activity by upright position, and make it possible to expand the indication of thoracic sympathicotomy to various cardiac diseases.

946-76 Assessment of Autonomic Function in Patients With Neurally Mediated Syncope

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The aim of the study was to evaluate the function of the autonomic nervous system (ANS) in patients with neurally mediated syncope. Holter recordings were obtained from 30 patients (21 men, age 50 ± 13) with a history of syncope and a positive tilt test (Group A) and 20 healthy controls (12 men, age 52 ± 15) (Group B) during tilt testing and over a further 24-hour period. We compared 1) spectral indexes (low and high frequency spectral power: LF and HF) of heart rate variability (HRV) and the ratio LF/HF during the periods 10 min before tilt at supine rest and 10 min immediately after tilt in the 60º head up position while all subjects were asymptomatic; 2) three time-domain indexes (SD, pNN50, rMSSD) and the spectral indexes of HRV for the whole 24-hour period.

There were no significant differences between the groups in any of the temporal or spectral indexes of HRV. In Group A, passive tilt caused a small but significant decrease in LF (4.88 ± 1.15 to 4.28 ± 1.04, $p < 0.02$) and HF (5.20 ± 1.40 to x.xx ± 1.44, $p < 0.05$), while the LF/HF ratio did not change significantly. In Group B there was a small but significant increase in LF (4.7 ± 0.8 to 5.63 ± 0.42, $p < 0.001$) and LF/HF (0.89 ± 0.15 to 1.19 ± 0.20, $p < 0.003$), and a significant decrease in HF (5.46 ± 0.68 to 4.65 ± 0.76, $p < 0.001$).

There is evidence from HRV data that patients with neurally mediated syncope have no chronic differences from normal subjects in ANS activity, but that these patients have a different pattern of response to the orthostatic stimulus.

947 Basic Mechanisms of Ventricular Arrhythmias

Tuesday, March 26, 1996, 9:00 a.m.–11:00 a.m.
Orange County Convention Center, Hall E
Presentation Hour: 9:00 a.m.–10:00 a.m.

947-22 Role of Purkinje Fibers in Induction of Early Afterdepolarizations and Triggered Ventricular Tachycardia in Isolated Rabbit Hearts

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To define the role of Purkinje fibers (PFs) in the genesis of early afterdepolarizations (EADs) and EAD triggered ventricular tachycardia (VT), we infused clofilium (5.5 μM), a delayed rectifier I_K blocker, in 16 isolated Langendorff-perfused rabbit hearts. The AV node was ablated and monophasic action potential recorded from the left ventricular epicardium. In group 1 (control, n = 10), clofilium infusion induced EADs and EAD triggered VT in all the 10 hearts (100%) at 7.2 ± 1.1 (M ± SEM) and 11.9 ± 2.0 min., respectively. Of note, there was a significant lengthening of action potential duration at 50% (APD_{50} 211 ± 10 → 276 ± 24 ms,) and at 90% (APD_{90} 299 ± 9 → 455 ± 39 ms) repolarization (both $P < 0.05$) along with prolongation of the RR interval (779 ± 66 → 1318 ± 14 ms, $P < 0.05$) before the onset of EADs. In group 2 (n = 6), the left and right ventricles were flushed with Lugol solution prior to infusion of clofilium. Lugol solution treatment resulted in disappearance of all spontaneous ventricular rhythms indicating destruction of PFs. All 6 hearts were paced from the right ventricle at a cycle length of 1500 ms. Clofilium (5.5 μM) infusion lengthened APD_{50} (194 ± 25 → 216 ± 33 ms, $P > 0.05$) and APD_{90} (300 ± 23 → 347 ± 28 ms, $P < 0.05$), but induced no EADs or EAD triggered VT.

We conclude that PFs play an essential role in the induction of EADs and EAD triggered VT in isolated rabbit hearts.

