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Differences Between Patients With Ventricular Tachycardia and Ventricular Fibrillation as Assessed by Signal-Averaged Electrocardiogram, Radionuclide Ventriculography and Cardiac Mapping

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This study examined 65 patients with ventricular tachycardia or fibrillation late after myocardial infarction to determine whether they differed with respect to duration of ventricular activation in sinus rhythm and left ventricular ejection fraction. Patients with spontaneous ventricular tachycardia had a longer ventricular activation time in sinus rhythm than did patients with spontaneous ventricular fibrillation. This difference was detected with the signal-averaged electrocardiogram (ECG) (tachycardia 181 ± 33 ms, fibrillation 152 ± 23 ms, $p < 0.001$) and at epicardial mapping (tachycardia 210 ± 17 ms, fibrillation 192 ± 17 ms, $p < 0.02$). Left ventricular ejection fraction was lower in patients with spontaneous ventricular tachycardia (0.22 ± 0.09) than in patients with spontaneous ventricular fibrillation (0.27 ± 0.09) ($p < 0.05$).

The patients with both spontaneous and inducible ventricular fibrillation had a shorter ventricular activation time on the signal-averaged ECG (129 ± 17 ms) and a higher ejection fraction (0.36 ± 0.05) than did either patients with spontaneous ventricular fibrillation and inducible ventricular tachycardia (158 ± 21 ms and 0.25 ± 0.08, respectively, each $p < 0.01$) or patients with both spontaneous and inducible ventricular tachycardia (181 ± 33 ms and 0.22 ± 0.09, respectively, each $p < 0.001$). Of the patients with inducible ventricular tachycardia, presentation with tachycardia rather than fibrillation was associated with a longer ventricular activation time on the signal-averaged ECG (181 ± 33 versus 158 ± 21 ms, $p < 0.02$) and a longer cycle length of inducible ventricular tachycardia (290 ± 61 versus 259 ± 44 ms, $p = 0.05$).

In conclusion, conduction delay during sinus rhythm and left ventricular dysfunction appear to be greatest in patients with spontaneous and inducible ventricular tachycardia, and least in patients with spontaneous and inducible ventricular fibrillation.

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Patients with spontaneous ventricular tachyarrhythmias occurring late after myocardial infarction in the absence of further ischemia have fragmented and delayed electrograms detectable during sinus rhythm. These can be recorded either directly from the heart at cardiac mapping or from the body surface using signal-averaging techniques (1–9). It is thought that these low amplitude delayed potentials arise from areas of myocardium that exhibit one of the prerequisites for reentry, namely, slow conduction during sinus rhythm (2,4,5,10). There is evidence from an endocardial mapping study (8) that the degree of conduction delay during sinus rhythm may be a factor determining whether patients have ventricular tachycardia or ventricular fibrillation.

The present study was designed to address the question of whether there are electrophysiologic and anatomic differences between patients with spontaneous ventricular tachycardia and ventricular fibrillation arising late after myocardial infarction. Duration of ventricular activation in sinus rhythm was measured noninvasively by the signal-averaged electrocardiogram and correlated with the findings at epicardial mapping. In addition, the degree of left ventricular

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dysfunction in patients with ventricular tachycardia and ventricular fibrillation was gauged by measurement of left ventricular ejection fraction. The study not only analyzed subgroups of patients categorized according to the spontaneous arrhythmia, but also examined subgroups classified according to the arrhythmia induced at programmed ventricular stimulation. Unlike the previous endocardial mapping study (8), no patients were on antiarrhythmic medications or beta-adrenergic blocking drugs while under investigation.

