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What Is the Best Predictor of Spontaneous Ventricular Tachycardia and Sudden Death After Myocardial Infarction?

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Background. **Death during the first year after myocardial infarction is most commonly due to spontaneous ventricular tachycardia (VT) or fibrillation (VF). The purpose of this study was to compare, in a single cohort of patients, the values of inducible VT, delayed ventricular activation, low left ventricular ejection fraction, high-grade ventricular ectopy, and ST segment displacement on exercise in predicting electrical events (witnessed instantaneous death and spontaneous VT or VF) during the first year after myocardial infarction.**

Methods and Results. **Three hundred sixty one patients aged less than 71 years underwent electrophysiological study, signal-averaged electrocardiogram, gated blood-pool scan, 24 hour ambulatory electrocardiographic monitoring, and exercise testing 1–2 weeks after myocardial infarction and were then followed up for at least 1 year. There were 34 deaths (eight witnessed instantaneous, 26 other), and nine patients survived one or more episodes of spontaneous VF or VT. Patients with inducible VT were 15.2 times more likely to suffer electrical events than patients without inducible VT. No proportional-hazards model excluding inducible VT was as good a predictor of electrical events as was inducible VT alone.**

Conclusions. **Inducible VT at electrophysiological study was the single best predictor of spontaneous VT and sudden death after myocardial infarction. (*Circulation* 1991;83:756–763)**

Death during the first year after myocardial infarction is most commonly due to spontaneous ventricular tachycardia or fibrillation, further myocardial infarction, or cardiac failure. Patients with inducible ventricular tachycardia after myocardial infarction[1–5] and/or delayed ventricular activation[4–8] through scarred myocardium are very much more likely to have spontaneous ventricular tachycardia during the first year after infarction than are patients without inducible ventricular tachycardia and without delayed ventricular activation. Patients with extensive myocardial infarction and low left ventricular ejection fraction[9] are more likely to die than are patients with less extensive myocardial damage and high left ventricular ejection fraction.

The purpose of this study was to compare, in a single cohort of survivors of acute myocardial infarction, the values of inducible ventricular tachycardia, delayed ventricular activation, low left ventricular ejection fraction, high-grade ventricular ectopy, and ST segment displacement on exercise and to assess their accuracy in predicting electrical events (witnessed instantaneous death and spontaneous ventricular tachycardia or ventricular fibrillation) and cardiac death (witnessed instantaneous death, death due to fresh myocardial ischemia or death due to cardiac failure).

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Methods

Between January 1985 and February 1987, 791 patients were admitted to the coronary care unit with acute myocardial infarction. Patients less than 71 years of age ($n=637$) who survived at least 5 days after acute myocardial infarction ($n=612$) and who were free of cardiac failure and persistent myocardial ischemia and were taking no cardioactive medications other than digoxin ($n=457$) were eligible for study.

The reasons for exclusion of patients from the study are summarized in Table 1. In addition, 96 eligible patients were not studied because the study was not offered by the attending physician ($n=92$) or

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TABLE 1. Summary of 155 Patients Excluded From Study Because of the Presence of One or More of the Exclusion Criteria

No. of patients (N=155)	Exclusion criterion: Failure (n=50)	Ischemia (n=38)	Drugs (n=112)
3	+	+	+
13	+	−	+
21	−	+	+
75	−	−	+
5	+	+	−
9	−	+	−
29	+	−	−

Failure, cardiac failure not controlled with fluid restriction and furosemide; Ischemia, postinfarctional angina pectoris requiring regular therapy; Drugs, cardioactive medications other than digoxin; N, total number of patients excluded; n, number of patients excluded in each category; +, presence of exclusion criterion; −, absence of exclusion criterion.

because consent was refused by the patient (n=4). When particular tests were omitted otherwise, it was for logistic reasons. The distribution of tests performed on 361 patients is summarized in Table 2. In the final analysis, all available data were used. The effect of restricting analysis to only those patients who underwent all tests was to reduce statistical, but not qualitative, differences.

