JACC Vol. 10, No. 4
October 1987:775–81

Signal Averaging of the Surface QRS Complex Predicts Inducibility of Ventricular Tachycardia in Patients With Syncope of Unknown Origin: A Prospective Study

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Forty patients with syncope of unknown origin underwent quantitative signal averaging of the surface QRS complex before invasive electrophysiologic testing with programmed ventricular stimulation. Of 34 patients without bundle branch block, 12 had inducible ventricular tachycardia (Group I) and 22 did not (Group II). The duration of low amplitude signals, the root mean square voltage of the terminal 40 ms and the signal-averaged QRS vector duration were measured in each case. One or more abnormal signal averaging variables were present in 92% of patients in Group I, but in only 27% of patients in Group II ($p < 0.005$). An abnormal root mean square voltage of the terminal 40 ms was the most significant distinguishing variable, being present in 83% of Group I patients and in only 14% of Group II patients ($p < 0.005$). The QRS vector duration was prolonged in 58% of Group I patients, but in only 9% of Group II patients ($p < 0.05$). Likewise, the duration of low amplitude signals was prolonged in 58% of Group I patients, but in only 19% of Group II patients ($p < 0.05$).

When compared with 24 hour ambulatory electrocardiographic monitoring, the presence of abnormal signal averaging variables was more predictive of inducible ventricular tachycardia. Seven (32%) Group II patients had ≥10 ventricular premature beats/h, couplets or episodes of nonsustained ventricular tachycardia; however, none had abnormal late potentials recorded. In contrast, three patients (25%) in Group I had <10 ventricular premature beats/h, although all in that group had one or more abnormal signal-averaged variables.

The sensitivity and specificity of the various signal-averaged variables ranged from 50 to 83% and from 82 to 91%, respectively. An abnormally low root mean square voltage of the terminal 40 ms had the highest sensitivity (82%) and specificity (91%) in distinguishing individuals with syncope of unknown origin who had inducible ventricular tachycardia. Thus, signal averaging of the surface QRS complex is a useful noninvasive technique for selecting patients with syncope of unknown origin who should undergo programmed ventricular stimulation.

(J Am Coll Cardiol 1987;10:775–81)

Which patients with syncope of unknown origin should undergo programmed ventricular stimulation remains unclear. Such testing is supported by a substantially higher mortality in patients with cardiac versus other causes of syncope (1). Whereas obstructive or hemodynamic causes of cardiac syncope can be detected by physical examination or noninvasive testing, or both, invasive electrophysiologic testing may be necessary to detect arrhythmic causes. In fact, nine major studies (2–10) found that invasive electrophysiologic studies may detect cardiac rhythm disturbances responsible for syncope in up to 79% of patients with syncope of unknown origin. More than 40% of these patients may have inducible ventricular tachyarrhythmias at electrophysiologic testing.

Late potentials detected by signal averaging of the surface QRS complex have been shown to reflect the substrate for reentrant ventricular tachycardia in the experimental animal model and human subjects (11,12). Abnormal signal-averaged QRS variables have been correlated with a higher occurrence of ventricular tachycardia and sudden death after myocardial infarction (13–15). Thus, we hypothesized that the presence of late potentials recorded from signal averaging of the surface QRS complex should correlate with the inducibility of ventricular tachyarrhythmias in patients with

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Manuscript received January 8, 1987; revised manuscript received March 11, 1987, accepted May 1, 1987.

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0735-1097/87/$3.50

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October 1987:775-81

syncope of undetermined origin. Here we report a prospective study performed with signal averaging of the surface QRS complex to predict the inducibility of ventricular tachyarrhythmias in such a group of patients.

Methods

Study patients. Forty consecutive patients with syncope of undetermined origin referred for electrophysiologic studies were evaluated. All of the patients had undergone prior neurologic evaluation and noninvasive cardiovascular assessment, including 24 hour ambulatory electrocardiographic monitoring. Six patients had a bundle branch block configuration on baseline electrocardiograms and were eliminated from the study. This report discusses the results of studies in the remaining 34 patients.