Methods

Study patients. The study group consisted of 65 of 72 consecutive patients with spontaneous sustained ventricular tachycardia or fibrillation occurring at a mean of 20 weeks (range 2 weeks to 14 years) after myocardial infarction. The seven patients excluded from the study included two patients with spontaneous ventricular fibrillation and five with ventricular tachycardia who did not have inducible arrhythmias at programmed stimulation. The spontaneous arrhythmias were not associated with antecedent chest pain or electrocardiographic (ECG) evidence of recurrent myocardial ischemia. At the time of these arrhythmias, two patients had been taking beta-adrenergic blockers, but none of the other patients had been taking antiarrhythmic medications. Thirty-three patients had presented with symptomatic sustained ventricular tachycardia that did not degenerate into ventricular fibrillation. Ventricular tachycardia had lasted ≥30 s and had been documented either on a monitor strip or on a 12 lead ECG. The remaining 32 patients had had a cardiac arrest with documented ventricular fibrillation. The mean age of the study group was 58 years (range 35 to 71). The site of old myocardial infarction (confirmed by ventriculography) was anterior in 33 patients (51%), inferior in 26 (40%) and both anterior and inferior in 6 (9%). Three patients had an intraventricular conduction defect, with surface QRS duration exceeding 120 ms. No patients had bundle branch block.

All 65 study patients had inducible ventricular tachyarrhythmias at programmed stimulation. The 65 patients underwent signal averaging of the surface ECG and radionuclide ventriculography, and 55 of the patients also had coronary arteriography. Twenty-six of the study patients (14 with ventricular tachycardia and 12 with ventricular fibrillation) also underwent cardiac mapping for medically refractory spontaneous ventricular tachyarrhythmias, enabling the configuration of the spontaneous arrhythmia to be correlated with epicardial ventricular activation time. Beta-adrenergic blockers, digoxin and other antiarrhythmic medications were stopped for at least 7 days before programmed stimulation, signal averaging and cardiac mapping. No patients had been taking amiodarone.

Programmed stimulation. The stimulation protocol has been described in detail previously (11). Drive trains of eight stimuli set as long as possible (usually 600 ms) were followed by single then paired extrastimuli. The extrastimuli were applied first at the right ventricular apex and then at the right ventricular outflow tract. This sequence was applied first at twice diastolic threshold (1 to 4 mA) and was then repeated at 20 mA. Stimuli were 2 ms rectangular pulses and were delivered from a Medtronic 5325 programmable stimulator. There was a 3 s delay between each pacing sequence.

This protocol, which uses a maximum of two extrastimuli, does not result in induction of ventricular fibrillation in patients with normal ventricles, even when high current stimulation is used (11). In patients with previous myocardial infarction who have not had spontaneous arrhythmias, the incidence of inducible ventricular fibrillation attributable to a 20 mA stimulation is only 8% (12). As in our previous studies (9,11), an inducible arrhythmia was said to be present if ventricular tachycardia or fibrillation lasting >10 s was induced before completion of the protocol because this was clearly an abnormal response with this protocol (11). Inducible arrhythmias lasting >10 s were terminated promptly by pacing or direct current cardioversion without neurologic or other significant sequelae.

The body surface Frank vectorcardiogram and intracardiac electrograms were recorded simultaneously at a paper speed of 250 mm/s. An induced arrhythmia was classified as ventricular tachycardia or ventricular fibrillation, depending on the initial QRS configuration (12). Ventricular tachycardia was said to be present if there was a well organized rhythm with a constant or nearly constant (±5%) cycle length and a stable surface ECG with a constant relation of inscription of the QRS complexes in the three leads. The classification of an arrhythmia as ventricular tachycardia was not altered if ventricular fibrillation subsequently supervened. Ventricular fibrillation was said to be present if, from the outset, there was a disorganized rhythm with irregularly timed endocardial electrograms and either no clearly defined QRS complexes on the surface ECG or the presence of QRS complexes of continuously varying configuration. Fast polymorphic arrhythmias were classified as ventricular fibrillation (12), and invariably required cardioversion for termination.

Eleven of 15 consecutive patients who were asked to undergo a second programmed stimulation study while taking no medications consented to have repeat stimulation 3 to 7 days after the initial study.