All protocols were approved by the research and ethics committees at the hospital, and informed consent was granted by all patients studied. Electrophysiological study, signal-averaged vectorcardiogram, radionuclide-gated heart-pool scan, and 24-hour ambulatory electrocardiogram (Holter) were performed just before hospital discharge, usually 7–10 days after infarction (range, 6–28 days). The exercise test was performed 1 week after discharge from hospital. Other clinical data recorded at entry (and tested for predictive value) included age, sex, previous infarction, cardiomegaly on chest x-ray, and heart failure on chest x-ray.

TABLE 2. Distribution of Tests Performed on 361 Patients

No. of patients (N=361)	EPS (n=313)	VCG (n=225)	GHPS (n=347)	Holter (n=358)	Ex test (n=284)
164	+	+	+	+	+
71	+	−	+	+	+
49	+	+	+	+	
37	−	−	+	+	+
15	+	−	+	+	−
10	+	+	−	+	+
8	−	−	+	+	−
2	+	+	−	+	−
2	−	−	−	+	−
1	+	−	+	−	+
1	−	−	+	−	+
1	+	−	+	−	−

EPS, electrophysiological study; VCG, signal-averaged vectorcardiogram; GHPS, gated heart-pool scan; Holter, 24-hour ambulatory electrocardiogram; Ex test, exercise test; N, total number of patients tested; n, number of patients participating in specific test; +, test performed; −, test not performed.

Electrophysiological Study

To determine whether ventricular tachycardia was inducible, 313 patients underwent programmed stimulation at electrophysiological study. Patients were fasted and sedated with 10 mg oral diazepam; they had taken no cardioactive medication (including conventional antiarrhythmic agents, β-blockers, and calcium antagonists) other than digoxin for at least 5 days before study. Two separate stimulation protocols were applied to each patient in random order with a 5–10-minute interval between completion of one protocol and commencement of the other. The first protocol (described below) was reported in earlier studies of programmed stimulation after myocardial infarction.[2,10] The second, which used increasingly aggressive stimulation, was chosen to permit a stepwise analysis of the predictive value of programmed stimulation and to identify an optimal protocol for programmed stimulation after myocardial infarction.

Protocol 1 comprised single and paired extrastimuli introduced in 10-msec decrements, from 300 msec to refractoriness, first at the right ventricular apex and then at the right ventricular outflow tract; the first stimulus was at twice diastolic current threshold and the second was at 20 mA current intensity.[2,10]

In protocol 2, up to five extrastimuli were introduced in 10-msec decrements from 300 msec to refractoriness. Each extrastimulus was applied three times at each coupling interval before decreasing to the next coupling interval.[11] With three extrastimuli, no sustained arrhythmia was inducible in 158 patients, ventricular tachycardia with cycle length of less than 230 msec was inducible in 23 patients, and ventricular flutter or fibrillation was inducible in 132 patients. If ventricular tachycardia was not induced with the nth extrastimulus, the nth extrastimulus was set 10 msec outside the ventricular effective refractory period, and a further extrastimulus was introduced from 300 msec in 10-msec decrements to ventricular refractoriness (maximum of five extrastimuli).

The end points for stimulation with both protocols were reached either when the protocol was completed without induction of ventricular tachycardia, ventricular flutter, or ventricular fibrillation, or when the protocol induced ventricular fibrillation, ventricular flutter, or at least 10 seconds of ventricular tachycardia. Only inducible ventricular tachycardia with cycle length greater than or equal to 230 msec was recorded as abnormal.[4] All other results (no inducible ventricular tachycardia, inducible ventricular tachycardia with cycle length less than 230 msec, ventricular flutter, or ventricular fibrillation) were recorded as normal.[4] Programmed stimulation was stopped if any of the end points (including ventricular flutter or ventricular fibrillation) was reached

irrespective of whether that end point represented a positive or a negative result.

All patients remained in bed overnight after the electrophysiological study. No study was complicated by any significant complication (death, stroke, cardiac tamponade, significant bleeding or thrombosis, infection, local nerve damage, or hematoma >10 cm). Cardioversion by countershock was not attended by any significant morbid sequelae. Most patients reported some discomfort during electrophysiological study, but a large majority said they would be prepared to undergo further study if required.