Signal-averaging technique. Signal averaging of the surface QRS complex was performed before electrophysiologic testing with the patient in the supine position. All antiarrhythmic agents were withheld for at least six half-lives before the study, and no patient had received amiodarone. Seven silver/silver chloride electrodes were attached after the skin was cleaned with alcohol to constitute three orthogonal bipolar electrodes as follows: 1) the horizontal electrodes (X) were positioned at the right and left midaxillary lines at the fourth intercostal space; 2) the vertical electrodes (Y) were positioned at the left parasternal second intercostal space and the V_3 position; and 3) the sagittal electrodes were positioned at the V_5 position anteriorly and at a corresponding posterior site. A ground electrode was positioned on the eighth rib in the right midaxillary line.

A high resolution electrocardiograph (Arrhythmia Research Technology, 101 System) with high gain amplification and bidirectional Butterworth filters (40 to 250 Hz) was used for signal averaging. Approximately 200 beats were amplified, filtered, digitally sampled, processed and printed with a Hewlett-Packard 7470A X-4 plotter. Signal averaging was performed within 24 hours of the electrophysiologic studies.

Electrophysiologic studies. After giving informed consent, all patients underwent electrophysiologic testing in the absence of antiarrhythmic agents and in the fasting state. Two quadripolar electrode catheters (USCI) were inserted percutaneously through the femoral vein and positioned under fluoroscopy in the high right atrium across the tricuspid valve for recording His bundle activity, and in the right ventricular apex and outflow tract. The distal poles were used for pacing, and the proximal poles were used for recording. Stimulation was performed with a programmed stimulator (Bloom Associates) that delivered impulses of 1.5 ms duration at a current that was twice threshold. Three electrocardiographic leads (I, II and V_1) were recorded, as well as intracardiac electrograms at filter settings of 30 to 500 Hz. Time lines generated at 40, 200 and 1,000 ms using a VR 12 multichannel recorder (Honeywell Electronics for Medicine) on thermal paper at a speed of 50 to 100 mm/s. Programmed atrial stimulation was performed using incremental pacing and premature stimulation, as previously described (16). Subsequently, all patients underwent programmed ventricular stimulation from the right ventricular apex using the following protocol:

1) *Incremental ventricular pacing at rates up to 240 beats/min.*
2) *Premature ventricular stimulation as follows:*
   a) *S_1S_2 method:* A single ventricular stimulus (S_2) was introduced after every eight ventricular paced beats (S_1S_1) at decreasing coupling intervals until ventricular muscle refractoriness.
   b) *$S_1S_2S_3$ method:* Two ventricular stimuli (S_2S_3) were introduced during a basic paced ventricular cycle (S_1S_1) as before, beginning with an S_1S_2 interval 50 ms longer than the effective refractory period of the ventricular muscle and an S_2S_3 interval equal to the S_1S_2 interval. The S_2S_3 interval was progressively decreased by 10 ms until S_3 was refractory. S_1S_2 was then decreased and S_3 reintroduced until S_3 captured the ventricle or S_2 became refractory.

None of the patients had S_4 stimulation or left ventricular stimulation. In our laboratory, the latter modes of stimulation are used only in patients with documented sustained ventricular tachycardia or out-of-hospital sudden death, or both, and not in patients with syncope of unknown origin. All patients underwent ventricular stimulation at two cycle lengths (600 and 450 ms). If stimulation of the right ventricular apex did not initiate ventricular arrhythmias, right ventricular outflow tract stimulation was performed using the same protocol.

Definition of terms. *Signal averaging:* A vector magnitude (V) was calculated for each point of the averaged QRS complex as $V = \sqrt{X^2 + Y^2 + Z^2}$. The following low amplitude signal measurements were recorded, computer calculated and confirmed visually:

1) *The duration of low amplitude signals* was recorded from the end of the signal-averaged QRS vector complex to the point at which signals measured 40 μV.
2) *The root mean square voltage (index of late potentials)* was measured for the terminal 40 ms of the QRS vector complex.
3) *The signal-averaged QRS vector duration (QRSd)* was the time (in ms) from the onset to end point of the vector complex.

Twenty-four hour ambulatory electrocardiographic monitoring. Couplets were defined as two repetitive ventricular premature contractions. *Nonsustained ventricular tachycardia* consisted of three or more successive wide complex ventricular beats at a rate of $\geq$120 beats/min, with atrio-

ventricular dissociation that lasted for ≤30 seconds and terminated spontaneously.

Electrophysiologic testing. Nonsustained ventricular tachycardia was defined as more than five repetitive ventricular responses present for ≤30 seconds. *Sustained ventricular tachycardia* was defined as tachycardia of ventricular origin that lasted >30 seconds or was associated with hemodynamic embarrassment.

