290–330 ms. It was 80% (4/5) at S_1-T delay of 290 ms, 80% (4/5) at S_1-T delay of 300 ms, 70% (44/66) at S_1-T delay of 310 ms, 78% (7/9) at S_1-T delay of 320 ms, 74% (17/23) at S_1-T delay of 330 ms and 0% (0/2) at S_1-T delay of 340 ms. In no patients incremental ventricular pacing or alternating current had to be used to induce VF.

We conclude that a low energy (1–2J) shock, when synchronized to the paced-T wave with S_1-T delays of 290–330 ms, induces VF in over 70–80% of the attempts. This precludes the need for prolonged ventricular bursts or the use of alternating current and should, thus, be the preferred modality of choice while inducing VF to check ICD function.

960-91 Influence of T-wave Shock Energy on Ventricular Fibrillation Vulnerability in Humans

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Delivery of low energy T-wave shocks may induce ventricular fibrillation (VF). Clinical use of this induction method is hindered due to the need for T-wave scanning with multiple shocks to identify the window of vulnerability (WV) to VF. The purpose of this study was to test the hypothesis that WV duration is directly related to shock strength. We studied the effect of shock strength on WV in 27 consecutive patients undergoing nonthoracotomy Medtronic 7219C/D PCD implantation or testing. WV duration was tested after a 3 pulse 400 ms drive by T-wave scanning between coupling intervals (CI) of 220–420 ms in 20 ms steps using randomized 0.6J and 2.0J truncated monophasic shocks. The results were (mean ± std):

	0.6J	2.0J	p
Min CI (ms)	270 ± 23	275 ± 28	N.S.
Max CI (ms)	312 ± 31	333 ± 30	<0.05
WV Duration (ms)	39 ± 25	56 ± 33	<0.05

The mean increase in WV duration with 2.0J shocks was 166 ± 134%, $p < 0.05$. Increased WV duration was due solely to persistence of VF inducibility further into diastole. Maximal VF induction efficacy was 20/27 (74%) patients using 2.0J at 300 ms CI. Optimal induction with 0.6J was 17/27 (63%) patients at a 280 ms CI.

We conclude that: 1) Window of vulnerability duration is energy dependent, 2) Increasing shock strength extends the vulnerable period, 3) Greatest induction efficacy for a single shock strength and CI is 74%, and 4) Need for T-wave scanning may be minimized by increasing shock energy.

961 Signal-Averaged Electrocardiography

Tuesday, March 21, 1995, Noon–2:00 p.m.
Ernest N. Morial Convention Center, Hall E
Presentation Hour: Noon–1:00 p.m.

961-76 Frequency Domain Analysis of the Signal-Averaged ECG in Patients with Arrhythmogenic Right Ventricular Dysplasia

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Signal-Averaged ECG (SAECG) was recorded in 28 patients (mean 38 ± 13 years, 19 men) with arrhythmogenic right ventricular dysplasia (ARVD) who had sustained ventricular tachycardia and 35 normal subjects (mean 35 ± 11 years, 30 men). The Fast-Fourier transform analysis of SAECG was performed using a Blackman-Harris window in a segment length of 120 msec from 20 msec before the end of the QRS complex. Area ratio 1 (area of 20–50 Hz)/(area of 0–20 Hz) and area ratio 2 (area of 40–100 Hz)/(area of 0–40 Hz) were calculated. The mean value of area ratio 1 and 2 in patients with ARVD was significantly higher than those of normal subjects (410 ± 340 vs 186 ± 28; $p < 0.001$, 174 ± 143 vs 41 ± 16; $p < 0.001$). Eighteen (64%) and 21 (75%) patients had a value greater than the mean ± 2SD value of normal subjects. These results suggest that the frequency domain analysis of SAECG is an available method for detecting late potentials in patients with ARVD who have sustained ventricular tachycardia of the right ventricular origin.

