Termination of Ventricular Tachycardia with Ventricular Stimulation: Salutary Effect of Increased Current Strength

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SUMMARY The effect of increased stimulus current strength on the right ventricular effective refractory period during sustained ventricular tachycardia and on the ability of single premature right ventricular stimuli to terminate ventricular tachycardia was evaluated. Studies were performed during 53 episodes of sustained ventricular tachycardia in 25 patients. Forty-four of 53 episodes were slowed by pharmacologic therapy. Current intensities of twice diastolic threshold, 5 mA and 10 mA were used. Increasing current from twice diastolic threshold (1.52 ± 0.66 mA) to 5 mA (32 episodes) shortened the mean ventricular effective refractory period from 213 ± 50 to 188 ± 43 msec (p < 0.001); and from twice diastolic threshold (1.53 ± 0.60 mA) to 10 mA (42 episodes), from 206 ± 55 to 176 ± 50 msec (p < 0.001). There was a direct correlation between cycle length of ventricular tachycardia and the ventricular effective refractory period measured at twice diastolic threshold (r = 0.77, p < 0.001). However, the cycle length of ventricular tachycardia and the amount of shortening of ventricular effective refractory period as current was increased were not significantly correlated (r = 0.40). Eleven episodes of ventricular tachycardia (21%) that could not be terminated by ventricular stimulation at twice diastolic threshold were terminated when increased current strength was used. The ability to terminate ventricular tachycardia was associated with a decrease in ventricular effective refractory period and long tachycardia cycle length. The mean cycle length of the 11 episodes terminated was 455 ± 50 msec, compared with 381 ± 63 msec in the 42 episodes not terminated at increased current.

SINGLE VENTRICULAR STIMULI can be used to terminate episodes of reentrant sustained ventricular tachycardia. Successful termination requires that an impulse penetrate a reentrant circuit and interrupt the circulating wave front. Refractoriness of the intervening tissue can limit the ability of an impulse to reach the reentrant circuit at the critical time necessary to terminate the rhythm. Thus, any manipulation that could shorten refractoriness of the intervening tissue and permit an impulse to reach and penetrate the circuit earlier might allow an increased success rate for termination of ventricular tachycardia by single ventricular stimuli.

Increased stimulus current strength has been used to shorten ventricular refractoriness in animal and human studies. Studies during atrial and ventricular pacing but not during sustained ventricular tachycardia. Studies using single ventricular stimuli to terminate ventricular tachycardia used current strengths, when specified, of twice diastolic threshold. Therefore, we studied the effect of increased current strength on right ventricular refractoriness during ventricular tachycardia, and the effect of shortening the right ventricular effective refractory period by increased stimulus current in facilitating termination of ventricular tachycardia by single right ventricular extrastimuli.

Patients and Methods

The patient population included 23 men and two women, ages 25–74 years. Twenty-one patients had atherosclerotic heart disease, one patient had mitral valve prolapse, one cardiomyopathy, one right ventricular dysplasia and one had no apparent heart disease. Fifty-three episodes of sustained ventricular tachycardia were induced with premature ventricular stimulation in these 25 patients by methods previously described. Forty-four of the 53 episodes of sustained ventricular tachycardia were induced while the patients were taking one or a combination of antiarrhythmic medications, including propranolol, lidocaine, quinidine, phenytoin, disopyramide, and amiodarone. Single ventricular stimuli at twice threshold failed to terminate all 53 episodes of ventricular tachycardia.

All patients gave written informed consent. They underwent electrophysiologic study using previously published techniques in a nonsedated, postabsorbptive state. Stimulation was performed from the distal and recording from the proximal pair of electrodes on a quadripolar catheter. Bipolar stimulation (distal electrode negative) was performed using an optically isolated, constant current source and a programmable stimulator (Bloom Associates, Ltd., Inc.). Rectangular pulses 1 msec in duration and current strengths from twice diastolic threshold (0.35–4 mA) to 10.0 mA were used.

We have found that there is no significant difference between the ventricular pacing threshold measured during sinus rhythm and that during ven-
tricular tachycardia (unpublished observations). Thus, threshold currents were measured before the induction of ventricular tachycardia. Ventricular tachycardia was diagnosed using standard criteria. After the induction of sustained ventricular tachycardia, single premature stimuli at twice diastolic threshold were introduced starting in late diastole and then at progressively shorter coupling intervals. The stimuli were synchronized to the right ventricular electrogram and were moved progressively earlier in diastole by 10-msec decrements until the stimuli failed to capture the ventricle (ventricular effective refractory period). In the first 17 episodes studied, the current was increased directly to 10 mA and the sequence repeated. In the next 36 episodes, the effects of current of 5 mA and 10 mA were studied.