Statistical analysis. This was performed utilizing Student's *t* test for unpaired and paired data, as well as two by two chi-square analysis. *Sensitivity* was defined as the percent of patients with the specified abnormal signal-averaged variable, who had inducible sustained or nonsustained ventricular tachycardia. *Specificity* was defined as the percent of patients with a specified normal signal-averaged variable, who had no inducible ventricular arrhythmias.

Abnormal signal-averaged variables. The abnormal signal-averaged QRS variables for our laboratory for 40 Hz high pass filtering are: 1) low amplitude signals >38 ms; 2) root mean square voltage of the terminal 40 ms <20 μV; and 3) QRS duration >114 ms. These values have been derived from 25 subjects without heart disease or ventricular arrhythmias. Values correspond to standard deviations above the mean for QRS duration and duration of low amplitude signals. Ninety-five percent of normal subjects had a root mean square voltage of the terminal 40 ms ≥ 20μV.

Results

On the basis of findings at the time of electrophysiologic studies, patients were divided into two groups. Group I consisted of 12 subjects who had inducible ventricular tachycardia with programmed ventricular stimulation (1 with single premature stimuli, 9 with two premature stimuli and 2 with burst ventricular pacing). Nine of these patients developed sustained monomorphic ventricular tachycardia, and three had reproducible nonsustained monomorphic ventricular tachycardia. Group II consisted of 22 patients who had no inducible ventricular tachycardia; 9 patients (41%) had no specific cardiac rhythm disturbances diagnosed by electrophysiologic studies and 13 (59%) did (Table 1), including paroxysmal supraventricular tachycardia, atrioventricular block and sinus node dysfunction.

The mean values for age did not differ between the groups with inducible and noninducible tachycardia (Group I = 54 ± 16 years; Group II = 60 ± 17 years; p = NS). Neither coronary artery disease nor cardiomyopathy was more prevalent in Group I than in Group II. Only two patients in Group I had a left ventricular aneurysm (Patients 6 and 8). Only 2 patients in Group I had recurrent syncope or presyncope, whereas 15 of those in Group II did.

Quantitative comparison of signal averaging. The root mean square voltage of the terminal 40 ms was significantly lower in patients in Group I than in Group II (20 ± 18 versus 74 ± 72 μV; $p < 0.05$), and the QRS duration was significantly longer in patients in Group I than in Group II (124 ± 22 versus 96 ± 25 ms; $p < 0.05$). The duration of low amplitude signals was longer in Group I than in Group II patients, but this difference was not statistically significant (41 ± 12 versus 30 ± 20 ms; $p < 0.1$).

Of the 12 patients with inducible ventricular tachycardia during electrophysiologic testing, 11 (92%) had one or more abnormal signal-averaged variables, whereas only 7 (32%) of those in Group II did ($p < 0.005$) (Table 1). The number of patients in each group with abnormal signal-averaged variables is shown in Table 2. As is evident, the root mean square voltage of the terminal 40 ms was abnormally low in 10 (83%) of Group I patients, but in only 2 (9%) of Group II subjects ($p < 0.005$). The mean QRS vector duration was prolonged in seven (58%) of Group I patients, but in only two (9%) of Group II patients ($p < 0.05$). Prolonged low amplitude signals were seen in seven (58%) of Group I patients and in three (14%) of Group II patients ($p < 0.05$).

Twenty-four hour electrocardiographic monitoring. Of the four grades of ventricular ectopic rhythm recorded during ambulatory electrocardiographic monitoring, only the presence of couplets differed between the two groups. Couplets were detected in eight Group I patients (67%) but in only six Group II patients (27%) ($p < 0.05$). The presence of nonsustained ventricular tachycardia did not distinguish either group.

In Group I, six patients (50%) had <10 ventricular premature complexes/h, six (50%) had ≥10/h and eight (67%) had couplets recorded. In contrast, among Group II patients, nine (41%) had <10 ventricular premature complexes/h, five (23%) had ≥10/h and six (27%) had couplets. Seven (32%) of Group II patients had ≥10 ventricular premature complexes/h, or couplets or episodes of nonsustained ventricular tachycardia. These patients might have been considered at high risk for inducible ventricular tachyarrhythmias; however, none had abnormal late potentials and only two had an abnormal mean QRS vector duration. Conversely, three patients (25%) in Group I demonstrated <10 ventricular premature complexes/h and might have been considered at low risk for ventricular tachyarrhythmias. They all had one or more abnormal signal-averaged variables.

