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Immediate Reproducibility of Electrically Induced Sustained Monomorphic Ventricular Tachycardia Before and During Antiarrhythmic Therapy

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The immediate reproducibility of sustained ventricular tachycardia induction was evaluated prospectively during 106 studies performed in 53 patients with clinical sustained monomorphic ventricular tachycardia. Programmed electrical stimulation was performed twice, using the same protocol during 53 drug-free studies and 53 subsequent studies on antiarrhythmic therapy.

Sustained monomorphic ventricular tachycardia was reproduced in 104 (98%) of the 106 studies. There was no significant difference in the incidence of reproducible tachycardia in the drug-free state compared with that observed during treatment with different classes of antiarrhythmic drugs. An increase in the number of extrastimuli was required to reinitiate the tachycardia in 9 (11%) of 83 studies in which single or double extrastimuli were initially required to induce the tachycardia. In 39 (37%) of 104 studies with reproducible tachycardia induction, the two tachycardias significantly differed in electrocardiographic (ECG) configuration and cycle length.

These observations suggest that the overall reproducibility of ventricular tachycardia induction is sufficiently high to provide a reliable marker for evaluating the efficacy of therapeutic interventions. However, specific tachycardia characteristics such as cycle length and ECG configuration are more variable even within the same study and may be less useful in assessing the effects of subsequent interventions.

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Programmed cardiac stimulation is widely used in the management of sustained ventricular tachycardia. Several studies (1–3) demonstrating the value of programmed cardiac stimulation in predicting the long-term efficacy of antiarrhythmic drug therapy have used the intravenous as well as oral route of drug administration. The immediate reproducibility of tachycardia induction by programmed stimulation is critical, especially in laboratories using acute intravenous drug testing immediately after baseline programmed stimulation to predict the future efficacy of oral drug therapy (4–6). Furthermore, because an antiarrhythmic drug may be considered partially effective and therefore clinically useful if it modifies ventricular tachycardia induction or slows the induced tachycardia rate without completely suppressing it (7–9), the reproducibility of induction modes and induced tachycardia characteristics such as cycle length before and after changes in drug therapy are clinically important. To date, few studies (10,11) have critically addressed the issue of immediate reproducibility of tachycardia induction and have done so only in the drug-free state.

The purpose of this prospective study was to: 1) determine the overall immediate reproducibility of sustained monomorphic ventricular tachycardia induced by programmed cardiac stimulation in patients presenting with documented clinical sustained ventricular tachycardia both in the absence and the presence of antiarrhythmic drug therapy; and 2) examine the reproducibility of specific tachycardia characteristics such as mode of induction, cycle length and surface electrocardiographic (ECG) pattern.

Methods

Study patients. A total of 106 electrophysiologic studies were conducted in 53 patients. There were 43 men and 10 women with a mean age of 60 ± 12 years. All patients underwent cardiac catheterization and coronary angiography. Coronary artery disease was present in 44 patients, dilated congestive cardiomyopathy in 3 and other underlying heart disease in 6. Of the 44 patients with coronary artery

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disease, there were 19 patients with transmural anterior wall infarction (11 with left ventricular aneurysm), 15 with transmural inferior wall infarction (2 with aneurysm), 9 with both anterior and inferior infarction (1 with an inferobasal aneurysm) and 1 with no transmural infarction. All patients had wall motion abnormalities, and the mean left ventricular ejection fraction was 31 ± 14%. The clinical presentation was sustained monomorphic ventricular tachycardia documented in all 53 patients.

Study design. One hundred six electrophysiologic studies were prospectively carried out in 53 consecutive patients who presented with spontaneous sustained monomorphic ventricular tachycardia documented by a 12 lead ECG and who met the following additional criteria: 1) sustained monomorphic ventricular tachycardia was induced by programmed cardiac stimulation in the absence of antiarrhythmic drug therapy in each patient; and 2) the induced ventricular tachycardia was terminated by ventricular pacing and did not require cardioversion by direct current countershock during that particular study. Patients in whom programmed cardiac stimulation resulted in ventricular fibrillation or ventricular tachycardia requiring direct current countershock for termination were excluded from this study to prevent repeated cardioversions for safety and humane considerations and to avoid possible postcardioversion electrophysiologic alterations that might interfere with immediate reproducibility of tachycardia induction. Patients with ventricular arrhythmias occurring in the setting of acute myocardial infarction or metabolic abnormalities were also excluded.

