Effects of High Stimulation Current on the Induction of Ventricular Tachycardia

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Programmed stimulation at 2 right ventricular sites with 1 to 3 extrastimuli was performed at current strengths of twice diastolic threshold (1.0 ± 0.2 mA, mean ± standard deviation) and 10 mA in 41 patients undergoing an electrophysiologic study because of sustained ventricular tachycardia (VT) (11 patients), nonsustained VT (19 patients) or unexplained syncope (11 patients). In 26 patients, VT was not induced by programmed stimulation at twice diastolic threshold. Programmed stimulation at 10 mA induced VT or ventricular fibrillation in 16 of these 26 patients (62%). In 4 of 16 patients, the coupling intervals of the extrastimuli that induced VT/ventricular fibrillation at 10 mA were all equal to or longer than the shortest coupling intervals resulting in ventricular capture at twice diastolic threshold. Fifteen patients had inducible VT at twice diastolic threshold. Programmed stimulation at 10 mA induced a similar VT in 12 of these patients, but resulted in no VT induction in 3 of 15 patients (20%), despite ventricular capture at the same coupling intervals that had induced VT at twice diastolic threshold.

This study shows that programmed stimulation at a high current strength may either facilitate or prevent induction of VT. Facilitation of VT induction usually is attributable to a shortening of ventricular refractoriness and the ability of extrastimuli at 10 mA to capture the ventricle at shorter coupling intervals than possible at twice diastolic threshold. However, in 25% of cases, the facilitation of VT induction by 10-mA stimuli is not explained by a shortening of ventricular refractoriness. In these cases, and in the patients in whom 10-mA stimuli prevent the induction of VT that was inducible at twice diastolic threshold, the effects of high current strength appear to be mediated through some other mechanism. Other possible mechanisms include an effect on temporal dispersion of refractoriness or on the pattern or extent of ventricular activation.

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Programmed ventricular stimulation is most frequently performed with a stimulus current strength 2 times the diastolic excitability threshold. However, a wide variety of other current strengths has also been used in some electrophysiology laboratories, ranging from 4 times the diastolic threshold to 20 mA.[1-5] Some reports indicate that programmed stimulation with current strengths greater than twice diastolic threshold result in an increased yield of clinically meaningful ventricular arrhythmias[4,5]; others indicate that high stimulation current simply increased the yield of nonclinical arrhythmias rather than those that are clinically significant.[6,7]

Aside from the issue of whether the induced arrhythmia is clinically meaningful, it is nevertheless clear that a high stimulation current more often results in the induction of ventricular tachycardia (VT) than does a current strength that is twice diastolic threshold. However, the mechanism by which a high stimulation current affects the results of programmed ventricular stimulation is not clear. Although prior studies have investigated the effects of graded stimulation currents on the induction of VT or ventricular fibrillation (VF) in animals,[8,9] the mechanism by which an increased stimulus strength affects the induction of VT in humans has not been investigated in a systematic fashion.

This prospective study compares the effects of high and low current strength on the induction of VT in an attempt to clarify how an increase in the stimulus cur-

rent strength affects the results of programmed ventricular stimulation.

Methods

The subjects in this prospective study included 41 patients who were referred to the University of Michigan Medical Center for an electrophysiologic study because of sustained VT (11 patients), nonsustained VT (19 patients) or unexplained syncope (11 patients). There were 29 men and 15 women, mean age 53 ± 16 years. Nineteen patients had coronary artery disease and a history of a myocardial infarction, 12 had an idiopathic dilated cardiomyopathy, and 10 had no identifiable structural heart disease.

After the patients gave informed consent, they underwent an electrophysiologic study in the fasting, unsedated state, at least 4 half-lives after discontinuation of antiarrhythmic drugs. Twelve patients were being treated with digoxin, which was not discontinued. Two quadripolar electrode catheters were inserted percutaneously into a femoral vein and positioned in the appropriate cardiac chamber. After the completion of atrial stimulation whenever clinically indicated, the electrode catheters were positioned against the apex and outflow tract of the right ventricle. Electrocardiographic leads V_1, I and III, and intracardiac right ventricular electrograms were displayed on an oscilloscope and recorded at a paper speed of 25 to 100 mm/s with an Electronics for Medicine VR 12 recorder. Pacing was performed with a programmable stimulator (Bloom Associates). The stimuli had a pulse width of 2 ms.

