One week later, rapid atrial pacing (400 bpm) was initiated and maintained until AF was sustained. 14 to 20 days later, the open chest ablation procedure was performed. Lesions were created as follows: #1 Bachmann's bundle, #2 isolation of the right and, #3 left atrial appendage, #4 superior to inferior vena cava, #5 isolation of the pulmonary veins, and #6 endocardial drag lesion in the coronary sinus.

Results: Ten dogs were in AF for 17 ± 14 days before ablation (table).

Conclusions: Using this model, which more closely resembles clinical AF than previously reported models, AF was successfully ablated in 70% of the experiments.

5:15

864-6 Changes in Atrial Refractoriness and Action Potential Duration After Sudden Loss of AV Synchrony

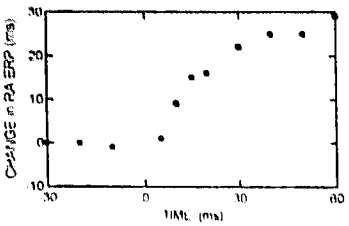
E.M. Kettunen, H.J. Parikka, L.K. Toivonen. *Helsinki University Central Hospital, Helsinki, Finland*

To understand the contribution of atrioventricular (AV) asynchrony to cardiac arrhythmias, atrial and ventricular electrophysiology was studied during normal and reversed AV interval. New automatic stimulation technique made it possible to monitor rapid changes in the effective refractory period (ERP).

Methods: Right atrial (RA) ERP using 2–4 ms stimulus interval increments, right atrial and ventricular (RV) monophasic action potential duration (APD) at 90% repolarization, peak RA pressure (rap) and mean arterial pressure (map) were determined simultaneously when the AV interval was suddenly changed from +80 to −40 ms in 5 anaesthetized pigs, 2–3 times in each.

Results: As the AV interval was made negative rap increased from 8 ± 3 to 14 ± 4 mmHg ($p < 0.001$) abruptly and map decreased from 86 ± 18 to 65 ± 21 mmHg ($p < 0.001$) more gradually. Parameters before and after 2 min of stabilization are shown in Table as mean ± SD of N determinations:

	N	AV +80 ms	AV −40 ms	Change	p-value
RA ERP (ms)	13	153 ± 24	175 ± 20	22 ± 15	< 0.001
RA APD (ms)	10	122 ± 24	163 ± 25	42 ± 12	< 0.001
RV APD (ms)	11	299 ± 9	296 ± 13	−3 ± 8	NS



Half of the total lengthening in RA APD occurred within 5 s. Respective change in RA ERP was noted within 15 s, the time course of which is shown in Figure. The ERP/APD relationship was modified by a lesser lengthening in RA ERP than in RA APD (Table, $p < 0.001$), facilitating reentrant mechanisms.

Conclusions: Loss of AV synchrony even for short periods markedly affects electrophysiological properties of the atria, which may contribute to the high incidence of atrial arrhythmias in sick sinus syndrome and in non-physiologic ventricular pacing modes.

865 Intracoronary Radiation and Other New Approaches to Revascularization

Tuesday, March 31, 1998, 4:00 p.m.–5:30 p.m.
Georgia World Congress Center, Lecture Hall 3

4:00

865-1 Intracoronary Radiation Therapy for Patients With In-stent Restenosis. Interim Report From a Randomized Clinical Study

R. Waksman, R.L. White, R.C. Chan, L.M. Gierlach, B.G. Bass, L.F. Satler, R. Mehran, M. Porrazzo, K.M. Kent, A.D. Pichard, J.J. Popma, G.S. Mintz, M.B. Leon. *Washington Hospital Center, Washington DC, USA*

The Washington Radiation for In-Stent restenosis Trial (WRIST) is an FDA approved double-blinded randomized trial in 130 patients with in-stent restenosis.

Methods: Patients with ≥1 episodes of in-stent restenosis (mean 3.5) underwent PTCA, laser ablation or rotational atherectomy, and or additional stents. A non-centered 5 Fr closed end lumen catheter was positioned at the stent site, and patients were randomly assigned to either a ribbon with 192-Ir seeds or a placebo ribbon with non radioactive seeds both delivered by hand. Different ribbon lengths were used to cover lesion length of up to 50 mm. The prescribed radiation dose was 15 Gy to a 2 mm radial distance from the center of the source. Patients with restenosis at follow-up were eligible to receive radiation therapy if initially were randomized to placebo.