Patients who had stopped an antiarrhythmic drug for more than five half-lives, or for at least 3 months in those previously taking amiodarone, were considered free of drug and underwent a programmed cardiac stimulation protocol. Similar studies were subsequently performed in the presence of antiarrhythmic drugs in 33 of these 53 patients who went on to have follow-up studies during antiarrhythmic drug therapy. A total of 53 drug trials were performed in 33 patients. The antiarrhythmic agent was procainamide during 16 trials, amiodarone during 14 trials, quinidine during 4 trials and disopyramide in 1. The remaining 18 trials were performed during treatment with a combination of drugs (procainamide and tocainide in 6, procainamide and mexiletine in 2, quinidine and mexiletine in 7, quinidine and tocainide in 2 and disopyramide and mexiletine in 1). Serial drug studies were not carried out in the remaining 20 patients for various clinical reasons, but mostly because of decisions in favor of nonpharmacologic methods of treatment. Eighteen of the 33 patients had 1 follow-up study on an antiarrhythmic drug regimen, 10 patients had 2 studies on 2 different antiarrhythmic drug regimens and 5 patients had 3 studies on 3 distinct regimens, with a total of 53 studies during antiarrhythmic drug therapy in 33 patients. With amiodarone therapy, programmed cardiac stimulation was performed after a period of 10 to 14 days of drug loading, with doses ranging from 1,200 to 1,800 mg/day.

Programmed stimulation protocol. The electrophysiologic studies were carried out in compliance with the guidelines of the Human Studies Committee at our institution. Patients were studied in the postabsorptive state after informed consent had been obtained. Multipolar electrode catheters were positioned under fluoroscopic guidance in the high right atrium, right ventricular apex and across the tricuspid valve anulus. During a serial drug study, a bipolar electrode catheter was positioned at the same site as the site of prior tachycardia induction. Programmed electrical stimulation using up to three extrastimuli was performed with a programmable stimulator (Medtronic model 5328) using rectangular stimuli with a pulse width of 2 ms at five times diastolic threshold. Catheter position was readjusted until the diastolic threshold was <1 mA before any programmed cardiac stimulation. Diastolic threshold was defined at the beginning of each study and redefined after the induction of the first tachycardia before repeat programmed cardiac stimulation.

Starting at the right ventricular apex, one, two and three extrastimuli were introduced at two drive cycle lengths (600 and 400 ms), with coupling intervals starting at an S_1S_2 interval of 20 ms + the effective refractory period and of 50 ms + the refractory period for S_2S_3 and S_3S_4 and decremented by 10 ms at a time until ventricular refractoriness was encountered for all extrastimuli. The end point of the stimulation protocol was induction of sustained ventricular tachycardia or completion of the stimulation protocol, including the use of a third extrastimulus (S_4) for all studies. If sustained ventricular tachycardia was not initiated from the right ventricular apex, the same stimulation protocol was repeated from the right ventricular outflow tract. Programmed left ventricular stimulation was not used in this study.

Immediately after the electrical induction of sustained monomorphic ventricular tachycardia, a 12 lead ECG was recorded in every case and ventricular pacing was tried to terminate the tachycardia. If cardioversion was not required, reproducibility of tachycardia induction was tested in the same setting by starting the identical stimulation protocol from the same site of stimulation without any change in catheter position after a rest period of approximately 10 min, during which any symptoms that might be present were allowed to resolve and vital signs allowed to return to preinduction values. As with the first stimulation protocol, the coupling intervals were decremented and the number of extrastimuli increased until either sustained ventricular tachycardia was reinitiated or the protocol using a maximum of three ventricular extrastimuli was completed.

