percutaneous approach for revascularization and replacement of transvenous pacemaker system is feasible, and can be an alternative to surgery for patients presenting with SVC syndrome due to chronic pacemaker leads.

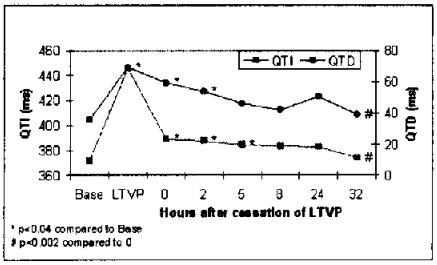
1115-110 Cardiac Memory: Effects on QT Interval and Dispersion

Maher C. Nahlawi, Michael Waligora, Stewart M. Spies, Robert O. Bonow, Alan H. Kadish, Jeffrey J. Goldberger, *Northwestern University Medical School, Chicago, Illinois.*

To evaluate the effects of long-term ventricular pacing (LTVP) on the QT interval (QTI) and QT dispersion (QTD), 12 subjects (8 women), mean age 68 ± 12 years, with dual chamber pacemakers, normal left ventricular function, and intact AV conduction were studied. Patients had standard 12 lead ECGs after 1 week of atrial pacing only (Base), after 1 week of AV pacing with an AV delay of 100 ms to ensure 100% ventricular pacing (LTVP), immediately after cessation of LTVP, and 2, 5, 8, 24, and 32 hours after cessation of LTVP. All data were acquired at a pacing rate of 80 bpm. QTI was measured in each lead, and the longest interpretable QTI was reported for each individual ECG. QTD was defined as the maximum QTI minus the minimum QTi.

Results: Persistent T wave inversions characteristic of cardiac memory were evident on all ECGs after cessation of LTVP (0-32 hours). The figure shows QTI and QTD. QTI and QTD were significantly prolonged during LTVP and after cessation of LTVP ($p<0.04$ versus Base) for up to 5-8 hours and gradually returned to baseline values.

Conclusion: After cessation of LTVP, the QTI and QTD are prolonged. These changes resolve before the T wave inversions characteristic of cardiac memory, suggesting that LTVP has multiple electrophysiologic effects on ventricular repolarization with different time courses of recovery.



POSTER SESSION

1116 Noninvasive Testing: Measuring Autonomic Tone

Monday, March 18, 2002, Noon-2:00 p.m.
Georgia World Congress Center, Hall G
Presentation Hour: 1:00 p.m.-2:00 p.m.

1116-103 Differential Catecholamine Response In Different Types of Neurocardiogenic Syncope

Steffen Loscher, Frank Mickley, Martin Ludewig, Andreas Hartmann, *Dept. of Cardiology, St. Georg Hospital, Leipzig, Germany.*

Background: Syncopal episodes are frequently caused by a neurohumoral mechanism. Tilt-table(TT) - testing has been established as a diagnostic tool to detect and differentiate neuro-cardiogenic syncope. Few data exist about the neurohumoral profile during TT-testing. It was the goal of this study to determine catecholamine profiles during TT- testing induced syncope.

Method: 60 patients (26 male, 34 female, mean age 60,4 ± 18,7 years) with a history of syncopal episodes of undetermined origin underwent a TT- testing protocol (70° tilt, 45 minutes). Syncope was inducible in 33,3 % of all patients. Episodes were classified as vasodepressant syncope (VS) or cardioinhibitory syncope(CS) .

22 Patients (mean age 58,9 ± 17,6 years) without a history of syncopal episodes served as controls.

Results: Catecholamine levels were determined at the begin of the test and at the time of syncope or at the end of the test if no syncope occurred. Catecholamine response is depicted in table I

	Adrenaline (ng/l)	Noradrenaline (ng/l)	Dopamine (ng/l)
VS - type(n = 10)			
Testbegin	47,7 ± 20	116,3 ± 84	18,1 ± 8
Syncope	82,1 ± 59	154,1 ± 107	32,1 ± 20
CS - type (n= 10)			
Testbegin	107,2 ± 68 **	348,1 ± 161 **	47,0 ± 32**
Syncope	258,6 ± 159++**	415,8 ± 188	54,6 ± 35
Non-inducible (n = 40)			
Testbegin	49,3 ± 28	171,2 ± 71	31,5 ± 24
During Test	66,2 ± 41	197,8 ± 51	48,1 ± 36
Control (n=22)			
Testbegin	34,7 ± 14	141,0 ± 49	21,7 ± 11
During Test	49,9 ± 18	180,3 ± 84	28,7 ± 14