Results: In the first 75 patients (60 native coronaries and 15 vein grafts) the radiation was delivered successfully and tolerated well with a mean dwell time of 22 ± 4 minutes. The angiographic residual stenosis post PTCA was 20% and following radiation 24%. Ten lesions required additional stenting following radiation. Thusfar, there have been no procedural, in hospital, or 30-days adverse cardiac events.

Conclusion: In WRIST, radiation therapy for patients with in-stent restenosis using an 192-Ir hand delivered system has been feasible, safe, and without early adverse cardiac events. Interim results of six months follow-up from this cohort will be available at presentation.

4:15

865-2 Reduction of the Hyperplastic Response Following Balloon Angioplasty by β-Radiation

D. Meerkin, R. Bonan, J.-C. Tardif, A. Arseneault, J.-F. Tanguay, M. Joyal, J. Crépeau, G. Gosselin, G. Lucier. *Montreal Heart Institute, Montreal, Canada*

A 30 patient (pt) pilot study was performed to assess the effect of β-radiation in the prevention of restenosis following PTCA.

Pts were enrolled between February-June '97 with de novo lesions in native coronary arteries and were treated with PTCA followed by 12, 14 and 16 GY of radiation randomly assigned using the Novoste Betacath system. Pt characteristics are: age = 57.4 yrs (34–77), male = 17, LAD = 12, RCA = 8, Cx = 10, lesion type A = 2, B_1 = 14 ≥ B_2 = 12, C = 2. All pts have undergone 3-month clinical follow-up. Two pts returned with symptoms, one at 1 week with a type D dissection which was stented and the other at 2 months was found to have a restenotic lesion and underwent repeat angioplasty. Thirteen pts have completed 6-month angiographic follow-up with automatic pull-back IVUS performed prior to PTCA, post-PTCA and radiation and at 6-month follow-up (FU). Repeat angioplasty was performed in a single pt as stated above. In these 13 pts, the results were:

	pre-PTCA	post	6 month FU
QCA MLD (mm)	0.9*	2.1	2.1
% stenosis	70.5*	28.7	26.3
IVUS lumen area (mm^2)	2.1*	6.5°	7.7
EEM area (mm^2)	14.4*	16.6	15.5
wall area (mm^2)	12.3*	9.9	7.8

* $p < 0.003$ pre vs post. ° $p = 0.04$ post vs FU. MLD minimum lumen diameter, EEM External Elastic Membrane

β-radiation prevents angiographic restenosis. This prevention appears to be at the level of the wall area without any remodelling.

We conclude that the principal mechanism of restenosis prevention of the β-radiation is the prohibition of the intimal hyperplastic response following balloon angioplasty.

4:30

865-3 Intracoronary Radiation Post PTCA Prevents Late Arterial Constriction: A QCA Analysis

R. Waksman, J.A. Condado, J.F. Saucedo, A. Lanski, J.J. Popma. *Centro Medico, Caracas, Venezuela, Washington Hospital Center, Washington DC, USA*

Vascular constriction (VC) following PTCA is a major component in the mechanism of restenosis. Intracoronary radiation (IR) showed reduction of neointima formation in animal models and lower restenosis rates in pilot clinical studies.

Methods: To determine the influence of IR on VC, angiograms from a radiation trial performed in human coronary arteries following PTCA were analyzed by quantitatve computerized angiography using CASS and NIH methods. In this trial 14 patients underwent balloon angioplasty to de-novo lesions followed by radiation therapy with 192-Iridium (192-Ir) at doses between 20–25 Gy. All patients with patent arteries at follow-up were studied, and the angiographic indices at 6 months and 24 months compared with the post PTCA outcome.

Results: The angiographic analysis showed larger minimal lumen diameter (MLD) at 6 months compared with the post procedural results in 8/14 (57%); the late loss, and the late loss index were both negative. The effect of the vascular favorable remodeling in-patients who received radiation therapy with 192-Ir was maintained at 2 years.