conduction, the later was felt to be arrhythmogenic, and radiofrequency (Rf) catheter ablation was proposed. Extensive multipolar catheter mapping using an encircling technique during fixed N.RA pacing revealed electrical propagation through the suture line at the level of the RA appendage *(n = 2)* or the low lateral RA *(n = 1)*. 4, 3 and 1 Rf pulses were delivered at the site of shortest conduction time between the pacing site and the ablation catheter when positionned across the suture line. Local electrogram morphology was double spikes *(n = 2)* or fragmented potential *(n = 1)*.

Results: Complete abolition of atrioatrial conduction was obtained in the 3 cases with a mean procedure duration of 4 hours and a mean fluoroscopy time of 27 min. No tachycardia was observed during a mean follow up of 9 months (range 5–15). A total disappearance of premature atrial contractions was seen at control 24 hours Holter recordings.

Conclusion: Although unfrequent, abnormal atrioatrial conduction may be seen after orthotopic heart transplantation and may result in clinically relevant G.RA arrhythmias. Rf catheter ablation at the level of impulse propagation across the suture line may provide a definite cure of arrhythmias in this selected group of patients.

928-27 Thermometry-Guided Ablation Using Commercial Systems

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Utilizing 3 commercial thermometry ablation systems (*I.* EP Technologies, *II.* Medtronic/Cardiorhythm, *III.* Cordis/Webster), we compared myocardial temperature profiles and lesion volumes during radiofrequency power *((P))* application adjusted to achieve and maintain an electrode temperature of 55° C for 120 seconds. Each electrode (8F, 4 mm) was placed into contact (10 gram force) with bovine myocardium mounted in a tank circulating a saline-dextrose solution whose physical properties were equivalent to blood. Two electrode orientations were evaluated: 1. perpendicular and 2. parallel to the myocardial surface. Three solution flow velocities (*F*, measured at the electrode-tissue interface) were evaluated: 0, 0.2 and 0.4 m/sec. Measurement of electrode (T_E) and myocardial temperature at 1 (T_1) and 3 (T_3) mm directly beneath the electrode-tissue interface was performed. Lesion volumes (*V*, mm³) were measured. *Results:* (Table: mean ± SD; • $p < 0.05$ vs $F = 0$):

System	*F*	Perpendicular				Parallel			
		P	T_1	T_3	*V*	*P*	T_1	T_3	*V*
I	0	9±3	46±3	46±3	3±3	4±1	43±3	44±3	3±8
I	0.2	8±2	53±4	52±3	43±24	17±5	63±8	62±6	309±101
I	0.4	31±7	61±11	63±9	762±196	40±5	82±9	80±11	465±95
II	0	4±1	44±1	47±3	0	3±1	46±3	46±2	0
II	0.2	12±4	56±5	63±3	99±27	14±4	59±3	63±4	128±30
II	0.4	27±6	67±6	79±5	408±135	28±5	71±6	81±5	339±48
III	0	4±1	45±4	41±3	0	3±1	48±2	46±4	0
III	0.2	12±3	59±5	56±5	76±42	11±2	67±7	67±6	122±61
III	0.4	19±5	61±5	64±6	254±97	14±3	74±9	76±7	163±70

Each system acted similarly. At $F = 0$, T_E was greater than T_1 and T_3, and *V* was small or absent. As *F* was increased, the *P* requirement also increased, resulting in progressive increases in T_1, T_3 and *V*. At $F = 0.2$, both T_1 and T_3 were greater than the 55° target for parallel but not perpendicular orientations; at $F = 0.4$, this was true for both orientations.

928-28 Electrode Size and Temperature Effects on Lesion Volume During Temperature-Controlled RF Ablation in Vivo

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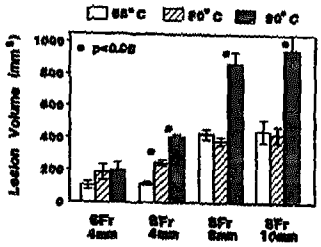
The correlation between electrode (*elec*) size or temperature (*temp*) with lesion size during radiofrequency (*RF*) ablation is good in vitro, but the effect of new *elec* geometries on this correlation using *temp* feedback power controlled RF delivery in vivo has not been tested.