Definitions. Sustained monomorphic ventricular tachycardia was defined as ventricular tachycardia with no beat to beat variation in rate and ECG configuration (QRS width and mean frontal and horizontal axes), lasting ≥100 beats or requiring pacing for termination. Nonsustained ventricular tachycardia was defined as ventricular tachycardia manifesting ≥3 and <100 beats, reverting to sinus rhythm spontane-

Table 1. Rate of Reproducibility of Ventricular Tachycardia (VT) Induction and Incidence of Similarity in Reproduced Tachycardia in the Drug-Free State and During Antiarrhythmic Drug Therapy (AAD)

AAD Therapy	Overall Reproducibility of VT Induction	Reproduced VT Similar	Reproduced VT Different
No AAD	51/53	32/51	19/51
(n = 53)	(96%)	(63%)	(37%)
P or Q or D	21/21	13/21	8/21
(n = 21)	(100%)	(62%)	(38%)
Combination	18/18	13/18	5/18
(P or Q or D + M or T)	(100%)	(72%)	(28%)
(n = 18)			
Amiodarone	14/14	7/14	7/14
(n = 14)	(100%)	(50%)	(50%)
Total	104/106	65/104	39/104
(n = 106)	(98%)	(63%)	(37%)

D = disopyramide; M = mexiletine; P = procainamide; Q = quinidine; T = tocainide.

Table 2. Differences in Rate and Morphology Between the First and Second Induced Ventricular Tachycardia (n = 104)

Similar rate and morphology	65 (63%)
Different morphology only	19 (18%)
Different rate and morphology	13 (13%)
Different rate only	7 (7%)

ously. Sustained ventricular tachycardia induction was considered reproducible if after the induction of the first sustained ventricular tachycardia, a second sustained ventricular tachycardia could be reinitiated using a maximum of three ventricular extrastimuli from the same site of stimulation, regardless of the number of extrastimuli required for the first induction and regardless of the similarities or differences in rate and surface ECG pattern between the two sustained ventricular tachycardias.

The rates of the two induced ventricular tachycardias in the same patient were considered similar if their cycle lengths differed by less than 40 ms. The morphologic similarity between different tachycardias was based on ECG criteria and not on activation sequence mapping. Two ventricular tachycardias were considered similar in ECG configuration if both their mean frontal and mean horizontal ECG axes differed by less than 45°, criteria previously used in our laboratory (12). Tachycardias manifesting both similar rates and similar ECG configuration were classified as similar. Otherwise they were considered to be different.

Statistical analysis. Fisher's exact test and McNemar's test for paired sample nominal-scale data (13) were used to compare reproducibility of tachycardia induction among different groups. The latter is a test of independence or, in this case, a test of whether the patients who have reproducible results during one study but not the other are evenly divided between the groups being compared. McNemar's test was also used to compare the proportions of similar and different ventricular tachycardias among various groups.

Results

Overall reproducibility (Table 1). In the 53 patients, sustained monomorphic ventricular tachycardia was induced for the second time by programmed cardiac stimulation after the first induction in 104 (98%) of the 106 studies. The site of stimulation for both the first and the second induction was the right ventricular apex in 92 studies and the right ventricular outflow tract in 12. In 2 (2%) of 106 studies, ventricular tachycardia could not be reinitiated by programmed cardiac stimulation. These two nonreproducible induced ventricular tachycardias were observed in two different patients who underwent their studies in a drug-free state. Thus, using our general definition of overall reproducibility for ventricular tachycardia induction (see Methods), the overall reproducibility was 98%. In the remaining two studies, the initial tachycardia was initiated by three extrastimuli and could not be reproduced using three extrastimuli during the second attempt.

In 53 studies performed in 33 patients off antiarrhythmic drug therapy, sustained ventricular tachycardia induction was reproducible in 51 (96%) (Table 1). The overall reproducibility of tachycardia induction for the 53 studies performed during antiarrhythmic drug therapy was 100%, regardless of the specific antiarrhythmic drug regimen used. Using either Fisher's exact test on the whole samples (unmatched) or McNemar's test for paired sample testing, there was no significant difference in reproducibility of tachycardia induction among the different categories listed in Table 1.