Sensitivity and specificity of signal-averaged variables. The sensitivity and specificity of the signal-averaged QRS variables ranged from 50 to 83% and from 82 to 91%, respectively, in distinguishing patients with syncope and inducible ventricular arrhythmias. The respective sensitivities and specificities of these variables were: 1) 83 and 91% for the root mean square voltage of the terminal 40 ms; 2) 50 and 86% for the duration of low amplitude signals; and 3) 58 and 82% for the duration of the QRS vector complex. Whereas 83% of patients with abnormally low root mean square voltage of the terminal 40 ms of the signal-averaged QRS complexes had inducible ventricular tachyarrhythmias,

Table 1. Clinical Characteristics, 24 Hour Electrocardiographic Monitoring Results, Quantitative Signal-Averaging Variables and Electrophysiologic Diagnoses in 34 Patients

Patient No.	Age (yr)	Sex	HD	24-Hour ECG Monitoring: Mean VPCs/hour ≤10/h	Mean VPCs/hour >10/h	Couplets/h	VTns/d	Signal Averaging: LAS (ms)	RMS (μV)	QRSd (ms)	EPS Dx
Group I (patients with inducible ventricular tachycardia)											
1	62	M	CAD	—	1,760	30	526	**55**	**12**	**123**	VTns
2	35	F	CM	6	—	1	2	**41**	**18**	**139**	VTs
3	65	M	CAD	3	—	0	0	38	**17**	113	VTns
4	44	M	CAD	3	—	0	0	19	75	100	VTs
5	66	M	CAD	—	12	<1	0	21	24	**141**	VTs
6	60	M	CAD	—	26	<1	0	**43**	**15**	**120**	VTs
7	45	M	NA	7	—	0	0	**45**	**18**	111	VTs
8	23	M	NA	2	—	0	0	35	**16**	105	VTs
9	57	M	CM	9	—	<1	0	37	**17**	96	VTs
10	60	M	CAD	—	147	1	4	**60**	**7**	**173**	VTs
11	46	F	CM	—	61	13	3	**50**	**14**	**122**	VTns
12	82	M	CAD	—	127	<1	0	**50**	**8**	**142**	VTs
Group II (patients without inducible ventricular tachycardia)											
1	61	M	CAD	3	—	0	0	34	23	108	HVT
2	62	M	NA	0	0	0	2	24	67	77	CSH
3	60	M	CM	—	1,079	38	1	14	104	81	—
4	62	M	CAD	—	184	11	0	35	21	105	—
5	46	F	MVP	0	0	0	0	28	81	93	—
6	45	M	NA	0	0	0	0	26	41	91	PAF
7	38	M	HBP	1	—	0	0	18	69	100	CSH
8	85	F	HBP	—	15	<1	0	25	23	**116**	PAF
9	76	F	NA	0	0	0	0	21	227	68	—
10	66	M	NA	—	421	<1	0	30	41	94	—
11	67	F	NA	4	—	<1	0	30	48	82	SSS
12	74	F	CAD	<1	—			26	48	**133**	SSS
13	70	M	CAD	2	—	0	0	**47**	**15**	105	—
14	70	M	CAD	—	341	1	0	24	43	**120**	SSS
15	70	M	NA	0	0	0	0	**110**	292	75	—
16	60	M	NA	1	—	0	0	**41**	17	96	—
17	22	F	MVD	0	0	0	0	19	58	86	HVT
18	82	M	NA	<1	—	0	0	20	64	102	—
19	67	M	HBP	>1	—	0	0	24	44	63	SSS
20	61	M	CAD	>1	—	0	0	17	65	86	CSH
21	23	M	MVP	0	0	0	0	24	173	**186**	AVB
22	47	M	CAD	0	0	0	0	17	65	58	CSH

