1160-109 Mechanisms Underlying the Reentrant Circuit of Ventricular Tachycardia in Isolated Canine Left Ventricular Preparation Using Optical Mapping

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The cellular mechanisms underlying the reentrant circuit of ventricular tachycardia (VT) after myocardial infarction (MI) have not yet been clearly understood. Using optical mapping and microelectrode recording in isolated canine left ventricular preparation, we characterized the reentrant circuit of VT and its relationship to action potentials recorded in the infarction border zone (BZ). The preparation was isolated from left ventricular free wall and perfused with Tyrode's solution through the left circumflex artery (LCX). MI was created by completely tightening one of the LCX branches. No VT was induced before MI by programmed extrastimuli (S1 to S4). However, 4 hrs after MI, sustained monomorphic VTs at cycle lengths of 375±12 ms were induced in all 6 preparations. Optical mappings during sustained VT showed 2 types of reentrant circuits as functional spiral waves reentry (n=4) using only part of the BZ and anatomic reentry rotating around the obstacle of infarction tissue (n=2). Low amplitude fractionated electrograms were obtained from site of slow conduction within the BZ. Compared to the non-ischemic normal tissue, action potentials recorded from the BZ (n=6) showed decrease in amplitude (87.5±2.5 vs. 62.2±4.5 mV, p<0.01), maximal diastolic potential (80.4±2.2 vs. 58.7±5.4 mV, p<0.05), dV/dt (156.6±12.7 vs. 26.4±6.5 V/sec, p<0.05), and ADP90 (208.3±7.2 vs. 145.2±7.4 ms, p<0.01), consistent with slow conduction and unidirectional block occurred in the BZ. In conclusion, sustained monomorphic VTs developed after MI were due to functional spiral wave reentry or anatomic macroreentry around the infarction area. Both types of reentries involved in the BZ with delayed conduction and unidirectional block.

1160-110 A Comparison of Left Atrial Volume Measured by Magnetic Endocardial Catheter Mapping Versus Two-Dimensional Transthoracic Echocardiography

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Background: Increased left atrial volume (LAV) is associated with a higher risk of recurrent atrial fibrillation (AF) and atrial arrhythmias. Two-dimensional (2-D) transthoracic echocardiography is validated as a reliable method by which to assess LAV compared to cine-computed tomography (cine-CT). Using the biplane method of disks, LAV measured by transthoracic echocardiography is well correlated with that obtained by cine-CT, but under estimates the LAV by 20-32%. Magnetic electroanatomic mapping (MEAM) is valuable for defining the anatomic location of catheter-based electrophysiologic recordings by creating a detailed shell of the endocardial anatomy in three-dimensional space that can help guide focal AF ablation. We sought to validate the accuracy of volume measurements by MEAM by comparing MEAM LAV measurements against those measured by two-dimensional transthoracic echocardiography.
Methods: Forty-seven patients underwent 2-D echocardiography and detailed MEAM of the left atrium (LA). The entire LA was mapped with 78-224 distinct points (mean 126 ± 37) acquired during atrial end-diastole. MEAM measurement of LAV was computed by using the built-in volume function of the Biosense™ system. The LAV was assessed using 2-D transthoracic echocardiography by the biplane methods of disks. The endocardial outlined was digitally traced in the apical 4-chamber and 2-chamber views at end-atrial diastole with exclusion of the pulmonic veins and appendage.
Results: The LAV by 2-D echocardiography was 92.7 ± 25.9 cc versus the LAV by MEAM which was 125.4 ± 28.4 cc. There is good correlation between the results of echocardiography and MEAM (r=0.90, p<0.001) for LAV, although the average value obtained by echocardiography is about 26% lower than that obtained by MEAM.
Conclusions: Magnetic electroanatomic catheter mapping appears to be a reliable method by which to assess LAV. The results of MEAM correlate well with the echocardiographic assessments of LAV. Echocardiography underestimates LAV by a similar percentage when compared to MEAM as it does when compared to cine-CT. MEAM may prove useful in tracking LAV with repeat mapping procedures and helping to plan post-ablative management.

POSTER SESSION

1161 Pathophysiology of Supraventricular Arrhythmias

Tuesday, March 19, 2002, 9:00 a.m.-11:00 a.m.
Georgia World Congress Center, Hall G
Presentation Hour: 10:00 a.m.-11:00 a.m.

1161-111 Coronary Sinus Ostium Size Is Associated With Longer Atrioventricular Conduction Times

