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Behavior of Late Potentials on the Body Surface During Programmed Ventricular Stimulation

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Objectives. **This study sought to evaluate the behavior of late potentials on the body surface by signal averaging during programmed stimulation and to correlate the findings with the cycle length of induced ventricular tachycardia.**

Background. **Clinically relevant late potentials may be concealed within the QRS complex and may be missed by the conventional signal-averaged electrocardiogram (SAECG). In contrast, some late potentials may arise from dead-end pathways or pathways not capable of supporting sustained ventricular tachycardia (VT). It has been shown that durations of late potentials in sinus rhythm correlate poorly with VT cycle length.**

Methods. **Signal-averaged electrocardiography during sinus rhythm, right ventricular pacing (S1) and introduction of a right ventricular extrastimulus (S2) was performed in 95 patients: 11 patients with a structurally normal heart and no inducible VT (Group I); 44 with a previous myocardial infarction (MI) and no inducible monomorphic VT (Group II); and 40 with a previous MI and inducible monomorphic VT (Group III).**

Results. **The best subset of SAECG variables and the best cut points for each variable to differentiate between patients with and without VT were first established for each rhythm studied. Total duration of the filtered QRS complex (QRSD) was found to be the only independent predictor of inducibility of VT. When late potentials were defined for these criteria (QRSD ≥113, ≥178 and ≥168 ms for the SAECG during sinus rhythm, S1 and S2, respectively), there was no difference in the incidence of false positive (16% vs. 18%) or false negative (30% vs. 26%) late potentials between sinus rhythm and S1. During S2, there were significantly fewer false positive late potentials (11% vs. 16%) and fewer false negative late potentials (17% vs. 30%) than with sinus rhythm. Compared with sinus rhythm, 31% of the false positive late potentials detected during sinus rhythm were lost, whereas 43% of the false negative late potentials became detectable after S2, resulting in improved sensitivity (83% vs. 70%), specificity (89% vs. 84%) and predictive accuracy (86% vs. 77%, $p < 0.05$). Among the patients with VT, QRSD during S2 achieved the best correlation with VT cycle length ($r = 0.74$) and was the only independent predictor of VT cycle length when all SAECG variables were considered.**

Conclusions. **Late potentials revealed by ventricular extrastimuli but concealed during sinus rhythm may be clinically relevant and may explain some of the false negative late potentials and reduced sensitivity of the conventional SAECG in predicting VT. In contrast, those late potentials that are detected during sinus rhythm but lost after ventricular extrastimuli are often clinically irrelevant and may account for the false positive late potentials and reduced specificity of the conventional SAECG.**

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The signal-averaged electrocardiogram (SAECG) during sinus rhythm has been shown to be a useful, noninvasive test for identifying patients with spontaneous (1–7) and inducible (8–10) ventricular tachycardia (VT) after infarction and to document predisposition to VT in patients with unexplained syncope (11–13). However, the overall predictive accuracy of the SAECG is still relatively poor. Slow conduction in sinus rhythm may occur not only in myocardium comprising part of a potential reentrant circuit, but also in other areas that are not associated with the capacity for reentrant VT. Such false positive late potentials may be recorded in patients with no history of VT. In contrast, clinically relevant late potentials may be buried within the QRS complex in patients with VT. It has been shown (14–16) that initiation of reentrant VT by premature ventricular pacing is dependent on a critical degree of conduction delay in electrograms recorded epicardially and intramyocardially in a canine model of chronic infarction and endocardially in humans. Sites demonstrating such critical delay during premature ventricular stimulation then go on to display middiastolic potentials during VT, presumably constituting part of the reentrant VT circuit. Fractionated potentials that persist during premature stimulation and span diastole during reentrant VT are likely to be part of a reentrant circuit (17–20).

The present study attempted to 1) detect late potentials on the body surface by signal-averaging during programmed stimulation; 2) evaluate the behavior of late potentials on the body

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Abbreviations and Acronyms

LAS40	=	duration of low amplitude signals >40 μV in the terminal portion of the filtered QRS complex
MI	=	myocardial infarction
QRSD	=	QRS duration
RMS40	=	root-mean-square voltage of the terminal 40 ms of the filtered QRS complex
SAECG	=	signal-averaged electrocardiogram, signal-averaged electrocardiographic
S1	=	regular right ventricular pacing
S2	=	introduction of single right ventricular extrastimulus
VT	=	ventricular tachycardia

surface during programmed stimulation; and 3) correlate the findings with the cycle length of induced VT.

Methods

Patients. Ninety-five patients with no bundle branch block gave written informed consent to be included in the study: *Group I* = 11 patients with no coronary artery disease or structural heart disease who underwent electrophysiologic study for assessment of palpitations or syncope of unknown etiology and who had no sustained monomorphic VT inducible (normal subjects). *Group II* = 44 patients with a previous myocardial infarction (MI) and no inducible sustained monomorphic VT (control patients). *Group III* = 40 patients with a previous MI and reproducibly inducible sustained monomorphic VT, cycle length ≥200 ms (cases patients). Patient characteristics are shown in Table 1. For Groups II and III, the diagnosis of a previous MI was confirmed by clinical history, 12-lead electrocardiography, coronary angiography and left ventriculography in each patient. Gated heart blood pool scan was also performed to quantify left ventricular ejection fraction in each patient. All patients had stopped antiarrhythmic drugs for at least 5 days at the time of study.

