acutely, but reverted to Afttr 3 days later. Histological analysis of the linear lesions tissues revealed marked heterogeneity of the healed lesions consisting of contiguous, collagenuos endocardial lesions (16  $\pm$  1 cm) with a raised, distening endocardial surface. The tissue had some regions showing nontransmural collagen formation under the ablation electrodes with tentacles of fibrous tissue penetrating transmurally.

Conclusion: Despite the heterogeneity and nontransmural character of some of the lesions, 6/7 hearts were converted to NSR or ODfiltr and 1 don converted to After 3 days post ablation. The predominance of After is likely due to the non-homogenous character of the linear lealons.

11:30 Radiofrequency Ablation of Bachmann's Bundle Terminates and Suppresses Atrial Fibrillation in Pigs 882-5

F, Abi-Samra, Z, Feng, S.K. Mobarek. Ochaner Medical Institutions, New Orleans, Louisiana, USA

Background; We hypothesized that radiofrequency ablation (RFA) of Bachmann's bundle (BB) could attenuate the induction of atrial fibrillation (AF).

Methods: Fifteen pigs were anesthetized with isoflurane\* and ventilated Sustained AF (lasting >30 min) was reproducibly induced using burst atrial pacing (50 msec intervals) during IV infusion of methacholine (M) (7.4  $\pm$  2.7 µg/kg/min). With M still infusing, RFA was performed at the region of BB in 10 plas (Gr I; 43,9 ± 3,2 kg). In the 5 controls (Gr II; 44,2 ± 2,3 kg), losions were applied in the right atrium (RA) along lines joining SVC to IVC, IVC to tricuspid annulus (TA), and TA to CS as using standard non-temperature controlled catheters with 5 mm tip (20-30 watts for 30-120 acc). The animals were convaleaced for 7-10 days, then re-tested for inducibility of AF. The hearts were then excised for gross anatomical examination.

Results: In Gr I, one pig died during M infusion prior to RFA, and one died after successful RFA prior to follow-up testing.

** ************************************	RFA of 8B	RFA of RA	Р	
AF	termination	8/9	05	0 001
AF reinduction - acute	1/9	-	wn	
AF induction - follow-up	1/8	5/5	- 0.001	
Number of RF leaions	61:43	18 : 1.4	- 0.001	

Atrial refractory periods were longer post-RFA (143  $\pm$  8 vs 158  $\pm$  17 msec in Gr I and 134  $\pm$  11 vs 146  $\pm$  13 msec in Gr II; p < 0.05). In Gr I, local AA intervals gradually organized and prolonged towards the end of each successful RF application

RF lesions were located at the region of BB in all 8 successul pigs. In the ninth Gr I pig, all lesions were found to have been inadvortently applied to the crista near the SVC. No perforations were noted.

Conclusions: 1) AF can be sustained with IV M; 2) In this model, RFA of BB terminates AF and prevents its reinduction while 3) extensive RFA of RA structures other than BB failed to do so.

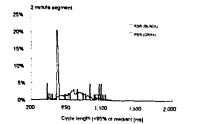


J.E.P. Waktare, K. Hnatkova, S.M. Sopher, F.D. Murgatroyd, A.J. Camm, M. Malik. St. George's Hospital Medical School, London, England

The initiation of paroxysmal atrial fibrillation (PAF) by a focal atrial tachycardia has been reported. Detection of atrial premature beats (APB) of a fixed coupling interval prior to AF onset would provide indirect support for this phenomenon.

Methods: Holter recordings (n = 177) from 60 patients with PAF were analysed by a validated process, generating computer files of RR intervals that differentiate AF from sinus rhythm, and ventricular ectopics from APB's. Sinus rhythm was divided into the 2 min preceding AF (PSR), the 2 min after AF, and that remote from AF (RSR). Those tapes where there were at least 15 APB's in both RSR and PSR were analysed.