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Background: Although the coronary sinus ostium is in close proximity to internodal pathways, the relationship between coronary sinus ostium size and atrioventricular (AV) conduction time has not been described.
Methods: We examined the morphological features of the coronary sinus in a cohort of 31 patients undergoing electrophysiologic testing for supraventricular tachycardia (SVT) using cineangiographic imaging. The diameter of the coronary sinus ostium was measured at the point of entry into the right atrium in left anterior oblique projection after contrast dye was injected through a lumen catheter. We also recorded baseline PR, PA, AH, and HV intervals, and the presence or absence of dual pathway physiology using single atrial extrastimuli to detect discontinuous antegrade AV conduction. Bivariate correlation and one-way analysis of variance tests were performed to evaluate the association between coronary sinus ostium diameter and AV conduction times, dual pathway physiology, and type of SVT.
Results: 15 patients had AV nodal reentry and 16 patients had either AV reciprocating tachycardia or atrial tachycardia. There were 23 (74%) tubular and 8 (26%) funnel shaped coronary sinus ostia (mean diameter: 12.5 ± 3.8 mm). There was a significant correlation between the coronary sinus ostium diameter and the AH (P<0.01) and PR intervals (P<0.01). The coronary sinus ostium diameter, regardless of morphology, had no significant correlation with the duration of the PA (P=0.49) or HV interval (P=0.78). Similarly, coronary sinus ostium diameter showed no correlation with the presence of dual pathway physiology (P=0.63) or type of SVT (P=0.42).
Conclusion: This study confirmed the previously described lack of association between coronary sinus size and type of SVT or presence of dual pathway physiology. In this cohort of patients, CSO diameter is significantly correlated to longer AV nodal conduction times (AH and PR intervals). This association may be due to a longer or thinner fast pathway associated with a larger CS ostium and/or delayed electrotonic influence to the fast pathway from a longer slow pathway.

1161-112 The Differences Between Posteroseptal and the Other Atrioventricular Accessory Pathways: The Coronary Sinus Morphology and the Conduction Over Accessory Pathways

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Backgrounds: There were few reports about the differences between posteroseptal and the other atrioventricular accessory pathways (APs) in anatomy and electrophysiology.
Methods: The size and shape of coronary sinus (CS) were measured in 21 patients with posteroseptal APs (11 right and 10 left posteroseptal wall), 83 with the other APs (63 left lateral, 13 right lateral, 7 right anteroseptal wall) and 25 control subjects after CS angiography. CS diameter and morphologic features were measured. In 38 patients with APs, we investigated the electrophysiological characteristics about anterograde and retrograde conduction over APs.
Results: The proximal CS in patients with posteroseptal APs was larger than in those with the other APs and the control (13.8 +/- 1.3 mm vs. 10.9 +/- 2.1 mm [p < 0.001] and 9.7 +/- 1.5 mm [p < 0.001], respectively). At a distance of 5 mm from the CS ostium, the CS measured 10.8 +/- 0.8 mm, compared with 8.9 +/- 1.9 mm [p < 0.05] and 8.2 +/- 1.8 mm [p < 0.01]. The dilatation persisted 10 mm into the CS, with the measurement of 8.6 +/- 1.2 mm, compared with 7.5 +/- 1.8 mm [p<0.05] and 7.2 +/- 1.7 mm [p<0.05]. There were no differences in these distal diameter. In 67% of patients with posteroseptal APs, the proximal CS had the wind-cone appearance. This morphology was found in 16% of patients with the other APs. In all of control subjects and 84% of patients with the other APs, the CS was the tubular. Only 1 patient with a posteroseptal AP had retrograde and anterograde decremental conduction over the AP. Three patients with posteroseptal APs, 2 with left lateral, 2 with right rateral and 1 with anteroseptal had only retrograde conduction. Posteroseptal APs with decremental conduction were located only in the right side.
Conclusions: The larger size of proximal CS was a structural characteristics in patients with posteroseptal APs. The appearance of proximal CS was like a wind-cone in these patients. Right posteroseptal APs were prone to have the high incidences of decremental conduction. These findings may have a clue to trace arrthythmia pathogenesis to its origin.

1161-113 Short-Term Rapid Atrial Pacing Produces Electrical Remodeling of the Sinus Node Function in Humans

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Background: Depression of sinus node function occurs in dogs, and in patients, following cessation of atrial flutter and fibrillation. We tested whether transient atrial pacing might produce similar changes.
Methods and Results: We studied the impact of short term rapid atrial pacing, simulating atrial tachyarrhythmia, on sinoatrial conduction time (SACT) and corrected sinus node recovery time (CSNRT) in 10 patients undergoing electrophysiologic study for supraventricular (6) or ventricular tachycardia (2), syncope (1) and pacemaker implant for heart block (1). None had structural heart disease, history of atrial fibrillation or flutter, autonomic dysfunction or any tachycardia for at least 24 hours prior to study. All cardiac drugs were discontinued ≥5 half lives prior to study. No patient had significant hypotension during atrial stimulation. SACT and CSNRT were measured at baseline and sinus node reset zone was determined. Pacing from high right atrium was started at the cycle length at which the reset zone was established (range 350-500 ms). Pacing was terminated after 10-15 minutes and within 30-60 seconds, SACT and CSNRT were measured again (ms) and showed a significant increase.

	Baseline	Post-pacing	Difference	P Value (paired t test)
SACT	80 ± 50	96 ± 53	16 ± 16	0.02
CSNRT	423 ± 208	491 ± 214	68 ± 41	0.001

Conclusion: Rapid atrial pacing for only 10-15 minutes, simulating atrial tachyarrhythmias, prolongs SACT and CSNRT in humans. Additional studies are needed to evaluate the mechanism, but the clinical implication is that even transient episodes of atrial tachyarrhythmias can cause sinus node remodeling in patients.