Programmed ventricular stimulation. Studies were performed in the postabsorptive, sedated state (diazepam, 10 mg, given orally 1 h before study). Quadrupolar electrode catheters were positioned in the right atrial appendage and right ventricular apex and a tripolar electrode catheter at the His bundle region. Pacing was performed at the right ventricular apex, using a rectangular pulse of 2 ms at twice diastolic current threshold. A baseline drive train of 8 beats with a single basic cycle length as close to 600 ms as possible was used. Extrastimuli were introduced in 10-ms decrements, from 300 ms to refractoriness. Each extrastimulus was applied three times at each coupling interval before decrementing to the next coupling interval. Up to four extrastimuli were used.

Table 1. Patient Characteristics

	Group I (n = 11)	Group II (n = 44)	Group III (n = 40)
Previous MI	No	Yes	Yes
Inducible SMVT	No	No	Yes
Mean (±SD) age (yr)	47 ± 18	59 ± 8	62 ± 10
Male/female (no.)	6/5	37/7	35/5
Mean (±SD) LVEF (%)	ND	51 ± 12 (n = 40)	35 ± 11 (n = 39)

LVEF = left ventricular ejection fraction by gated heart blood pool scan. MI = myocardial infarction; ND = gated heart blood pool scan was not performed in this group with no coronary artery disease and structurally normal hearts on echocardiography; SMVT = sustained monomorphic ventricular tachycardia.

For patients with inducible VT, the stimulation protocol was applied three times, with a 10-min rest period between each induction. This protocol has been performed in our unit as part of a project to study reproducibility of VT induction. The cycle length of VT was measured as the average of three consecutive cycle lengths (to the nearest 10 ms), 10 s after the onset of VT, using a paper speed of 100 mm/s. *Ventricular tachycardia cycle length* was defined as the longest cycle of any of the three induced VT for that patient. *Number of extrastimuli* to induce VT was defined as the least number of extrastimuli required to induce the VT with the longest cycle length.

Signal-averaged ECG. The SAECG was acquired at the same visit as the electrophysiologic study. Unipolar X, Y and Z leads referenced to Wilson's central terminal were used (21). Signals were amplified, filtered (0.05 to 500 Hz), sampled at 1 kHz/channel and digitized to 12-bit resolution. The SAECGs were acquired during sinus rhythm, right ventricular pacing (S1) and introduction of a ventricular extrastimulus (S2).

Signal acquisition during sinus rhythm. The SAECG was recorded during sinus rhythm for 250 s (~250 to 350 cardiac cycles).

Signal acquisition during regular right ventricular pacing. Two quadpolar catheters, one in the high right atrium and one in the right ventricular apex, which had been inserted for electrophysiologic study, were utilized for SAECG acquisition during ventricular pacing. To prevent any atrial depolarization signals (P wave) from mimicking or masking late potentials in the T wave during ventricular pacing, the right atrium was paced simultaneously with the ventricle so that any atrial signals would be buried within the earlier portion of the broad left bundle branch blocklike complex.

Pacing was performed at the right ventricular apex using a basic cycle length of close to 600 ms. Pacing pulses were 2 ms at twice diastolic threshold. The SAECG was recorded for 180 to 240 s during regular ventricular pacing so that approximately the same number (250 to 350) of cardiac cycles as during sinus rhythm was recorded. The pacing signal output from the stimulator was used to produce the fiducial mark for signal averaging.

Signal acquisition during single extrastimulus. A single extrastimulus was delivered at a coupling interval of 300 ms after a drive train of 4 to 6 beats. This sequence was repeated 300 times with no pause between drive trains. If bundle branch reentrant beats or unstable ventricular responses were encountered, the basic drive cycle or the number of beats in the drive train, or both, was adjusted until stable ventricular capture and response were achieved. Signal acquisition was commenced

only when a stable response was obtained. Each extrastimulus pacing signal was used to trigger data acquisition during the succeeding 600 ms. In this way, only the ventricular response to the extrastimulus was acquired. Approximately 300 premature ventricular complexes were acquired for averaging.

Editing of acquired signals. All recordings were recalled in 2-s segments and displayed on an oscilloscope (model 2245A, Tektronix) for editing. For studies during sinus rhythm, atrial and ventricular premature contractions, noisy segments and obvious baseline drifts were marked manually for exclusion from averaging. For studies during regular ventricular pacing, in addition to the above, all atrial and ventricular premature contractions as well as any subsequent sinus and fusion beats resulting from perturbation of the regular pacing were marked manually for exclusion from averaging. For studies after an extrastimulus, editing of the extrastimulus response was the same as that for regular ventricular pacing. In addition, because the drive trains were not recorded, any drive trains with irregular capture were marked by two observers in real time. The extrastimulus response corresponding to that drive train was subsequently excluded from averaging, even though the electrogram may have appeared identical to the other extrastimulus responses.

