Editorial Comment

Clinical Application of Rapid Ventricular Burst Pacing Versus Extrastimulation for Induction of Ventricular Tachycardia*

MASOOD AKHTAR, MD, FACC
Milwaukee, Wisconsin

Programmed electrical stimulation of the heart is increasingly employed for the management of patients with cardiac arrhythmias. Induction of ventricular tachycardia in the controlled environment of an electrophysiology laboratory has gained widespread acceptance for both diagnostic and therapeutic purposes in high risk groups of patients (1–7). Over the last few years, a variety of pacing protocols have been developed and utilized. With the passage of time and experience, many of these protocols have already been changed or modified. These evolutionary aspects of programmed electrical stimulation have undoubtedly been a source of confusion to many, particularly those not intimately familiar with cardiac electrophysiologic studies. Even among those who perform such studies routinely, the search for a more clinically relevant and standardized pacing protocol continues.

Standard Extrastimulus Technique

The most frequently employed programmed electrical stimulation protocol for the induction of ventricular tachycardia incorporates introduction of ventricular premature beats (V₂) during sinus or paced atrial rhythm and paced ventricular rhythms, usually from a site in the right ventricle. The coupling interval between the V₂ and the preceding ventricular beat (usually termed V₁) is progressively shortened until the effective refractory period of the ventricular myocardium is encountered. If the V₂ fails to initiate ventricular tachycardia, an additional premature beat (V₃) is introduced. During scanning with V₃, the V₁V₂ interval is kept constant at a given coupling interval and the V₂V₃ interval is progressively decreased to the point of ventricular effective refractory period. Similarly when more premature beats (such as V₄ and V₅) are added to the protocol, all of the preceding intervals are kept unaltered. This type of programmed electrical stimulation is generally referred to as the extrastimulus technique and is the most widely used method for tachycardia induction. The maximal number of extrastimuli used (that is, V₂, V₃, V₄ and so forth) as well as the duration of the paced atrial or ventricular basic cycle length (V₁V₁) varies among laboratories, but this aspect of the technique will not be detailed here.

Rapid Ventricular Burst Pacing Technique

The other frequently employed method of programmed electrical stimulation for ventricular tachycardia induction consists of a series of ventricular stimuli of short cycle lengths (usually <350 ms) introduced during the prevailing rhythm. When applied in short salvos this type of protocol is referred to as rapid ventricular burst pacing. The cycle length of such pacing is progressively decreased until: A) ventricular tachycardia is initiated, B) the effective refractory period of the ventricle is encountered with every other stimulus, or C) the shortest predetermined cycle length of burst pacing is achieved. The latter end point (C) is used in preference to B in some laboratories. Certain fundamental differences exist between the extrastimulus technique and rapid ventricular burst pacing as currently employed in clinical electrophysiology laboratories and are outlined as follows.

1) In contrast to the extrastimulus technique, the first beat of burst pacing generally falls randomly within the cardiac cycle and, therefore, the coupling interval between the last QRS complex of spontaneous rhythm and the first beat of burst pacing may vary each time the pacing is introduced. Depending on whether the first beat of burst pacing falls within the relative refractory period of the last spontaneous QRS complex or late in diastole, the elicited responses may be different and may not be comparable with extrastimulation. This problem of a variable relation between the last spontaneous QRS complex and the first beat of rapid ventricular burst pacing could be a source of poor reproducibility. However, it can be obviated by triggering the stimulation of the spontaneous QRS complex (an option available with some programmable digital stimulators).

2) During rapid ventricular burst pacing in most instances, the cycle lengths of all the beats are identical. Therefore, when the cycle length in rapid ventricular burst pacing is changed, it results in an alteration of intervals between all of the successive beats. This also is in contrast with the extrastimulus technique, in which the only altered interval is the one between the newly introduced premature beat and the preceding QRS complex; the events preceding that are not changed and remain comparable with the events previously scanned.

3) The duration of rapid ventricular burst pacing is highly variable among the various laboratories, ranging from 3 to...
12 beats and sometimes even more, which lends itself to less standardization as compared with extrastimulation.

Because of these differences, there could be different electrophysiologic sequelae of rapid ventricular burst pacing compared with extrastimulation. This in turn would cause difficulty in the interpretation of responses during programmed electrical stimulation with the two techniques and, consequently, could differentially influence clinical decision-making.

Experimental Data

In this issue of the Journal, El-Sherif et al. (8) address some of these important issues in an experimental animal model. Utilizing a 64 channel multiplexer in canine hearts 1 to 5 days after infarction, the authors display the analysis of isochronal maps of ventricular (epicardial) activation during initiation of ventricular reentry with programmed electrical stimulation. They demonstrate that both with rapid ventricular burst pacing and with extrastimulation, successive beats result in progressively larger arcs of functional block and slower circulating wave fronts. This allows reexcitation of the myocardial zones on the proximal side of the arc of block, thereby initiating reentry. However, for reentry to be manifested during rapid ventricular burst pacing, the stimulation had to be stopped with the beat producing the critical degree of conduction delay and block, otherwise the reentry would remain concealed by the subsequent paced beat. The latter could also rapidly advance to the sites of slow conduction and result in conduction block and interrupt circulating excitation, terminating the reentry process. Discontinuing stimulation after this critical beat during rapid ventricular burst pacing would not initiate reentry, but continuation of pacing would set up a new cycle of progressive delay and block with successive cycles. Because the critical number of burst pacing beats necessary to initiate reentry is difficult to anticipate and can vary among experiments or the cycle length of pacing within the same experiment, El-Sherif et al. (8) lean toward abandoning the random rapid ventricular burst pacing in favor of multiple extrastimuli for induction of ventricular tachycardia.