Methods: Seven mongrel dogs were anesthetized and the femoral artery and vein were cannulated. Four catheters were used with 6 Fr. 4 mm, 8 Fr. 4 mm, 8 Fr. 8 mm, or an 8 Fr. 10 mm *elec* tip (EP Technologies). Serial lesions were made in the RV and LV with *temp* feedback power control (up to 150 W, EPT) at target *temps* of 65, 80, and 90° C. The animals were sacrificed and the lesions stained for gross examination.

Results: 60 lesions were identified. Mean power for all *temps* were 11.2 ± 6.2, 17.4 ± 12.9, 47.3 ± 33.7 ($p = 0.0001$), and 60.4 ± 32.4 W with increasing *elec* size. Lesion size increased with both *elec* size and *temp* (graph). With the 8 and 10 mm *elecs* char was noted in 5 and popping in 3 of 39 lesions. There was 1 case of impedance rise. All but one event occurred at *temp* = 90°C.



Conclusion: Using *temp* feedback power control in vivo, lesion size may be predictably increased with higher preset *temps* and large elec tip sizes. Temps of 90° C may have excess popping and charring.

929 Arrhythmia Mechanisms

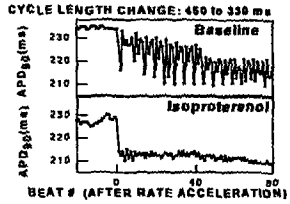
Monday, March 25, 1996, 3:00 p.m.–5:00 p.m.
Orange County Convention Center, Hall E
Presentation Hour: 3:00 p.m.–4:00 p.m.

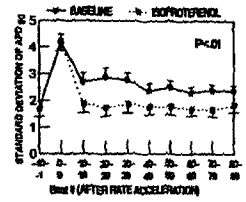
929-54 Oscillations in Human Ventricular Repolarization After Abrupt Rate Acceleration and Beta-Adrenergic Stimulation

Philip T. Sager, Steve W. Koh. *West Los Angeles VAMC/UCLA, Los Angeles, CA*

Oscillations in ventricular repolarization and refractoriness may play an important role in the genesis of spontaneous clinical VT/VF but oscillations of human ventricular action potentials have not been studied. We examined the beat to beat APD_{90} during steady state RV pacing at a cycle length (CL) of 450 ms and following acute acceleration to a CL of 330 ms in 18 pts. To determine the effects of beta-adrenergic stimulation, the measurements were repeated during steady state isoproterenol (ISOP; 35 ng/(kg·min)). Oscillations were analyzed by examining the mean standard deviation of APD_{90} for every 10 beats.

Oscillations ranged from 2–21 ms, variability was greatest ($p < 0.05$) immediately after rate acceleration, and were increased compared to pre-acceleration in the baseline group. Quasi-periodic APD oscillations were observed. After the first 10 beats of rate acceleration, ISOP reduced APD oscillations ($p < 0.01$).





Thus, APD oscillations following abrupt increases in heart rate occur in humans, variability is enhanced acutely after rate acceleration, and are reduced during ISOP in this study. Oscillations may be important in arrhythmia initiation and termination.

929-55 Mechanisms of Ventricular Tachycardia Termination in the Human Heart

Steven M. Pogwizd, Mina K. Chung[1], Michael E. Cain. *Washington Univ., St. Louis, MO;[1] The Cleveland Clinic, Cleveland, OH*

To define the electrophysiologic mechanism(s) by which ventricular tachycardia (VT) terminates, three-dimensional cardiac mapping was performed in 6 patients with healed myocardial infarction undergoing surgery for VT. Data from 43 nonsustained (3 to 39 beats) VTs (NSVT) and 6 sustained VTs (SuVT) were analyzed. The total activation times (140 ± 6 ms) and coupling intervals (312 ± 12 ms) for the terminal beats of NSVT were similar to those measured from beats during SuVT (162 ± 14 and 278 ± 29 ms, $p = 0.16$ and 0.28, respectively). Termination of VT was due to either: 1) activation from multiple subendocardial or subepicardial sites that were discordant from the sites initiating SuVT (45%); 2) repetitive firing of sites discordant from those initiating SuVT, which were at times preceded by oscillation in total activation time or coupling interval (24%); or 3) repetitive activation from sites concordant with those initiating SuVT which either failed to shift to initiation sites required for maintenance of SuVT or which stopped suddenly (31%). Electrode density was sufficient to define the mechanism for 57 beats of

MONDAY POSTER