Signal averaging. Signal averaging was performed on a DEC PDP 11/73 computer. Each signal-averaged QRS complex was analyzed with two different highpass filtering frequencies (25 and 40 Hz, four-pole Butterworth filter). For QRS complexes obtained from sinus rhythm studies, the filter was applied bidirectionally, as described by Simson (7). For QRS complexes obtained during pacing, the filter only processed the signal backward in time up to the pacing spike, which was automatically set at 0 ms. The filtered signals from the X, Y and Z unipolar leads were combined into a vector magnitude $(X^2 + Y^2 + Z^2)^{1/2}$, termed the *filtered QRS complex* (7). For sinus rhythm studies, the QRS end points were determined by computer algorithm, as described by Simson (7). For paced QRS complexes, Simson's algorithm was also used to determine the offset, whereas the onset was defined as the pacing spike on the ECG. While the times to commence the forward search for QRS onset in sinus rhythm studies and backward search for QRS offset in all three studies were manually nominated, no human overreading was performed once the onset or offset point was identified.

For sinus rhythm studies, an iterative cross-correlation procedure was used to optimize temporal (horizontal) alignment (22). The mean voltage of a 100-ms window in the horizontal ST segment was used for vertical alignment. For signal-averaging during regular ventricular pacing and during introduction of an extrastimulus, the acquired SAECG was temporally aligned at the pacing stimulus, and no cross-correlation was used. The segment used for vertical alignment was a 100-ms window beginning 150 ms after the pacing spike, usually a relatively straight-sloping segment of the inverted T wave.

For each filtered QRS complex, three standard SAECG variables were obtained: total QRS duration (QRSD); root-mean-square voltage of the terminal 40 ms (RMS40); and duration of low amplitude signals <40 μV in the terminal portion (LAS40). The mean numbers of cardiac cycles averaged and the mean noise (mean voltage measured over a 40-ms segment after the QRS offset) for each of the rhythms studied are shown in Table 2.

Table 2. Cardiac Cycles Averaged and Noise Levels

	SR (mean ± SD)	S1 (mean ± SD)	S2 (mean ± SD)
No. of beats averaged			
Group I	300 ± 50	301 ± 50	280 ± 42
Group II	313 ± 60	305 ± 55	267 ± 75
Group III	302 ± 62	307 ± 55	273 ± 38
Noise levels (μV)			
Group I	0.4 ± 0.3	0.7 ± 0.4	0.7 ± 0.4
Group II	0.5 ± 0.3	1.0 ± 0.5	1.0 ± 0.5
Group III	0.5 ± 0.1	0.8 ± 0.5	0.8 ± 0.3

SR = sinus rhythm; S1 = regular right ventricular pacing; S2 = right ventricular pacing with single extrastimulus.

Statistical methods. Results are shown as mean value ± SD. Unpaired *t* tests were used to compare patient characteristics and SAECG variables between patients with and without inducible VT. Paired *t* tests were used to compare variables from signal averaging during the different rhythms within patient. SAECG results were analyzed in an exploratory data analysis. There was no single primary outcome variable. Rather, six well documented and validated variables (namely, QRSD, RMS40 and LAS40 each at two highpass filtering frequencies of 25 and 40 Hz) were considered as possible explanatory variables of case-control status or of VT cycle length. Univariate logistic regression analyses of case-control status were used to identify cut points for each variable under each filtering frequency and pacing regime (23). Using these cut points the sensitivity, specificity and predictive accuracy of each variable was calculated for each filtering frequency and pacing regime. Multivariate logistic regression analyses were used to determine whether two or more possible explanatory variables could improve the fit obtained by the best fitting univariate model of case-control status (defined as the univariate model with the lowest deviance). The fit was said to be significantly improved if a statistically significant decrease in deviance was seen when the two-variable model was compared with the univariate model. *Sensitivity* for each rhythm studied was the percentage of case patients with positive late potentials using the criteria for late potentials for that rhythm. *Specificity* for each studied rhythm was the percentage of control patients with no detectable late potentials using the criteria for late potentials for that rhythm. *Predictive accuracy* was the percentage of cases and control patients correctly identified by the test. McNemar's test was used to compare the differences in predictive accuracies between different methods of signal averaging. A p value <0.05 was considered statistically significant. The linear regression analyses were performed in the same order: first univariate under each filtering and pacing

Table 3. Sensitivity, Specificity and Predictive Accuracy of Individual Signal-Averaged Electrocardiographic Variables

