EXERCISE CAPACITY AND NEUROENDOCRINE RESPONSE IN FOUR PACING MODES - WITHIN PATIENT COMPARISON

Christopher Travill MRCP, Meurig Williams MRCP, Rafique Ahmed MD, Ann Ingram, Stafford Lightman FRCP, Richard Sutton DScMed FACC. Westminster Hospital, London, UK.

5 patients with complete atrioventricular block received a dual chamber pacemaker with dynamic A-V delay (Paragon, Pacesetter). 4 exercise tests were performed in modes VVI, VVIR, DDD and DDD+AV in which the A-V delay shortens at faster rates. Blood for plasma atrial natriuretic peptide (ANP), plasma renin activity (PRA) and arginine vasopressin (AVP) was drawn at rest and after a period of exercise equivalent to the peak achievement in VVI ("peak VVI"). The initial mode was VVI to establish the exercise time frame, the other modes were randomised and double-blind. VVIR was imposed by use of an external Activitrax.

Mode	Duration (min)	Rest ANP (pmol/l)	Peak ANP (pmol/l)
VVI	11.0±1.0*	102±33	179±39**
VVIR	12.2±1.0	80±18	115±16***
DDD	12.5±1.0	29±8	71±18
DDD+AV	13.0±1.0	42±14	66±20

* p<0.05 vs VVIR, DDD and DDD+AV
** p<0.05 vs DDD and DDD+AV
*** p<0.05 vs DDD and DDD+AV

No significant change in PRA or AVP.

Conclusions: VVIR, DDD and DDD+AV resulted in significantly longer exercise duration than VVI. Lower levels of ANP at equivalent amount of exercise (= peak VVI) is consistent with improved haemodynamics in VVIR, DDD and DDD+AV, when compared with VVI. Levels of ANP at "peak VVI" are lower in both DDD and DDD+AV compared with VVIR but there is no difference between DDD and DDD+AV.

A NEW LOW THRESHOLD PLATINIZED EPICARDIAL PACING ELECTRODE: INITIAL EXPERIENCE IN CHILDREN.

Peter P Karpawich, M.D., Mehdi Hakimi, M.D., F.A.C.C., Eduardo Arciniegas, M.D., F. C.C., Dianne L. Cavitt, C.C.V.T., Kenneth B. Stokes. Children's Hospital of Michigan, Wayne State University, Detroit, MI.

Epicardial (epi) pacing is limited by electrode (E)-tissue interface inflammation (I) and fibrosis (F) causing elevated threshold (thld) and exit block. Previous animal studies have shown the benefits of a new platinized (Pl)-platinum (P) E design with a fine textured, high microscopic surface area, in reducing I and F. We present the first clinical experience with this new Pl-P epi E design (Medtronic model 4951P) in 5 children (ages 1-9y) followed up to 1 year post-implant. Efficacy of the new E was evaluated by weekly thld analysis acutely and by chronic thld comparisons with other epi E implanted in 35 children: Medtronic models 6917A-35T (n=24), 4951 (n=3) and Telectronic model 030-170 (n=8).

Acute mean thld (msec) ± standard error (sem) at 5V of the Pl-P E showed negligible changes from implant:

	implant	1wk	2wk	3wk	4wk	5wk	6wk
msec	.05	.05	.07	.08	.08	0.1	.09
sem	0	.02	.03	.02	.02	.02	.05

Comparative chronic mean thld/± sem showed significantly lower values for the new Pl-P than other E:

E model	1mo	3mo	6mo	12mo
4951P	.08/.02	.07/.04	.10/.006	.10/.001
6917A-35T	.10/.05	.18/.17*	.27/.21*	.20/.19*
4951	.15/.07	.15/.07	.45/.21*	---
030-170	.28/.20*	.18/.09*	.18/.09*	.14/.05*

(*p < .05 compared to model 4951P E)

Conclusion: The Pl-P E design limits thld changes and improves epi pacing capabilities in children.

DDD MODE SURVIVAL IN PATIENTS WITH DUAL CHAMBER PACEMAKERS.