Signal-Averaged Vectorcardiogram

Frank vectorcardiographic recordings (5 minutes) were digitized (1,000 samples/sec) and averaged.[12] The duration of ventricular activation was expressed as the interval between the earliest onset of ventricular activation in any lead and the latest offset of ventricular activation in any lead.[12] Ventricular activation times greater than or equal to 120 msec were recorded as prolonged.

Holter Monitoring

Ambulatory electrocardiograms (two channels) were recorded for 24 hours before discharge. The frequency and nature of ventricular ectopic beats were noted and summarized as follows[13]: grade 0, no ventricular ectopic beats; grade 1, less than 30 ventricular ectopic beats/hr; grade 2, 30–60 ventricular ectopic beats/hr; grade 3, greater than 60 ventricular ectopic beats/hr; grade 4a, paired ventricular ectopic beats; grade 4b, salvos of three or more ventricular ectopic beats; grade 5, R-on-T ventricular ectopic beats. Grades 3–5 were recorded as abnormal.

Gated Heart-Pool Scan

Left ventricular ejection fraction was measured at radionuclide-gated heart-pool scan. In addition, ventricular aneurysms (regions of systolic expansion) were noted. Ejection fractions less than or equal to 0.40 were recorded as low.[9]

Exercise Test

Symptom- or sign-limited exercise tests (graded-treadmill, modified Naughton protocol[14,15]) were performed at 1 week and at 7 weeks after discharge from the hospital.[16] ST segment displacement of at least 2 mm at either test during exercise was recorded as abnormal; this classification is consistent with our previous work wherein the prognostic value of ST segment displacement of 2 mm was greater than that for displacement of only 1 mm.[16]

Follow-up

Patients were followed up for as long as 775 days (median, 740 days; lower quartile, up to 511 days) or until death. Spontaneous ventricular tachycardia was defined as documented sustained ventricular tachycardia, in the absence of chest pain or other prodrome, causing hemodynamic embarrassment and requiring urgent treatment. Modes of death were established from written records and by interview of witnesses and attending physicians. Patients who were observed to die virtually instantaneously, in the absence of any prodrome, were said to have suffered witnessed instantaneous death. Patients who complained of chest pain before death were said to have died of fresh myocardial ischemia. Patients whose deaths were primarily due to progressive left ventricular dysfunction were said to have died of heart failure.

Statistics

Survival data were analyzed using a statistical package for interactive data analysis (SPIDA[17]). This permitted life-table analysis (Kaplan–Meier) and univariate and multivariate analyses using proportional hazards models (Cox regressions).

Therapy

Patients with persistent ischemia, evident at exercise test or elsewhere, were treated with medications, angioplasty, or surgical revascularization as necessary. During follow-up, 136 of the 313 patients who underwent electrophysiological study received β-blockers at some stage, according to individual physician preference. However, β-blockers were not prescribed routinely to patients with or without inducible ventricular tachycardia or complex ventricular ectopy in the absence of evidence of spontaneous ventricular tachycardia or further ischemia. β-Blocker therapy, when prescribed, was not guided by the results of electrophysiological study.

The incidence of β-blockade was not significantly different between those who did (nine of 27) and those who did not (127 of 287) have inducible ventricular tachycardia at electrophysiological study, or between those who did (five of eight) and those who did not (131 of 305) subsequently exhibit electrical events. Therapy for spontaneous ventricular tachycardia during follow-up was directed by serial electrophysiological studies.

Results

Three hundred sixty-one survivors of acute myocardial infarction who were less than 71 years of age were studied. During follow-up, there were 34 deaths (eight witnessed instantaneous, 12 fresh myocardial ischemia, seven cardiac failure, three post–surgical revascularization, two unwitnessed, two other), and nine patients survived one or more episodes of spontaneous ventricular fibrillation or ventricular tachycardia.