Limitations of the experimental data. There are several limitations and criticisms of the experimental data obtained by El-Sherif et al. (8). These are:

1) The more thoroughly studied human model of ventricular tachycardia is that of the chronic, recurrent, sustained variety. The experimental model used by El-Sherif et al. may not be exactly comparable with this clinical model. El-Sherif et al. did not elaborate on how often the sustained ventricular tachycardia was induced during the experiments. From the available data, including the figures, it appears that in many instances only short runs of nonsustained ventricular tachycardia may have been induced.

2) A monomorphic ventricular tachycardia was induced in only three experiments, thus providing limited data concerning the reproducibility of isochronal maps in this infarction model. Since the polymorphic ventricular tachycardia (induced more often in this study) can represent a nonspecific response, the in-depth analysis of isochronal maps in these settings, although of interest, may not be as meaningful or reproducible.

3) Only one cycle length of burst pacing was employed by El-Sherif et al., whereas during clinical electrophysiologic studies, several such cycle lengths are utilized until ventricular tachycardia is initiated or the shortest cycle length of pacing is achieved. It is conceivable that data obtained during a given cycle length of burst pacing may be more reproducible compared with those from another cycle length.

4) The possibility still exists that some form of clinical ventricular tachycardia may be induced only with burst pacing from either reentrant or nonreentrant but inducible mechanisms.

Clinical Applications and Considerations

For the reasons presented and also as pointed out by El-Sherif et al. (8), it does seem that the use of random rapid ventricular burst pacing would be less desirable than the extrastimulus technique in clinical settings as well. Random burst pacing is a less reproducible method for ventricular tachycardia induction and is difficult to standardize in terms of its duration and cycle length. From a clinical perspective, however, an ideal method of programmed electrical stimulation, which reproducibly induces only prognostically significant arrhythmias in all patients at risk of spontaneous life-threatening tachyarrhythmias but not in patients without such a risk, does not exist. An induced arrhythmia is considered clinically relevant if its inducibility correlates with previous as well as future occurrences and if its control predicts reliability of the tested therapy. The sensitivity of programmed electrical stimulation proportionately increases with the complexity of the pacing protocol but this invariably results in compromise of specificity (9–12). Recent data (9–14) have indicated that a significant number of patients without clinical ventricular tachyarrhythmias may develop repetitive ventricular responses, polymorphic nonsustained ventricular tachycardia or fibrillation with the extrastimulus technique. The incidence of polymorphic nonsustained ventricular tachycardia or fibrillation increases as the number of extrastimuli is increased, reaching 40% with three ventricular extrastimuli (11). Therefore, the clinical significance of induced polymorphic nonsustained ventricular tachycardia or fibrillation in patients without previously documented arrhythmias (such as syncope) is difficult to interpret regardless of whether the arrhythmia is initiated with rapid ventricular burst pacing or multiple premature extrastimuli. Even in patients with documented monomorphic sustained ventricular tachycardia, the induction of such arrhythmias may not represent a reliable end point and
could also lead to false negative predictions of drug efficacy (9–15).

On the other hand, less aggressive programmed electrical stimulation protocols have low sensitivity (16). For example, a single extrastimulus introduced during ventricular paced rhythms has a relatively poor yield in terms of ventricular tachycardia induction. At this time, it seems that two extrastimuli delivered during several paced ventricular drives offer a good balance between sensitivity and specificity. Monomorphic nonsustained ventricular tachycardia less frequently represents a nonspecific response to the extrastimulus technique (10,11). Whether this will hold true with rapid ventricular burst pacing as well is not entirely clear. Recent data (10) have also demonstrated that a sustained monomorphic ventricular tachycardia is rarely initiated in patients without documented or suspected ventricular tachycardia utilizing the extrastimulus technique even when multiple extrastimuli are used and it, therefore, represents a specific and clinically significant response. Similarly, when a sustained monomorphic ventricular tachycardia is initiated with rapid ventricular burst pacing in patients with prior documented ventricular tachycardia, it is considered clinically relevant especially if it represents the patient’s own tachycardia. However, it has not been documented whether rapid ventricular burst pacing can induce a sustained monomorphic ventricular tachycardia in patients without spontaneous arrhythmia of this type.

Aside from the differences between rapid ventricular burst pacing and extrastimulation elucidated by El-Sherif et al., addition of other variables such as another site of stimulation (outflow, left ventricle, for example), use of high current strength and drugs like isoproterenol significantly complicates interpretation of the data (17–20). Development of reliable animal models for ventricular arrhythmias and elaborate mapping techniques such as those utilized by El-Sherif et al. should help a great deal toward better understanding of the electrophysiology of ventricular tachycardia.

Meanwhile from the clinical laboratories, data are becoming available that more critically examine the sensitivity, specificity and reproducibility of programmed electrical stimulation protocols for patients with and without documented or suspected ventricular tachycardia. This type of information for rapid ventricular burst pacing may be difficult to obtain, however, because many of the laboratories have already abandoned the routine use of rapid ventricular burst pacing in favor of additional extrastimuli. However, the application of rapid ventricular burst pacing may sometimes be necessary in patients with previously documented ventricular tachycardia in whom the extrastimulus technique fails to reproduce the arrhythmia.

References


