group ($p < 0.05$) but there was no difference in heart rate, deceleration time, E wave height or LA size.

	Peak A (cm/sec)	Peak E	VTIA	E decel (msec)	LA size	HR
Dof500	40.3	94.0	3.9	215	4.6	68
Placebo	32.3	90.4	2.9	205	4.6	72

Comparison among patients randomized to dofetilide 125 bid, or 250 bid showed a dose-response effect of A wave height, maximum at 250–500 mg bid.

Conclusion: Immediately following cardioversion dofetilide increases A wave height. The absence of corresponding changes in heart rate and deceleration time indicate that this is a true atrial positive inotropic effect and not due to an effect on ventricular hemodynamics.

1174-165 Prevalence and Determinants of Non-rheumatic Atrial Fibrillation (AF) in Patients With Primary Aldosteronism

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Background: Primary aldosteronism (PA) associates two recognized risk factors for AF: hypertension and hypokalemia. The prevalence of AF during PA has not been determined yet.

Methods: The prevalence of AF has been calculated in a serie of 124 consecutive patients with PA identified between 1994 and 1996, and in 475 essential hypertensive patients (HT) matched for age (mean age 52 ± 10), sex and blood pressure (BP) (mean BP 130 ± 15 mmHg). Results are summarized in the following table.

	HT	PA
Kalemia (mmol/l)	4.4 ± 0.3	3.4 ± 0.3*
ECG-LVH (Sokolow, mm)	25 ± 8	29 ± 10†
History of myocardial infarction (%)	1.5	4.0§
Atrial fibrillation (%)	0.6	7.3*

*$p < 0.001$. †$p < 0.005$. §$p < 0.01$

Univariate analysis of the population with PA indicated that age ($p < 0.005$), duration of hypokalemia ($p < 0.01$), and cardiac hypertrophy ($p < 0.01$) were the only parameters associated with the presence of AF. In multivariate analysis, only duration of hypokalemia and cardiac hypertrophy were still significantly correlated.

Conclusion: These results indicated that the prevalence of AF is significantly higher in patients with PA. This report suggests that a renin-aldosteron work up should be performed in every hypertensive patient who present an unexplained AF.

1175 Pacemaker Therapy: Programming

Wednesday, April 1, 1998, 9:00 a.m.–11:00 a.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 10:00 a.m.–11:00 a.m.

1175-167 Incidence of Paroxysmal Atrial Fibrillation in Dual Chamber Pacing: Relevance of Lower Rate Programming

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Background: It is known that pacing can reduce the incidence of paroxysmal vagal-induced atrial fibrillation (AF). It has not yet been investigated if different lower rate programming can influence paroxysmal AF in a pacemaker (PM) patient group with known intermittent AF.

Methods: Twenty patients (5 women, 15 men; mean age 64 ± 9 y) with dual chamber pacing were prospectively randomised to a lower rate of 50 or 75 beats/min. After six weeks the PM patients were programmed to the alternate rate. Every 2 weeks the incidence of mode switch (MS) episodes/24 h were documented using the PM mode switch counter. In addition, the incidence of atrial stimulation (AP) or sensing (AS) and ventricular pacing (VP) or sensing (VS) were registered. The preexisting antiarrhythmic drug regime had to be stable during the cross-over period.

Results: At a lower rate of 50/min the incidence of AP was reduced to 27% as compared to 62% at 75/min. The mean incidence of mode switch episodes/24 h was 7.2 ± 4.7 at 50 beats/min vs 5.6 ± 3.9 at 75/min, indicating a tendency to less MS episodes with a lower rate of 75/min, however statistical significance could not be reached. The MS episodes were not significantly correlated to the amount of AP ($p = 0.48$; $r = 0.11$) or AS ($p = 0.47$; $r = -0.116$). Even including the ventricular events no significant correlation could be documented: AS/VS: $p = 0.13$, $r = 0.24$; AS/VP: $p = 0.09$, $r = -0.26$, AP/VS: $p = 0.55$, $r = -0.1$; AP/VP: $p = 0.48$, $r = 0.12$.

