JACC Vol. 8, No. 5
November 1986:1035–40

Ventricular Arrhythmias Initiated by Programmed Stimulation in Four Groups of Patients With Healed Myocardial Infarction

PEDRO BRUGADA, MD, BERND WALDECKER, MD, YVO KERSSCHOT, MD, MANFRED ZEHENDER, MD, HEIN J. J. WELLENS, MD, FACC

Maastricht, The Netherlands

Programmed electrical stimulation of the heart was prospectively used in 160 patients with healed myocardial infarction to study the incidence and characteristics of ventricular arrhythmias induced. Thirty-five patients had neither documented nor suspected ventricular arrhythmias (Group A); 37 patients had documented nonsustained ventricular tachycardia (Group B); 31 patients had been resuscitated from ventricular fibrillation (Group C); and 57 patients had documented sustained monomorphic ventricular tachycardia (Group D). No electrophysiologic differences were found between patients in Group A and Group B, but patients in both groups differed significantly from patients in Group C and Group D. In the last two groups, sustained monomorphic ventricular tachycardia was more frequently induced, the cycle length of the induced ventricular tachycardia was slower and a lesser number of premature stimuli was required for induction. No differences were found in the incidence, rate or mode of induction of nonsustained monomorphic ventricular tachycardia, but nonsustained polymorphic ventricular tachycardia and ventricular fibrillation were more frequently induced in Groups A and B.

It is concluded that the substrate for sustained ventricular arrhythmia is present in at least 42% of patients after myocardial infarction. The electrophysiologic characteristics of the substrate for ventricular tachycardia seem to be the major determinant of the clinical occurrence of sustained ventricular arrhythmia. Changes in the electrophysiologic properties of the substrate of ventricular tachycardia, either spontaneously with time or induced by ischemia or antiarrhythmic drugs, can contribute to the clinical occurrence of sustained ventricular arrhythmias in patients with an old myocardial infarction.

(J Am Coll Cardiol 1986;8:1035–40)

Ventricular tachycardia and ventricular fibrillation constitute a major cause of morbidity and mortality in patients discharged from the hospital after myocardial infarction (1–3). Available evidence (4–6) supports the concept that sudden death after the acute phase of myocardial infarction is caused in the majority of cases by the spontaneous occurrence of a sustained, rapid ventricular arrhythmia. Many studies have been undertaken to gain understanding of the determinants and predictors of sudden death and the development of ventricular tachycardia after myocardial infarction. The results of these studies (7–13) have demonstrated that the degree of left ventricular dysfunction, the density of ambient ectopic activity and the potential for new ischemic events are important determinants of the patients' prognosis and the spontaneous occurrence of sustained ventricular arrhythmias. The initiation and perpetuation of a sustained ventricular arrhythmia require, however, the presence of a substrate able to initiate and perpetuate this arrhythmia. Both experimental and clinical studies (14–17) have provided support for the hypothesis that myocardial infarction creates the anatomic functional basis or substrate for a reentrant tachycardia in the diseased ventricle.

The purpose of our study was to evaluate the incidence and characterize the electrophysiologic properties of the substrate for ventricular arrhythmias in patients after myocardial infarction with and without spontaneously occurring ventricular arrhythmias. For this purpose, the results of programmed electrical stimulation of the heart were analyzed prospectively in four groups of patients with healed myocardial infarction.

Methods

Study patients. One hundred sixty consecutive patients with a well documented myocardial infarction were studied

From the Department of Cardiology, Academic Hospital, University of Limburg, Maastricht, The Netherlands.

Manuscript received October 29, 1985; revised manuscript received May 14, 1986; accepted May 27, 1986.

Address for reprints: Pedro Brugada, MD, Clinical Electrophysiology Laboratory, Department of Cardiology, Academic Hospital, University of Limburg, Maastricht, The Netherlands.