Boldface numbers represent abnormal values. AVB = atrioventricular block; BP = hypertensive heart disease; CAD = coronary artery disease; CM = cardiomyopathy; couplets = total number of paired ventricular premature complexes recorded per hour; CSH = carotid sinus hypersensitivity; EPS Dx = results of programmed ventricular stimulation; HD = underlying heart disease; HVT = hypervagal state; LAS = duration of low amplitude signals; NA = no apparent heart disease; NI = five or fewer repetitive ventricular responses; PAF = paroxysmal atrial fibrillation; QRSd = duration of the QRS vector complex; RMS = root mean square voltage of the terminal 40 ms; SSS = sick sinus syndrome; ≤10 VPCs = % of total hours monitored with ≤10 ventricular premature complexes/h; >10 VPCs = % of total hours monitored with >10 ventricular premature complexes/h; 24 hour ECGm = 24 hour ambulatory electrocardiographic monitoring; VHD = valvular heart disease; VPC = ventricular premature complex; VTns/d = number of episodes of nonsustained ventricular tachycardia per day, defined as five or more sequential ventricular premature complexes lasting <30 seconds; VTns = nonsustained ventricular tachycardia (>five repetitive ventricular responses), as defined in text; VTs = sustained ventricular tachycardia (>30 seconds in duration or requiring termination), as defined in text.

Table 2. Incidence of Abnormal Signal-Averaged Variables in the Two Patient Groups

	RMS	LAS	QRSd
Group I	10 (83%)	7 (58%)	7 (58%)
Group II	2 (9%)	3 (14%)	4 (18%)
p Value	<0.005	<0.05	<0.05

Abbreviations as in Table 1.

only 9% did if this variable was normal. Thus, the presence or absence of an abnormally low root mean square voltage of the terminal 40 ms of the signal-averaged complex (index of late potentials) provided the highest sensitivity and specificity in identifying individuals with syncope of unknown origin who had inducible ventricular arrhythmias.

Examples of signal-averaged QRS complexes are shown

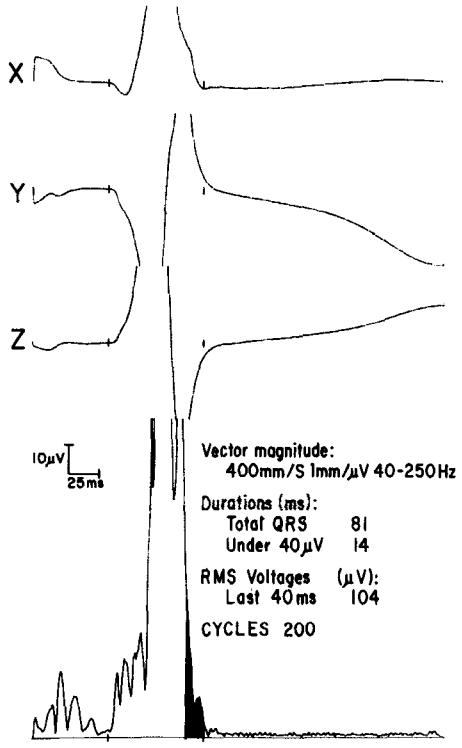


Figure 1. Patient 3, Group II. The signal-averaged QRS complex shows that all variables were normal. The duration of low amplitude signals is represented by the **shaded area.** Cycles = number of QRS complexes processed by signal averaging; RMS = root mean square voltage of the terminal 40 ms of the QRS complex.

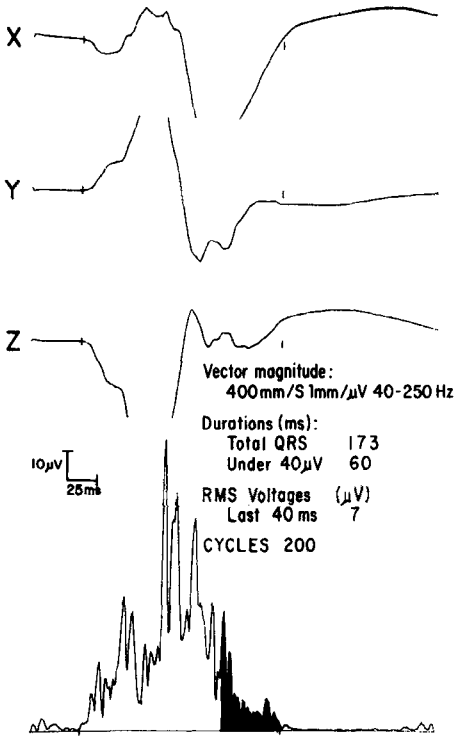
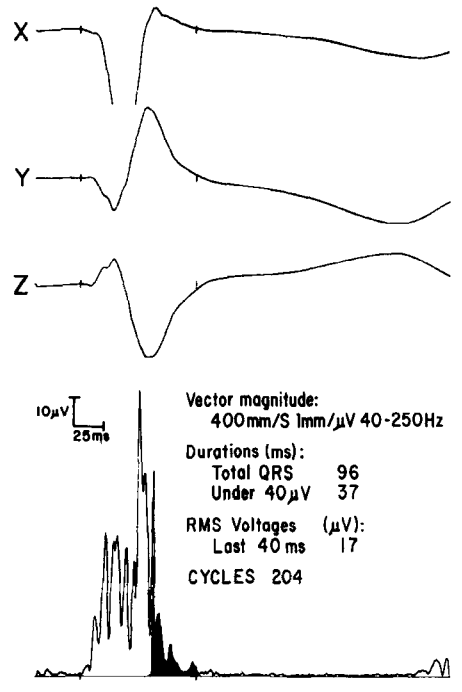