Sustained VT was defined as VT lasting 30 seconds or longer or requiring direct-current countershock or overdrive pacing for termination. Nonsustained VT was defined as VT at least 4 beats in duration, terminating spontaneously within 30 seconds.

The following stimulation protocol was used. Stimulation was initially performed with a current strength that was 2 times the diastolic stimulation threshold. The mean current strength was 1.0 ± 0.2 mA (range 0.6 to 1.5). Using a drive train of 6 to 8 stimuli at a cycle length of 600 or 500 ms, programmed stimulation was performed with a single extrastimulus (S_2), then double extrastimuli (S_2S_3) at the right ventricular apex. This was repeated using a drive cycle length of 400 ms. Single and double extrastimuli were then introduced at the right ventricular outflow tract using the same 2 drive cycle lengths that were used at the right ventricular apex. Programmed stimulation was then performed using 2 drive cycle lengths at the right ventricular apex with triple extrastimuli ($S_2S_3S_4$) with S_2 and S_3 initially positioned 30 ms beyond their respective points of ventricular refractoriness, and S_4 positioned 300 ms beyond S_3. Diastole was scanned with $S_2S_3S_4$ in 10-ms steps. Triple extrastimuli were then introduced at the right ventricular outflow tract. The endpoint for the stimulation protocol performed at a current strength of twice diastolic threshold was either 1 induction of VT or VF requiring direct-current countershock for termination, or 2 or more inductions of sustained VT not requiring direct-current countershock.

After completion of programmed stimulation at a current strength of twice diastolic threshold, the current strength was increased to 10 mA and the stimulation protocol previously described was repeated without changing the positions of the catheters. Because of time constraints, in some patients, programmed stimulation at 10 mA was performed at only 1 right ventricular site. The endpoints for the stimulation protocol at a current strength of 10 mA included the same endpoints used with a current strength of twice diastolic threshold; in addition, to minimize the need for direct-current countershock, the stimulation protocol at 10 mA was discontinued after 2 inductions of nonsustained polymorphic VT longer than 3 seconds in duration.

Whenever 1 of the arrhythmia endpoints was reached during stimulation at 10 mA, the coupling intervals of the extrastimuli that resulted in VT induction were held constant, and stimulation was repeated after the current strength was decreased back to twice diastolic threshold.

Results

Programmed stimulation at a current strength of 10 mA in patients without inducible ventricular tachycardia at twice diastolic threshold: In 26 patients, VT was not induced by programmed stimulation at a current strength of twice diastolic threshold. Programmed stimulation at a current strength of 10 mA resulted in the induction of nonsustained polymorphic VT in 6 patients (23%), sustained polymorphic VT in 2 patients (8%), sustained unimorphic VT in 3 patients (12%), and VF in 5 patients (19%) (Table I). Among these 16 patients in whom VT or VF was induced only by stimulation at a current strength of 10 mA, double extrastimuli were required in 3 patients and triple extrastimuli in 13.

In 4 patients (nos. 1 to 4), the coupling intervals of the extrastimuli that induced VT or VF at a current strength of 10 mA were all either equal to or longer than the shortest coupling intervals that resulted in ventricular capture at a current strength of twice diastolic threshold (Table I). After VT or VF was induced at a current strength of 10 mA, stimulation with the same number of extrastimuli and the same coupling intervals at twice diastolic threshold resulted in ventricular capture without the induction of VT or VF in 3 of the 4 patients (Fig. 1). In 1 of the 4 patients (no. 4), when the current strength was lowered to twice diastolic threshold, the extrastimuli did not all capture the ventricle.

In 12 patients (nos. 5 to 16), the coupling intervals of the extrastimuli that induced VT or VF at a current strength of 10 mA were shorter than the shortest coupling intervals that had resulted in ventricular capture during initial stimulation at twice diastolic threshold (Table I). A return to twice diastolic threshold with the same coupling intervals that had induced VT at a current strength of 10 mA resulted in incomplete ventricular capture, and consequently no induced VT, in 10 patients. However, in 2 patients (nos. 15 and 16), extrastimuli at coupling intervals that had initially failed to completely capture the ventricle did so after stimulation at 10 mA had induced VT. In these 2 patients, repeat stimulation at twice diastolic threshold with coupling intervals identical to those that had induced VT at 10 mA resulted in the induction of VT in 1 patient, but not the other.