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0735-1097/86/$3.50

Table 1. Clinical and Angiographic Characteristics of 160 Patients Studied

	Group				
	A	B	C	D	Total
No. of patients	35	37	31	57	160
Men/women	26/9*	31/6	29/2	54/3	140/20
Mean age (yr)	57 ± 9	56 ± 10	56 ± 9	58 ± 9	57 ± 9
Mean LVEF (%)	50 ± 14†	43 ± 13‡	34 ± 9	36 ± 9.8	41 ± 13
Mean no. of diseased vessels (≥ 70% stenosis)	1.94 ± 0.9	2 ± 0.75	2.2 ± 0.85	1.7 ± 0.9§	1.98 ± 0.8
Anterior MI	17	21	16	21	75
Inferior MI	16	12	10	27	65
Multiple MI	2	4	5	8	19
Subendocardial MI	0	0	0	1	1

*$p < 0.01$ versus Group D; †$p < 0.01$ versus Group C, $p < 0.001$ versus Group D; ‡$p < 0.05$ versus Groups A and D, $p < 0.01$ versus Group C; §$p < 0.05$ versus Group C. Group A = no documented or suspected ventricular arrhythmias; Group B = documented nonsustained ventricular tachycardia; Group C = documented ventricular fibrillation; Group D = documented sustained monomorphic ventricular tachycardia; LVEF = left ventricular ejection fraction; MI = myocardial infarction.

prospectively (Table 1). Thirty-five patients (Group A) had neither suspected nor documented nonsustained or sustained ventricular arrhythmias before study. Thirty-seven patients (Group B) had documented nonsustained ventricular tachycardia (three beats or more, but lasting less than 30 seconds and not associated with syncope) during long-term electrocardiographic ambulatory monitoring in the hospital 4 to 12 days after myocardial infarction. Thirty-one patients (Group C) had been resuscitated from ventricular fibrillation either in or out of the hospital, 2 weeks to 3 years after myocardial infarction. Fifty-seven patients (Group D) had documented sustained regular, monomorphic ventricular tachycardia 2 weeks to 12 years after myocardial infarction. There were 140 men and 20 women, with Group A having significantly more women than Group D. The mean age was 57 ± 9 years, without significant differences among the four groups.

Myocardial infarction was diagnosed in these patients by all three of the following criteria during the acute admission: 1) chest pain lasting for at least 30 minutes, with 2) evolving ST-T electrocardiographic abnormalities, and 3) a typical rise of cardiac serum enzymes to at least twice the normal values. The ST-T electrocardiographic abnormalities, development of new Q waves and wall motion abnormalities on left ventricular contrast angiography or gated blood pool scan were used to characterize the infarction as anterior, inferoposterior, multiple or subendocardial. There were no differences in the distribution of the location of the myocardial infarct among the four groups. Left ventricular ejection fraction was calculated angiographically or by radionuclide techniques, or both. There was a significant difference in the mean left ventricular ejection fraction among the four groups; patients in Group A had the highest ejection fraction, and patients in Group B had a higher ejection fraction as compared with Patients in Group C and Group D (Table 1). No significant difference was present between Groups C and D. The mean number of diseased vessels (≥70% stenosis) was 1.98 ± 0.8.

Electrophysiologic study. After the patients gave informed consent, programmed ventricular stimulation was performed with patients at rest in the postabsorbtive state under light sedation with oral diazepam (10 mg). In Groups A and B, programmed stimulation was performed 21 to 28 days after myocardial infarction. In Groups C and D, it was performed at the time the patients presented with sustained ventricular arrhythmia, an interval that varied from 12 days to 12 years (median 6.5 months) after infarction. Programmed stimulation was performed without antiarrhythmic drug treatment. Patients under long-term treatment with amiodarone before study were excluded. A uniform stimulation protocol, consisting of 12 consecutive steps (Table 2), was used in all patients. Stimulation was discontinued

Table 2. Standardized Programmed Ventricular Stimulation Protocol

Step	Pacing Mode (RVA, 2× threshold, 2 ms duration)
1	1 VPD given during sinus rhythm
2	2 VPDs given during sinus rhythm
3	1 VPD given during pacing at 100 beats/min
4	2 VPDs given during pacing at 100 beats/min
5	1 VPD given during pacing at 120 beats/min
6	2 VPDs given during pacing at 120 beats/min
7	1 VPD given during pacing at 140 beats/min
8	2 VPDs given during pacing at 140 beats/min
9	3 VPDs given during sinus rhythm
10	3 VPDs given during pacing at 100 beats/min
11	3 VPDs given during pacing at 120 beats/min
12	3 VPDs given during pacing at 140 beats/min

RVA = right ventricular apex; VPD = ventricular premature depolarization.

after the stimulation steps had been performed or after initiation of a syncopal ventricular arrhythmia requiring direct current (DC) shock for termination. All stimulation was bipolar using pulses of 2 ms duration given at twice diastolic threshold, with the stimulation site confined to the right ventricular apex.