Figure 3. Patient 10, Group I. The duration of low amplitude signals of the signal-averaged QRS complex (**shaded area**) was abnormally long, as was the QRS vector complex duration, while the root mean square voltage (RMS) of the terminal 40 ms (RMS) was abnormally low.

Figure 2. Patient 9, Group I. The signal-averaged QRS complex shows that although the duration of low amplitude signals (**shaded area**) was normal, the root mean square voltage of the terminal 40 ms (RMS) was abnormally low.



in Figures 1 to 3. The recording in Figure 1 is taken from Patient 3 in Group II. This patient had had frequent episodes of near syncope as well as one episode of true syncope. Frequent ventricular premature complexes, couplets and nonsustained ventricular tachycardia were present on 24 hour ambulatory electrocardiographic monitoring. However, all signal averaging variables were within normal limits. No ventricular tachyarrhythmias were induced, and the patient's symptoms were attributed to transient increased vagal states. His symptoms have been relieved with the use of probanthine.

In contrast, Figure 2A demonstrates an abnormally low root mean square voltage of the terminal 40 ms of the signal-averaged QRS complex in Patient 9 of Group I. This 57 year old man had had mitral valve surgery 2 years earlier for mitral regurgitation and had mildly impaired left ventricular systolic function. After two episodes of syncope, ambulatory electrocardiographic monitoring revealed a moderate number of ventricular premature beats and occasional couplets. However, sustained ventricular tachycardia was induced with two premature ventricular stimuli. This patient has done well without further syncopal episodes on treatment with quinidine gluconate and tocainide hydrochloride.

An example of a 60 year old man (Patient 10, Group I), 10 years postmyocardial infarction, who had had syncope

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and had abnormalities of all three signal-averaged variables, is shown in Figure 3. This patient had induction of sustained ventricular tachycardia while undergoing electrophysiologic testing and has been free of recurrences on treatment with amiodarone hydrochloride.

Discussion

The results of this study demonstrate that signal averaging of the surface QRS complex may be a useful noninvasive technique for selecting patients with syncope of undetermined origin who should undergo programmed ventricular stimulation. Low amplitude, high frequency potentials occurring at the end of the signal-averaged QRS complex correspond to delayed and fractionated electrical activity recorded from endocardial and epicardial surfaces in human subjects (11,12). These may serve as a marker for the underlying substrate responsible for many ventricular tachyarrhythmias, because they in turn result from inhomogeneous conduction in localized areas of ventricular myocardium. In fact, late potentials recorded with signal-averaging techniques have been associated with ventricular tachycardia in the postmyocardial infarction period as well as with an increased incidence of sudden death (13–15). Such fractionated diastolic activity, as recorded from signal averaging of the surface QRS complex, has been eliminated after endocardial resection (17–19). Further validation of the technique of signal averaging of the QRS complex to detect late potentials in humans is corroborated by the finding of a good correlation of signal averaging of the surface QRS complex with signal averaging of intracardiac and epicardial ventricular depolarizations (20).