ECG signal averaging. Signal averaging was performed on the same day as programmed stimulation. Leads X, Y and Z of the Frank vectorcardiogram were recorded for 5 min, filtered (0.05 to 500 Hz) and then digitized simultaneously at 1,000 samples/s. Signal averaging was performed using a PDP 11/34 computer (Fortran averaging program) after an iterative cross-correlation procedure was utilized to optimize QRS alignment (13). The QRS onset and offset in the averaged recordings were determined manually by display-

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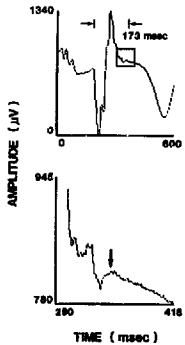


Figure 1. Signal-averaged Y lead traces from a patient with ventricular tachycardia. **Upper trace,** Signal-averaged Y lead trace at low amplification. Ventricular activation time in this lead was 173 ms, the determinations of QRS onset and offset (**arrows**) being made at high amplification. **Lower trace.** The QRS offset shown at high amplification of the **boxed segment** in the **upper trace.** The QRS offset (**arrow**) occurs at the end of low amplitude signals that extend into the ST segment. These signals were well above the noise level of 0.5 μV.

ing segments of each trace at high amplification. For each patient, ventricular activation time (ms) was measured as the total time from the earliest QRS onset in any lead to the latest QRS offset in any lead. The QRS offset was measured to the end of any delayed, low amplitude, high frequency signals extending into the ST segment (delayed potentials) (Fig. 1) provided they had an amplitude more than twice that of the simultaneously displayed noise level, which was usually 0.5 to 1.0 μV. Determination of ventricular activation time by this method does not result in significant interobserver variability (14). For patients with ventricular tachyarrhythmias, the standard error of a single estimate of ventricular activation time on any day by one observer was 9 ms (14).

Cardiac mapping. Intraoperative cardiac mapping was performed with the patient on cardiopulmonary bypass at normothermia. Standard limb electrodes and a posterior chest electrode were used to record body surface electrograms (0.05 to 500 Hz). Bipolar reference electrodes (interelectrode distance of 1 mm) were sewn onto the right ventricular epicardium, and hand-held bipolar probe electrodes (interelectrode distance of 1 mm) were used to record local electrograms (50 to 500 Hz).

As described in detail previously (15), amplified body surface electrograms and epicardial electrograms were transmitted through a delay circuit to a Tektronix 5103N dual beam, eight channel storage oscilloscope. The right ventricular reference electrogram was used to trigger the oscilloscope, and the delay was adjusted so that the onset of the body surface QRS complex coincided with a reference pulse exactly 50 ms after the beginning of each sweep across the oscilloscope. Heart rhythm was monitored using a second oscilloscope (Hewlett Packard HP1209A). The exact timing of local cardiac electrograms was determined by a programmable digital clock, which could be started by one cardiac electrogram or reference pulse and stopped by another cardiac electrogram or reference pulse (16). Baseline stability for making these measurements was achieved by using a 50 Hz high pass filter.

Mapping during sinus rhythm was performed at 25 stan-

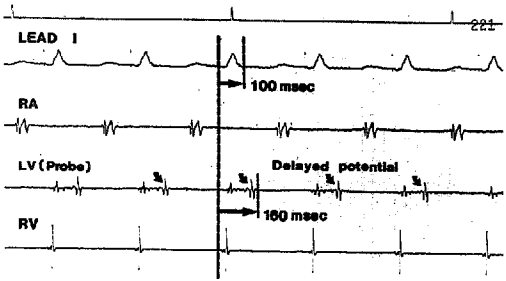


Figure 2. Delayed ventricular activation observed at epicardial mapping. Lead I of the surface ECG is depicted together with epicardial electrograms recorded from the right atrium (RA), left ventricle (LV) by means of a probe and right ventricle (RV). The QRS duration on the surface ECG was 100 ms, but the ventricular activation time was prolonged to 160 ms because of a delayed potential (**curved arrows**) at the left ventricular site from which the probe electrogram was recorded.