Responses to programmed ventricular stimulation were classified as follows (18,19): 1) repetitive ventricular responses—two to five nonstimulated beats of ventricular origin; 2) nonsustained ventricular tachycardia—six or more nonstimulated ventricular beats with a rate of 100/ml or greater, lasting less than 30 seconds and not associated with loss of consciousness; 3) sustained ventricular tachycardia—lasting more than 30 seconds or producing loss of consciousness; 4) polymorphic ventricular tachycardia—ventricular tachycardia with continuous (at least every six beats) changes in QRS axis or configuration, or both; 5) monomorphic ventricular tachycardia—ventricular arrhythmia with constant QRS axis and configuration; and 6) ventricular fibrillation—ventricular arrhythmia with totally disorganized electrical activity in the surface electrocardiographic leads and no recognizable QRS complexes.

Statistical analysis. This was performed using the chi-square test and the Student's *t* test for paired and unpaired data.

Results

Intergroup Comparisons of Ventricular Arrhythmias Initiated

Repetitive ventricular responses and nonsustained ventricular arrhythmias (Table 3). With the stimulation protocol used, repetitive ventricular responses were initiated in all 160 patients. There were no significant differences among the four groups in relation to the mean number of premature stimuli required to initiate repetitive ventricular responses, the mean number of stimulation steps required or the duration of the repetitive ventricular responses.

Nonsustained polymorphic ventricular tachycardia was initiated in 79 of the 160 patients. This arrhythmia was more frequently initiated in patients in Groups A and B as compared with those in Groups C and D. The mean number of premature stimuli required to initiate nonsustained polymorphic ventricular tachycardia and the mean ventricular tachycardia cycle length did not differ between Groups A and B. However, in these two groups, the mean number of premature stimuli required was greater and the mean ventricular tachycardia cycle length shorter as compared with Groups C and D.

A nonsustained monomorphic ventricular tachycardia was initiated in 27 of the 160 patients, without significant differences among the four groups in the mean number of premature stimuli required to initiate the arrhythmia or in the cycle length of tachycardia.

Sustained ventricular arrhythmias. Three types of sustained ventricular arrhythmias were initiated (Table 4). Sustained polymorphic ventricular tachycardia was initiated in 8 patients, ventricular fibrillation was initiated in 24 patients and sustained monomorphic ventricular tachycardia was initiated in 104 patients. No significant differences occurred in the incidence, mean number of premature beats needed to induce the arrhythmia or the mean cycle length of sustained polymorphic ventricular tachycardia among the four groups. The mean number of extrastimuli required to initiate ventricular fibrillation was lower in Group C than in Groups A and D; the difference was only of borderline significance in comparison with Group B.

A sustained monomorphic ventricular tachycardia was more frequently initiated in patients in Group D as compared with the other three groups. The mean number of extrastimuli required to initiate sustained monomorphic ventricular tachycardia was less and the mean cycle length of the arrhythmia was longer in Groups C and D as compared with Groups A and B.

Syncopal arrhythmias and need of DC shock. To terminate a syncopal ventricular arrhythmia, DC shock was required in 73 of the 160 patients. There were no significant

Table 3. Repetitive Ventricular Responses and Nonsustained Ventricular Arrhythmias Initiated

