however might also occur during systole. We assessed the significance of isolated systolic potentials (ISP) appearing as potentials at the end of electrical systole at sites with concealed entrainment (CE). We hypothesized that ISPs analogous to IDPs matching the stimulus-QRS (S-QRS) interval during CE might also identify critical sites within the reentry circuit.

Methods: In 23 pts (20 men; 66 ± 13 years, EF 0.29 ± 0.12) with CAD and hemodynamically stable VT, 36 VTs (CL 461 ± 83 msec) were targeted with radiofrequency (RF) ablation at 72 sites. Only sites with CE were included. Pacing (4-polar catheter, pacing: poles 1/3, recording: poles 2/4) was performed at a cycle length (CL) 30–50 msec shorter than the VT CL.

Results: Thirty-three/36 VTs could be successfully ablated. At 7/72 sites ISPs matching the S-QRS interval during CE could be identified. Using logistic regression analysis a successful ablation was associated with the presence of ISPs ($p < 0.001$) and with a match of electrogram (EGM)-QRS with the S-QRS interval (20/72 sites), ($p < 0.001$). Sites with ISPs had a longer EGM-QRS interval compared to other sites (310 ± 71 vs 116 ± 63 msec, $p < 0.001$) When sites with ISPs were compared to other successful sites their S-QRS/VTCL ratio was longer (0.67 ± 014 vs 0.38 ± 0.27, $p = 0.01$).

Conclusion: The presence of ISPs matching the S-QRS interval at sites with CE is indicative of activation within a critical zone of the reentry circuit. In selecting appropriate sites for RF ablation of VT, the late systolic period should also be inspected for ISPs, especially if there is a long S-QRS interval during CE.

1202-174 PVCs as a Harbinger of Sustained Ventricular Arrhythmias in a Canine Model of Ischemia

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The purpose of this study was to determine incidence, location and coupling interval (CI) of PVCs induced by ischemia (isch) and to relate these findings with the occurrence of sustained ventricular tachyarrhythmias.

Methods: Ten dogs underwent placement of 100 plunge needles containing 420 electrodes in both ventricles. Isch was created by thrombotic occlusion induced by anodal current of the circumflex artery which was monitored with a Doppler flow probe. The heart was divided into three regions by local electrogram morphology: 1) non-isch, 2) isch, and 3) border zone. Origin and CI of all PVCs were determined for 5 min epochs at 2 times: 1) the start of the current in the artery (control), 2) 5 min following complete occlusion (tot occ).

Results: Of 10 animals, 6 developed ventricular tachycardia followed by fibrillation (VT-VF). For VT-VF animals, PVCs increased in all 3 regions during isch with the largest increase ($p < 0.05$) in the isch area (1.2 PVCs/min control vs. 12.5 PVCs/min tot occ). For non VT-VF animals, the increase in PVCs was not markedly different ($p > 0.05$) in any of the 3 regions (0.7 PVCs/min control vs. 2.2 PVCs/min tot occ in the isch region). In VT-VF animals, CIs of the PVCs from the isch region were shorter ($p < 0.05$) in the VT-VF animals (267 ± 54 msec) than in the non VT-VF animals (285 ± 41 msec).

Conclusion: Following tot occ, the increase in PVCs is much larger and the CIs of the PVCs are shorter in those animals destined to develop VT-VF. These findings may help predict VT-VF in acute ischemia.

1202-175 Time to Termination of Post-Infarct Ventricular Tachycardia by Radiofrequency Catheter Ablation Suggests Superficial Endocardial Reentry

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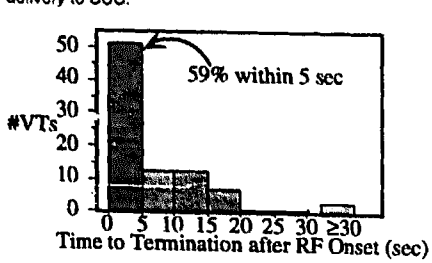
Background: Post-infarct ventricular tachycardia (VT) has been difficult to cure with radiofrequency (RF) catheter ablation (CA), possibly due to a non-endocardial location of critical circuit components (CCC) for reentry, or the insulative properties of endocardial scar prevents effective energy delivery to CCC.