Prediction of Electrical Events

Electrical events were defined as spontaneous ventricular fibrillation or ventricular tachycardia ($n=9$) or witnessed instantaneous death ($n=8$). The relative risks of electrical events for each of the variables are summarized in Table 3. Inducible ventricular tachy-

TABLE 3. Relative Risk of an Electrical Event After Acute Myocardial Infarction

	Risk	*p*
Univariate analysis		
EPS	15.2	<0.001
LVEF	4.8	0.002
Heart size	4.5	0.002
VCG	4.4	0.003
Holter	3.1	0.08
Aneurysm	1.8	0.4
Exercise test	1.0	1.0
Failure	2.0	0.2
Previous AMI	2.0	0.2
Female sex	1.0	1.0
Multivariate analysis		
EPS	10.4	<0.001
LVEF	3.2	0.05

An electrical event is defined as spontaneous ventricular fibrillation, ventricular tachycardia, or witnessed instantaneous death. EPS, inducible ventricular tachycardia with a cycle length ≥230 msec at electrophysiological study; LVEF, low left ventricular ejection fraction at gated heart-pool scan; Heart size, cardiomegaly on chest x-ray; VCG, prolonged ventricular activation time on signal-averaged vectorcardiogram; Holter, high-grade ectopy on 24-hour ambulatory electrocardiogram; Aneurysm, ventricular systolic expansion at gated heart-pool scan; Exercise test, abnormal ST displacement on exercise test; Failure, pulmonary venous congestion or pulmonary edema on chest x-ray; Previous AMI, previous acute myocardial infarction.

cardia, low ejection fraction, and delayed ventricular activation were all associated with a greatly increased risk of electrical events. At multivariate analysis, inclusion of variables other than inducible ventricular tachycardia and low ejection fraction did not significantly improve the predictive value of any proportional-hazards model including inducible ventricular tachycardia and low ejection fraction. No model that excluded inducible ventricular tachycardia predicted electrical events as well as any model that did include inducible ventricular tachycardia.

To examine the potential impact of using low ejection fraction as a screening test before testing for inducible ventricular tachycardia, the predictive value of inducible ventricular tachycardia was assessed in the subset of patients with low ejection fractions (n=95). Whereas seven of 13 electrical events during the first year would have been predicted by inducible ventricular tachycardia alone, only one of those 13 electrical events would have been missed had inducible ventricular tachycardia been assessed only in patients with low ejection fraction. The one patient with normal ejection fraction and inducible ventricular tachycardia who exhibited an electrical event during the first year of follow-up had spontaneous ventricular fibrillation requiring cardioversion by countershock. Life-table analysis of the 95 patients with low ejection fraction showed that the probability of 1-year survival without an electrical event for patients with inducible ventricular tachycardia (n=13) was 54%, and for those without ventricular tachycardia (n=82), the probability was 95%.

TABLE 4. Prediction of Electrical Events Within 1 Year After Acute Myocardial Infarction

	Sensitivity (%)	Specificity (%)	Predictive accuracy	
			Positive (%)	Negative (%)
EPS	58	95	30	98
LVEF	71	74	11	98
VCG	57	82	17	97
Holter	82	40	6	98
Aneurysm	14	93	7	96
Exercise test	20	83	2	98

EPS, inducible ventricular tachycardia at electrophysiological study; LVEF, low left ventricular ejection fraction at gated heart-pool scan; VCG, prolonged ventricular activation time on signal-averaged vectorcardiogram; Holter, high-grade ventricular ectopy on 24-hour ambulatory electrocardiogram; Aneurysm, ventricular systolic expansion at gated heart-pool scan; Exercise test, abnormal ST displacement on exercise test.

Table 4 compares the predictive value, at 1 year, of inducible ventricular tachycardia, low ejection fraction, delayed ventricular activation, high-grade ventricular ectopy, presence of ventricular aneurysm, and positive exercise test in terms of sensitivity, specificity, predictive accuracy of a positive test, and predictive accuracy of a negative test. To standardize the presentation of data, the indexes shown in this table were derived from contingency tables for up to 1 year of follow-up. For the same period, in the subset of patients with low ejection fraction, the sensitivity, specificity, positive predictive accuracy, and negative predictive accuracy (of inducible ventricular tachycardia for witnessed instantaneous death or spontaneous ventricular tachycardia or fibrillation) were 67%, 91%, 46%, and 96%, respectively.