	Cut Point	Sensitivity (%)	Specificity (%)	Predictive Accuracy (%)
Sinus rhythm				
25 Hz				
QRSD (ms)	≥113	70	84	77
RMS40 (μV)	≤38	75	52	63
LAS40 (ms)	≥34	52	73	63
40 Hz				
QRSD (ms)	≥112	73	77	75
RMS40 (μV)	≤22	73	57	64
LAS40 (ms)	≥43	55	80	68
Regular RV pacing				
25 Hz				
QRSD (ms)	≥178	74	82	78
RMS40 (μV)	≤22	63	75	70
LAS40 (ms)	≥40	51	84	69
40 Hz				
QRSD (ms)	≥175	71	84	78
RMS40 (μV)	≤12	58	64	61
LAS40 (ms)	≥75	41	86	65
Premature RV pacing				
25 Hz				
QRSD (ms)	≥168	83	89	86*
RMS40 (μV)	≤24	64	64	64
LAS40 (ms)	≥40	54	79	67
40 Hz				
QRSD (ms)	≥167	78	84	81
RMS40 (μV)	≤11	59	55	56
LAS40 (ms)	≥64	62	74	68

*$p < 0.05$ versus sinus rhythm. LAS40 = duration of low amplitude signals <40 μV in terminal portion of filtered QRS complex; QRSD = total QRS duration; RMS40 = root-mean-square voltage of terminal 40 ms; RV = right ventricular.

Table 4. Mean (±SD) Value for Each Signal-Averaged Electrocardiographic Variable in the Three Study Groups

	Group I	Group II	Group III
Sinus rhythm			
25 Hz			
QRSD (ms)	90 ± 10	101 ± 13	121 ± 19
RMS40 (μV)	92 ± 72	63 ± 52	32 ± 36
LAS40 (ms)	23 ± 12	35 ± 27	37 ± 19
40 Hz			
QRSD (ms)	87 ± 10	100 ± 13	119 ± 19
RMS40 (μV)	85 ± 36	27 ± 14	20 ± 22
LAS40 (ms)	27 ± 12	34 ± 25	46 ± 24
Regular RV pacing			
25 Hz			
QRSD (ms)	151 ± 17	165 ± 13	190 ± 19
RMS40 (μV)	31 ± 12	31 ± 15	22 ± 16
LAS40 (ms)	27 ± 20	26 ± 19	44 ± 31
40 Hz			
QRSD (ms)	149 ± 18	164 ± 13	187 ± 20
RMS40 (μV)	16 ± 6	17 ± 9	13 ± 9
LAS40 (ms)	50 ± 25	53 ± 33	67 ± 38
Premature RV pacing			
25 Hz			
QRSD (ms)	142 ± 18	153 ± 13	183 ± 19
RMS40 (μV)	37 ± 21	33 ± 16	21 ± 13
LAS40 (ms)	27 ± 14	27 ± 19	47 ± 26
40 Hz			
QRSD (ms)	141 ± 18	154 ± 12	183 ± 19
RMS40 (μV)	19 ± 11	16 ± 9	11 ± 9
LAS40 (ms)	46 ± 28	50 ± 26	73 ± 28

Abbreviations as in Table 3.

regime to determine individual correlations with VT cycle length and then multivariate to determine whether the best fitting univariate model could be improved.

Results

Cut points derived for each of the SAECG variables and the associated sensitivity, specificity and predictive accuracy achieved by using each of these cut points alone as a criterion to differentiate between Groups II and III are summarized in Table 3. The mean values for each of the SAECG variables obtained during the different study rhythms are summarized in Table 4. When all SAECG variables were considered, multiple logistic regression analysis identified QRSD (25 Hz) as the only independent predictor of VT. The scattergrams for QRSD (25 Hz) obtained during the different study rhythms are shown in Figure 1. The QRSDs during all three rhythms for Group I were significantly shorter than those for Group II ($p = 0.002$, 0.001 and 0.028 for sinus rhythm, S1 and S2, respectively). Because the most difficult task for SAECG is to differentiate between patients with and without VT among those with a previous MI, comparisons were therefore only drawn between Groups II and III. The cut points that best differentiate between Groups II and III were 113 ms for sinus rhythm, 178 ms for regular right ventricular pacing and 168 ms for premature ventricular stimulation. Because RMS40 and LAS40 did not contribute any independent predictive value to case-control status, the presence of a late potential is defined as QRSD ≥113 ms for sinus rhythm, ≥178 ms during regular right ventricular pacing and ≥168 ms during ventricular extrastimuli.

Using these criteria, SAECG during sinus rhythm correctly identified 70% and 84% of patients with and patients without VT, respectively. Late potentials were detected in 16% of the patients without VT (false positive late potentials), whereas among patients with VT, late potentials were not detected in 30% (false negative late potentials). During regular ventricular pacing, the incidence of false positive (18%) and false negative late potentials (26%) was similar to that during sinus rhythm. Thus, there was no difference in sensitivity (70% vs. 74%), specificity (84% vs. 82%) or predictive accuracy (77% vs. 78%) between SAECG during sinus rhythm and during regular ventricular pacing.

