Transient Entrainment of Tachycardias

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About 15 years ago, Waldo and associates first described that a tachycardia could be transiently accelerated to a faster rate by overdrive pacing without fundamentally altering the underlying tachycardia, a phenomenon they called transient entrainment. That first description of transient entrainment applied to atrial flutter but was soon extended to ectopic atrial tachycardias, atrioventricular reentrant tachycardia, and ventricular tachycardia. Three basic criteria of transient entrainment were recognized: 1) acceleration of the tachycardia to the pacing rate with constant fusion of the ECG complex at constant pacing rates except for the last paced complex, which is unfused, 2) progressive fusion of the ECG complex with different degrees of constant fusion at different overdrive pacing rates, and 3) termination of the arrhythmia associated with sudden shortening of the conduction time from the pacing site to the captured ECG complex and reversion to the ECG morphology characteristic of pacing that site in the absence of an arrhythmia. Later, a fourth criterion was recognized, an electrogram equivalent of progressive fusion. Remarkably, and to his great credit, the explanations for the criteria of transient entrainment proposed by Waldo and his coworkers have been validated by direct experimental studies virtually without modification or refinement and stand as a testament to the power of his deductive reasoning. It is also fitting to recall that Waldo’s first recognition of the phenomenon of transient entrainment occurred in the course of providing clinical care to patients with tachycardia, a classic example of the bedside practice of medicine leading to a fundamental breakthrough in the understanding of disease mechanisms.

Waldo et al also proposed that the finding of transient entrainment of a tachycardia meant that the tachycardia mechanism was reentry with an excitable gap. Subsequently, it was recognized that the site of pacing relative to the reentry circuit influenced the ability to demonstrate transient entrainment, a property that could be used to map reentrant circuits. It was also recognized that transient entrainment could be used to probe the conduction properties of the components of reentrant circuits and to assess the mechanisms of drug action. While these applications of transient entrainment have been applied to reentrant circuits that probably utilize fixed anatomic pathways, the elegant article in this issue of Circulation by Waldecker and associates extends these principles to reentrant arrhythmias utilizing functional circuits.

In their article, Waldecker and coworkers have analyzed the effects of overdrive stimulation on a model of reentry involving functional reentrant circuits resulting from recent coronary artery ligation. They obtained high resolution activation maps of the functional reentrant ventricular tachycardia circuits that form in the thin rim of muscle surviving on the epicardial surface of the infarct and studied the effects of overdrive pacing during ventricular tachycardia. This model essentially eliminates the need to map in three dimensions so that complete activation maps can be obtained from the epicardial border zone. Most importantly, they have demonstrated that functional reentrant circuits can have a fully excitable gap and that they can be transiently entrained. Just as was deduced for arrhythmias caused by reentrant circuits utilizing fixed anatomic pathways, constant fusion of the QRS pacing at constant rates faster than the ventricular tachycardia (transient entrainment criterion 1) occurred when the stimulated impulses activated the ventricles concurrently with a previous stimulated impulse leaving the reentrant circuit at a different site. Progressive fusion (transient entrainment criterion 2) occurred when the stimulated wave front captured more of the ventricular mass, moving the line of collision closer to the site of exit from the reentrant circuit. Blocking of impulses with shortening of conduction times and termination of reentry (transient entrainment criterion 3) occurred when the cycle length of the pacing shortened to a critical value. Thus, the major tenets of transient entrainment have been directly validated for functional reentrant circuits.

The present study also sheds important insights into the mechanism whereby the first postpacing cycle length may be longer than the overdrive cycle length. The mapping showed that even though the first postpacing cycle length in the circuit was equal to the pacing cycle length, the exit from the reentrant circuit was delayed, and therefore the onset of the QRS was delayed. Thus, analysis of only the surface ECG complex can be misleading regarding events in the reentrant circuit.

It is striking how persistent and stable the lines of functional block were in this model of functional reentry, but that should not come as a surprise. After all, it is well known that human ventricular tachycardia, which must often be due to functional reentry, shows a striking propensity to repeatedly form the same reentrant circuit as demonstrated by the repeated development of the same QRS morphology of the ventricular tachycar-
ventricular tachycardia. Furthermore, studies have shown that human ventricular tachycardia related to prior myocardial infarction can usually be transiently entrained. The clinical challenge is to now develop the mapping tools that can localize the essential elements of the reentrant circuits of human ventricular tachycardia. Almost certainly, if we had better mapping arrays, we would be in a position to offer effective catheter ablation therapy to what is now the Achilles' heel of clinical electrophysiology—ventricular tachycardia.

References

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