Table 1. Differences Between Patient Groups With Spontaneous Ventricular Tachycardia and Spontaneous Ventricular Fibrillation

	Spontaneous VT	Spontaneous VF	p Value
No. of patients	33	32	
Anterior infarction	18 (55%)	15 (47%)	NS
Presentation 2 to 8 weeks after MI	15 (45%)	13 (41%)	NS
Age (yr)	58 ± 6	56 ± 6	NS
Male (no.)	26 (79%)	29 (91%)	NS
QRS duration (ms)	98 ± 30	102 ± 10	NS
QT interval (ms)	377 ± 46	380 ± 40	NS
QTc (ms)	445 ± 40	457 ± 34	NS
No. of diseased coronary vessels	1.9 ± 1.1 (n = 26)	1.9 ± 1.0 (n = 29)	NS
Ventricular activation time (ms)			
Signal-averaged ECG	181 ± 33	152 ± 23	<0.001
Epicardial mapping	210 ± 17 (n = 14)	192 ± 17 (n = 12)	<0.02
LV ejection fraction	0.22 ± 0.09	0.27 ± 0.09	<0.05
LV aneurysm	19 (58%)	12 (38%)	NS

ECG = electrocardiogram; LV = left ventricular; MI = myocardial infarction; NS = not significant; QTc = QT interval corrected for heart rate; VF = ventricular fibrillation; VT = ventricular tachycardia. Values are reported as mean ± standard deviation.

dard epicardial sites (15), with the probe electrogram being amplified by a factor of 1,000 to facilitate detection of fractionated electrograms extending past QRS offset into the ST segment (delayed potentials) (Fig. 2). For a particular patient, ventricular activation time was measured as the time from QRS onset to the end of the latest high frequency electrographic component at any of the 25 sites mapped.

Radionuclide ventriculography. Left ventricular ejection fraction at rest was quantitated during sinus rhythm using technetium-99m radionuclide ventriculography and computer-generated time-activity curves. This method has been found to be reliable for identifying left ventricular aneurysms and for quantifying ejection fraction in the presence of an aneurysm (17). The normal range of left ventricular ejection fraction was 0.50 to 0.65. A left ventricular aneurysm was said to be present if there was dyskinetic wall motion.

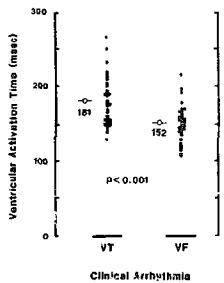
Cardiac catheterization. Significant coronary artery disease at coronary arteriography was defined as >50% reduction in luminal diameter of the left main coronary artery or >75% reduction in luminal diameter in any other major coronary artery.

Statistics. Paired and unpaired *t* tests were used to compare continuous variables, with the Bonferroni procedure being applied for multiple simultaneous comparisons (18). For discrete variables, a Fisher's exact test was used. Statistical significance was defined as a p value <0.05.

Results

Spontaneous arrhythmias. Table 1 shows the differences between the patient groups with spontaneous ventricular tachycardia and spontaneous ventricular fibrillation. Patients with spontaneous ventricular tachycardia had a longer ventricular activation time during sinus rhythm than did patients with spontaneous ventricular fibrillation. This was observed when ventricular activation time was measured from the signal-averaged ECG (Fig. 3) and at epicardial mapping. The differences in ventricular activation times between patients with ventricular tachycardia and ventricular fibrillation were due to differences in delayed potentials because QRS durations were almost identical in the two groups of patients (Table 1). In the 26 patients undergoing cardiac mapping, ventricular activation time obtained from the signal-averaged ECG underestimated epicardial ventricular activation time (by approximately 30 ms) (Fig. 4). The number of epicardial sites at which delayed potentials were recorded was 9.2 ± 5.3 for patients with ventricular tachy-

Figure 3. Ventricular activation time determined from the signal-averaged ECG in patients whose clinical arrhythmia was ventricular tachycardia (VT) (n = 33) or ventricular fibrillation (VF) (n = 32).



