

**A NEW COMPREHENSIVE VENTRICULAR TACHYARRHYTHMIA CONTROL DEVICE: EFFICACY OF HIERARCHICAL THERAPY.**

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An investigational pacemaker (P), cardioverter (C), defibrillator (D), the Medtronic PCD device was implanted in 12 patients (pts) to terminate recurrent sustained ventricular tachyarrhythmias (VTA). The follow-up period was 1-16 mths (total 100 pt-mths). Three pts received a nonthoracotomy lead system. Acute defibrillation threshold at implant was 5-18 J. Seven pts had therapies for spontaneous VTA: 6 VTA were treated as ventricular tachycardia (VT), 9 VTA as ventricular fibrillation (VF). 7 problematic therapies were delivered for atrial fibrillation. Antitachycardia pacing (ATP) was the initial therapy in 4 pts, C in 3 pts. ATP failed in 4/27 episodes (15%) and C in 5/22 (22%) using an energy of 1-6 J. When ATP failed, automatic backup C was successful using an energy of 3-16 J (problematic episodes excluded). When initial C failed, backup C with higher energy (3-15 J) was successful. Overall 6 of 7 pts needed staged therapy, 4 of the ATP pts and 2 of the C pts. Initial VF therapies (18-24 J) were successful in all cases. All pts remain alive and no hospital admissions were necessary for VTA or device related problems. In conclusion: backup C effectively terminates spontaneous VT not responding to ATP. ATP combined with hierarchical C offers safe VT therapy. By providing increasingly aggressive therapy automatically the PCD device represents an important improvement in the therapy of VTA.

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Poster Displayed: 2:00PM-5:00PM

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Hall F, West Concourse

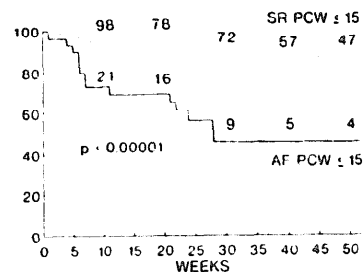
**Pathophysiology and Epidemiology of Heart Failure****ADVERSE PROGNOSIS OF ATRIAL FIBRILLATION IN ADVANCED HEART FAILURE: A STUDY OF 390 PATIENTS**

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In advanced heart failure, the impact of atrial fibrillation (AF) on survival is controversial. The prognostic significance of AF was examined in 390 consecutive heart failure pts hospitalized for heart transplant evaluation, and discharged on medical therapy. AF pts (n=75) compared with sinus rhythm (NSR) pts (n=315) had similar LV ejection fraction (0.21 vs 0.19, p=0.04), and pulmonary capillary wedge (PCW) pressure on therapy (16 vs 15 mmHg, p=NS). For AF vs NSR pts, 1 yr actuarial survival without transplantation was 52% vs 71%

(p=0.001), and sudden death free survival was 69% vs 82% (p=0.001). By Cox analysis, higher PCW on therapy, lower LV ejection fraction, and AF were independently associated with total mortality and sudden death. When stratified for PCW  $\leq$  or  $>$  15 mmHg, AF vs NSR was associated with a worse 1 yr survival (44% vs 83%, p < 0.0001), and sudden death free survival (60% vs 92%, p=0.0001) in the low, but not the high, PCW group. **Conclusion:** In pts who are well compensated despite severe LV dysfunction, AF is an important predictor of mortality and sudden death. Variable prognostic significance in previous studies may be due to the interdependence with hemodynamic status.

**TRENDS IN CARDIAC FAILURE - INCIDENCE AND CAUSES OVER THREE DECADES IN THE FRAMINGHAM STUDY**

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Framingham data comparing congestive heart failure (CHF) incidence for the decades of the 1950's, 60's and 70's in subjects aged 49 - 62 years, indicate no major decline in incidence despite a massive increase in anti-hypertensive therapy and a fall in cohort average blood pressure. Examination of the prevalence of CHF etiologies comparing the 3 decades was undertaken seeking an explanation. A major improvement in the cardiovascular risk profile over the 3 decades should have resulted in less CHF. There was a 40% decrease in smoking and a 30% reduction in hypertension.

Coronary Heart Disease as a cause of failure increased from 44% to 50% in men, but in women did not change significantly (27% vs. 26%). Rheumatic heart disease declined from 15% to 3% in men and from 21% to 15% in women. Only diabetes has risen substantially as a CHF etiology: from 6% to 31% in men and from 16% to 26% in women. Thus we have no good explanation for the failure of CHF incidence to decline despite improvements in hypertension and CHD occurrence. It is apparently not due to saving more CHD victims from early death or to a major increase in cardiomyopathies other than possibly diabetic cardiomyopathy.

**IMPORTANCE OF ABNORMAL LUNG PERFUSION IN EXCESSIVE EXERCISE VENTILATION IN CHRONIC HEART FAILURE**

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Whether excessive ventilatory response to exercise is related to distribution of lung perfusion was examined in 23 pts with heart failure and 13 age-matched normal subjects. Using technetium 99m-macroaggregated albumin, we assessed the resting distribution of pulmonary blood flow by scintigraphic counts ratio of upper to lower lung fields. The ventilatory response to exercise was assessed by the slope of the relation between minute ventilation and carbon dioxide production ( $\dot{V}_E$ - $\dot{V}CO_2$ ). We also measured maximal reduction in physiological dead space/tidal volume ratio ( $V_d/V_t$ ) during exercise. 8 pts (Group A) had  $\dot{V}_E$ - $\dot{V}CO_2 \leq 33$ , which is the upper limit of normal range and 15 had  $\dot{V}_E$ - $\dot{V}CO_2 > 33$  (Group B).

	Peak $\dot{V}O_2$ (ml/kg/min)	$V_d/V_t$ change (%)	U/L (%)
Normal	24 $\pm$ 6	-21 $\pm$ 6	35 $\pm$ 7
Group A	18 $\pm$ 4 *	-23 $\pm$ 7	38 $\pm$ 7
Group B	16 $\pm$ 4 *	-6 $\pm$ 12†	60 $\pm$ 20†

(\* p < 0.01 vs. Normal, † p < 0.01 vs. Normal and Group A) Peak oxygen uptake (Peak  $\dot{V}O_2$ ) was comparably reduced in Group A and in Group B.  $V_d/V_t$  in Group B failed to decrease normally during exercise, indicating an increase in physiological dead space. The lung perfusion in Group B was characterized by a relative increase in the blood flow to the upper lung, while the distribution in Group A was similar to that in normal subjects. **Conclusion:** Maldistribution of pulmonary blood flow is closely related to excessive ventilation during exercise, suggesting the importance of pulmonary vascular abnormalities in ventilatory response in chronic heart failure.