During pacing with an extrastimulus, there were fewer false positive late potentials (11% vs. 16%) and fewer false negative late potentials (17% vs. 30%) than with sinus rhythm (S2 vs. sinus rhythm). Compared with sinus rhythm, 31% of the false positive late potentials detected during sinus rhythm were lost,

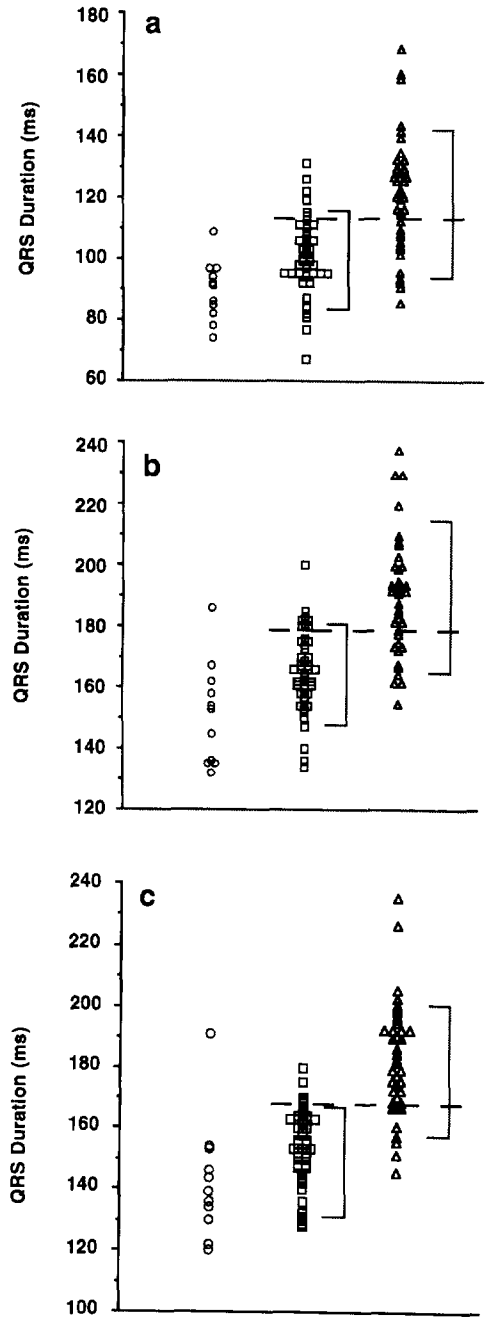

Figure 1. Total QRSD (25-Hz highpass filter) in patients with (**triangles**) and without (**squares**) inducible VT. **a,** During sinus rhythm, there was a moderate number of false positive and false negative late potentials and a large degree of overlap between the 10th and 90th percentiles (**brackets**) of those with and without inducible VT. **b,** During regular right ventricular pacing, there was little improvement. **c,** During right ventricular pacing with single extrastimulus, there were fewer false positive and false negative late potentials. **Circles** = "normal" group; *dashed lines* = cut points derived from discriminant analysis.

whereas 43% of the false negative late potentials became detectable during pacing with an extrastimulus, resulting in improved sensitivity (83% vs. 70%), specificity (89% vs. 84%) and predictive accuracy (86% vs. 77%, S2 vs. sinus rhythm, $p < 0.05$).

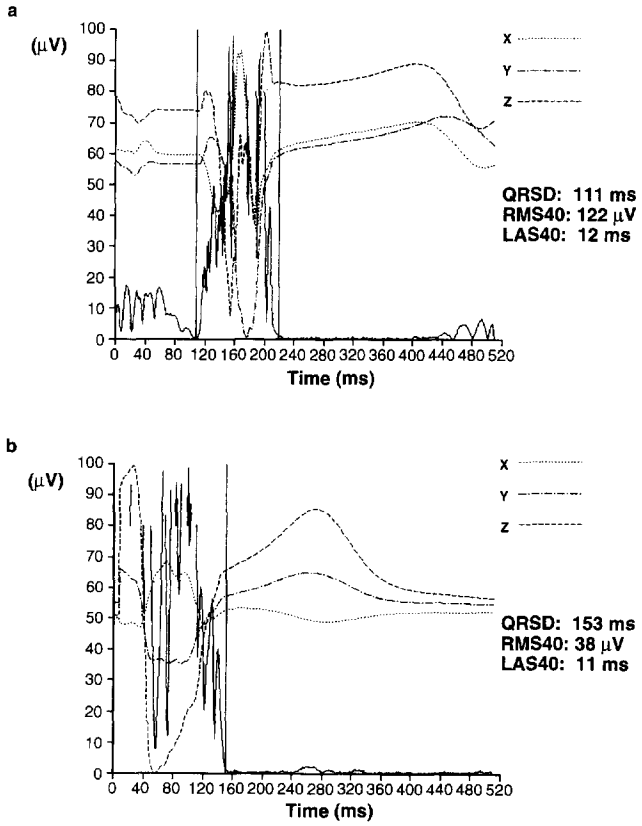

Figure 2. Signal-averaged ECGs from a patient with no spontaneous or inducible VT. **a,** During sinus rhythm. **b,** During right ventricular pacing with single extrastimulus. In both studies, there was a sharp, clean falloff in potentials, with absence of low amplitude signals at the end of the filtered QRS complex. Both studies were negative for late potentials and correctly predicted the noninducibility of VT in this patient. The filtered (25 Hz) combined vector magnitude is displayed as a **continuous line** at the base of each panel. **Vertical lines** mark the onset and offset of the QRS complex as located by computer algorithm (see text). X, Y, Z = SAECG leads.