Programmed stimulation at a current strength of 10 mA failed to induce VT in 10 patients who did not have inducible VT at twice diastolic threshold. When the 16 patients with inducible VT were compared with the 10 patients without inducible VT at a current strength of 10 mA, there was a higher prevalence of coronary artery disease in the former group (8 of 16 vs 1 of 10, $p <0.05$).

TABLE I   Ventricular Tachycardia Induction at a Current Strength of 10 mA in Patients Without Inducible Ventricular Tachycardia at Twice Diastolic Threshold

Pt	Heart Disease	Shortest CIs at 2X DT (ms)*	CIs that Induced VT at 10 mA (ms)	VT Induced at 10 mA	Capture at 2X DT†	VT Induced at 2X DT†
1	CAD	230/180/180	240/190/200	P, 7 sec	+	0
2	CAD	250/210/210	260/220/280	U, S	+	0
3	IDC	230/160/150	230/160/180	VF, S	+	0
4	NSHD	230/190/160	230/200/160	P, 12 sec	0	0
5	CAD	240/220	230/160	P, 12 sec	0	0
6	NSHD	290/230/190	260/220/210	P, 24 beats	0	0
7	NSHD	220/180/160	220/160/140	P, 9 beats	0	0
8	CAD	270/230	270/220	P, S	0	0
9	IDC	290/270/240	290/250/230	U, S	0	0
10	CAD	280/240/230	280/220/220	U, S	0	0
11	NSHD	250/180/180	240/190/170	VF, S	0	0
12	CAD	220/190/190	230/180/170	VF, S	0	0
13	CAD	220/170/130	240/150/130	VF, S	0	0
14	IDC	230/170/160	220/130/130	VF, S	0	0
15	CAD	270/240	260/180	P, S	+	P (S)
16	NSHD	260/210/190	260/190/180	P, 9 beats	+	0

* Shortest coupling intervals ($S_1S_2/S_2S_3/S_3S_4$) associated with ventricular capture at current strength of twice diastolic threshold, before programmed stimulation at 10 mA. The drive cycle length in each case was the same as that used to induce ventricular tachycardia (VT) at 10 mA.

† Refers to ventricular capture or VT induction at twice diastolic threshold, after induction of VT/ventricular fibrillation at 10 mA, with the same number of extrastimuli and coupling intervals that induced VT/ventricular fibrillation at 10 mA.

