

effect of pacing site (S1) on the ULV for a single shocking electrode configuration (S2). In 10 open-chest pigs with an elastic epicardial mapping sock, we determined the ULV for: LV-apex (LVA), RV outflow tract (RVOT), and posterior LV-base (LVB) S1 sites for S2 shocks using an SVC catheter to small LVA-patch electrode configuration. For each S1 site, shocks (6/6 ms biphasic exponential) were delivered after 20 paced beats (S1S1 at 80% intrinsic RR interval) to scan the T-wave (measured through the S2 electrodes) in 10 ms steps beginning at the peak (Tpk). S2 shocks were delivered starting at 60V leading-edge voltage (LEV) and incremented in 40V steps until VF could not be induced at any point in the T-wave.

**Results:** There was no significant difference in LEV ( $p = 0.48$ ), leading-edge current (LEI) ( $p = 0.83$ ), or total energy (TE) ( $p = 0.91$ ) at the ULV for the three S1 sites. However, the timing of the S2 shock relative to Tpk (S2-Tpk) at which VF was induced for one step below the ULV varied with S1 site. For both LVA and LVB, VF was induced later than Tpk while VF was induced earlier than Tpk for RVOT. S2-Tpk for LVA and LVB was significantly ( $p < 0.001$ ) different from RVOT. There was no significant difference for LVA vs LVB ( $p = 0.10$ ).

	LEV	LEI	TE	S2-Tpk
LVA	336 ± 63V	2.8 ± 0.72A	6.0 ± 2.5J	32.4 ± 17 ms
LVB	344 ± 69V	2.8 ± 0.67A	6.1 ± 2.7J	20.6 ± 21 ms
RVOT	356 ± 74V	2.7 ± 0.60A	6.2 ± 2.5J	-19.1 ± 19 ms

**Conclusion:** For a single shocking electrode configuration, the ULV obtained by scanning the T-wave does not vary with ventricular pacing site. However, the position in the T-wave at which VF is induced just below the ULV is dependent upon pacing site. These results support the critical point hypothesis for the ULV. Additionally, they show that there is no one fixed point for shock delivery in the T-wave for determining the absolute ULV for all S1 sites.

**945 Methods in Diagnostic Exercise Testing**

Tuesday, March 21, 1995, 9:00 a.m.–11:00 a.m.

Ernest N. Morial Convention Center, Hall E

Presentation Hour: 9:00 a.m.–10:00 a.m.

**945-61 A New Approach to Management of Low Risk Patients Presenting to the Emergency Room with Chest Pain: The Immediate Exercise Treadmill Test**

J. Douglas Kirk, Samuel Turnipseed, William R. Lewis, Ezra A. Amsterdam.  
*University of California, Davis*

To enhance clinical and cost-effective management of low risk patients (pts) with chest pain who traditionally require admission to a monitoring unit to rule out a coronary event, we have applied immediate exercise treadmill testing (IETT) directly in the emergency department (ED) in this subset. Based on our initial pilot experience with IETT suggesting its safety and utility, we have extended this approach to a large pt population:  $n = 192$ , 103 men (M), mean age 46 yr. (28–79) and 89 women (W), mean age 49 yr. (31–89). Low risk pts were defined by: chest pain, a normal or nondiagnostic electrocardiogram, no history of coronary artery disease (CAD) and a negative screening evaluation for acute pulmonary and noncardiac vascular processes. Most recently we have included selected pts with known CAD. IETT entailed symptom-limited testing (prior to report of cardiac isoenzymes). Positive (Pos) IETT:  $\geq 1.0$  mm ST-segment  $\downarrow$  at 80 msec; Nondiagnostic (NonDx) IETT: negative (Neg) test at  $<85\%$  age-predicted maximum heart rate. **Results:**

IETT	Men	P	Women
Negative	70% (69/103)	n.s.	53% (47/89)
Nondiagnostic	22% (23/103)	n.s.	31% (28/89)
Positive	10% (11/103)	n.s.	16% (14/89)

There were no adverse effects of IETT. Based on further studies in 18 Pos pts (coronary angiography — 10, stress scintigraphy — 4, stress echocardiography — 4), predictive accuracy of Pos IETT was 67% (12/18; M 67% [6/9], W 67% [6/9]). Preliminary data revealed no adverse clinical events in Neg pts 1 mon. after IETT and selected NonDx pts received further evaluation. IETT is safe in selected pts with chest pain who have traditionally required admission to rule out a coronary event. Pos rate is low but true Pos are not rare and are not readily identifiable by traditional clinical criteria. These results do not differ significantly in M and W. The high negative rate of IETT indicates a potential for major savings if this approach were utilized in the criteria for admission of this pt population.