947-23 Increase of Intracellular Systolic Calcium by Single Pulse Premature Ventricular Stimulation

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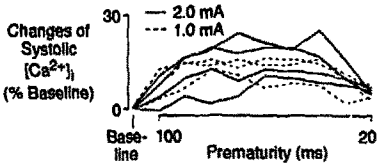
Cardiac vulnerability to ventricular fibrillation (VF) is frequently evaluated by VF threshold (VFT). However, electrical stimulation used for VFT determination has been shown to release norepinephrine which may perturb VFT interpretation. Similar perturbation may arise from Ca^{2+} which has been proposed to be a determinant of vulnerability to VF. Therefore, we tested whether VFT determination by a conventional single pulse method affects intracellular Ca^{2+} ($[Ca^{2+}]i$). For this purpose, we measured $[Ca^{2+}]i$ in isolated intact rat hearts during VFT determination by the single pulse method. Single pulses at increasing prematurity were introduced after every eighth sinusbeat (10 ms increments starting at 100 ms to scan the vulnerable period). Systolic $[Ca^{2+}]i$ of cardiac cycles preceding premature beats was measured and plotted against prematurity. Only arrhythmia-free sequences were included in this analysis. $[Ca^{2+}]i$ was assessed by surface fluorometry after indo-1 loading.



We found that VFT determination by increasing prematurity of a single pulse led to a rise in systolic $[Ca^{2+}]i$ reaching a maximum around 40 ms prematurity. This rise was independent of stimulation intensity ($p > 0.37$ at all prematurity states; paired t-test). Interestingly, the maximal rise coincided with the reported vulnerable period in rat hearts. This rise could be a contributing factor in the induction of VF by the single pulse method facilitating nonuniform dispersion of refractoriness. Moreover, it could perturb VFT interpretation in studies using Ca^{2+} antagonists.

947-24 Influence of Refractory Period on Ventricular Fibrillation Cycle Length

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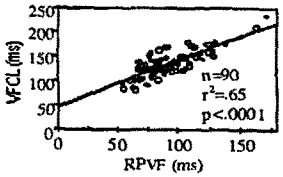
We have previously shown that an excitable gap (EG) is present in experimental ventricular fibrillation (VF). To evaluate how refractory period (RP) affects cycle length (CL) in a model of reentry with an EG, we studied VF in 15 dogs. Six dogs (group A) were studied at control, 5 (group B) 5 days, and 4 (group C) 8 weeks after MI. Effective RPs at paced CL of 300 ms (RP300) were determined at 112 anterior LV (infaction) sites. VF was induced by pacing and VFCL determined at each site. At selected sites the RP in VF (RPVF) was determined by analysis of local activation and block. *Results:* RPVF could be determined at 90 sites. Mean VFCL was 135 ± 27 ms, mean RP300 175 ± 21 ms, and mean RPVF 94 ± 23 ms. The table shows linear regression R^2 values for each variable pair and the figure VFCL-RPVF correlation for the total group.

	Group A	Group B	Group C	Overall
VFCL-RPVF	0.45+	0.62+	0.46+	0.65+
VFCL-RP300	0.37+	0.07	0.40+	0.02
RP300-RPVF	0.29*	0.001	0.29*	0.05

*$p < 0.05$; +$p < 0.001$



Conclusions: 1) VFCL correlates with RPVF despite an EG, perhaps because the RP determines the length of lines of functional block and thus of reentrant circuits. 2) VFCL correlates with RP300 in normals and chronic MI, because RP300 is correlated with RPVF. In sub-acute MI, VFCL does not correlate with RP300, due to lack of a consistent effect of CL on RP at different sites.

947-25 Nearfield Delay Is the Major Determinant of Ventricular Conduction Latency Occurring With Premature Stimulation in Chronic Heart Failure

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To determine the relative contribution of nearfield (NF) and distant (DIS) conduction delay (CD) to apparent ventricular conduction latency observed with premature ventricular stimulation (VPC), 8 dogs with pacing-induced cardiomyopathy (CMP) and 6 control dogs were studied using a 12 bipolar electrode plaque. Apparent CDs from a central co-axial bipolar pacing site (S) to the proximal electrode at 3 mM (PE) and the distal electrode at 11 mm