CAD = coronary artery disease; CI = coupling interval; IDC = idiopathic dilated cardiomyopathy; NSHD = no structural heart disease; P = polymorphic; S = sustained; VF = ventricular fibrillation; VT = ventricular tachycardia; U = unimorphic, 2X DT = twice diastolic threshold; + = yes; 0 = no.
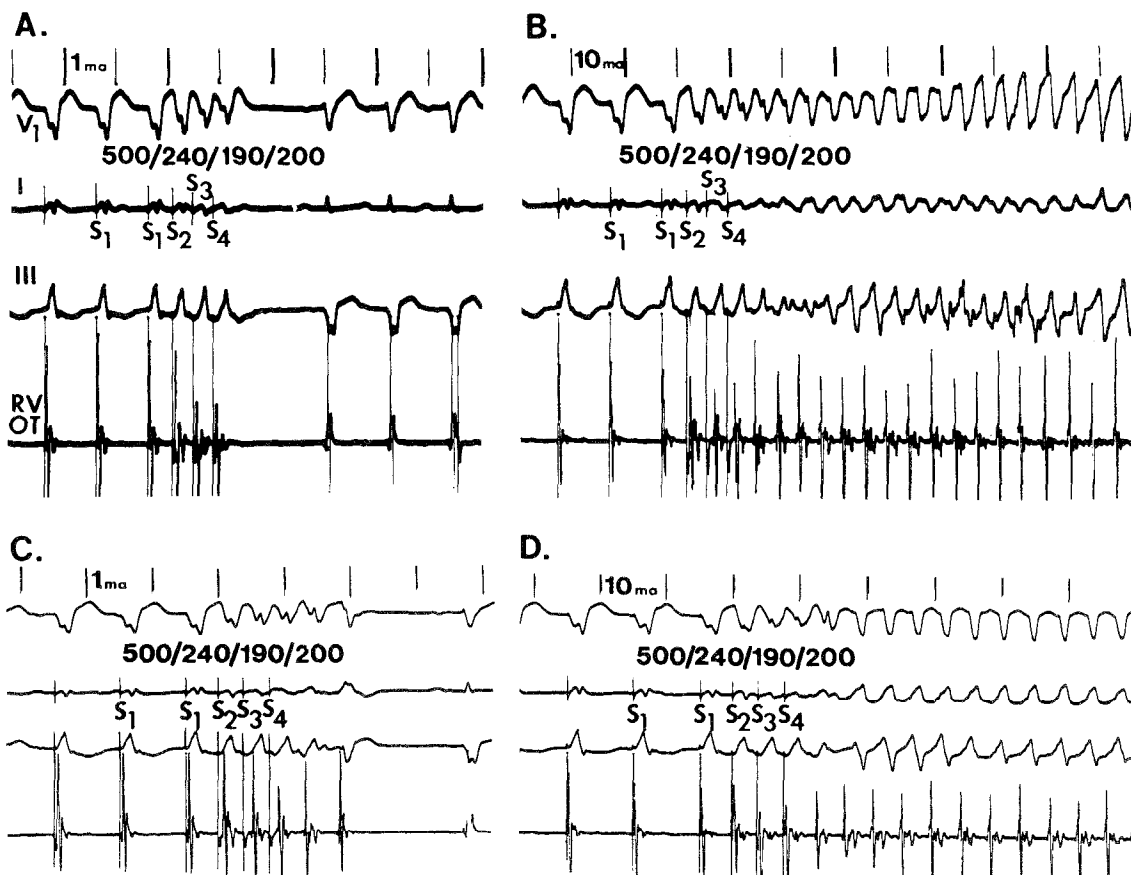
FIGURE 1. Programmed stimulation at the right ventricular outflow tract in patient 1, at a drive cycle length (S_1S_1) of 500 ms, and with $S_1S_2/S_2S_3/S_3S_4$ intervals of 240/190/200 ms. **Top** to **bottom**, leads V_1, I and III, and an electrogram recorded at the right ventricular outflow tract (RVOT). Time lines represent 500-ms intervals. **A,** when the current strength was twice diastolic threshold (1 mA), ventricular tachycardia (VT) was not induced. **B,** after the current strength was increased to 10 mA, 7 seconds of polymorphic VT (mean cycle length 220 ms), was induced. **C,** when the current strength was decreased back to 1 mA, there was again ventricular capture by all of the extrastimuli, but VT was not induced. **D,** an increase in current strength to 10 mA again resulted in the induction of nonsustained polymorphic VT lasting 5 seconds.

**TABLE II   Ventricular Tachycardia Induction at a Current Strength of 10 mA in Patients
With Inducible Ventricular Tachycardia at Twice Diastolic Threshold**

Pt	Heart Disease	CIs that induced VT at 2X DT (ms)	VT Induced at 2X DT	CIs at 10 mA (ms)*	VT Induced at 10 mA
17	NSHD	250/210/200†	P, 13 beats	250/190/170	0
18	CAD	260/210/210	RBBB, S	260/190/200	0
19	CAD	240/200	RBBB, 13 beats	240/180	0
20	NSHD	230/180/170	P, 8 sec	230/180/170	P, S
21	CAD	220/160/140	P, 9 beats	230/180/210	P, 21 beats
22	IDC	140/130	P, 5 sec	200/140/140	P, S
23	CAD	220/160/150	P, 8 beats	220/160/150	P, 8 sec
24	CAD	250/190/180	P, S	250/180/170	P, 4 sec
25	CAD	240/190	P, S	240/190	P, 7 sec
26	CAD	260/230	RBBB, 20 sec	260/260	RBBB, 18 sec
27	IDC	260/190/170	RBBB, 12 sec	260/190/170	RBBB, 15 sec
28	NSHD	230	LBBB, 11 sec	200	LBBB, 7 sec
29	CAD	290/280	LBBB, S	290/250	LBBB, S
30	CAD	270/220/210	LBBB, S	280/210/200	LBBB, S
31	CAD	270/210/180	RBBB, S	270/200/170	RBBB, S

* Coupling intervals that resulted in induction of ventricular tachycardia or when ventricular tachycardia was not induced were the shortest coupling intervals associated with ventricular capture.
† $S_1S_2/S_2S_3/S_3S_4$ intervals in milliseconds.
LBBB = left bundle branch block configuration; RBBB = right bundle branch block configuration; other abbreviations as in Table I.