Jay Gross, M.D., Stuart Moser, M.D., Z. Michael Benedek, M.S.E.E., Carolyn Andrews, RN-C, Seymour Furman, M.D., F.A.C.C., Montefiore Medical Center, Bronx, NY

489 consecutive initial transvenous implants between 12/83 & 12/88 with a mean follow-up of 33 mos were assessed for DDD mode survival. Patients (pts) were 54.7% male, mean age 71.9 years, 39% had dominant SA and 61% dominant AV nodal disease. 19 pts (4%) required secondary intervention, including 11 for lead dislodgment, 6 for lead or pacer malfunction and 2 for infection. During follow-up, 87 (18%) were permanently programmed out of their initial dual chamber settings and 10 others (2%) required temporary reprogramming out of DDD. Reasons included atrial fibrillation (AF) - 48 pts (10%), (5 returned to sinus rhythm permanently, 6 temporarily); loss of atrial sensing - 26 pts (5%) (4 had limited atrial sensitivity pulse generators, 3 had atrial leads which deteriorated in situ), recurrent "endless loop" tachycardia (ELT) 5 pts (1%), diaphragmatic stimulation - 5 pts (1%), loss of atrial capture - 5 pts (1%), lead dislodgment without repositioning - 4 pts (1%), pulse generator malfunction - 1 (1%), and 5 others (1%). Two of the 5 pts with ELT had units with limited programmability, 2 were suspected to be tracking supraventricular rhythms, and 1 was managed effectively with reprogramming to VAI. A DDD survival table is shown below:

Cumulative DDD Survival Rate

6 mos	12 mos	18 mos	24 mos	36 mos	48 mos	60 mos
92.6%	91.2%	90.4%	89.5%	85.9%	81.4%	80.6%

Conclusions: 1) 4% of DDD implants required a secondary surgical intervention. 2) 81% remained in the DDD mode; 3) Onset of AF or loss of atrial sensing accounted for 73% of the mode changes. 4) 23% of patients with AF could be at least transiently programmed to DDD during subsequent follow-up. 5) 10% of the permanent mode changes were directly related to earlier pacemaker technology (limited sensing atrial refractory programmability or defective leads) and are preventable with current pacing systems.

THE IMPACT OF STEROID ELUTING LEADS ON LONG TERM PACING IN THE ATRIUM AND VENTRICLE

Richard Sutton DScMed FACC, Sema Guneri MD. Westminster Hospital, London, UK.

129 right ventricular (Medtronic (MD) 4003) and 115 right atrial (MD 4503) steroid eluting leads have been implanted 1983-9. Complications: 1 displacement of MD 4503 (0.9%), nil else. 73 MD 4003 and 51 MD 4503 were selected for comparison with 73 MD 4011 and 41 MD 4511 non-steroid eluting leads, on the basis of programmability of the implanted generators (Pacesetter 233/285, Biotronik 04/05, MD 7005) offering data on P/R amplitude and stimulation threshold. Implant data was measured on MD 5309/5311. No significant difference in any parameters was detected between steroid and non-steroid leads at implant. Both short (one week) and long term (6-10 months) atrial thresholds were improved 1.10±0.55 vs 1.65±0.94 V (p<0.01) and 0.92±0.04 vs 1.07±0.28 V (p<0.001) respectively (data given for Pacesetter units). Medium term (one month) ventricular thresholds were also improved 0.91±0.07 vs 1.19±0.47 V (p<0.01). Short and medium term P wave amplitude was improved 2.36±1.63 vs 1.71±0.86 mV (p<0.05) and 3.16±2.16 vs 1.97±0.97 mV (p<0.05) respectively and long term R wave amplitude was improved 10.0±1.91 vs 8.61±2.85 mV (p<0.01). In 92 atrial leads existence of sinus node disease did not influence P wave amplitude. In conclusion steroid eluting leads offer possibility of safe long term pacing at 2.5 V output or even lower and better sensing characteristics in both chambers