The ventricular deflection on the right ventricular electrogram during tachycardia is referred to as $V_1$ and that resulting from the premature stimulus (S) as $V_2$. The relative refractory period was defined as the longest $V_1S$ that resulted in a prolongation of $SV_2$ compared to that $SV_2$ resulting from a stimulus introduced in late diastole. The local functional refractory period is determined by the relative refractory period and is defined as the shortest $V_1V_2$ at any $V_1S$.

If the ventricular tachycardia was not terminated by single premature ventricular stimuli at any current strength, double or triple premature stimuli were introduced or bursts of rapid ventricular pacing with current at twice diastolic threshold were used. If pacing failed or acceleration of the tachycardia causing hemodynamic compromise ensued, DC cardioversion was used.

The $t$ test for paired data and linear regression analysis were used for analysis of the data.

Results

Effect of Current Strength on the Ventricular Effective Refractory Period

Measurements of the right ventricular effective refractory period were performed during 53 episodes of sustained ventricular tachycardia in 25 patients. The ventricular pacing threshold ranged from 0.35–2.0 mA (mean 0.76 ± 0.33 mA). The ventricular effective refractory period determined at twice threshold ranged from 150–310 msec (mean 227 ± 42 msec).

In 32 episodes of ventricular tachycardia, an increase in current strength from twice diastolic threshold (1.52 ± 0.66 mA) to 5 mA resulted in a decrease in the mean ventricular effective refractory period, from 213 ± 50 msec to 188 ± 43 msec ($p < 0.001$). In 42 episodes of ventricular tachycardia increasing the current from twice diastolic threshold (1.53 ± 0.60 mA) to 10 mA resulted in a decrease in the mean ventricular effective refractory period, from 206 ± 55 msec to 176 ± 50 msec ($p < 0.001$). In 27 episodes of ventricular tachycardia, increasing the current strength from 5 to 10 mA resulted in a decrease in the mean ventricular effective refractory period, from 185 ± 47 msec to 175 ± 45 msec ($p < 0.001$) (fig. 1). The ventricular effective refractory period was shortened by 10–50 msec in most episodes of ventricular tachycardia when the current strength was increased from twice threshold to 10 mA (fig. 2).

Relationship of Cycle Length of Ventricular Tachycardia to Ventricular Effective Refractory Period Measurements at Different Current Strengths

The ventricular effective refractory period measured at twice diastolic threshold varied directly with the cycle length of ventricular tachycardia, i.e., the ventricular effective refractory period during ventricular tachycardia was less in episodes of ventricular tachycardia with shorter cycle lengths (fig. 3) ($r = 0.77, p < 0.001$). However, the amount of shortening in ventricular effective refractory period produced by an increase in current was not related to ventricular tachycardia cycle length ($r = 0.40$).

Termination of Ventricular Tachycardia at Increased Current Strength

Eleven episodes of ventricular tachycardia induced in seven patients were terminated by single premature ventricular stimuli at increased current strength (5 or 10 mA) when stimulation at twice diastolic threshold was unsuccessful. Increasing current strength from twice diastolic threshold (1.85 ± 1.40 mA) to 5 mA resulted in the termination of four episodes of ventricular tachycardia in three patients. An increase in current strength from twice diastolic threshold (1.83 ± 1.49 mA) to 10 mA resulted in termination of seven episodes of ventricular tachycardia in four patients. In two episodes of ventricular tachycardia that were terminated with a stimulus current strength of 10 mA, stimulation was not performed at 5 mA; therefore, the effect of the lower current strength (5 mA) on ventricular tachycardia termination could not be assessed. The ability of ventricular stimulation at higher current strengths to terminate ventricular tachycardia was, in
The shortening in VERP before termination of VT associated with increased current (5 or 10 mA) is plotted. As much as 70 msec of shortening in the VERP did not result in termination in some episodes of VT, whereas in others only 10 msec of shortening preceded termination.

The effect of increasing current strength on termination of sustained ventricular tachycardia with a cycle length of 430 msec is shown in figure 4. The $V_1V_2$ current is increased at the ventricular apex. (A) During sustained ventricular tachycardia a premature stimulus ($S$) is introduced in the $V_1V_2$ 300 msec after the preceding beat ($V_1$) and captures the ventricle ($V_2$). (B) The stimulus is moved earlier in diastole until it fails to capture the ventricle at 290 msec (ventricular effective refractory period). (C) The current strength is increased to 5 mA, and the ventricle is captured from 290 to 250 msec. (D) The stimulus fails to capture the ventricle at a coupling interval of 240 msec (ventricular effective refractory period at 5 mA). (E) The stimulating current strength is increased to 10 mA, and the ventricle is captured at a coupling interval of 240 msec, resulting in termination of ventricular tachycardia. The $V_1V_2$ is shortened from 370 msec at twice threshold to 300 msec at 10 mA along with the ventricular effective refractory period.

is shortened along with the effective refractory period. Another example is shown in figure 5.

