GORDON K. MOE LECTURE

Resetting and entrainment of reentrant ventricular tachycardia associated with myocardial infarction

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Resetting and entrainment are specific responses to programmed stimulation that are useful in determining the mechanism of ventricular tachycardia (VT), localizing critical components of a reentrant circuit to guide ablation, determining how antiarrhythmic drugs affect the tachycardia, and developing antitachycardia pacing modalities. While resetting and entrainment have certain things in common, they differ significantly in the ability to characterize the properties of VT. Only resetting, which is the interaction of a single extrastimulus with the tachycardia, can characterize the properties of the VT itself. Entrainment assesses the effect of overdrive pacing on a reset circuit, not the VT itself. The terms for these techniques are often incorrectly used interchangeably. The present review details the characteristics and uses of both stimulation techniques.

KEYWORDS Tachycardia; Resetting; Reentry; Pacing; Entrainment

ABBREVIATIONS **ATP** = antitachycardia pacing; **ECG** = electrocardiogram; **VT** = ventricular tachycardia

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Resetting and entrainment are specific responses to the introduction of 1 or more stimuli during reentrant tachycardia.[1–10] While similar stimulation may affect focal tachycardias, what distinguishes reentrant tachycardia from focal tachycardias are the specific responses to a single or sequential stimuli interacting with the tachycardia. The hallmark of a reentrant rhythm is the presence of *fusion*, whether it occurs on the surface electrocardiogram (ECG) or during the assessment of intracardiac electrograms.[1,6,7,9,10]

Resetting of ventricular tachycardia

Resetting is the interaction of a premature wavefront with a tachycardia, resulting in either advancement or delay of the subsequent tachycardia beat. While focal mechanisms may be reset, the ability to reset with fusion, either on the surface ECG or on intracardiac recordings, is diagnostic of reentry. For resetting to occur, an excitable gap must exist in the reentrant circuit such that there is excitable tissue between the head of the preceding tachycardia wavefront and the tail of refractoriness. During reentry, a premature stimulated wavefront enters the excitable gap and collides antidromically with the preceding tachycardia wavefront and conducts orthodromically through excitable tissue in the circuit to produce an early complex. Termination occurs when the premature impulse collides antidromically with the preceding wavefront and blocks orthodromically owing to encroachment on the refractory period of the prior wavefront (Figure 1). The ability to reset the tachycardia depends on the site of stimulation, which is also a site of sensing for stimulation during the tachycardia. The closer the stimulus to the entrance site in the tachycardia circuit, the easier it is to reset the tachycardia. It would therefore be expected that stimulation of the left ventricle would facilitate the resetting of a left ventricular tachycardia (VT). If the stimulation site, which is usually the right ventricle, is distant from the tachycardia circuit, the sensing of the tachycardia wavefront limits the prematurity with which the stimulus can be introduced. The prematurity with which right ventricular extrastimuli can reach the tachycardia circuit is affected by the refractory period at the site of stimulation and the recovery of the intervening tissue from the prior wavefront. This can result in the inability of a single extrastimulus to reach the tachycardia circuit in time to reset it. In that instance, the use of double extrastimuli is extremely useful. The first extrastimulus acts as a conditioning extrastimulus and reverses the wavefront of activation in the intervening tissue between the pacing site and the VT circuit. It also shortens the refractory period at the stimulus site. Both these

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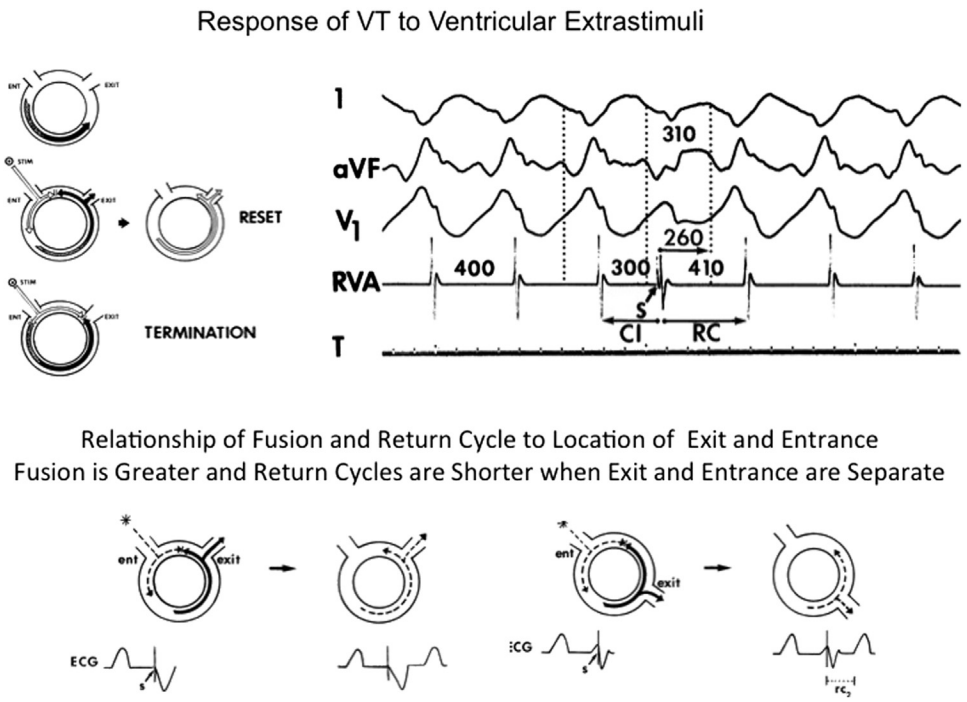