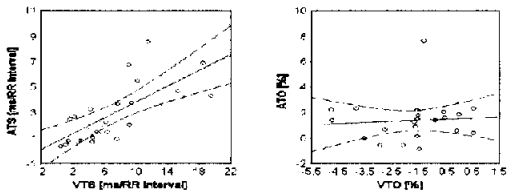
(++ p<0,005 test begin versus syncope, ** p<0,005 CS-type versus VS-type, syncope non-inducible and control group)

Conclusion: In conclusion, a different catecholamine response was found in CS-type patients as compared to VS-type patients suggesting a different degree of sympathetic stimulation and a different pathophysiological mechanism. This finding may have implications in patient selection for beta-blocker therapy of neurocardiogenic syncope.

1116-104 Heart Rate Turbulence Can Be Detected After Atrial Premature Beat

Irina Savelieva, Dan Wichterle, Azad Ghuran, Maggie Meara, John Camm, Marek Malik, *St George's Hospital Medical School, London, United Kingdom.*

Heart rate turbulence (HRT) describes a bi-phasic chronotropic response of sinus rhythm to a ventricular premature beat (VPB). The aim of the study was to investigate whether an atrial premature beat (APB) can produce HRT and whether it can be described using ventricular HRT quantifiers (turbulence onset, TO, and turbulence slope, TS). Methods: Thirty-seven subjects (54±16 years) referred for electrophysiologic evaluation for ventricular tachyarrhythmias underwent a stimulation protocol consisting of 3 series of single atrial and ventricular extrastimuli delivered from the high right atrium and right ventricular apex prior to programmed electrical stimulation. Atrial and ventricular TO and TS (ATO, ATS, VTO, VTS) were calculated separately using a dedicated computer algorithm. Results: Expressed as mean±SD, VTO -2.03±0.42%, ATO 1.37±0.35% ($p<0.001$); VTS 8.32±1.9 ms/RR, ATS 3.95±0.99 ms/RR ($p<0.008$). There was a strong correlation between VTS and ATS ($r=0.71$, $p=0.0002$; Figure, right) but not between VTO and ATO ($r=0.83$, $p=0,074$; left). Conclusions: HRT phenomenon is present after an APB, suggesting that similar baroreceptor mechanisms operate following both VPB and APB. After an APB, the late phase response (rhythm deceleration) is significantly attenuated and the early phase response is different than that after a VPB, indicating the presence of alternative mechanisms. Atrial HRT can be measured using existing technique but algorithm adjustments are desirable.



1116-105 Characteristics of Heart Rate Recovery After Maximal Exercise

Jeffrey J. Goldberger, Prince J. Kannankeril, Francis K. Le, Alan H. Kadish, *Northwestern University Medical School, Chicago, Illinois.*

Ten normal subjects (5 men, age 33±5 years) underwent maximal exercise (Ex) testing on a bicycle ergometer. On day 1, subjects exercised to maximum tolerated workload (140±29 Watts, 175±16 bpm) using a graded protocol with 5 minutes at peak workload. On day 2, subjects performed the identical Ex protocol as on day 1; one minute into the maximum Ex stage, atropine (0.04 mg/kg) was administered. Heart rate (HR) was recorded each minute during recovery for 10 minutes. Each subject's HR recovery data was fit to a monoexponential decay of the form $HR = a+b * \exp(-t/\tau)$, where t is time in recovery, τ is the time constant for HR recovery, and a and b are constants. Results: All subjects demonstrated a monoexponential decay in HR during recovery on each day (r^2 0.94-1.0 [mean 0.98±0.02] on day 1 and 0.95-0.99 [mean 0.99±0.01] on day 2). τ was 1.4±0.4 min on day 1 and 1.6±0.5 min on day 2 (p=NS). HR recovery (HR at peak Ex minus HR 1 minute into recovery) was 42±9 bpm on day 1 and 22±8 bpm on day 2 ($p<0.0003$). The table shows HR on each day at 0, 1, 5, and 10 minutes of recovery. There was a negative correlation between HR recovery at 1 minute and τ (day 1: $r^2=0.51$, $p<0.02$; day 2: $r^2=0.46$, $p<0.03$). Conclusion: In normal subjects, approximately half the HR recovery is attributable to reactivation of parasympathetic effects. The time constant for HR recovery is inversely correlated with 1 minute HR recovery. As τ provides a more complete characterization of HR recovery, this parameter may provide even better prognostic information than 1 minute HR recovery.