The cycle length of ventricular tachycardia had a profound effect on the ability to terminate the arrhythmia. The mean cycle length in the 11 episodes of ventricular tachycardia that could be terminated by increasing current strength was longer (455 ± 50 msec) than the mean cycle length (381 ± 63 msec) of the 42 episodes of tachycardias not terminated at any current strength (fig. 3).
performed on intact mammalian hearts\textsuperscript{8, 10, 12} and isolated heart muscle\textsuperscript{77} has revealed that at a low current, small changes in current cause large changes in refractory periods. In contrast, at high current strengths, similar changes in current cause minimal changes in the refractory period. This study has demonstrated qualitatively similar effects of current on the measurement of the ventricular effective refractory period during sustained ventricular tachycardia. An increase in current strength from twice diastolic threshold to 5 and 10 mA resulted in a significant shortening in the ventricular effective refractory period. Furthermore, the decrease in effective refractory period when increasing the current from twice diastolic threshold to 5 mA was greater than that when the current was increased from 5 to 10 mA. Time limitations prevented us from performing full strength-interval curves during sustained ventricular tachycardia, which may have confirmed the similarity to those previously reported.

It has also been shown in animal\textsuperscript{14, 19} and human\textsuperscript{14, 19} studies that the ventricular effective refractory period is directly related to the cycle length at which it is measured. Although we could not perform refractory period measurements at different cycle lengths in the same patient, we did note a correlation ($r = 0.77$) between the cycle length of ventricular tachycardia and the effective refractory period measured at twice diastolic threshold.

Our observations were made in patients on antiarrhythmic medications. This was necessary to slow the tachycardia cycle length so that the patients could tolerate the arrhythmia long enough to allow study. Animal studies have not shown significant alterations in the strength-interval relationship due to procainamide or quinidine,\textsuperscript{20} and similar results for procainamide were found in humans.\textsuperscript{19} Nevertheless, in the absence of drugs, the effect of increasing current on the ventricular effective refractory period during ventricular tachycardia and the relation of ventricular tachycardia cycle length to the ventricular effective refractory period may be different from what we observed.

We measured the ventricular effective refractory period in the right ventricle (presumably distant from the tachycardia circuit in most instances). Because the premature stimulus was not moved later in diastole with each increase in current strength, we could not evaluate the right ventricular relative and functional refractory periods. However, we did observe a decrease in $V_{1}V_{2}$ parallel to the decrease in effective refractory period in the 11 episodes of tachycardia terminated with increased current (figs. 4 and 5). This observation is highly suggestive of a concomitant decrease in the right ventricular relative and functional refractory periods.

The amount of shortening in the right ventricular effective refractory period in those episodes not terminated (0–70 msec) by increased current was similar to the decrease observed before termination (0–60 msec) in the episodes interrupted with increased current (fig. 2). Thus, the amount of shortening in the ventricular effective refractory period per se resulting
from increased current was not predictive of termination.

In the 11 episodes of ventricular tachycardia terminated with increased current strength, the amount of shortening in the ventricular effective refractory period before termination was inversely related to the tachycardia cycle length. Thus, more shortening of the ventricular effective refractory period was required to terminate faster, as opposed to slower, episodes of ventricular tachycardia. Recognizing the limitations of studying a small patient population, this finding is consistent with a smaller window in faster tachycardias. Since those episodes of ventricular tachycardia with longer cycle lengths did not demonstrate more shortening in ventricular effective refractory period with increased current than episodes with shorter cycle lengths, the ability to terminate slower ventricular tachycardia was probably due to a larger window.

Presumably, in episodes that were not terminated, either the window was too small to be entered with stimuli of any current or penetration was limited by the characteristics of the intervening tissue, i.e., the impulse never reached the circuit earlier at increased current because of a conduction delay in the intervening tissue. We did not measure the functional refractory period of tissue adjacent to the reentrant circuit; thus, we cannot comment on the relative contribution of each measurement.

Clinical Implications

Ventricular pacing has been used to terminate ventricular tachycardia in the electrophysiology laboratory,1-4,21 in the coronary care unit21 and chronically using implanted ventricular pacemakers.1,7,22-24 Single premature ventricular stimulation is widely accepted as the pacing method least likely to result in detrimental arrhythmias, i.e., acceleration of ventricular tachycardia or ventricular fibrillation. Most implanted ventricular pacemakers deliver impulses at 5-10 mA. Therefore, the combination of antiarrhythmic medications that slow the rate of ventricular tachycardia plus testing higher current strengths that can increase the success rate of single ventricular stimuli to terminate ventricular tachycardia may make “underdrive pacing” a feasible treatment in more patients.1,7

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References

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