QRSD (25 Hz) during pacing with an extrastimulus was the only independent predictor of case-control status, there being no two possible explanatory variables which when jointly fitted in a model significantly improved the univariate model fit.

An example of a patient in whom VT was not inducible and in whom SAECG study results were normal during both sinus rhythm and premature ventricular pacing is shown in Figure 2. An example of a patient with inducible VT and abnormal SAECG results during both sinus rhythm and premature ventricular pacing is shown in Figure 3. An example of a patient with inducible VT and normal SAECG results during sinus rhythm is shown in Figure 4. During pacing with an extrastimulus, the QRS duration of 220 ms was well beyond the cut point of 168 ms, and there were low amplitude signals at the end of the paced QRS complex.

An example of a patient with inducible ventricular tachycardia and increasing delay in late potentials is shown in Figure 5. Although late potentials were already present during sinus rhythm and regular ventricular pacing, there was a further

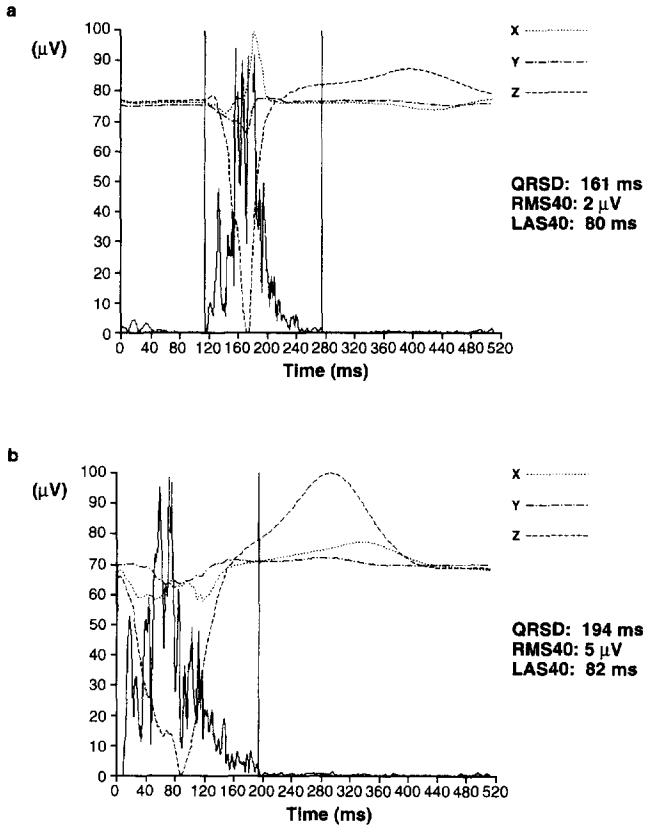

Figure 3. Signal-averaged ECGs from a patient with spontaneous and inducible VT. **a,** During sinus rhythm. **b,** During right ventricular pacing with single extrastimulus. In both studies, there were low amplitude signals at the end of the filtered QRS complex. Both studies were positive for late potentials and correctly predicted the inducibility of VT in this patient. Format as in Figure 2.

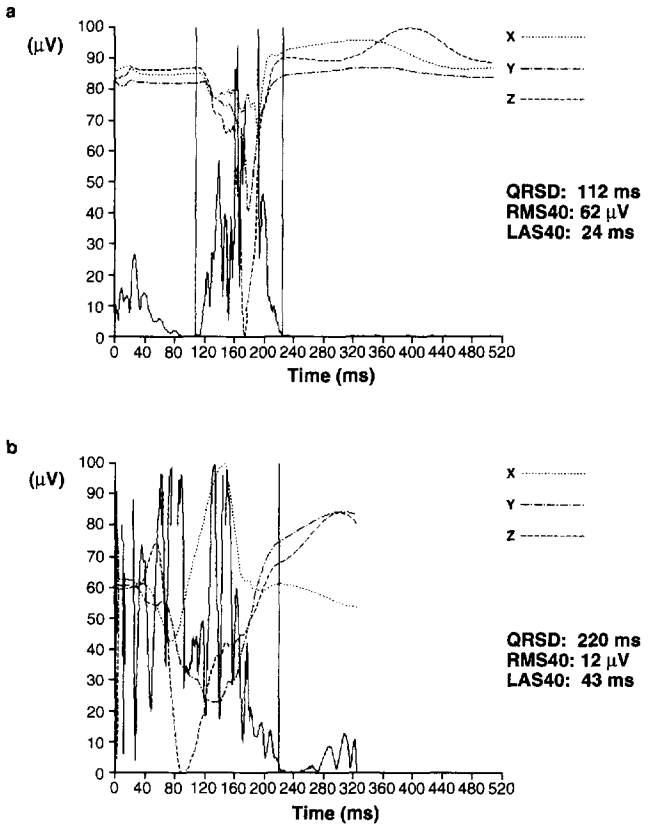

Figure 4. Signal-averaged ECGs from a patient with spontaneous and inducible VT. **a,** During sinus rhythm, there were few low amplitude signals at the end of the QRS complex; QRSD, RMS40 and LAS40 were all within the normal limits. **b,** During right ventricular pacing with single extrastimulus, there were low amplitude signals at the end of the QRS complex; QRSD was well beyond the cut point of 168 ms. The sinus rhythm study was therefore falsely negative for late potentials, whereas that during ventricular premature pacing correctly identified this patient with inducible VT. Format as in Figure 2.

increase in low amplitude signals and QRS duration during premature ventricular pacing.