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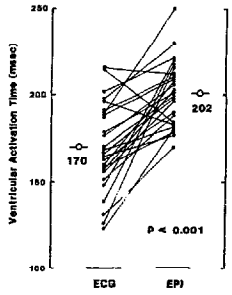


Figure 4. Comparison of ventricular activation times obtained from the signal-averaged electrocardiogram (ECG) and epicardial mapping (EPI) in 26 patients.

cardia and 6.2 ± 4.0 for patients with ventricular fibrillation ($p = 0.13$).

Patients with spontaneous ventricular tachycardia had a lower mean ejection fraction than did patients with spontaneous ventricular fibrillation (Table 1).

Correlation between configuration of spontaneous and inducible arrhythmias (Table 2). Of the 33 patients with spontaneous ventricular tachycardia, 32 (97%) had inducible monomorphic ventricular tachycardia. Of the 32 patients with spontaneous ventricular fibrillation, 25 (78%) had inducible monomorphic ventricular tachycardia and 7 (22%) had inducible ventricular fibrillation. The incidence of inducible ventricular fibrillation was similar for patients presenting 1 to 8 weeks after myocardial infarction (3 [11%] of 28) and for patients presenting >8 weeks after infarction (5 [14%] of 37).

Of the 11 patients who consented to have a repeat programmed stimulation study, 10 had inducible monomorphic ventricular tachycardia and 1 had inducible ventricular fibrillation at the initial study. At repeat study, ventricular tachycardia was again inducible in the 10 patients with tachycardia at initial study, and inducible ventricular fibrillation was reproducibly initiated in the remaining patient.

Table 2. Arrhythmias Inducible at Programmed Stimulation

	Spontaneous Arrhythmia		
Inducible Arrhythmia	VT	VF	Total
VT	32	25	57
VF	1	7	8
	33	32	65

Abbreviations as in Table 1.

Inducible arrhythmias. Table 3 shows the analysis for patients grouped according to the configuration of the inducible arrhythmia. Excluded from this analysis is the one patient with spontaneous ventricular tachycardia and inducible ventricular fibrillation. In this patient, spontaneous ventricular tachycardia was always self-terminating and ventricular fibrillation was inducible at two electrophysiologic studies. The remaining 64 patients were grouped according to whether they had both spontaneous and inducible ventricular tachycardia (n = 32), spontaneous ventricular fibrillation and inducible ventricular tachycardia (n = 25) and both spontaneous and inducible ventricular fibrillation (n = 7).

Of the 57 patients with inducible ventricular tachycardia (Table 3), the 32 who presented with spontaneous ventricular tachycardia had a longer mean ventricular activation time and a longer mean cycle length of inducible ventricular tachycardia than did the 25 patients who presented with ventricular fibrillation. The small group of seven patients with both spontaneous and inducible ventricular fibrillation appeared to represent a different group than the other two groups, having a low incidence of anterior infarction and left ventricular aneurysm, a higher mean left ventricular ejection fraction and a lower mean ventricular activation time.

Correlation between cycle length of inducible ventricular tachycardia, left ventricular ejection fraction and ventricular activation time. Possible interrelations among cycle length of inducible ventricular tachycardia, left ventricular ejection fraction and ventricular activation time were investigated in the 57 patients with inducible ventricular tachycardia. In the 10 patients who had induction of monomorphic ventricular tachycardia at two studies, the cycle length of tachycardia at the initial study was analyzed. No attempt was made to examine the cycle length of the spontaneous arrhythmias because the cycle length of the spontaneous arrhythmias was not as accurately documented as that of the inducible arrhythmias.

The cycle length of inducible ventricular tachycardia in the 57 patients with this rhythm induced was weakly correlated ($r = -0.34$, $p < 0.02$) with left ventricular ejection fraction ($y = -0.053x + 38$) (Fig. 5). However, the cycle length of inducible ventricular tachycardia did not correlate with ventricular activation time determined either from the signal-averaged ECG or at epicardial mapping. Ventricular activation time measured at epicardial mapping correlated weakly ($r = -0.45$, $p < 0.05$) with left ventricular ejection fraction ($y = 0.224x + 72$), but this correlation was not observed when ventricular activation time was measured from the signal-averaged ECG.