HR at various time points in recovery (in bpm)

	0 min	1 min	5 min	10 min
Day 1	174±17	133±18	104±21	98±22
Day 2*	178±14	156±16	134±18	130±18

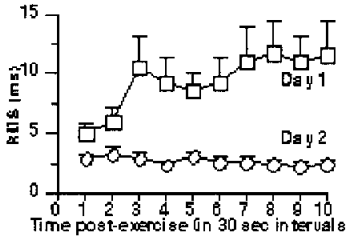
* $p<0.05$ versus day 1

1116-117 Heart Rate Variability After Exercise Is Related to Recovery of Parasympathetic Tone

Francis K. Le, Prince J. Kannakeril, Alan H. Kadish, Jeffrey J. Goldberger, *Northwestern University, Chicago, Illinois.*

To assess the recovery of heart rate variability (HRV) after exercise (Ex) and its relationship to recovery of parasympathetic (P) effect (PE), 10 normal subjects (5 men, age 33±5 yrs) exercised on a bicycle ergometer. On day 1, subjects exercised for 14.2±2.3 minutes, to a peak workload of 140±29 Watts and peak HR of 175±16 bpm. On day 2, subjects exercised using their same day 1 protocol; atropine (0.04 mg/kg) was infused over 2 minutes during the maximum Ex stage. Upon termination of Ex on each day, a 5 minute ECG recording was made. RR interval tachograms were generated and in each 30 second segment, linear regression analysis was performed. HRV was defined as the root

mean square residual (RMS) from the linear regression analysis; RMS quantifies the deviation of the RR interval from a pure linear change over the 30 sec period. PE was defined as the difference in the 30 sec average RR intervals without (day 1) and with (day 2) atropine. Results: RMS dramatically increases after 1 minute of recovery. P blockade attenuates the increase in RMS. Day 2 RMS was less than day 1 RMS for all time periods ($p<0.03$). There was a correlation between day 1 RMS and PE ($R2 = 0.63$, $p< 0.0001$) Conclusion: RMS is a new measure of HRV that is related to recovery of PE after Ex. RMS may help differentiate whether the recently demonstrated prognostic significance of HR recovery after Ex testing is due to P recovery or sympathetic withdrawal, both of which decrease HR. If P recovery is more important, RMS may provide better risk stratification than HR recovery.



1116-118 **Effects of Nesiritide and Dobutamine on Heart Rate Variability in Patients With Decompensated Heart Failure**

Doron Aronson, Darlene P. Horton, Andrew J. Burger, *Rambam Medical Center, Haifa, Israel, Beth Israel Deaconess Medical Center, Boston, Massachusetts.*

INTRODUCTION: Congestive heart failure (CHF) is characterized by sympathetic overactivity and parasympathetic withdrawal. Clinical trials in patients (pts) with decompensated CHF have shown that nesiritide (brain natriuretic peptide) is associated with beneficial hemodynamic effects and symptomatic improvement. Recent studies suggest that nesiritide exerts a favorable effect on autonomic dysfunction in CHF. We compared the effect of nesiritide and dobutamine (Dob) on heart rate variability (HRV) in decompensated CHF.

METHODS: We studied 185 pts admitted for decompensated CHF requiring intravenous vasoactive therapy. Baseline 24-h Holters were obtained prior to initiation of the study drugs (nesiritide or Dob) and continued for an additional 24-h after initiation of therapy. The study population was divided into 2 groups: highly depressed HRV (SDNN < 50 ms) and moderately depressed HRV (SDNN = 50-100 ms). HRV time domain indices were compared from baseline and treatment Holter recordings.