Programmed stimulation at a current strength of 10 mA in patients with inducible ventricular tachycardia at twice diastolic threshold: Fifteen patients had inducible VT at a current strength of twice diastolic threshold. The VT was polymorphic in 8 patients and unimorphic in 7; it was induced by a single extrastimulus in 1 patient, double extrastimuli in 2 patients, and triple extrastimuli in 12 patients. Programmed stimulation at a current strength of 10 mA also resulted in the induction of VT in 12 of these 15 patients (Table II). However, in 3 patients who had inducible VT at a current strength of twice diastolic threshold, VT was not induced by programmed stimulation with up to 3 extrastimuli at a current strength of 10 mA (Fig. 2). In each of these 3 patients, the shortest coupling intervals associated with ventricular capture at 10 mA were all equal to or shorter than the coupling intervals that had resulted in VT induction at twice diastolic threshold.

Among the other 12 patients with inducible VT at twice diastolic threshold, programmed stimulation at 10 mA induced VT that was similar in configuration and cycle length, with the same number of extrastimuli and similar coupling intervals.

Discussion

The results of this study indicate that programmed stimulation with a high-current strength (10 mA) induces VT or VF in a large proportion (52%) of patients who do not have inducible VT or VT at a current strength of twice diastolic threshold. In many patients (75%), this discordant response to programmed ventricular stimulation may be explained by the ability of extrastimuli at 10 mA to capture the ventricle at shorter coupling intervals than is possible when the current strength is twice diastolic threshold.

In accord with the above-mentioned finding are the results of prior experimental studies.[8,9] Michelson et al[8] reported that the use of increased current strength facilitated the induction of VT in a chronic canine model of myocardial infarction by its effects on ventricular refractoriness; the induction of VT by pacing stimuli of high-current strength was always attributable to ventricular capture at coupling intervals that could not be attained when the current strength was twice diastolic threshold. Also, Hamer et al[9] reported that the ability to capture the ventricle at shorter coupling intervals was almost always the mechanism by which pacing stimuli of high-current strength induced VF in the normal canine heart in which VF could not be induced when the stimuli were twice diastolic threshold.

In 25% of patients in this study in whom VT or VF was induced by extrastimuli of 10 mA but not twice diastolic threshold, the discordant response was not attributable to a difference in coupling intervals. This observation suggests that at least in some patients, a high current strength facilitates the induction of ventricular arrhythmias by a mechanism other than simply allowing extrastimuli to capture the ventricle at shorter coupling intervals. At least 2 other mechanisms are possible. First, a high current strength may facilitate the initiation of VT or VF by an effect on recovery of excitability. In the open-chest dog, Spear et al[10] showed that an increase in current intensity increased the temporal dispersion in recovery of excitability in the myocardium surrounding the stimulation site, thereby lowering the VF threshold. A second mechanism by which a high-current strength may facilitate the induction of VT or VF is by depolarizing more myocardial cells than does a stimulus of twice diastolic threshold. This might alter the pattern of ventricular activation in such a way as to favor the initiation of reentry, or might allow for depolarization of a region of myocardium containing a potential substrate for reentry.