Discussion

Differences between patients with ventricular tachycardia and fibrillation. This study suggests that there are different electrophysiologic and anatomic abnormalities responsible

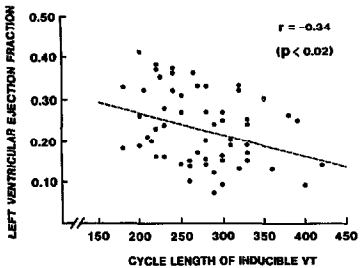
Table 3. Subdivision of 64 Patients According to Configuration of Spontaneous and Inducible Arrhythmias

	Spontaneous VT and Inducible VT	Spontaneous VF and Inducible VT	Spontaneous VF and Inducible VF
No. of patients	32	25	7
Anterior infarction	17 (53%)	13 (52%)	1 (14%)
Age (yr)	58 ± 6	57 ± 5	52 ± 5
Male (no.)	25 (78%)	22 (88%)	6 (86%)
QRS duration (ms)	98 ± 30	102 ± 10	99 ± 12
QT interval (ms)	385 ± 40	382 ± 40	379 ± 38
QTc (ms)	450 ± 45	455 ± 35	457 ± 39
Cycle length of inducible VT (ms)	290 ± 61	259 ± 44	
		$p = 0.05$	
Arrhythmia inducible only at 20 mA	5 (16%)	6 (24%)	2 (29%)
No. of diseased coronary vessels	2.0 ± 1.0	1.9 ± 1.0	1.6 ± 0.8
Ventricular activation time (ms) on signal-averaged ECG	181 ± 33	158 ± 21	129 ± 17
		$p < 0.05$	$p < 0.01$
		$p < 0.001$	
LV ejection fraction	0.22 ± 0.09	0.25 ± 0.08	0.36 ± 0.05
		NS	$p < 0.01$
		$p < 0.001$	
LV aneurysm	19 (59%)	11 (44%)	1 (14%)

Unless otherwise stated, differences between groups were not significant (NS). Values are reported as mean ± standard deviation. Abbreviations as in Table 1.

for ventricular tachycardia and ventricular fibrillation occurring spontaneously late after myocardial infarction. We have shown that, compared with patients with ventricular fibrillation, patients with ventricular tachycardia have a longer ventricular activation time during sinus rhythm (measured either from the body surface ECG or by cardiac mapping) and a lower left ventricular ejection fraction. These findings are in agreement with studies of inducible arrhythmias in chronic canine infarct models (19–21) and with recent patient studies (7–9). However, these latter studies were deficient in that they included patients without myocardial infarction (7), considered only inducible arrhythmias in patients with myocardial infarction (9) or included patients who were taking antiarrhythmic medications (8).

Figure 5. Correlation between left ventricular ejection fraction and cycle length of inducible ventricular tachycardia (VT) in 57 patients.



Although the programmed stimulation protocol in the present study used high current stimulation rather than multiple drive cycle lengths to promote arrhythmia induction (22), seven of the eight patients who had inducible ventricular fibrillation had their clinical arrhythmia reproduced at programmed stimulation. These seven patients with both spontaneous and inducible ventricular fibrillation had the least conduction delay detected by the signal-averaged ECG and best preserved ventricular function. Patients with a longer conduction delay on the signal-averaged ECG usually had inducible ventricular tachycardia, and their presenting arrhythmia was related to the degree of conduction delay in sinus rhythm, such that more marked conduction delay was associated with spontaneous tachycardia rather than spontaneous fibrillation. As has been noted previously (23–25), patients presenting with ventricular fibrillation often have a preceding period of ventricular tachycardia or ventricular flutter, which then degenerates into ventricular fibrillation. Our finding that the mean cycle length of inducible ventricular tachycardia was shorter for patients with cardiac arrest than for patients with spontaneous ventricular tachycardia is consistent with the observation that many patients presenting with ventricular fibrillation may have had fast ventricular tachycardia as the initiating, albeit undetected, arrhythmia. We found that although the degree of conduction delay

during sinus rhythm was related to the configuration of both the spontaneous and inducible arrhythmias, it was not closely related to the cycle length of inducible ventricular tachycardia, a finding also noted in a previous study (26) using left ventricular endocardial mapping.