When each SAECG acquisition technique was considered alone, the only independent predictor of VT cycle length by regression analysis was QRSD (25 Hz) (Fig. 6). QRSD during ventricular extrastimuli achieved the best correlation with VT cycle length ($r = 0.74$) compared with that during regular ventricular pacing ($r = 0.54$) or sinus rhythm ($r = 0.68$). When all three SAECG acquisition techniques were considered together, QRSD (25 Hz) during ventricular extrastimulus was the only independent predictor of VT cycle length. There was no correlation ($r = 0.01$) between left ventricular ejection fraction and VT cycle length. Gender and age were not associated with VT cycle length. The number of extrastimuli required to induce VT was found to be correlated with VT cycle length ($r = 0.37$, $p = 0.02$). QRSD and number of extrastimuli required to induce VT were independent predictors of VT cycle length.

Discussion

The SAECG has shown promise in differentiating patients with inducible VT among those with cardiac arrest and syncope of unknown etiology (24). It is also useful in stratifying patients at risk of spontaneous VT or sudden death after acute myocardial infarction (24). However, the overall predictive accuracy is relatively poor. Specificity is limited because false positive late potentials are common in patients in the early postinfarction period as well as in the normal population (25). Sensitivity is limited in patients with VT because potential sites of origin of VT may sometimes be activated early in sinus rhythm. These areas of delayed activation may therefore fall within the normal QRS duration and not be detected by time domain analysis.

Signal-averaging during rapid atrial pacing in an attempt to improve the predictive accuracy of the SAECG has been described (26,27). However, the results were no better than analysis in sinus rhythm.

Ventricular pacing and premature stimulation may increase conduction delay, thereby exposing concealed late potentials. In contrast, late potentials from electrical "dead ends" may fail to conduct during premature ventricular stimulation. In the 4-day old canine postinfarction model of reentry, El-Sherif et