	Group				
	A	B	C	D	Total
RVR (no.)	35 (100%)	37 (100%)	31 (100%)	57 (100%)	160 (100%)
Nonsust PVT (no.)	26 (74%)*	24 (65%)†	11 (35%)	18 (31%)	79 (49%)
Mean extr (no.)	2.5 ± 0.6‡	2.4 ± 0.6§	1.8 ± 0.75	2.1 ± 0.8	2.3 ± 0.7
Mean CL (ms)	209 ± 27‖	216 ± 18	221 ± 20	228 ± 27	217 ± 22
Nonsust MVT (no.)	5 (14%)	5 (13%)	5 (16%)	12 (21%)	27 (16%)
Mean extr (no.)	2.4 ± 0.5	2.4 ± 0.5	2 ± 0.7	2.2 ± 0.7	2.2 ± 0.6
Mean CL (ms)	268 ± 34	247 ± 37	296 ± 112	309 ± 90	288 ± 82

*$p < 0.01$ versus Group C, $p < 0.001$ versus Group D; †$p < 0.01$ versus Group D, $p < 0.02$ versus Group C; ‡$p < 0.01$ versus Group C; §$p < 0.05$ versus Group C; ‖$p < 0.05$ versus Group D; CL = cycle length; Extr = extrastimuli; Nonsust MVT = nonsustained monomorphic ventricular tachycardia; Nonsust PVT = nonsustained polymorphic ventricular tachycardia; RVR = repetitive ventricular responses.

Table 4. Sustained Ventricular Arrhythmias Initiated

	Group				
	A	B	C	D	Total
Sus MVT (no.)	17 (48%)*	15 (40%)*	19 (61%)*	53 (93%)	104 (65%)
Mean extr (no.)	2.47 ± 0.6†	2.26 ± 0.45‡	2.05 ± 0.6	1.8 ± 0.7	2.06 ± 0.7
Mean CL (ms)	214 ± 35§	232 ± 45‖	274 ± 58**	315 ± 79	279 ± 77
Sus PVT (no.)	4 (11%)	1 (2%)	1 (3%)	2 (3.5%)	8 (5%)
Mean extr (no.)	2.75 ± 0.5	2	2	2.0 ± 0.5	2.25 ± 0.7
Mean CL (ms)	202 ± 30	200	210	185 ± 35	198 ± 29
VF (no.)	10 (28%)††	6 (16%)	5 (16%)	3 (5%)	24 (15%)
Mean extr (no.)	2.7 ± 0.5‡‡	2.7 ± 0.5	2.2 ± 0.4	3§§	2.6 ± 0.5

*$p < 0.001$ versus Group D; †$p < 0.05$ versus Group C, $p < 0.001$ versus Group D; ‡$p < 0.05$ versus Group D; §$p < 0.001$ versus Groups C and D; ‖$p < 0.02$ versus Group C, $p < 0.001$ versus Group D; **$p < 0.02$ versus Group D; ††$p < 0.01$ versus Group D; ‡‡$p < 0.05$ versus Group C; §§$p < 0.01$ versus Group C. Sus MVT = sustained monomorphic ventricular tachycardia; Sus PVT = sustained polymorphic ventricular tachycardia; VT = ventricular tachycardia; other abbreviations as in Table 3.

differences in the requirement of DC shock because of a syncopal ventricular arrhythmia among the four groups. However, when only DC shock required for a syncopal sustained monomorphic ventricular tachycardia was considered (excluding ventricular fibrillation and sustained polymorphic ventricular tachycardia), a significantly lower incidence of DC shock was found in patients in Group D.

Intragroup Comparisons

To uncover differences in inducibility of nonsustained or sustained ventricular arrhythmia within the four groups, intragroup comparisons were made for clinical, angiographic and electrophysiologic variables. In Group A, a borderline ($p = 0.05$) significant difference in age was found between patients who had an inducible sustained ventricular arrhythmia (mean age 48.9 ± 13 years) and those who did not (mean age 52.8 ± 15 years). None of the remaining variables analyzed differed between patients within the four groups who exhibited an inducible or noninducible nonsustained or sustained ventricular arrhythmia.