Neither the frequency of ventricular premature complexes nor the presence of nonsustained ventricular tachycardia distinguished the two groups. Yet 67% of the patients with inducible ventricular tachyarrhythmias did not have nonsustained ventricular tachycardia on continuous 24 hour electrocardiographic monitoring. Furthermore, only 9% of patients without noninducible ventricular tachycardia had nonsustained ventricular tachycardia recorded before signal averaging or electrophysiologic testing. Interestingly, of the seven patients (32%) in Group II with >10 ventricular premature complexes/h, ventricular couplets or nonsustained ventricular tachycardia, only two (29%) had abnormal signal averaging variables. Thus, signal averaging of the surface QRS complex detected 92% of the patients with inducible ventricular tachycardia, whereas ambulatory monitoring detected only 67% of these patients. Difficulty in identifying patients at risk for ventricular tachyarrhythmias from continuous electrocardiographic monitoring has been reported previously (21,22).

Role of coronary artery disease and bundle branch block. Seven (32%) of the patients in Group II had coronary artery disease. Although none of these seven patients had inducible ventricular tachyarrhythmias, four did have one or more abnormal signal-averaged variables. However, in Group I, seven (58%) of the patients had coronary artery disease; six of these patients had one or more abnormal signal-averaged variables and all had inducible ventricular tachycardia. Thus, the sensitivity and specificity of abnormal signal-averaged variables for inducible ventricular tachyarrhythmias in patients with coronary artery disease were 86% and 57%, respectively.

Although patients with bundle branch block and syncope may have transient atrioventricular block as an underlying etiologic factor, ≥25% of such patients with coronary artery disease develop ventricular tachyarrhythmias during electrophysiologic studies (23,24). However, the significance of signal averaging in such patients is unknown, because no standards have been established for what constitutes abnormal quantitative signal-averaged variables in the presence of bundle branch block. As a result of asynchronous ventricular activation, late potentials may occur in the absence of a substrate for ventricular tachyarrhythmias. Alternatively, the intrinsically prolonged QRS durations have been reported to obscure abnormal late potentials. Thus, we excluded patients with bundle branch block from this analysis. In addition, patients who had not completed a full year after myocardial infarction were excluded from study, because preliminary studies suggest that signal-averaged variables may fluctuate during this time period.

Previous studies. Two recent studies (25,26) have suggested a similar role for signal averaging of the surface QRS complex in evaluating patients with syncope of unknown origin. However, one of these studies (25) did not prospectively assess in all cases the value of programmed ventricular stimulation in predicting ventricular tachycardia. Rather, in 63% of patients the presence of more than three repetitive ventricular responses on Holter monitoring was taken as a criterion for the diagnosis of ventricular tachycardia. Because short runs of nonsustained ventricular tachycardia on electrocardiographic monitoring may not be significant, we performed programmed ventricular stimulation on all patients. This might account for the higher sensitivity in our study. Although the other study (26) was similar in design, fewer patients were included and the methodology used was less quantitative and thus the data are more likely to be open to subjective interpretation. Furthermore, S_4 stimulation was used, which, as pointed out by Morady et al. (6), may be less specific than double premature stimulation. In addition, neither of these studies carefully presented or analyzed the electrophysiologic data in comparison with the presented Holter monitoring results.

The system that we employed to perform signal averaging of the QRS complex represents computerized quantitative analysis, which is far superior to earlier, less exact, qualitative analysis. It enables substantial reduction of noise, and with the use of bidirectional Butterworth filters, prevents

filter "ringing." The noise levels in our studies were low (Fig. 1 to 3), and all computerized measurements were validated visually. For better accuracy, we utilized normal values determined in our laboratory with our equipment.

The absence of recurrent syncope in patients with inducible ventricular tachyarrhythmias who are treated accordingly and of subsequent ventricular arrhythmias in patients with normal signal averaging supports the utility of signal averaging as a screening tool to determine which patients with syncope of unknown origin should undergo programmed ventricular stimulation.

Implications. Our study suggests that signal averaging of the QRS complex, which is a noninvasive technique and free of morbidity, may be a useful procedure for the evaluation of syncope of unknown origin. Although signal averaging may select patients who do not need programmed ventricular stimulation, electrophysiologic studies may still be needed to determine other causes of syncope, such as paroxysmal supraventricular tachycardia, atrioventricular block and sinus node dysfunction. This is supported by the finding that 59% of the patients in our study had cardiac rhythm disturbances other than ventricular tachyarrhythmias diagnosed from electrophysiologic testing.

We thank Valentin Fuster, MD for his encouragement and support, Philip Barreca for technical assistance and Patricia A. Ettrick for secretarial assistance.

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