Reproducibility of Ventricular Tachycardia Characteristics (Tables 1 and 2)

Rate and ECG configuration. The mean cycle length of all induced ventricular tachycardias was 370 ± 85 ms (range 200 to 600). The first and second induced ventricular tachycardia analyzed in 104 studies of reproducible induction were similar in both rate and ECG configuration in 65 studies (Table 1), resulting in a 63% incidence of similarity for reproduced sustained monomorphic ventricular tachycardia. For the remaining 39 studies (37%), the first and the second induced tachycardias were different because of a difference in ECG configuration (18%), rate (7%) or both (13%) (Table 2). In the 20 studies in which the rate of the first and second ventricular tachycardia was different (Table 2), the mean difference in cycle length was 103 ± 74 ms.

Hemodynamic tolerance. This was a function of tachycardia cycle length; there was no difference in hemodynamic tolerance between the first and second tachycardia when similar tachycardias were induced. Ventricular pacing was uniformly successful in terminating ventricular tachycardia after the second induction of ventricular tachycardia, as with

Table 3. Number of Extrastimuli (ES) Used to Initiate Ventricular Tachycardia (VT) During the First and Second Programmed Electrical Stimulation (PES)

		VT During 2nd PES (n = 104)				
	VT Initiated With	1 PES (n = 27)	2 PES (n = 55)	3 PES (n = 24)	Overall Reproducibility	Reinduced by Same No. of PES as First
VT during 1st PES (n = 109)	1 PES (n = 33)	27	6	—	33/33 (100%)	27/33 (82%)
	2 PES (n = 50)	—	47	3	50/50 (100%)	47/50 (94%)
	3 PES (n = 23)	—	—	21	21/23 (91%)	21/23 (91%)
	Total (n = 106)	—	—	—	104/106 (98%)	95/104 (91%)

the first when a similar ventricular tachycardia was reinduced. Cardioversion with countershock was required to terminate the second ventricular tachycardia in 10 studies.

Effect of drug on rate and ECG configuration (Table 1). The incidence of similar and different ventricular tachycardias during the first and second induction in the same patient was separately determined for the drug-free state and different modes of antiarrhythmic drug therapy. The incidence of similar ventricular tachycardias was 63% in the drug-free group and nearly identical (62%) for all studies conducted on antiarrhythmic drug therapy. The incidence of similar tachycardias was highest (72%) during therapy with the combination of a class IA and class IB agent and lowest (50%) with amiodarone. However, there were no significant differences in the proportion of similar ventricular tachycardias among the groups compared either by Fisher's exact or McNemar's paired sample test.

The 33 patients in whom both a drug-free baseline study and subsequent studies on antiarrhythmic drugs were conducted were analyzed separately. The incidence of similar tachycardias during reproducible baseline inductions was 64% compared with 60% for all studies performed on drugs in these 33 patients. Comparison of drug-free baseline studies with studies on specific drugs resulted in the following incidence rates of similar ventricular tachycardia in individual patients: 62% and 62% for baseline versus class IA; 58% versus 50% for baseline versus amiodarone and 67% versus 72% for baseline versus class IA and class IB combination.

Location of scar or aneurysm. In 44 patients with coronary artery disease, the location of scar and the presence of left ventricular aneurysm did not appear to affect the incidence of similar or different ventricular tachycardias during successive inductions. The incidence of similar tachycardias was 58% in patients with anterior infarction, 60% in patients with inferior infarction, 56% in patients with both anterior and inferior infarction and 66% in patients without ischemic heart disease. The incidence of similar tachycardias was 57% in 14 patients with left ventricular aneurysm and 60% in 30 patients with ischemic heart disease but without a left ventricular aneurysm.

Reproducibility of Induction Characteristics (Table 3)

Number of extrastimuli. In 95 (91%) of the 104 studies with reproducible induction, sustained ventricular tachycardia was reinitiated using the same number of extrastimuli and in the remaining 9 (9%), an additional extrastimulus was required. Sustained ventricular tachycardia was initially induced with a single extrastimulus in 33 studies, and in 27 (82%) of these, the second induction of ventricular tachycardia was also initiated with a single extrastimulus. Sustained ventricular tachycardia was initiated with double extrastimuli in 50 studies, and in 47 (94%) of these, tachycardia could be reinitiated by two extrastimuli. Thus, to reproduce sustained ventricular tachycardia, an increase in the number of extrastimuli was required in 9 (11%) of 83 studies in which one or two extrastimuli were initially required to induce the tachycardia. In the two studies where three extrastimuli induced the first but not the second tachycardia, more than three extrastimuli were not used to test the reproducibility during the second induction attempt because stimulation through three extrastimuli was an end point for our protocol.