Figure 1 Mechanism of resetting of ventricular tachycardia (VT). On the left is a schema of the VT circuit that has anatomic dimensions with separate entrance and exit sites. The solid arrow is the wavefront of VT, and the time between the head and tail is the excitable gap. At long coupling intervals, stimuli enter the circuit and propagate orthodromically to advance the VT. Termination occurs when the stimulus falls on the refractory part of the wavefront. The upper right panel shows VT from an inferoposterior infarction being reset by a single extrastimulus delivered after the onset of the VT QRS (dotted lines). On the bottom is a schema to show that the further apart the entrance and exit sites, the shorter the return cycles (RCs) and greater the component of the VT-QRS to fusion. ECG = electrocardiogram; Ent = entrance; RVA = right ventricular apex.

allow later coupled extrastimuli to reset the tachycardia circuit (Figure 2). The use of double extrastimuli therefore allows one to achieve a greater degree of prematurity in entering the tachycardia circuit at comparable coupling intervals, which in turn can eliminate the refractoriness of the intervening tissue from interfering with the assessment of the postpacing intervals that follow the extrastimulus. This is critical because delay in intervening tissue refractoriness can affect the timing of the return cycle following a premature beat and lead to misinterpretation that the slowing is due to slower conduction in the orthodromic limb of the tachycardia circuit.

Fusion during the stimulated wavefront may be manifested on the ECG or it may only be seen in the left ventricular electrogram. Resetting with manifest fusion on the ECG in response to a single extrastimulus occurs in approximately 50% of reentrant VTs owing to prior infarction. Recognition of fusion is not always easy at any stimulated coupling interval. With progressively premature coupling intervals, a change in the QRS complex (QRS) morphology of the paced complex suggests that a fusion complex is present. One can also demonstrate that the stimulated QRS is a hybrid between the VT-QRS and a purely paced impulse. Another important observation that proves fusion is when a stimulated impulse resets the tachycardia despite being delivered after the onset of the tachycardia QRS (*prestimulus fusion*). In that case the stimulated impulse depolarizes the myocardium after parts of the ventricle are already depolarized by the VT. The determinants of surface fusion include the following: (1) the site of pacing relative to the VT circuit; if the stimulation site is distant from the VT circuit exit, fusion is more likely (as the stimulation site and the exit site capture different parts of the myocardium); (2) antidromic capture of the exit site and presystolic electrograms makes it impossible for fusion to occur because the

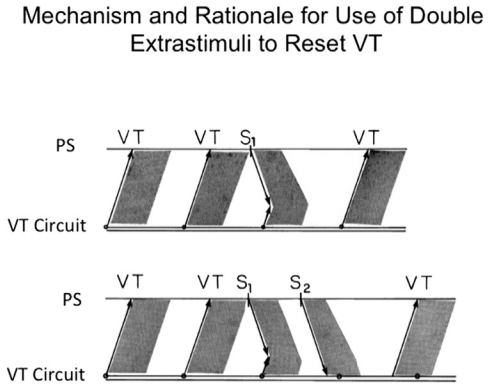

Figure 2 Mechanism and rationale for the use of double extrastimuli to reset ventricular tachycardia (VT). The ladder diagram shows that the refractory period of the intervening tissue between the tachycardia circuit and the pacing site (PS) limits the ability of a single extrastimulus to reach and reset the tachycardia. Double extrastimuli that shorten local refractoriness (shaded area) and alter the wavefront of activation in the intervening tissue allow even late extrastimuli to reset VT. S1 = the conditioning stimulus; S2 = the premature stimulus engaging the tachycardia circuit.