961-77 The Anatomic Basis of Late Potentials in Patients with 'Idiopathic' Right Ventricular

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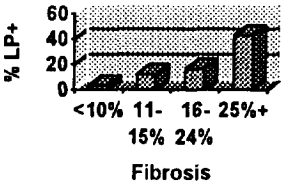
Background: A significant number of patients with 'idiopathic' right ventricular tachycardia (VT) have abnormal histology on endomyocardial biopsy and positive late potentials on signal-averaged electrocardiogram (SAE).

Aims: This study attempts to relate the histological abnormalities on endomyocardial biopsy to the presence of late potentials.

Methods: Patients with 'idiopathic' VT were studied by time domain SAE and endomyocardial biopsy. Biopsies were examined by a specialist cardiac histologist and the occurrence of abnormality related to the presence of late potentials.

Results: Fifty-two patients, mean age 34.6 ± 13.8 years were studied. Six patients had endocardial thickening, 31 had interstitial fibrosis (quantified by morphometric methods), 27 had fibrosis isolating myocardial cells into islets, 4 had leucocyte infiltration and 26 had adipose infiltration. Of these abnormalities, interstitial fibrosis was associated with the presence of late potentials on SAE (Table). There was a strong relationship between the degree of interstitial fibrosis and the prevalence of late potentials (Figure). Adipose infiltration was not related to the presence of fibrosis and not associated with the presence of late potentials on signal-averaged ECG.

	LP −/+	LP −/+	
No fibrosis	19/1	No adipose infiltr	19/6
Fibrosis	22/7	Adipose infiltr	22/2



Conclusions: The presence of increasing degrees of interstitial fibrosis relates to increasing prevalence of late potentials on signal-averaged ECG, whereas these do not appear to relate to any of the other histological abnormalities examined. This strongly suggests that interstitial fibrosis is involved in the genesis of late potentials.

961-78 Importance of Left Ventricular Ejection Fraction and Signal Averaged Electrocardiogram but not of Coronary Artery Patency nor Holter Monitoring to Predict Severe Arrhythmic Events After a First Myocardial Infarction in the Thrombolytic Era

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We followed-up 244 consecutive patients (210 men, mean age = 56 ± 9 yrs) who survived the acute phase (alive after day 7) of a first anterior (N = 102) or inferior (N = 142) myocardial infarction (MI) with a mean follow-up (FU) delay of 57 ± 18 months. In the acute phase, 97/244 patients (40%) received a thrombolytic therapy. Within the second and third week after admission, all patients underwent a Holter ECG monitoring graded by the Lown classification, a signal averaged electrocardiogram (SAECG) and a coronary angiography. Three parameters were measured by SAECG (predictor system, 40 Hz high-pass filter): total QRS duration (QRSd), root mean square voltage of the last 40 ms (RMS) and duration of the terminal low (<40 uV) amplitude signal (LAS). The number of diseased vessels as well as the infarct related artery (IRA) patency was evaluated by TIMI grading (TIMI 2 or 3 = patent) and left ventricular ejection fraction (LVEF) was measured angiographically. Cox proportional hazards model was used for the statistical analysis.

Results: We observed 18 arrhythmic events (AE): 10 sudden cardiac death and 8 ventricular tachycardia during the FU period. Statistical analysis identified 3 independent factors predictive of the occurence of an AE: 1) LVEF, with a risk multiplied by 1.9 for each 0.10 decrease in the LVEF value, 2) LAS, with a risk multiplied by 1.3 for each 5 ms increase in the LAS value and 3) absence of thrombolysis, with a risk multiplied by 3.9.

Conclusions: After MI in the thrombolytic era the Holter ECG monitoring and the results of the coronary angiography do not predict the risk of an AE. LVEF, SAECG and absence of thrombolysis are the 3 independent predictors of such a risk.

961-79 Ventricular Late Potentials and Left Ventricular Function After Early ACE-Inhibition in Myocardial Infarction

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Myocardial infarction may be followed by development of ventricular late potentials (LP) and by impairment of left ventricular function.

The purpose of the present double-blind placebo-controlled study was to