Number of extrastimuli versus similarity of induced tachycardia. The effect of the number of extrastimuli on the similarity and difference between the twice-induced ventricular tachycardias was assessed in those studies in which the number of extrastimuli for the first and the second induction was the same. Similar ventricular tachycardias were initiated in 63% of studies using a single extrastimulus for both inductions, in 66% of studies using double extrastimuli for both inductions and in 57% of studies using triple extrastimuli for both inductions. These differences were not statistically significant.

Discussion

Reproducibility of induced sustained ventricular tachycardia. Management of recurrent sustained ventricular tachycardia frequently involves antiarrhythmic drug therapy

guided by electrophysiologic testing. Reports from this (6) and other (4,5) laboratories have shown that suppression of arrhythmia induction by acute intravenous drug administration predicts, with varying degrees of success, subsequent tachycardia suppression by the same or other antiarrhythmic drugs. The prognostic significance of the response to programmed cardiac stimulation and assessment of antiarrhythmic efficacy require that the results are reproducible. This prospective study demonstrates that in a selected subgroup of patients presenting with documented sustained ventricular arrhythmias, the immediate reproducibility of sustained ventricular tachycardia induction by programmed cardiac stimulation is very high in the drug-free state and during antiarrhythmic drug therapy. Furthermore, the mode of stimulation required to induce sustained ventricular tachycardia does not change from the first to the second induction in the vast majority of cases (91%).

Studies in the absence of antiarrhythmic drug therapy. Previous studies (14–16) addressing day to day rather than immediate reproducibility reported changes in induction mode in up to 32% of the cases, but some of these studies also included nonsustained ventricular tachycardia as an end point. Two other studies (10,11) more comparable with ours, addressing the immediate reproducibility of the response to programmed cardiac stimulation, reported similar high overall reproducibility rates. De Buitleir et al. (10) found 91% immediate reproducibility during a second induction trial of what they regarded as clinical ventricular tachycardia. In a recent study using a unique protocol with up to five extrastimuli in a smaller group of patients, Cooper et al. (11) showed 100% sensitivity and immediate reproducibility of tachycardia induction. However if their results using only up to three extrastimuli are considered, their reproducibility of induction becomes very similar to ours. These investigators further demonstrated that 94% of the tachycardias requiring two extrastimuli for initiation during the first induction could be reproduced with the use of two extrastimuli during the subsequent trial, same as our 94% (Table 3) despite the differences in protocol between the studies.

The overall reproducibility of induction mode or the number of extrastimuli required for tachycardia initiation, was higher in our study (89%) compared with the previously reported value of 81% by De Buitleir et al. (10) and 74% by Cooper et al. (11) if only their first two of three inductions are considered. Differences in patient groups and the stimulation protocols used may account for these differences among the studies in reproducibility of induction mode.

Changes in rate and ECG morphology. Although reproducibility of ventricular tachycardia induction is tested in many laboratories, analysis of QRS configuration during ventricular tachycardia has frequently been limited to a few surface ECG leads, precluding careful assessment of changes in frontal and horizontal axes. The present study bases the similarities in configuration between separate tachycardia episodes on characteristics of the 12 lead ECG in all studies. This approach, previously used in our laboratory (12), was not intended to describe similarity in site of origin or activation sequence, but to compare the ventricular tachycardia pattern by using an easily accessible clinical tool, the 12 lead ECG, without any implication regarding the underlying mechanisms.

