effect of CRT on LV end-diastolic diameter (LVEDD), LV end-systolic diameter (LVESD), and LV capture threshold. Random assignment to CRT or pacing off for six months allowed the comparison of end-points. Electrocardiographic determination of LVESD and LVEDD was done at baseline and at 6 months, as were LV stimulation threshold checks. RESULTS: CRT reduced LVEDD, LVESD, and threshold compared to control. There was a significant correlation between the change in LVEDD and LVESD (r=0.77, P<0.001).

Baseline  Baseline 6 Month 6 Month LV LVEDD LVESD LVESD threshold LVEDD threshold pacemaker off  6.46±0.97  4.22±0.90  2.02±1.43  0.51±1.16  7.52±1.05  2.27±1.17 pacemaker on  6.29±1.01  3.02±0.97  2.00±1.65  0.56±1.38  6.86±1.15  1.74±0.78

P  0.3  0.48  0.8  0.0001  0.0025  0.0064

CONCLUSION: Chronic CRT reduces ventricular dimensions and ventricular capture thresholds. We speculate that decreasing ventricular diameters reduce wall stress, a known contributor to increased ventricular stimulation threshold.

**1138-110** Biventricular Pacicing Improves Measures of Exercise in Patients With Atrial Fibrillation and Heart Failure

Chetan Varma, Sanjaya Sharma, Sam Finizio, William McKenna, Jean-Claude Daubert, St Georges Hospital Medical School, London, United Kingdom.

Background: Multisite pacing is used to treat advanced heart failure. Atrial fibrillation is common in these patients. Cardiopulmonary exercise testing provides a reliable assessment of changes in functional capacity. We assessed the effect of active biventricular (BiV) pacing compared to inactive BiV pacing upon exercise in patients enrolled in the AF arm of the multicentre MUSTIC (Multisite Stimulation in Cardiomyopathies) trial - a prospective, randomised, blinded crossover study.

Methods: Patients enrolled (NYHA class III on maximal medical therapy, paced QRS duration > 200 ms) and 6-minute walk test < 450 m were implanted and randomised to active (BiV) and inactive BiV (VVIR 70) pacing with 3 months of each. Detailed 15 second data of ventilation, inspired oxygen, expired carbon dioxide and heart rate were recorded (n=300) and exercise indices analysed if respiratory quotient > 1 (n=25).

Results: Peak oxygen consumption (V02) and duration of exercise (ET) as well as submaximal measures of exercise: anaerobic threshold (AT) and ventilatory efficiency (VE/VO2 slope) were compared between baseline, inactive and active phase.

Results:

<table>
<thead>
<tr>
<th>Baseline</th>
<th>Inactive BiV</th>
<th>Active BiV</th>
<th>% Change</th>
<th>p value (paired t)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak V02</td>
<td>3.3±1.3</td>
<td>3.3±1.2</td>
<td>0.05</td>
<td>0.09</td>
</tr>
<tr>
<td>ET seconds</td>
<td>314±132</td>
<td>334±140</td>
<td>3.3±148</td>
<td>0.001</td>
</tr>
<tr>
<td>VE / V02</td>
<td>40±11</td>
<td>43±10</td>
<td>37±9</td>
<td>0.03</td>
</tr>
<tr>
<td>AT</td>
<td>0.68±0.13</td>
<td>0.70±0.24</td>
<td>0.75±0.22</td>
<td>0.21</td>
</tr>
</tbody>
</table>

Conclusion: BiV pacing in patients with AF and heart failure can significantly improve measures of peak and submaximal exercise. Benefit in VE/VO2 suggesting normalization in some patients of the increased ventilatory drive seen with congestive cardiac failure. Antiventricular synchrony may not be the mechanism of benefit in this group.

**POSTER SESSION**

**1160 T Wave Alternans, Reentry, and Electroanatomy**

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.

**1160-106** Mechanism of Fibrillation Induction by Upper Limit of Vulnerability Shocks: An Optical Mapping Study in Isolated Swine Hearts

Nipon Chittaranjan, Isabell Barville, Raymond E. Ideker, Richard A. Gray, University of Alabama at Birmingham, Birmingham, Alabama.