Methods: We analyzed time from onset of RF to termination (T) of 87 VTs in 43 post-infarct patients (total VTs = 119). Only RF attempts causing T/permanently eradicating the VT were analyzed.

Results: T occurred in the first 5 sec of power in 51 (59%) VTs; another 19 (14%) had T in 5–10 sec (see figure).

Conclusion: These data, combined with data from explanted infarcted hearts at transplant showing minimal lesion depth after even 30 sec of RF power, suggest that: 1) Cure of most post-infarct VT is feasible with RF-CA. 2) The CCC is usually in superficial endocardial layers, not deeper. 3) Failure to cure post-infarct VT with RF CA is usually not due to inaccessibility of CCC by the ablating catheter, but rather poor tissue contact or poor mapping.

1202-176 Quantitative Analysis of Left Ventricular Endocardial Activation During Progressive Ventricular Fibrillation

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Background: Multiple wavefronts are known to be simultaneously active during ventricular fibrillation (VF). However, attempts to quantitate endocardial events during VF have been limited by difficulty obtaining multiple simultaneous endocardial surface electrograms analogous to epicardial surface mapping.

Methods: Full surface, real-time, LV endocardial activation mapping was performed during VF in 6 normal dogs using a non-contact multi-electrode array balloon catheter (Endocardial Solutions, St. Paul, MN). Computer reconstructed endocardial electrograms were dynamically displayed on a 3 dimensional model of the LV by color-coding the amplitude of the reconstructed electrograms at 3360 individual endocardial sites.

Results: Fast Fourier Transform (FFT) analysis of endocardial electrograms demonstrated a peak frequency in the power spectrum at 7.2 Hz, similar to the dominant frequency seen by surface ECG analysis (6.8 Hz) Wavefront activity was quantitated by counting the number of simultaneous non-contiguous wavefronts (SNCwaves) present every 10 msec for a 2 sec period beginning 10 sec after VF onset (Wiggers' stage II) and again 5 min later. The number of distinct wavesfronts present at any given time decreased from 2.4 ± 1.1 at 10 sec of VF to 1.8 ± 0.8 at 5 min ($p < 0.05$). FFT of the SNCwaves, which provides an index of the "turnover" rate of new wavefronts, had a peak frequency of 9.0 Hz at 10 sec and 7.9 Hz 5 min later ($p = 0.26$).

Conclusion: After 5 min of untreated VF, fewer wavefronts are present, but they emerge and dissipate at the same rate as in early VF.

1202-177 Subendocardial Temperature Differences During Radiofrequency Catheter Ablation: Normal vs Scar Tissue

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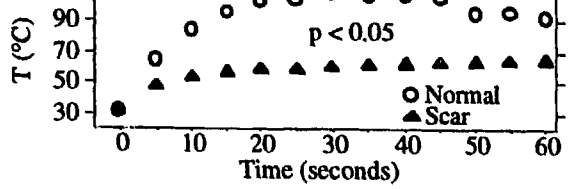
Radiofrequency (RF) catheter ablation of post-infarct ventricular tachycardia (VT) is difficult, perhaps due to inadequate temperature (T) achieved in subendocardial regions. We performed in vitro RF ablation in freshly explanted human hearts with prior transmural infarcts (obtained at time of transplantation). A total of 17 RF applications were made in regions of scarred (S) and normal (N) myocardium targeting a catheter tip T of 70°C for 60 seconds. Subendocardial T was measured with a needle thermocouple at a depth of 2 mm. There was no significant difference in *catheter tip T* (maximum/mean = 64 ± 8/60 ± 8 for S, 67 ± 9/61 ± 9 for N, p = NS) or power delivery (maximum/mean = 45 ± 7/31 ± 13 for S, 47 ± 1/32 ± 10 for N, p = NS) in S and N myocardium. *Subendocardial Ts* are shows below and are significantly lower in S than N myocardium.



Conclusions: 1) Deep tissue heating is difficult to achieve in S myocardium using standard RF catheters. 2) The reported clinical success of RF ablation in post-infarct VT suggests that the critical components of *most* VT circuits are located in, or just below, the endocardium. 3) VT circuits not amenable to RF catheter ablation may have critical components located deeper in the subendocardium.

WEDNESDAY POSTER