



A Reentry Story

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Cardiac arrhythmias may stem from abnormalities of impulse formation (such as triggered activity) or of impulse conduction (such as reentry). Extensive experimental work has facilitated the establishment of certain rules that characterize these different mechanisms of arrhythmia. The application of these data to the clinical arena has been quite successful, although "real life" conditions tend to create more complex situations than the artificial environment of the laboratory.

Sustained monomorphic ventricular tachycardia is the most prevalent type of VT. It mostly occurs in patients with structural heart disease generally after a previous myocardial infarction, and is almost always due to a reentrant mechanism [1]. Reentry in these patients results from the circulation of an electrical impulse around an obstacle (generally a scar) with repetitive excitation of the ventricles. Initiation of reentry depends on blocking conduction in one direction so that uninterrupted conduction can occur in the opposite direction. For sustenance, a protected zone of slow conduction is required (again, generally in between scar tissue), which allows electrical recovery of the myocardial tissue that constitutes the reentry circle. Since scar tissue results mostly from MI, it is not surprising that sustained monomorphic VT generally strikes patients with coronary artery disease.

Interestingly, a sizable minority of these arrhythmias also afflicts patients with no evidence of structural heart disease [2]. There are two main types of sustained monomorphic VT that may occur in the absence of structural heart disease and they are termed "idiopathic VTs."

The most common type of idiopathic VT arises from the right ventricular outflow tract demonstrating a typical electrocardiographic pattern of left bundle branch block and inferior electrical axis. The VT is focal, generally originating from below the pulmonary valve, although recently a supraventricular origin from the pulmonary artery was also described [3]. Right ventricular outflow tract VT is caused by triggered activity, which arises from membrane potential oscillations (termed after-depolarizations) that occur during (early AD) or immediately following (delayed AD) an action potential.

Triggered activity depends by definition on (and is triggered by) a previous action potential.

Right ventricular outflow tract VT seems to be caused by delayed AD. These delayed membrane oscillations result from intracellular calcium overload that initiates a transient inward sodium current. This current causes membrane depolarization that might trigger a new action potential and could lead to self-sustaining triggered activity and tachycardia. Catecholamines may cause intracellular calcium overload [4] and, in fact, right ventricular outflow tract VT is often (but not always) associated with exercise or induced by isoproterenol infusion during the electrophysiologic study. Less commonly, VT may originate in the left ventricular outflow tract or the aortic sinuses of Valsalva.

Idiopathic outflow VTs are generally considered to be benign. When symptomatic, they can be targeted during radiofrequency ablation with an overall success rate of about 90%.

The other type of idiopathic VT is the one described in this issue of *IMAJ* by Topilski, Glick and Belhassen [5]. The typical ECG pattern is of a right bundle branch block with superior axis, reflecting the fact that the VT originates in the low apical left inter-ventricular septum. Initially it was believed that this VT was due to triggered activity, but now most investigators agree that it is a reentrant arrhythmia [6]. How and where can a reentrant circle exist within a normal myocardium? The answer to this question is not entirely clear.

Evidence based on endocardial mapping and sophisticated maneuvers during electrophysiologic studies suggests that all [7] or at least the anterograde part of the reentry circle of the VT [8] is confined to the left posterior fascicle and the Purkinje network. Based on this, many authors coined this type of idiopathic VT "fascicular VT." However, this concept has recently been challenged [9].

A protected area of slow conduction (possibly the fascicle that is insulated from the ventricular myocardium) apparently exists, but tends to be short and difficult to identify anatomically [8]. Nevertheless, several authors have identified slowly conducting tissue as part of the tachycardia circuit [10]. Moreover, sharp mid- and late diastolic potentials, assumed to represent depolarization of the posterior fascicle or of the Purkinje fibers (the P potentials), have been identified along the tachycardia circuit during VT. The

VT = ventricular tachycardia
MI = myocardial infarction
AD = after-depolarization

mid-diastolic potentials are conceivably recorded from the protected area of slow conduction while the late ones represent the exit point to the myocardium. In fact, application of radiofrequency energy during VT to P potentials (particularly the mid-diastolic ones) is associated with a high cure rate of the VT.

Another term used quite often to describe this entity is "verapamil-sensitive VT," which alludes to the ability of intravenously administered verapamil to terminate the arrhythmia. This unusual reaction of a VT to verapamil, which is much more typical of supraventricular tachycardias, was first described more than 20 years ago by Belhassen et al. [11] and remains intriguing to this day [12]. The likely explanation for this VT responsiveness to a calcium channel blocker is that the area of slow conduction distal to the mid-diastolic potential is calcium channel-dependent [12].

Dr. Belhassen's group should be congratulated for their contribution to the recognition of this clinical entity, for the detailed and accurate study of their patients, as well as for the excellent results in curing them using radiofrequency energy. They have now reconfirmed the importance of ablating the mid-diastolic potential for achieving cure of the arrhythmia. For Dr. Belhassen, it is a reentry story of closing a circle from identification to cure of an intriguing clinical entity.

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