In our study, the reproducibility of the characteristics of the induced ventricular tachycardia was substantially lower (63%) than the reproducibility of induction (98%). The most common difference between the two successively induced tachycardias, occurring in 18% of studies, was a difference in ECG configuration with a similar cycle length. We cannot comment on the mechanism underlying this phenomenon because detailed mapping data were not available for any of the studies. Using a definition of similarity between ventricular tachycardias similar to the definition used in our study, Cooper et al. (11) found a 36% incidence of different tachycardia configuration during immediate reproducibility testing, almost identical to the 37% incidence of dissimilar ventricular tachycardias observed in our study.

Limitations. This prospective study was carried out in a selected group of patients who presented with documented sustained monomorphic ventricular tachycardia. Our results should not be generalized to patients presenting with out of hospital cardiac arrest, nonsustained ventricular tachycardia or syncope because they may manifest different types of induced arrhythmias and induction characteristics than the homogeneous group we describe in this study.

Although the reproducibility of sustained ventricular tachycardia was extensively evaluated in this study, it was not tested in patients requiring cardioversion for several reasons. In addition to the safety and humane considerations, we could not rule out the possibility that direct current countershock may reversibly alter myocardium long enough to interfere with immediate reproducibility testing. Regardless of the reasons, however, as a result of our policy, we could not assess the reproducibility of induction for tachycardias with relatively rapid rates (average tachycardia rate in our study 165 beats/min), which frequently required cardioversion, or of ventricular tachycardias, which regardless of the rate, were refractory to termination by ventricular pacing. Reproducibility of ventricular tachycardias with relatively short cycle lengths remains an unresolved but clinically important topic that needs further investigation, especially because hemodynamic collapse is more likely to occur with rapid tachycardias.

In our study, the two tachycardias that could not be induced a second time manifested rates comparable with the average rate. However, because these tachycardias represented an extremely small subgroup precluding any statistical analysis, it is not known with certainty whether the rate of induced ventricular tachycardia is a factor that correlates with reproducibility of induction. If so, the overall reproducibility of all induced ventricular tachycardias, including the very rapid ones requiring cardioversion, may differ from the values reported in this study.

Clinical implications. The high incidence of immediate reproducibility of tachycardia induction in this highly selected group of patients provides a rational basis and acceptable confidence limits for acute intravenous antiarrhythmic drug testing in the electrophysiology laboratory in similar groups of patients. Our study shows that persistence of induced tachycardia during antiarrhythmic drug therapy or failure to suppress the induced tachycardia is also a highly reproducible phenomenon, lending further support to the rationale behind acute drug testing in similar patients.

In the absence of complete suppression of induced ventricular tachycardia, management may be influenced by factors such as modulations in the rate of the induced tachycardias and changes in the number of extrastimuli required to induce them (7–9). Naccarelli et al. (8) found a higher recurrence rate when ventricular tachycardia was initiated with fewer extrastimuli in patients on amiodarone. Breithardt et al. (7) demonstrated a significant 1 year reduction in recurrent ventricular tachycardia or sudden death when tachycardia was either completely suppressed or more difficult to induce. Furthermore, changes in induced ventricular tachycardia cycle length are sometimes used as a marker of antiarrhythmic and proarrhythmic drug effects. Waller et al. (9) suggested that drug-associated slowing of the induced ventricular tachycardia rate predicted a low incidence of sudden death during long-term antiarrhythmic drug therapy. Such changes, however, should be interpreted with caution because in 20% of our studies, induced tachycardia rates were substantially different (>40 ms change in cycle length) between the first and the second induction, even without alterations in drug therapy. These data on reproducibility of cycle length during the same study should be taken into consideration when making clinical decisions.

Conclusions. The overall immediate reproducibility of induction of sustained monomorphic ventricular tachycardia in patients presenting clinically with this arrhythmia is extremely high, although in a minority of patients, an increase in the number of extrastimuli is required to reinitiate the tachycardia. This finding supports the reliability of ventricular tachycardia induction as a marker against which future therapeutic interventions may be assessed. However, significant differences in specific surface ECG configuration or cycle length occur in 37% of the studies, even in a ... setting. These latter observations suggest that caution must be used in attributing clinical or prognostic significance to changes in induced tachycardia cycle length or ECG pattern after therapeutic interventions, particularly if baseline variability has not been defined.

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