Discussion

Determinants of the spontaneous occurrence of ventricular arrhythmias. Understanding the factors that predict and determine the spontaneous occurrence of sustained ventricular arrhythmias and sudden death after myocardial infarction requires studies to uncover the factors triggering these arrhythmias. In addition, detailed analysis must be made of the substrate that makes initiation and perpetuation of the fatal arrhythmic event possible. Arrhythmias in the chronic phase of myocardial infarction are most likely due to a reentrant mechanism, and can be reproducibly initiated by the technique of programmed electrical stimulation of the heart (16–19). Application of this technique to patients not suffering clinically from ventricular arrhythmias after myocardial infarction has shown that a sustained ventricular arrhythmia can be initiated in approximately one-third of patients. However, the predictive value of this technique for identifying patients prone to arrhythmic complications is still controversial (20–23). Both the stimulation protocol and the definitions used to classify the induced arrhythmias vary from institution to institution, making comparisons difficult. However, the initial enthusiastic reports have not been confirmed, whatever the definitions used. The lack of predictive accuracy might be the result of many factors, including the general lack of clear documentation of the cause of sudden death in patients studied prospectively after myocardial infarction.

The predictive value of noninvasive studies (clinical variables, long-term electrocardiographic monitoring or exercise testing) has not been better than the predictive value of invasive studies (angiography and programmed stimulation). Although one may fail to identify the real cause of sudden death after myocardial infarction in all patients, evidence has been presented that a rapid ventricular arrhythmia plays a role in the fatal event, and that this arrhythmia is based on a reentrant mechanism.

By comparing the incidence and characteristics of the ventricular arrhythmias initiated by programmed stimulation in these four groups of patients after myocardial infarction, we tried to obtain a better idea of the substrate for the arrhythmia (the reentrant circuit). Our study demonstrated marked and significant differences not only in the incidence of induction, but also in the mode of initiation and the rate of ventricular arrhythmias among the four groups.

Arrhythmias induced. Patients without documented or suspected ventricular arrhythmias and those with documented nonsustained ventricular tachycardia after myocardia infarction showed a higher incidence of nonsustained polymorphic ventricular tachycardia initiated by programmed stimulation as compared with patients with documented ventricular fibrillation or sustained monomorphic ventricular tachycardia. However, this was the result of our

stimulation protocol and end points during the study, and was consistent with our previous observations (18,19) showing that this arrhythmia is a nonspecific response to stimulation protocols using more than two extrastimuli. Interestingly, nonsustained polymorphic ventricular tachycardia had the shortest cycle length (that is, the fastest rate) in patients in Groups A and B (these groups excluded patients with documented ventricular fibrillation or sustained monomorphic ventricular tachycardia). This suggests that in these patients, the arrhythmic substrate has the fastest conduction velocity and the slowest duration of the refractory period within the involved ventricular tissues. Patients in Groups A and B had the highest ejection fraction, consistent with a larger amount of normal myocardium in this functional circuit.

Although no significant differences were found in the incidence, rate and mode of induction of nonsustained ventricular tachycardia and sustained polymorphic ventricular tachycardia, marked differences were observed in the incidence and mode of induction of sustained monomorphic ventricular tachycardia. As discussed for nonsustained polymorphic ventricular tachycardia, ventricular fibrillation represents a nonspecific response to aggressive programmed electrical stimulation protocols (19). The most obvious example of the possible functional nature of ventricular fibrillation is, without doubt, its occurrence in a normal heart during electrocution or during studies to determine the fibrillation threshold in individuals with a normal heart. Also, other experimental studies (24) have demonstrated the possibility of initiating ventricular fibrillation in the normal dog heart when enough premature stimuli are given. The results observed in our patients must be interpreted in the light of these studies. In our patients, programmed stimulation was continued until a sustained ventricular arrhythmia requiring DC shock occurred or the stimulation protocol was completed. Patients without documented sustained ventricular arrhythmias had a lower incidence of induction of sustained monomorphic ventricular tachycardia.

The higher incidence of ventricular fibrillation in these groups was therefore related to the high mean number of premature stimuli that had to be given. A lower mean number of premature stimuli was required to initiate ventricular fibrillation in Group C, but the incidence of ventricular fibrillation was higher in Group A. Whether ventricular fibrillation initiated in patients of Group C had the same mechanism as that suffered clinically by those patients is impossible to investigate using our present methods.

