ONE OF THE MOST deeply ingrained tenets of contemporary medical practice is the prevention or abolition of ventricular tachycardia. Medical personnel throughout the world have been trained to approach this arrhythmia with both awe and alarm for the patient's safety. The necessity for its prompt eradication has been unanimously proclaimed from the highest cardiologic pulpits. The introduction of laboratory techniques (originally described by Wellens et al.) to provoke ventricular tachycardia in man appears at first appraisal to violate fundamental medical tenets. In this issue of Circulation, the reports of Horowitz et al. and Mason and Winkle describe how these techniques have been used to evaluate the drug treatment regimens for patients with recurrent ventricular tachycardia (RVT). These studies are a logical sequence to the important observations of Wu et al. in the use of serial electrophysiologic testing in patients with recurrent supraventricular tachycardia.

The lethal potential of RVT is well appreciated, and its treatment is often exasperating both to patient and physician. The sporadic occurrence of this arrhythmia, for example, seriously limits the use of ambulatory monitoring to guide management. Moreover, judging from my own experience and numerous articles submitted to Circulation, there appears to be a dramatic increase in the incidence of RVT, especially in patients with ischemic heart disease. An interesting conjecture is that the advent of coronary care units, mobile coronary flying squads, and perhaps coronary bypass surgery have increased the survival rate of a subset of patients afflicted with RVT/fibrillation. The majority of patients with RVT referred to our laboratory are those successfully resuscitated after cardiac arrest.

Formulation and testing within hours or days of a drug regimen that will effect long-term control of tachycardia for these frequently desperately ill patients is an important new approach to the problem. However, as with any procedure, the clinician must carefully weigh the benefit/risk ratio before subjecting patients to this type of testing. Induction of life-threatening cardiac arrhythmias can never be taken lightly. In the experience of Mason and Winkle, for example, 52% of the patients required emergency direct current cardioversion because of hemodynamic instability.

In addition, the basic technique involves induction of the ventricular tachycardia. Horowitz et al. reported successful induction of tachycardia in all patients in their series, while Mason and Winkle, using similar techniques, had an 83% success rate. The latter experience appears to be more representative of reports from other laboratories and in accord with previous reports from the same group. The discrepancy may be related in part to the site of ventricular stimulation. We recently studied a patient with RVT in whom right ventricular overdrive pacing, including insertion of double extrastimuli, failed to induce the tachycardia, but tachycardia was induced when the same pacing protocol was applied during endocardial left ventricular pacing. Obviously, repeated left ventricular pacing challenge is contraindicated. Moreover, the proposed pacing challenge tests the ability of a given drug (or combination of drugs) to suppress tachycardia in response to stimulated ventricular depolarizations. A drug that is effective in abolishing trigger premature ventricular depolarizations may prove to be clinically effective, yet fail to prevent tachycardia induction in the laboratory. Nevertheless, it is reassuring that in both studies reported in this issue, long-term suppression of clinical ventricular tachycardia occurred despite the presence of persistent premature ventricular depolarizations.

Another potential limitation of this technique is demonstrated by the fact that the vast majority (90% or more) of patients in both series reported in this issue had organic cardiac disease. The role of laboratory testing in predicting long-term suppressibility of RVT in the presence of progressive cardiac disease (i.e., worsening ischemia or heart failure) is unknown.

The issue of repeated control stimulation studies after each drug trial is an important practical question, because this approach prolongs the total length of the trial, and hence patient discomfort and risk. In the carefully designed protocol by Horowitz et al., tachycardias were always reproduced between drug trials, thus confirming the reproducibility of this technique. Now that the method has been validated, it seems prudent and expeditious to avoid testing between drug trials in the usual clinical setting.

In spite of the limitations alluded to and in spite of the highly selected patient groups studied, both reports document an exciting and important advance in our approach to patients with RVT. My assessment of the risk/benefit ratio of this technique leads to the following conclusions:

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1) For patients who present initially with symptomatic ventricular tachycardia, it is reasonable to initiate therapy with a so-called type I antiarrhythmic agent (procainamide, quinidine or disopyramide), based on the results of the studies reported by Horowitz et al. and Mason and Winkle. The technique of electrode catheter induction should be considered if the treated patient subsequently suffers ventricular tachycardia (in an effort to define an effective (higher) dose for a given drug or to establish effective drug combinations). In addition, such studies should be considered in patients with RVT and aborted sudden death.

2) No deaths or serious sequelae have been reported from the many centers throughout the world4, 7-10 in which techniques for induction of ventricular tachycardia are used, confirming the care and diligence of the investigators. Nevertheless, the proposed technique has serious potential risks, and should not be undertaken in a setting in which only occasional intracardiac electrophysiologic studies are performed. These studies should be performed under the supervision of experienced cardiologists in either electrophysiologic laboratories or in coronary care units staffed with persons who are both knowledgeable and experienced in the management of cardiac emergencies. As suggested by Mason and Winkle, it is desirable to have continuous direct systemic arterial pressure monitoring throughout the test procedure.

3) The approach is not clinically indicated for patients with frequent premature ventricular depolarizations or short, unsustained bouts of asymptomatic ventricular tachycardia. The use of this technique in patients with ventricular arrhythmias of lesser magnitude to define a subset of patients at high risk of subsequent development of malignant arrhythmias is a vital issue requiring further research. Similarly, the clinical value of this approach for patients with ventricular tachycardia that are exercise-induced or related to mitral valve prolapse, prolonged repolarization syndrome, or primary ventricular fibrillation has not been defined adequately.

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