Although a current strength of 10 mA usually facilitated the induction of VT, the opposite was also observed. When the current strength was increased to 10 mA, VT was no longer inducible in 20% of patients who had inducible VT at twice diastolic threshold, despite ventricular capture at the same coupling intervals
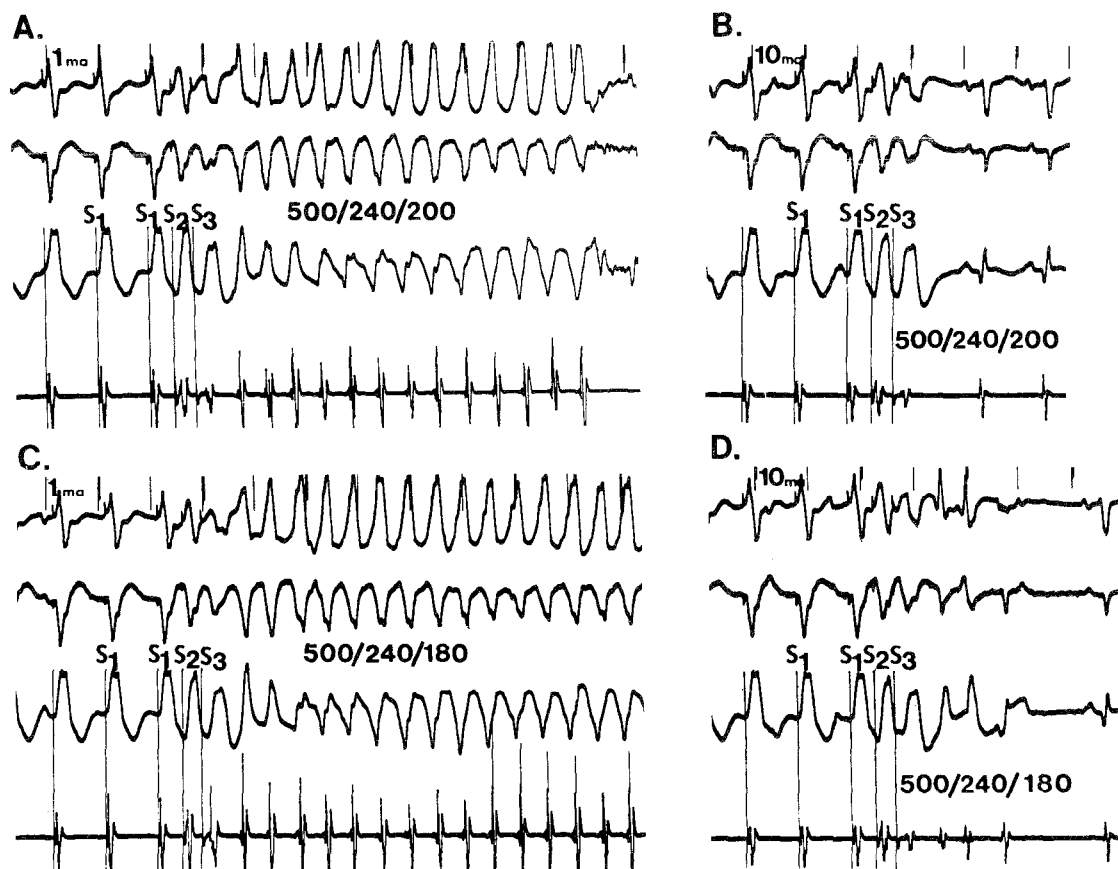
FIGURE 2. Programmed stimulation at the right ventricular outflow tract in patient 17, at a drive cycle length (S_1S_1) of 500 ms. **Top to bottom,** leads V_1, I, and III, and an electrogram recorded at the right ventricular outflow tract. Time lines represent 500-ms intervals. **A,** when the current strength was twice diastolic threshold (1 mA) and the S_1S_2/S_2S_3 intervals were 240/200 ms, a 13-beat run of polymorphic ventricular tachycardia (VT) (mean cycle length 270 ms), was induced. **B,** an increase in current strength to 10 mA resulted in ventricular capture, but VT was not induced. **C,** at current of 1 mA, S_1S_2/S_2S_3 intervals of 240/180 ms also induced polymorphic VT (24 beats). **D,** an increase in current strength to 10 mA again resulted in ventricular capture without the induction of VT.

at both levels of current strength. This observation provides additional evidence that an increase in current strength may affect VT induction in some way other than simply shortening ventricular refractoriness. An effect on temporal dispersion in recovery of excitability or on the pattern of ventricular activation, while facilitating the induction of reentry in some patients, may also prevent the initiation of reentry in some patients who have the appropriate substrate for reentry when the current strength is twice diastolic threshold. Brugada et al,[7] in a preliminary report, found that in a group of 20 patients with clinically documented VT, VT was induced by stimulation only at twice diastolic threshold, and not by stimulation at 20 mA, in 9 patients (45%).