Conclusion: Lower rate programming of 75 beats/min did significanly increase the amount of atrial paced events, as compared to 50 beats/min. However, in a PM patient cohort with known intermittent AF, randomised cross-over programming of these two different lower rates did not significantly influence the incidence of mode switch episodes.

1175-168 Does Stimulation Mode Influence Atrial Function? Answer From a Randomized Prospective DDD vs. VVI Study

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Retrospective data reported an increased mortality and morbidity in patients with sick sinus syndrome and VVI pacemaker since 1986. Ever since the discussions about the right stimulation mode in sick sinus syndrome did not stop. No prospective data comparing the physiologic DDD to VVI stimulation mode have not been available.

The aim of our randomized prospective cross-over trial was to determine the risk for atrial fibrillation (AF) in both stimulation modes in patients with pre-existing paroxysmal AF before pacemaker implantation in an intra and inter individual comparison.

Fifty four patients were randomized to either VVI or DDD stimulation mode for a one years follow-up with a consecutive cross-over. At 1, 3, 6, 9, 12, 15, 18, 21 and 24 months cardiac rhythm was controlled by a holter-ECG, the data of the inherent holter function and a questionnaire. Echocardiography was performed to evaluate ejection fraction and atrial diameters.

Results: Episodes of paroxysmal AF were highly significant more often on VVI than on DDD stimulation ($n = 345$ vs. 70, $p < 0.001$) as well as the number of patients that showed paroxysmal AF ($n = 47$ vs. 23, $p < 0.001$). Recurrence of AF was more often in patients with sick sinus syndrome than in AV-block ($p < 0.01$) and did very well correlate to atrial size. Mean atrial diameter on DDD was 39 ± 3 vs. 45 ± 5 mm on VVI stimulation with an increase in diameter in 38 patients.

Conclusion: Patients with paroxysmal AF exhibited significantly more episodes of AF on VVI than on DDD stimulation increasing their mean atrial diameter significantly. Physiologic DDD is superior to VVI mode in patients with pre-existing paroxysmal AF.

1175-169 DDD Pacing With Optimal AV Delay Versus AAI Pacing in Patients With AV Block I Degree

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Background: AAI pacing has been reported to be hemodynamically superior to DDD pacing in patients (pts) with normal native AV conduction. In case of AV block I-st degree (AVB I) however the preferable pacing mode is not clearly established. The purpose of this study is to evaluate the relative hemodynamic benefit of AAI and DDD with optimal AV delay pacing modes in pts with AVB I.

Method: 9 pts, (6 men, aged 68) with AVB I and dual chamber pacemakers were studied. Using Pulse Doppler Echocardiography the Aortic Flow Velocity Integral (AFVI) was measured. After determining of the optimal AV delay in DDD pacing mode for each patient, comparison between AAI mode and DDD mode with optimal AV delay was performed by measuring of AFVI while pacing at rates of 70 bpm and 90 bpm.

Results: The native AV conduction (AR interval during AAI) ranged 240–460, (311 ± 70) ms for rate 70bpm and 280–520, (378 ± 110) ms for rate 90 bpm. The optimal AV delay ranged 120–200, (164 ± 24) ms. At rate 70 bpm in all pts with AR $\leq$270 ms AFVI was higher in AAI than in DDD mode (0.164 ± 0.06 m vs. 0.142 ± 0.05 m, $p < 0.05$, $n = 4$), while all the pts with AR $\geq$315 ms had higher AFVI in DDD than in AAI mode (0.174 ± 0.03 m vs. 0.146 ± 0.03 m, $p < 0.05$, $n = 5$). At rate 90 bpm all of the pts had AR $\geq$280 ms and showed higher AFVI in DDD than in AAI mode (0.163 ± 0.03 m vs. 0.132 ± 0.03 m, $p < 0.05$, $n = 8$).

Conclusions: The results suggest that in pts with AVB I the relative benefit of AAI and DDD pacing modes depends on the of native AV conduction and on the pacing rate.