The changes in the relative risk of an electrical event with a decade increase in age and a 10% increase in left ventricular ejection fraction was 1.24 and 0.58, respectively.

Prediction of Cardiac Death

Cardiac death was defined as witnessed instantaneous death (n=8), death due to fresh myocardial ischemia (n=12), or death due to cardiac failure (n=7).

The relative risks of death for each of the variables are summarized in Table 5. Delayed ventricular activation, inducible ventricular tachycardia, and low ejection fraction were all associated with a highly significantly increased risk of death during follow-up. At multivariate analyses, these were the only variables that exhibited independent predictive value. The addition of other variables did not improve the predictive value of any proportional-hazards model including any two of the following: delayed ventricular activation, inducible ventricular tachycardia, and low ejection fraction.

TABLE 5. Relative Risk of Cardiac Death After Myocardial Infarction

	Risk	*p*
Univariate analysis		
VCG	7.0	<0.001
EPS	5.6	<0.001
LVEF	5.2	<0.001
Heart size	2.8	0.01
Aneurysm	2.9	0.04
Holter	2.2	0.08
Exercise test	0.9	0.6
Failure	1.9	0.09
Previous AMI	2.2	0.06
Female sex	0.6	0.4
Multivariate analysis groupings		
VCG	5.6	0.001
EPS	4.2	0.01
LVEF	4.7	0.001
EPS	3.5	0.01
VCG	4.5	0.003
LVEF	3.1	0.03

Cardiac death is defined as witnessed instantaneous death, death due to fresh myocardial ischemia, or death due to cardiac failure. VCG, prolonged ventricular activation time on signal-averaged vectorcardiogram; EPS, inducible ventricular tachycardia with a cycle length ≥230 msec at electrophysiological study; LVEF, low left ventricular ejection fraction at gated heart-pool scan; Heart size, cardiomegaly on chest x-ray; Aneurysm, ventricular systolic expansion at gated heart-pool scan; Holter, high-grade ectopy on 24-hour ambulatory electrocardiogram; Exercise test, abnormal ST displacement on exercise test; Failure, pulmonary venous congestion or pulmonary edema on chest x-ray; Previous AMI, previous acute myocardial infarction. In each of the three multivariate analyses shown, the addition of other variables (beyond the two depicted in each case) did not improve the predictive values of the models.

Table 6 summarizes, at 1 year, the predictive value of inducible ventricular tachycardia, low ejection fraction, delayed ventricular activation, high-grade ventricular ectopy, presence of ventricular aneurysm, and positive exercise test in terms of sensitivity, specificity, predictive accuracy of a positive test, and predictive accuracy of a negative test. As for electrical events (above), to standardize the presentation of data, the indexes shown in this table were derived from contingency tables for up to 1 year of follow-up.

TABLE 6. Prediction of Cardiac Deaths Within 1 Year After Myocardial Infarction

	Sensitivity (%)	Specificity (%)	Predictive accuracy	
			Positive (%)	Negative (%)
EPS	25	94	17	96
LVEF	75	76	16	98
VCG	71	83	22	98
Holter	81	40	8	97
Aneurysm	20	93	15	95
Exercise test	23	83	6	96

EPS, inducible ventricular tachycardia with a cycle length ≥230 msec at electrophysiological study; LVEF, low left ventricular ejection fraction at gated heart-pool scan; VCG, prolonged ventricular activation time on signal-averaged vectorcardiogram; Holter, high-grade ectopy on 24-hour ambulatory electrocardiogram; Aneurysm, ventricular systolic expansion at gated heart-pool scan; Exercise test, abnormal ST displacement on exercise test.