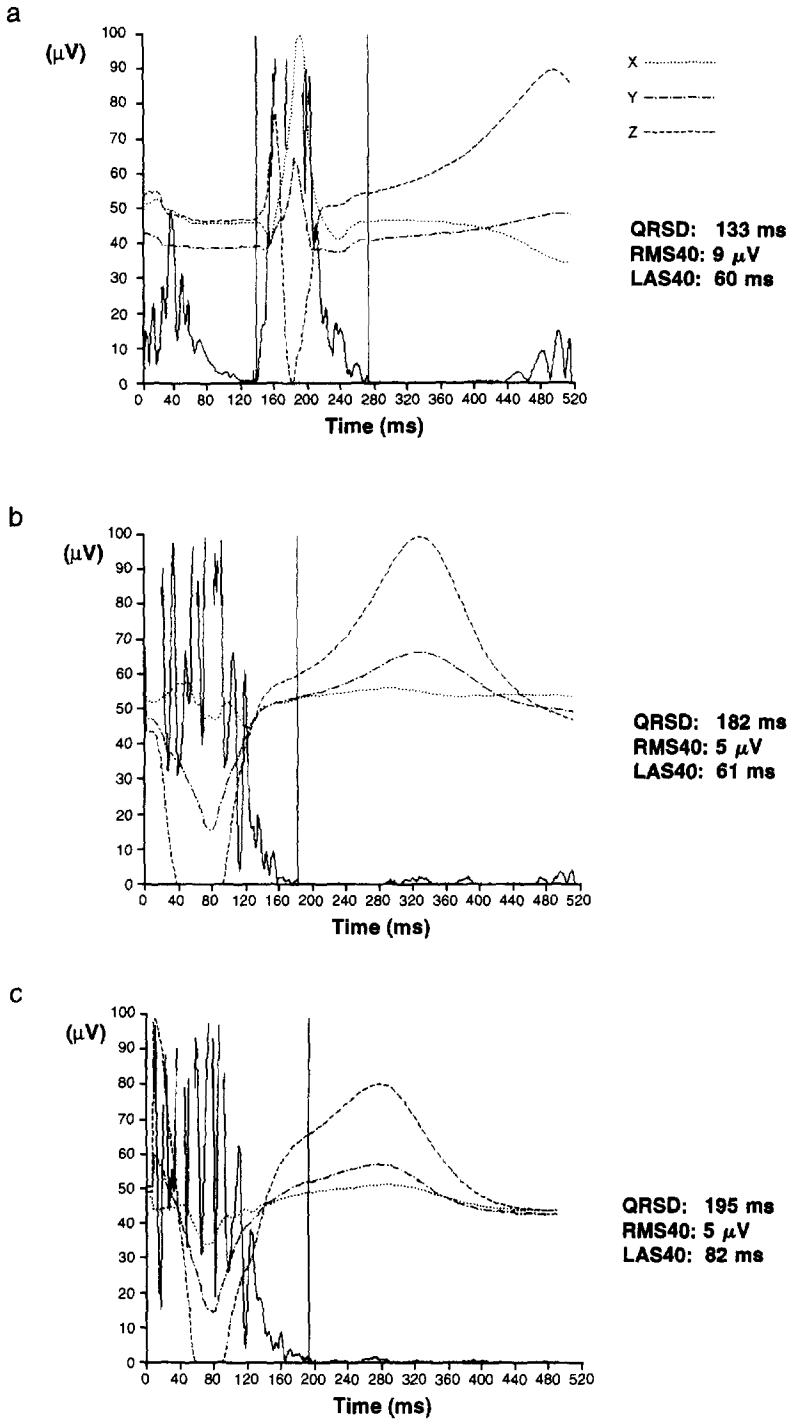

Figure 5. Signal-averaged ECGs from a patient with inducible VT and late potentials during sinus rhythm (**a**), regular ventricular pacing (**b**) and premature ventricular pacing (**c**). Note the increase in total QRSD and low amplitude signals during premature ventricular stimulation compared with that during regular ventricular pacing. Format as in Figure 2.

al. (18) showed that regions showing marked conduction delay in a 1:1 pattern, or showing Wenckebach or 2:1 conduction patterns during sinus rhythm, usually blocked during single premature pacing and did not participate in the reentrant process. The present study showed that late potentials can be detected on the body surface by signal-averaging during premature ventricular stimulation. There were less false positive and false negative late potentials during premature ventricular stimulation than during sinus rhythm. This improvement over that achieved during sinus rhythm suggests that right ventricular extrastimuli must be exposing late potentials that are important for VT in some patients while eliminating those not responsible for VT in others.

The present study showed that QRSD obtained from signal-averaging during sinus rhythm, right ventricular pacing and ventricular extrastimuli were all significantly correlated with VT cycle length. However, QRSD during ventricular extrastimuli achieved the strongest correlation with VT cycle

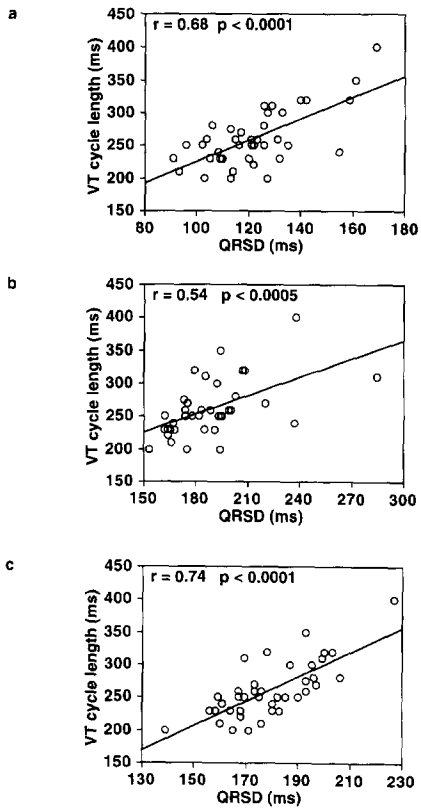

Figure 6. Relation between VT cycle length and total QRSD (25 Hz) obtained during (**a**) sinus rhythm, (**b**) regular right ventricular pacing and (**c**) right ventricular pacing with single extrastimulus.

length. It was also the only independent predictor of VT cycle length among all SAECG variables analyzed. The strength of our study was the large cohort of patients with spontaneous VT, and who also had reproducibly inducible VT. To our knowledge, the present study is the only one in which monomorphic VT was induced multiple times so that the VT with the longest cycle length could be evaluated. Our findings were in accord with those of Freedman et al. (28) and Cassidy et al. (29), which showed poor correlation between local electrogram duration determined by catheter mapping during sinus rhythm with induced VT cycle length.

Conclusions. Late potentials can be detected on the body surface by signal averaging during regular right ventricular pacing and during introduction of a ventricular extrastimulus. During regular right ventricular pacing, there was a similar incidence of false positive and false negative late potentials compared with those measured during sinus rhythm. During ventricular extrastimuli, there were significantly less false positive and false negative late potentials than those measured during sinus rhythm. Moreover, in patients with a previous MI and inducible VT, total QRS duration obtained during ventricular extrastimuli was better correlated with the cycle length of VT than that obtained during sinus rhythm. Those late potentials revealed by ventricular extrastimuli but concealed in sinus rhythm explain some of the false negative SAECG results. In contrast, those late potentials that are lost after ventricular extrastimuli are less likely to be able to sustain VT and explain some of the false positive SAECG findings. These observations explain the suboptimal sensitivity and specificity of conventional methods of SAECG for detecting increased risk for VT.

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