Editorial Comment

Programmed Electrical Stimulation—Before When, How?*

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Programmed electrical stimulation has gained increasing acceptance as a tool for investigating patients with clinical tachyarrhythmias (1-4). Indeed this technique has even been applied to assess arrhythmic potential in patients who have not had but may be at increased risk for ventricular tachyarrhythmias (5-7). In this issue Mitchell et al. (8) and Duff et al. (9) address two issues of critical, but underemphasized, importance to the clinical application of programmed ventricular stimulation. The first issue is the selection of the most appropriate stimulation variables to enhance sensitivity of studies with minimal loss of specificity (8). The second is the identification of features affecting the reproducibility of the technique (9).

Procedural factors that may affect electrophysiologic variables. Such factors, which also affect the sensitivity and specificity of programmed ventricular stimulation, are analyzed in a well conceived and executed study by Mitchell et al. (8). These investigators started with the reasonable supposition that a measure of propagated impulses—the ventricular functional refractory period—is more important for the induction of clinical tachyarrhythmias than is the more easily obtained measure of nonpropagated impulses—the ventricular effective refractory period. The effects of five procedural factors—stimulus intensity, stimulus duration, number of extrastimuli, drive train cycle length and proximity of recording and stimulation sites—on the ventricular functional and ventricular effective refractory periods were analyzed. The latter three factors were found to have proportional effects on both refractory periods. However, increases in stimulus intensity and duration, beyond twice diastolic threshold and 2 ms, respectively, decreased the effective refractory period but did not decrease the functional refractory period. Because increases in stimulus intensity and duration did not affect the functional refractory period—the shortest propagated coupling interval—these changes would not be expected to enhance inducibility of clinical tachyarrhythmias. This was the conclusion of Mitchell et al., although that conclusion should be confirmed in patients with documented clinical tachyarrhythmias not inducible with stimuli of twice diastolic threshold and 2 ms duration.

Maneuvers that decrease the effective refractory period without changing the functional refractory period are necessarily associated with increased local tissue conduction delay, termed ΔL2 by Mitchell et al. An increase in local tissue conduction delay could be associated with the production of nonclinical tachyarrhythmias. We (10) recently reported that high stimulus intensity (10 mA) stimulation is associated with an unacceptably high incidence of ventricular fibrillation, not seen with twice diastolic threshold stimulation. This arrhythmia was always preceded by evidence of local tissue conduction delay and was often preceded by self-terminating polymorphous ventricular tachycardia. In contrast to Mitchell et al., we found more local conduction delay associated with S3 than with S2 with 10 mA stimulation but not with twice diastolic threshold stimulation. Our experience emphasizes the clinical importance of the observation that changes in arrhythmia inducibility over a several day period may be attributable in part to the use of in situ catheter placement and may be obviated by replacing the catheter daily. We have noted changes in inducibility with serial drug testing which can mimic drug exacerbation of arrhythmia (11) as well as drug effectiveness (12). Other features that may affect reproducibility as well as sensitivity and specificity of programmed ventricular stimulation may include metabolic and neurohumoral changes during serial electrophysiologic studies.

Reproducibility of programmed ventricular stimulation. A second important feature, that of reproducibility of programmed ventricular stimulation, is addressed in the companion article by Duff et al. (9). The authors point out that changes in arrhythmia inducibility over a several day period may be attributable in part to the use of in situ catheter placement and may be obviated by replacing the catheter daily. We have noted changes in inducibility with serial drug testing which can mimic drug exacerbation of arrhythmia (11) as well as drug effectiveness (12). Other features that may affect reproducibility as well as sensitivity and specificity of programmed ventricular stimulation may include metabolic and neurohumoral changes during serial electrophysiologic studies.

Importance of uniformity of stimulation protocols. Programmed ventricular stimulation has clearly gained an important role in assessing patients with clinical tachyarrhythmias. For optimal investigative and clinical usefulness a standardized stimulation protocol would be valuable. On the basis of their data, Mitchell et al. have suggested a reasonable protocol designed to optimize the relation between sensitivity and specificity. Those procedural factors that reduce the effective refractory period but do not affect the functional refractory period, stimulus intensity and duration, are kept at the lower values of twice diastolic thresh-
old and 2 ms. Changes in the functional refractory period are accomplished by sequentially changing drive cycle length, increasing the number of extrastimuli and changing the site of stimulation. Fortunately, these recommendations are consonant with the practices of most, but not all, electrophysiology laboratories. Mitchell et al. are to be congratulated for beginning to provide a rational basis for adoption of uniform stimulation protocols. Adoption of these recommendations will facilitate evaluation of other stimulation protocol variables such as the number of extrastimuli and the sites of stimulation. In addition, such uniformity of stimulation protocols will improve our ability to compare and contrast reports using programmed ventricular stimulation in different patient groups including patients in the post-myocardial infarction period.

References