RESULTS: In the Dob group, pts with moderately depressed HRV at baseline (n = 20) displayed a reduction in indices of total variability such as SDNN (82 ± 4 to 71 ± 4 ms, p = 0.01) and SDANN5 (69 ± 3 to 60 ± 4 ms, p = 0.01). Indices of parasympathetic modulation, pNN50 (7.2 ± 1.5 to 6.6 ± 1.7 %, p = 0.04) and RMSSD (29 ± 4 to 27 ± 4 ms, p = 0.05), also decreased. With severely depressed HRV (n = 38), no significant changes occurred with Dob in indices of overall HRV or parasympathetic modulation. However, in the nesiritide group, pts with severely depressed HRV (n = 75) displayed a significant increase in SDNN (35 ± 1 to 40 ± 2 ms, p = 0.002), SDANN5 (30 ± 1 to 34 ± 1 ms, p = 0.02), and RMSSD (13 ± 1 to 15 ± 3 ms, p = 0.01). No significant changes occurred in HRV indices with moderately depressed HRV at baseline (n = 53).

CONCLUSION: In pts with relatively preserved HRV, Dob reduces overall variability and parasympathetic modulation. With severely depressed HRV, the effect of Dob on HRV is minor, presumably due to β-adrenergic receptor down-regulation or saturation. In contrast, nesiritide improves indices of overall HRV and parasympathetic modulation in pts with severely depressed HRV and has no adverse effect on pts with relatively preserved HRV.

1116-119 **Altered Fractal Behavior and Heart Rate Variability in Daily Life in Neurally Mediated Syncope**

Bonpei Takase, Haruhiko Hosaka, Yoshihiro Matsushima, Takashi Akima, Syuuichi Katsushika, Toshihiko Nishioka, Fumitaka Ohsuzu, Akira Kurita, *National Defense Medical College, Tokorozawa, Japan, Self Defense Forces Central Hospital, Tokyo, Japan.*

Background: The fractal dimension is an index to the complexity of cardiovascular control system and autonomic function is important in the pathogenesis of neurally mediated syncope (NMS). However, it is still not fully understood if the derangement of the autonomic nervous system exists in NMS in daily life. **Methods:** Thus, we performed 24-hour ECG monitoring (AECG) and measured traditional heart rate variability indices (HRV) along with long fractal scaling exponent (b) in 36 pts (NMS, 24±3 y/o) with NMS diagnosed by head-up tilt testing (HUT, 80? tilt) and 11 age-matched normal controls (NL). AECG was performed within 48 hours before HUT procedure. For HRV, SDANN, SD, rMSSD, pNN50, low frequency (0.04-0.15Hz, LF) and high frequency (0.15-0.40 Hz, HF) were measured. All measurements were analyzed in three different periods such as total 24-hour, awake (09:00-15:00) and sleep (00:00-06:00) phases. The ratio of each measurements in awake over sleep phase (A/S ratio) was also calculated. **Results:** Several 24-hour HRV showed significantly higher values in NMS than those in NL (SD, 91±32 vs 65±11 ms; rMSSD, 60±31 vs 38±11 ms; pNN50, 30±17 vs 14±7%; LF, 35±15 vs 26±5 ms; HF, 27±11 vs 14±7 ms: $p<0.05$) while 24-hour b was not different (0.95±0.13 vs 0.95±0.14). In contrast, although A/S ratio of any HRV showed no differences, A/S ratio of b revealed significantly higher value in NMS than in NL (1.7±0.49 vs 1.2±0.15, $p<0.05$). The awake and sleep values of b in NMS were also significantly different from those in NL. **Conclusion;** Overall autonomic activity in daily life is exaggerated in NMS. Since in most of the time pts are upright during awake and are supine during sleep phase, the significant differences between awake and sleep values of b, and abnormal A/S ratio of b suggest that deteriorated fractal behavior exits in NMS. These findings may be associated with the mechanisms of orthostatic intolerance in NMS.