In 2 patients (nos. 15 and 16), stimuli at twice diastolic threshold captured the ventricle at the same coupling intervals that induced VT at 10 mA, but only after VT had been induced by the 10 mA stimuli. The explanation for this phenomenon is unclear; one possibility is that the induction of VT by 10-mA stimuli results in the release of catecholamines that shorten ventricular refractoriness, thereby enabling stimuli at twice diastolic threshold to capture the ventricle. In patient 15 the extrastimuli at twice diastolic threshold did subsequently induce sustained polymorphic VT,

similar to the VT that had been induced by stimulation at 10 mA. In this patient VT induction clearly was dependent only on ventricular capture at critical coupling intervals and not on the stimulus current strength per se. However, in patient 16, extrastimuli at twice diastolic threshold did not induce VT even after ventricular capture became possible at the same coupling intervals that resulted in VT induction at 10 mA. This result indicates that when high current strength facilitates the induction of VT at coupling intervals that did not capture the ventricle at twice diastolic threshold, the possibility is not ruled out that a mechanism other than shortening of ventricular refractoriness is responsible for the induction of VT.

Among the patients without inducible VT at twice diastolic threshold, the only discernible difference between the patients in whom a current strength of 10 mA did and those in whom it did not facilitate VT induction was that there was a higher prevalence of coronary artery disease in the group that did have inducible VT at 10 mA. This finding suggests that the facilitation of VT induction by the use of a high stimulus current strength may be more common in patients who have coronary artery disease. This possibility is consistent with the finding that coronary occlusion in the dog augments the

increase in temporal dispersion in recovery of excitability that occurs as the stimulus current strength increases.[10]

When programmed stimulation with a current strength of both twice diastolic threshold and 10 mA induced VT, the configuration of the VT was similar at both current strengths, as were the number of extrastimuli and the coupling intervals that induced the VT. Unimorphic VT was induced in patients with documented unimorphic VT and was thought to be a clinically meaningful response. However, the polymorphic VT was induced either in patients with documented unimorphic VT or in those with unexplained syncope, and was believed to be a laboratory artifact rather than a clinically significant arrhythmia. Among the patients in whom polymorphic VT was induced at a current strength of twice diastolic threshold, an increase in the current strength to 10 mA never resulted in the induction of unimorphic VT. Furthermore, in only 3 of 16 patients who had an inducible arrhythmia at 10 mA and not twice diastolic threshold was the induced arrhythmia a unimorphic VT. Although this study was not intended to address the issue of relative sensitivity and specificity of stimulation protocols with high and low current strength, these observations suggest that the use of a stimulus current strength of 10 mA may compromise the specificity of stimulation protocols to a much greater degree than it enhances sensitivity. Preliminary reports on the sensitivity and specificity of programmed ventricular stimulation protocols using current strengths of 10 or 20 mA have found this to be the case.[6,7]

The principal limitation of the present study is the inability to guarantee the reproducibility of all induced arrhythmias. Arrhythmias requiring direct-current countershock were induced only once at a given current strength to avoid subjecting patients to multiple shocks. Therefore, a discordant response to stimulation at twice diastolic threshold and 10 mA may be attributable to nonreproducibility of arrhythmia induction instead of a change in the stimulus current strength. To minimize the likelihood of this possibility, discordant responses to stimulation at the high and low current strengths were confirmed whenever feasible by immediately alternating between the 2 current strengths.

In conclusion, this study has demonstrated that programmed stimulation at a high current strength may either facilitate or prevent the induction of VT. Facilitation of VT induction is most often attributable to a shortening of ventricular refractoriness and the ability of extrastimuli at 10 mA to capture the ventricle at shorter coupling intervals than is possible at twice diastolic threshold. However, in at least 25% of cases, the facilitation of VT induction by 10 mA stimuli cannot be explained by a shortening of ventricular refractoriness. In these cases, and in the patients in whom 10-mA stimuli prevented the induction of VT that was inducible at twice diastolic threshold, the effects of high current strength appear to be mediated through some other mechanism. Other possible mechanisms include an effect on temporal dispersion of refractoriness or on the pattern or extent of ventricular activation.

A clinical implication of this study is that programmed ventricular stimulation at a high current strength should always be preceded by stimulation at twice diastolic threshold. The immediate use of pacing stimuli of high current strength may not only result in a high incidence of arrhythmias that are not clinically meaningful, but also may prevent the induction of VT in some patients who have inducible VT at a current strength of twice diastolic threshold.

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