Reentry is thought to be responsible for ventricular fibrillation (VF) induction by upper limit of vulnerability (ULV) shocks. However, recent studies using only near-ULV shocks found no reentry after successful VF induction (VF). Instead, rapid repetitive focal activations were always observed before degeneration into VF. It has been debated that post-shock reentry exists but was not detected due to technical limitations and limited spatial resolution in electrical mapping. To test this hypothesis, high spatial resolution optical mapping was performed in 5 isolated pig hearts. Methods and Results: In each heart, 10 near-ULV shocks (RV apical-RA, biphasic, 6/4 ms) were delivered after a train of 10 S1s. Activations on the anterior and posterior ventricular epicardium (~8000 sites) before and after each shock were mapped using 2 CCD cameras. Postshock activation pattern, intercycle interval (ICI), an interval between 2 successive cycles), and wavefront conduction by (WCT, the total time for each cycle to propagate across the entire heart) were determined for 5 cycles after each shock. Activations always arose from the apex and propagated focally during these cycles. No reentry was found. ICI was shorter and WCT was longer from cycles 3 to 5 in VF vs. No VF episodes. Conclusions: A focus is the pattern of VF initiation by near ULV shocks. Trigger waves of activation arising from the apex cause short ICI and long WCT, resulting in slow propagation and block of subsequent cycles eventually degenerating into VF.

**1160-107** Out of Phase T Wave Alternans in Left Ventricular Hypertrophy: Its Role in Ventricular Arrhythmogenesis

Gan-Xin Yan, Ramgop S. Lankelila, Ying Wu, Roger A. Mannox, Pater R Kowey, Main Line Health Heart Center, Wynnewood, Pennsylvania.

T wave alternans (TWA) is characterized by beat to beat changes in T wave morphology, amplitude or polarity on the ECG and serves as an important prognostic marker of ventricular arrhythmias. It is hypothesized that ventricular action potential duration (APD) may alternate "out of phase" with contraction force, i.e. APD and force increase and decrease in opposite phase, under conditions of electrical remodeling with a robust sarcolemmal sarcoplasmic reticulum (SR) function. This was tested using an arterially perfused left ventricular wedge isolated from rabbits with left ventricular hypertrophy (LVH). Transmembrane action potentials from epicardial (Epi), endocardial (Endo) and subendocardial (Subendo) were simultaneously recorded with isometric contraction and a transmural ECG. LVH (renovascular hypertensive model) resulted in "out of phase" TWA in 26% of the preparations (6/23), under slow pacing rates (BCLs: 1000-2000 ms), TWA was largely determined by APD alteration in Endo and Subendo, thus leading to significant beat-to-beat changes in QT interval and transmural dispersion of repolarization (TDR), di-Souza (0.01-0.03 mm) and 4-aminohippuride (2 mm) exaggerated "out of phase" TWA and increased its incidence (13/23), whereas acceleration of pacing rate (BCLs: 500-1000 ms) attenuated it. Phase 2 early afterdepolarization (EAD) could be generated from Endo or Subendo during a beat with longer APD during TWA, leading to an "R on T" extrasystole and polymorphic ventricular tachycardia (VT) in the absence of APD prolonging agents. Verapamil and ryanoid at 1 mm reduced contraction markedly and abolished TWA, EAD and extrasystoles. Interestingly, charybdotoxin (10 µM), a specific inhibitor of Ca2+ activated K+ current, inhibited TWA without significant influence on contraction, indicating a strong feedback of [Ca2+]i to membrane currents involved in "out of phase" TWA. In conclusion, Bradycardia-dependent TWA in LVH, probably secondary to alteration in intracellular Ca2+ handling and Ca2+-dependent iconic currents, is associated with marked beat-to-beat changes in QT interval and TDR that predispose to the genesis of EADS and R on T extrasystoles of initiating polymorphic VT.