1116-120 **Right Atrial Size May Be a Primary Determinant of Atrial Conduction Time of the Sinus Node Impulse**

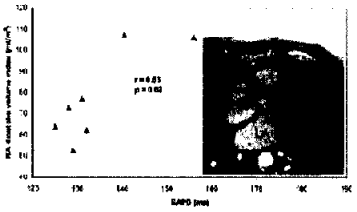
Susette K. Therkelsen, Bjoern A. Groenning, Ulrik Dixen, Gorm B. Jensen, *Department of Cardiology, Copenhagen University Hospital Hvidovre, Copenhagen, Denmark, Danish Research Center of Magnetic Resonance, Copenhagen University Hospital Hvidovre, Denmark.*

Background: Prolonged signal-averaged p-wave duration (SAPD) measured by a signal-averaged ECG is thought to represent a delay in the intra-atrial conduction of the electrical impulse. Prolonged SAPD is a prognostic marker of development of atrial fibrillation (AF). In addition, atrial dilatation in patients with previous AF has been associated with risk of relapse of AF. However, the relation between duration of atrial conduction of the electrical impulse and atrial dimensions is not clear. Our aim was to evaluate the correlation between atrial dimensions measured by magnetic resonance imaging (MRI) and the intra-atrial conduction time measured by SAPD.

Methods: In 7 subjects with no history of cardiac disease, we measured the total filtered SAPD. Atrial dimensions were evaluated by cinematographic breathhold MRI scans.

Results: Right atrial (RA) diastolic volume index (mean±SD=77±21ml/m^2; r=0.83, p=0.02), systolic volume index (49±17ml/m^2; r=0.76, p=0.05) and stroke volume index (29±6.7ml/m^2; r=0.77, p=0.04) were positively associated with SAPD (135±10.6ms). Left atrial (LA) diastolic (64±7.1ml/m^2) and systolic (39±7.3ml/m^2) volume indices and stroke volume index (24±1.0ml/m^2) were completely unrelated to SAPD. The combined RA and LA diastolic (141±21.6ml/m^2; r=0.80, p=0.03) and systolic (87.8±17.0ml/m^2; r=0.70, p=0.08) volume indices correlated positively with SAPD.

Conclusion: Our results suggest that RA size may be a primary determinant of atrial conduction time measured by SAPD.



ORAL CONTRIBUTIONS

826 External and Implantable Defibrillators: Clinical Studies

Monday, March 18, 2002, 2:00 p.m.-3:30 p.m.
Georgia World Congress Center, Room 254W

2:00 p.m.

826-1 **New Therapeutic Option for Patients With Time-Dependent Risk of Sudden Cardiac Arrest: Application of Novel Wearable Cardioverter-Defibrillator**

Arthur M. Feldman, Helmut Klein, Patrick Tchou, The WEARIT & BIROAD Investigators, *University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, Otto Von Guericke University, Magdeburg, Germany.*

Background: Outpatient populations with temporary risk for sudden cardiac arrest (SCA), such as patients awaiting cardiac transplant, post-myocardial infarction patients (p-MI) or post-CABG patients (p-CABG), often rely on emergency medical services (EMS) for defibrillation therapy. Success rates for EMS resuscitation are poor. A new therapeutic option, the completely automatic (no bystander intervention) wearable cardioverter-defibrillator (WD), may provide better protection for these populations.

Methods: A multicenter trial studied the use of WD (WCD® system, LIFECOR Inc., Pittsburgh). WD provides automatic detection and treatment of ventricular tachycardia and fibrillation (VT/VF) using unique non-stick ECG electrodes and self-gelling therapy electrodes. WEARIT sub-population: patients on the cardiac transplant list or equivalent in cardiac status. WEARIT subjects used WD until transplanted, hospitalized for transplant or circulatory assist device, or receiving an active ICD. BIROAD sub-population: patients not receiving an ICD who were p-MI or p-CABG and had either VT/VF within 48 hours, an ejection fraction < .30 or Killip class > II after 72 hours, or syncopal VT/VF after 48 hours. BIROAD used WD for ~4 months. The effectiveness objective was >25% resuscitation success with 90% confidence. The safety objective was <2.3% false shocks per patient-month with 90% confidence (500 patient-months minimum).

Results: There were 8 VT/VF SCA events in 6 patients (4 BIROAD, 2 WEARIT). 6 were successfully treated. 2 were unsuccessful with VT/VF detected but treatment prevented by incorrect patient assembly of WD (therapy electrode in pocket backward, gel released away from body). A redesign occurred to prevent this from reoccurring. 6 false shock episodes occurred in 873 patient-months. The most frequently reported adverse event (20) was temporary skin rash under electrodes. 285 patients were enrolled over 3 years. Daily