The cycle length of inducible ventricular tachycardia was, however, found to be related to left ventricular ejection fraction, such that a longer cycle length of inducible ventricular tachycardia was associated with a lower left ventricular ejection fraction, which presumably reflected more extensive scarring.

Anatomic correlates of conduction delay in sinus rhythm. The anatomic substrate for conduction delay during sinus rhythm is patchy scar tissue interdigitating with normal myocardium (19,27). In canine infarction, these viable muscle bundles interdigitating with scar tissue are much larger in dogs with inducible ventricular tachycardia than in dogs with inducible ventricular fibrillation, and the infarct edge in dogs with ventricular tachycardia is more irregular (21). Thus, infarct anatomy may help to determine the degree of localized conduction delay present during sinus rhythm and, hence, the configuration of an induced arrhythmia. However, although the localized conduction delay may be important in arrhythmogenesis, it may have little influence on the cycle length of ventricular tachycardia because the reentrant circuit may also involve distant tissues, at least in the case of macroreentry (10,28). Thus, both the infarct size and the anatomic features of the infarct at sites other than those with conduction delay during sinus rhythm may help determine cycle length of ventricular tachycardia. With more extensive scarring, ventricular tachycardia may be slower because of increased potential circuit size.

Correlation between signal-averaged ECG and cardiac mapping. The present study used both the signal-averaged ECG and epicardial mapping to detect conduction delay during sinus rhythm. Epicardial mapping was used because electrical signals detected from the body surface should more accurately reflect epicardial rather than endocardial signals. To be detectable from the body surface, delayed ventricular activation of either endocardial or epicardial origin must outlast normal ventricular activation (5). In the present study, we noted that, although the signal-averaged ECG underestimated the conduction delay detectable at cardiac mapping, the difference in ventricular activation time between patients with ventricular tachycardia and those with ventricular fibrillation was still detectable from the body surface.

There are several possible explanations for the differences in ventricular activation time determined by signal averaging and epicardial mapping in the present study. In addition to differences in bandpass filtering, there were differences in timing as the two recordings were not made on the same day. We previously noted that 25% of patients with delayed potentials detected on the signal-averaged ECG have day to day changes in ventricular activation time >20 ms (14). However, there may have been a larger difference between the two methods if more than 25 epicardial sites had been mapped because mapping additional sites would increase the chance of finding a site with a longer epicardial activation time.

Clinical implications. Patients with ventricular tachycardia and those with ventricular fibrillation associated with previous myocardial infarction appear to have a different electrophysiologic and anatomic basis for their arrhythmias. The degree of conduction delay in sinus rhythm appears to be an important factor influencing the morphology of both spontaneous and inducible ventricular tachyarrhythmias occurring late after myocardial infarction. Patients presenting with the potentially more devastating arrhythmia, ventricular fibrillation, usually do not have as marked electrophysiologic abnormalities as do patients presenting with ventricular tachycardia. A small group of patients presenting with ventricular fibrillation (7 of 32 [22%] in our series) also have inducible ventricular fibrillation, and these patients with potentially lethal ventricular fibrillation are characterized by having relatively well preserved ventricular function and minimal conduction delay in sinus rhythm. Although inducible ventricular fibrillation carries no adverse prognostic implications in patients who have not had spontaneous arrhythmias (12), the findings of inducible ventricular fibrillation in patients with spontaneous ventricular fibrillation appear to have more sinister implications (29), even though the electrophysiologic and anatomic abnormalities in these patients with ventricular fibrillation are less marked than in most postinfarction patients with spontaneous ventricular tachyarrhythmias. Therefore, patients with ventricular tachyarrhythmias after myocardial infarction should not be considered as a homogeneous group during clinical evaluation.

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