Determinants of induction of a sustained monomorphic ventricular tachycardia. In a previous study (18), we showed that a variety of ventricular arrhythmias can be initiated by "aggressive" electrical ventricular stimulation protocols in patients not suffering clinically from ventricular arrhythmias. In that study, however, a sustained regular monomorphic ventricular tachycardia was never initiated in patients not having the substrate for the arrhythmia. A myocardial infarction creates an anatomic and functional substrate in which reentry can be initiated and perpetuated. When reentry occurs and the revolution time and exit point from the reentrant circuit remain constant, a regular monomorphic ventricular tachycardia results. The ability to initiate and perpetuate this arrhythmia depends on an accurate balance of the electrophysiologic properties of the pathways involved in reentry. The triggering factor (extrasystole) has to result in the occurrence of unidirectional block in one of the reentrant pathways, enough slow conduction over an alternative pathway and re-excitation of the formerly blocked pathway in a retrograde direction. Reentrant circuits with pathways having short refractory periods require very closely given, multiple premature stimuli to create unidirectional block. Short conduction times might make re-excitation of the formerly blocked pathway difficult because of refractoriness of this pathway at the time of arrival of the retrograde impulse by way of the alternative pathway.

The results of programmed stimulation in our 160 patients demonstrated that a potential reentrant circuit able to perpetuate a sustained regular monomorphic ventricular tachycardia was present in at least 42% of patients with a healing or healed myocardial infarction. This was demonstrated by initiating sustained monomorphic ventricular tachycardia by programmed stimulation.

In patients in whom sustained monomorphic ventricular tachycardia was not induced, the substrate for the arrhythmia might also be present. However, either the stimulation protocol was inappropriate or the properties of the reentrant circuit made it impossible to initiate the arrhythmia. Induction of sustained monomorphic ventricular tachycardia required a lower mean number of premature stimuli in patients in Groups C and D, and the arrhythmia was slower. These observations suggest that the reentrant circuit had longer refractory periods and revolution times in these patients as compared with patients in Groups A and B.

Relevance for antiarrhythmic drug treatment. Many patients with an old myocardial infarction receive antiarrhythmic drugs because of complex ectopic activity while they are not suffering from spontaneous sustained ventricular tachycardia. Most antiarrhythmic drugs exert their effects by prolonging refractoriness, slowing conduction velocity or most commonly, a combination of both. In patients without clinically sustained ventricular arrhythmias, such drugs might prolong refractoriness in the reentrant circuit and slow conduction velocity. This could bring the electrophysiologic properties of the reentrant circuit of these patients closer to those of the reentrant circuit in patients with clinically sustained ventricular arrhythmias, thereby facilitating the spontaneous occurrence of sustained ventricular arrhythmias and sudden death. In light of our observations, it seems appropriate to reevaluate the wisdom of giving antiarrhythmic drugs to patients after myocardial infarction

if they do not have clinically sustained ventricular tachycardia.

Limitations. There are several obvious limitations to our study. First, as previously commented on, persistence of inducibility was not assessed in Groups A and B. However, at least 42% of these patients had the substrate for sustained monomorphic ventricular tachycardia 3 weeks after myocardial infarction. Whether this incidence increased, decreased or remained the same during the follow-up period is unknown. Second, our findings in these patients have been explained on the basis of reentry as a mechanism of ventricular arrhythmias after myocardial infarction. Although there is much evidence supporting this hypothesis, identification of the exact mechanism of spontaneous ventricular arrhythmias remains difficult, and other mechanisms might have been operating in our patients. However, our observations can be most easily explained by the reentry theory. Third, we did attempt to treat patients in Groups A and B with antiarryhthmic drugs and evaluate whether such treatment would have resulted in the spontaneous occurrence of sustained ventricular arrhythmias. We had no clinical reason to treat patients in either of these two groups with antiarrhythmic drugs, nor did we consider it ethical to perform such a study. Therefore, whether antiarrhythmic drugs would have resulted in the predicted effects, such as the spontaneous occurrence of a sustained ventricular tachycardia or sudden arrhythmic death remains unknown. These limitations do not, however, invalidate the findings that programmed stimulation with a standardized stimulation protocol uncovers marked differences in incidence, mode of induction and characteristics of sustained monomorphic ventricular tachycardia between patients not suffering and those suffering from spontaneously sustained monomorphic ventricular tachycardia or ventricular fibrillation.

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