TABLE 7. Stepwise Analysis of Prediction of Electrical Events After Acute Myocardial Infarction

	Sensitivity (%)	Specificity (%)	Predictive accuracy	
			Positive (%)	Negative (%)
EPS 1				
Low current	27	96	20	97
High current	27	95	18	97
EPS 2				
1	8	100	100	96
2	25	98	30	97
3	58	95	30	98
4	58	94	28	98
5	58	93	26	98

EPS 1, electrophysiological study protocol 1 (see "Methods" for discussion of protocols); EPS 2, electrophysiological study protocol 2; 1–5, extrastimuli introduced in 10-msec decrements from 300 msec to refractoriness.

The changes in the relative risk of cardiac death with a decade increase in age and a 10% increase in left ventricular ejection fraction were 1.14 and 0.49, respectively.

Stepwise Analysis of the Predictive Value of Programmed Stimulation

Table 7 summarizes the stepwise analysis of the value of programmed stimulation to predict electrical events up to 1 year after infarction in terms of sensitivity, specificity, positive predictive accuracy, and negative predictive accuracy. Ventricular tachycardia with a cycle length greater than or equal to 230 msec was always considered abnormal,[4] irrespective of the current intensity or number of extrastimuli required to induce ventricular tachycardia. Seven of 23 patients with ventricular tachycardia inducible with three extrastimuli experienced an electrical event. With four extrastimuli, two more patients had inducible ventricular tachycardia, neither of whom exhibited an electrical event subsequently. With five extrastimuli, another two patients had inducible ventricular tachycardia, neither of whom exhibited an electrical event subsequently. Two hundred sixty-six patients were tested with three extrastimuli, 158 patients were tested with four extrastimuli, and 92 patients were tested with five extrastimuli.

The specificity and negative predictive accuracy of programmed stimulation were high irrespective of the current intensity or the number of extrastimuli. However, maximal sensitivity and positive predictive accuracy were achieved when three extrastimuli were introduced at twice diastolic current threshold. When more than three extrastimuli were used, sensitivity was maintained, but positive predictive accuracy began to fall.

Discussion

This study confirms previous reports from ourselves[2,4] and others[1,3,5] that patients with inducible ventricular tachycardia after myocardial infarction are much more likely to die suddenly or to exhibit spontaneous ventricular tachycardia or fibrillation (electrical events) than are patients without inducible ventricular tachycardia. In the present study, patients with ventricular tachycardia inducible with three extrastimuli ($n=23$) were 15 times more likely to suffer electrical events than patients without inducible ventricular tachycardia ($n=290$).

Electrophysiological Study as a Prognostic Indicator

The present study comprises a fresh cohort of patients, none of whom was included in our previous series.[2,4] In our studies throughout the last 10 years, we have reported our experience with programmed stimulation in a total of 881 patients (three separate cohorts) after myocardial infarction. Our consistent finding that inducible ventricular tachycardia is associated with subsequent spontaneous ventricular tachycardia and sudden death suggests that our patient samples are indeed representative of survivors of uncomplicated myocardial infarction (no heart failure, no postinfarctional angina requiring regular therapy, and no cardioactive medications other than digoxin).

Spontaneous ventricular tachycardia in the absence of fresh myocardial ischemia is due to reentry through patchy scar tissue at the edge of the infarct.[18,19] This patchy scar tissue represents the substrate for reentrant ventricular tachycardia. Our studies suggest that inducible ventricular tachycardia not only is an indicator for this substrate but also is an indicator for predisposition to spontaneous ventricular tachycardia, even in patients who have never experienced spontaneous ventricular tachycardia previously.

The results of the present study suggest that our earlier protocol (protocol 1) of programmed stimulation[2,10] is no longer sufficiently aggressive to obtain adequate sensitivity to predict sudden death or spontaneous ventricular tachyarrhythmias. Our protocol 2 with three extrastimuli applied three times at each coupling interval appears to offer the optimal balance of positive and negative predictive accuracy. Why have other investigators[20–24] not confirmed our findings? The reasons for this may be complex and have been discussed previously in detail.[25] However, the following points are important. The overall incidence of electrical events during the year after infarction is now less than 5%. This lower mortality nowadays, compared with that found previously,[2] may be due to a change in the nature of the infarct population or due to variations in therapy that may alter infarct morphology and reduce predisposition to ventricular tachycardia (e.g., thrombolytic therapy). Also, reentrant ventricular tachycardia, although the most common cause,[26] is not the only cause of sudden death late after myocardial infarction. Cardiac rupture, aortic dissection, bradycardia, stroke, and thromboembolism may all occur.