Ventricular coupling interval of APB's was plotted graphically against relative frequency, and distributions classified by two observers



Results: 27 tapes (19 pts, age 56.6 ± 11.9) fulfilled inclusion criteria. Three broad categories of distributions were seen: Distinct frequency peaks both in RSR and PSR (52%); no distinct peaks (22%); or peaks in PSR only (26%, see figure).

Conclusion: Unifocal atrial ectopica may have a role in the initiation of AF. Thus Holter screening may identify AF pts suitable for limited radio-framency ablation procedures.

## 883 Stress Echonal diography: Relation Between Ischemia and Coronary Flow

Wednesday, April 1, 1998, 10:30 a.m.-Noon Georgia World Congress Center, Room 367W

10:30

## 883-1 Effect of Abnormal Coronary Flow Reserve and **Coronary Endothelial Dysfunction on Dobutamine** Stress Echocardiography: Pilot Phase Data From the NHLBI Women's Ischemia Syndrome Evaluation (WISE)

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Background: Ischemia related wall motion abnormalities during dobutamine stress ochocardiography (DSE) are a sensitive marker for severe epicardial coronary stenosis. However, impaired coronary dilation (or constriction) due to endothelial dysfunction or reduced firm reserve may also cause ischemia without epicardial stenosis.

Methods: Thirty-nine women with chest pain syndromes and atherosclerosis risk factors had DSE and core lab quantilative coronary angiography (QCA) analysis. Endothelial function was assessed by QCA following acetylcholing infusion (Ach) and coronary reserve (CFR) by Doppler flow velocity after adenosine infusion

Results: Endothelial dysfunction (failure to dilate with Ach) and/or abnormal CFR (<2.0) were identified in 67% (26 of 39) women. Of these 26 with coronary function abnormalities, only 4 had abnormal DSE (3 with severe CAD >50% stenosis by angiography and 1 without)

Conclusion: Thus, in the absence of severe CAD, wall motion abnormalities during DSE are rarely (1 of 23) observed in women with coronary endothelial dyslunction or abnormal microvascular flow reserve.

10:45

## 883-2 **Relation Dobutamine Induced Wall Motion** Abnormality and Coronary Flow Reserve in Patients With Angina Pectoris

T. Kajiura, R. Kuroda, K. Kurogane, Y. Funada. Takatsuki General Hospital, Takatski city, Japan

This investigation studied the relation ischemic threshold (DIT) and the magnetude of wall motion abnormality (WMA) during dobutamine (DOB) stress and coronary flow reserve (CFR) in patients with angina pectoris.

Methods: We studied 36 patients with significant coronary stenosis and without WMA at rest. DOB was infused 5-40 µg/kg/min until severe ischemia developed. Left ventricular wall motion was assessed using a 16-segment model, echocardiographic image quality was assessed with a five point scale. When regional dyssynergy developed, DIT was identified. WMA were detected in 20 patients (Group AP) and were not in 16 (Group N) during DOB infusion. Group AP patients were divided into 2 groups, with tow DIT (<20  $\mu$ g/kg/min; Group L, N = 10) and with high (>20; Group H, N = 10)

Results: 1) Mean stenoses of Group AP patients were more severe (97.2 vs 69.9%; p < 0.001) and CFR decreased in Group AP (1.15 vs 2.22; p < 0.001). There were significant correlation between wall motion score index (WMSI) at a maximal dose and CFR (r  $\approx$  -0.584; p < 0.05). 2) DIT was lower in Group L (16 vs 37  $\mu$ g/kg/min; p < 0.0001), heart rate at that dose was lower in Group L (95 vs 134 bpm; p < 0.0001). Wall motion score (WMS) and WMSI at a maximal dose increased in Group L than in Group H (WMS: 10 vs 6; p < 0.01, WMSI: 1.52 vs 2.56; p < 0.01). CFR decreased in Group L (1.01 vs 1.28; p < 0.01).

Conclusions: There were significant relation between ischemic threshold, extent and magnetude of DOB induced WMA and CFR.