**945-62 Impaired Heart Rate Response to Graded Exercise: Prognostic Implications of Chronotropic Incompetence**

Michael S. Lauer, Peter M. Okin, Martin G. Larson, Jane C. Evans, Keaven M. Anderson, Daniel Levy. *Cleveland Clinic Foundation, Cleveland, Ohio; Framingham Heart Study, Framingham, Massachusetts*

**Background and Methods:** To determine the associations between exercise heart rate response, mortality and coronary heart disease (CHD) risk, 1617 male participants in the Framingham Offspring Study underwent treadmill exercise testing (Bruce Protocol) and were followed for 7.7 years. Heart rate response was assessed in three ways: 1) failure to achieve age-predicted target heart rate, 2) absolute increase in heart rate with exercise, and 3) percent increase in heart rate to stage 2 of exercise. The two main endpoints were all-cause mortality and incident CHD in subjects without CHD at baseline. **Results:** During follow-up there were 60 deaths and 95 incident CHD events. 1) Failure to achieve target heart rate was associated with increased mortality (Hazards ratio [HR] = 2.63, 95% CI = 1.57–4.39,  $P = 0.0002$ ) and increased CHD risk (HR = 3.49, 95% CI = 2.02–4.82,  $P = 0.0001$ ). 2) Absolute increase in heart rate with exercise was independently and inversely related to mortality risk (HR for 12 beats per minute decrease 1.22, 95% CI 1.03–1.46,  $P = 0.02$ ) and CHD risk (HR = 1.29, 95% CI 1.12–1.49,  $P = 0.0004$ ) even after adjusting for age, ST segment response, baseline heart disease (for mortality), and traditional coronary risk factors. 3) Percent increase in heart rate to stage 2 of exercise was also independently and inversely predictive of mortality (HR for 25% decrease 1.30, 95% CI 1.00–1.70,  $P = 0.05$ ) and incident CHD risk (HR = 1.31, 95% CI = 1.06–1.63,  $P = 0.013$ ) even after adjusting for age, exercise capacity, and (for CHD risk) traditional coronary risk factors. **Conclusion:** An attenuated heart rate response to exercise, a manifestation of chronotropic incompetence, is predictive of increased mortality and coronary heart disease incidence.

**945-63 Cigarette Smoking, Exercise Capacity, and Chronotropic Incompetence in Men with and without Coronary Artery Disease**

Michael S. Lauer, Fredric Pashkow, Claire E. Snader, Sharon A. Harvey, Thomas H. Marwick, James D. Thomas. *Cleveland Clinic Foundation, Cleveland, Ohio*

**Background and Methods:** To determine the associations between cigarette smoking, exercise capacity, and exercise heart rate response, data on 2323 men who had not undergone prior invasive cardiac procedures and who were referred for symptom-limited treadmill thallium studies were analyzed. Structured interviews yielded data on smoking habits, prior cardiac events, standard coronary risk factors, medication use, and other chronic diseases including lung disease. Aerobic impairment was defined as inability to achieve 85% of the age-predicted exercise capacity in metabolic equivalents (METs). Chronotropic incompetence was defined as failure to achieve 85% of the age-predicted maximum heart rate. **Results:** There were 385 regular smokers, 909 non-smokers, and 1029 ex-smokers, of whom 653 had quit more than 9 years before ("remote ex-smokers"). Aerobic impairment was present in 24% of non-smokers, 38% of smokers (Odds ratio [OR] = 1.98, 95% CI 1.53–2.56), and 27% of remote ex-smokers (OR = 1.18, 95% CI 0.94–1.48). These associations remained unchanged even after adjusting for age, thallium abnormalities, and clinical characteristics. Compared to non-smokers, smokers had an age-adjusted exercise capacity of one MET less ( $P < 0.0001$ ), irrespective of thallium abnormalities. Chronotropic incompetence was present in 10% of non-smokers, 21% of smokers (OR = 2.52, 95% CI 1.81–3.50), and 14% of remote ex-smokers (OR = 1.53, 95% CI 1.12–2.09). These associations remained largely unchanged even after adjusting for age, thallium abnormalities, exercise capacity in METs, and clinical characteristics. Furthermore, age-adjusted heart rates were lower at all levels of sub-maximal exercise in smokers compared to non-smokers ( $P < 0.02$  by ANOVA). In subset analyses of subjects who underwent coronary angiography similar independent associations of smoking with impaired aerobic capacity and chronotropic incompetence were noted, irrespective of the severity of coronary disease. **Conclusions:** Cigarette smoking is an independent predictor of impaired aerobic capacity and chronotropic incompetence. Smoking cessation may be associated with improvement of these exercise characteristics.

**945-64 Repeated Standardized Exercise Testing in Cardiopulmonary Patients Using Implanted Hemodynamic Monitors**

Ake Ohlsson, Rolf Nordlander, Lars Ryden, Hans Åström, Barbro Kjellström, Brian Lee, Mark Claxton, Frans Ottenhoff, Tom Bennett. *Karolinska Hospital, Stockholm, Sweden; Pacing New Indications Department, Medtronic, Inc., Minneapolis, MN*

**Introduction.** Exercise testing is often a valuable diagnostic procedure in patients with chronic cardiopulmonary diseases. Easily repeated measurement

TUESDAY AM