Even in patients with a history of spontaneous ventricular tachycardia in the context of chronic myocardial infarction, the likelihood of recurrence within a year is probably only about 35%.[26,27] Therefore, to obtain any useful information from programmed stimulation, one must study relatively large numbers of representative patients and accept that a positive predictive accuracy of the order of 35% is the maximum to be expected from any single test after infarction. Furthermore, at electrophysiological study, adequately aggressive programmed stimulation must be used. The present data suggest that an optimal protocol for programmed stimulation should include three extrastimuli. As we have suggested before,[4] an inducible ventricular tachycardia cycle length of 230 msec or more sustained for at least 10 seconds should be considered abnormal. Ventricular tachycardia cycle length of less than 230 msec, ventricular flutter, ventricular fibrillation, or completion of the protocol with no inducible ventricular fibrillation or tachycardia should all be considered normal in patients 2–4 weeks after myocardial infarction.[2,4]

Other Prognostic Indicators

Given that dense scar tissue is electrically inert and that slow conduction through patchy scar tissue at the edge of the infarct is a necessary condition for reentry, it is probable that the incidence of spontaneous ventricular tachycardia would be highest in patients with more rather than less patchy scar tissue. This is supported by the observation that nonspecific markers for extensive myocardial damage such as low ejection fraction at gated heart-pool scan, cardiomegaly on chest x-ray, delayed ventricular activation at the signal-averaged vectorcardiogram, and high-grade ventricular ectopy on Holter monitoring are all associated with a three to five times increased risk of electrical events after infarction.

However, if slowly conducting viable muscle fibers at the edge of the infarct are not in electrical continuity with other muscle fibers to complete a reentrant circuit, the presence of delayed potentials will not be a sufficient condition to indicate the substrate for ventricular tachycardia.

This study suggests that although the presence of delayed potentials in sinus rhythm is a useful predictor of electrical events, the presence of delayed potentials is even more useful for identifying patients at risk for cardiac death. In the analysis of the value of various tests to predict cardiac death, the relative risks were similar for delayed potentials, inducible ventricular tachycardia, and low ejection fraction. Each of these variables correlates with myocardial damage. At multivariate analysis, inducible ventricular tachycardia or low ejection fraction, along with delayed ventricular activation, accounted for nearly all of the increased risk for cardiac death. Further

studies will be required to confirm the results of the present multivariate analysis.

Exercise testing did not predict outcome. This was probably because patients with reversible myocardial ischemia that was revealed at exercise testing were successfully treated with medical therapy, angioplasty, or surgical revascularization as required.

Clinical Implications

Two important clinical questions are raised by this study: 1) Which patients should be evaluated by electrophysiological study after infarction? We suggest that all patients with low ejection fraction, provided there is no uncontrolled cardiac failure or persistent myocardial ischemia, should be eligible to enter into prospective clinical trials of treatment strategies to prevent spontaneous ventricular tachycardia and sudden death (e.g., drug therapy, automatic implantable defibrillator, myocardial resection, and cryoablation). 2) How should patients with inducible (but without spontaneous) ventricular tachycardia be treated? We have previously shown that class I antiarrhythmic therapy (not electrophysiologically directed) does not reduce the risk of spontaneous ventricular tachycardia or sudden death in patients with inducible (but without previous spontaneous) ventricular tachycardia after myocardial infarction.[28] There is a pressing need for prospective studies to evaluate the ability of electrophysiologically directed medical therapy to prevent ventricular tachycardia and sudden death in patients with inducible (but without previous spontaneous) ventricular tachycardia after myocardial infarction.

Conclusion

We conclude that inducible ventricular tachycardia at electrophysiological study is the best single predictor of spontaneous ventricular tachycardia and sudden death after myocardial infarction. Further studies are required to identify therapies to reduce the risk of sudden death in patients with inducible ventricular tachycardia after myocardial infarction.

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