MHz Pedof probe. Resistance was closely related to OA with an indirect curvilinear relationship (logy = 0.19–0.55x, r = −0.93, see 0.13, p < 0.00001. n = 87), whilst the continuity area was directly and linearly related although with a systematic overestimation by the continuity equation (y = 1.13 + 0.79x, r = 0.90, see 0.23; p < 0.00001. n = 87). By correcting using the regression line, good agreement was shown using a Bland Altman plot with 95% limits of −0.41 to +0.42 cm^2 (resistance) and −0.43 to +0.43 cm^2 (Continuity equation). The empirical effective orifice area (EOA) formula based on resistance was EOA = 0.35 − 1.8 × log(mean ΔP/flow).

We conclude that resistance shows promise as a method of describing function in replacement valves in the mitral position whilst the continuity equation appears less accurate.

964 Cardiac Pacing: Clinical Observations

Tuesday, March 26, 1996, Noon–2:00 p.m.
Orange County Convention Center, Hall E
Presentation Hour: Noon–1:00 p.m.

964-101 Natural History of Atrial Arrhythmia in DDD Paced Patients Using the Permanent Holter Function of the Device

Serge Cazeau, Stephane Guarrigue, Philippe Ritter, Arnaud Lazarus, Jacques Mugica. *CC du Val d'Or, Saint-Cloud, France*

The incidence of atrial arrhythmia (AA) is probably underestimated because of paroxysmal asymptomatic episodes and intermittent ECG evaluation. In 213 chronically DDD paced patients (pts) – (70 ± 13 y, F 62%, M 38%) we used the extended memories of the available Holter function of the device — Chorus Ela Medical — to permanently monitor AA occurrence during follow-up (FU). Pacemaker indication was: High-degree AV block (30%), trifascicular block with long HV interval (21.%), Brady-tachy syndrome (21%), isolated sinus node dysfunction (10%), carotid sinus syndrome (6%), other indications (12%). Clinical, echo, electrophysiological data and pacing variables were recorded (multivariate analysis).

48.5% of the pts experienced AA during FU (361 ± 281 days — 3 to 1030). AA duration was < 24 hours in 31.1% asymptomatic pts (81.5% of the episodes); 24 hours < AA < 8 days in 10.3% pts; permanent AA > 7 days occurred in only 7.5% pts always after paroxysmal episodes. AV Block (p = 0.02), atrial premature complexes (p = 0.004), male gender (p = 0.008) appeared as independent risk factors. The more the atria were paced, the less we found AA episodes (p = 0.02). Implantation for trifascicular block was independently associated with a lower incidence of AA (p = 0.008). Age, existence of a cardiopathy, atrial size, ejection fraction, pacing and sensing thresholds, antiarrhythmic drugs were not predictive factors. 67 of the 154 pts without AA prior to implantation presented at least 1 episode after 207 ± 203 days of FU. In 37 of the 59 pts with AA before implantation AA recurred, much earlier (127 ± 0.113 days — p < 0.01). These data continuously collected modify our opinion about the natural history of AA and the true incidence of episodes in chronically paced DDD patients.

964-102 The Telectronics 330–801 Accufix™ Atrial Lead: Extraction Experience and Complications

Margaret A. Lloyd, David L. Hayes, David R. Holmes, Jr., Michael J. Osborn, Anthony W. Stanson, Raul E. Espinosa. *Mayo Clinic, Rochester, MN*

The Telectronics 330–801 Accufix™ Atrial Lead was withdrawn from the market after reports of fracture of the "J"-shaped retention wire, with subsequent extrusion of the wire and perforation of cardiac and vascular structures. As of April 1, 1995, we extracted 70 Accufix atrial leads (17 normal retention wires, 4 indeterminate wires, 24 fractured wires without protrusion, 25 fractured wires with protrusion). There were 43 males and 27 females; the average age was 65.1 ± 2.0 years. Average lead placement was 29.8 ± 2.0 months.

All patients underwent preoperative evaluation, including chest x-ray, ECG, and hematology, electrolyte, and coagulation panels. Patients had blood typed/screened prior to extraction, and extractions were performed with cardiac surgery available. Pacing dependency was assessed to determine need for temporary pacing.

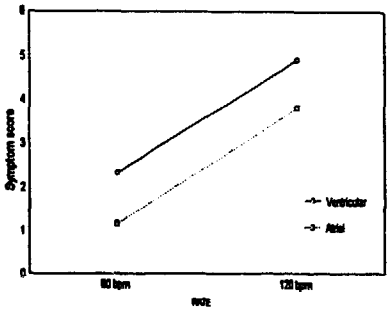
Forty-two leads were removed with simple traction only, and 6 leads were removed utilizing locking stylet/telescoping sheaths. The Cook workstation was employed in the removal of 20 leads via the femoral vein. The retention wire was removed leaving the lead intact in 1 patient, and the retention wire was removed prior to lead extraction in another. The average fluoroscopy time was 14.7 ± 2.3 min. Complications included asymptomatic innominate vein perforation (1), femoral vein bleeding (no transfusion required) (2), and ventricular lead dislodgment (2). In 4 patients, the entire pacing system was moved to the contralateral side after extraction because of inability to insert the new lead into the ipsilateral subclavian vein. There have been no life-threatening complications.

In experienced hands, the Telectronics 330–801 Accufix™ atrial lead can be extracted safely with minimal morbidity.

964-103 Palpitation: How Does It Originate?

Sergio L. Pinski, Rainer Meierhenrich, Elena B. Sgarbossa, Richard G. Trohman. *Cleveland Clinic Foundation, Cleveland, OH*

Palpitation (palp) is a common symptom in clinical practice. However, its pathogenesis is largely unexplored. We investigated the determinants of palp intensity in 26 pts (15 men, age 68 ± 13) with permanent pacemakers during short-term pacing at different rates and modes. Pts recorded the intensity of palp on a visual analogue interval scale at baseline and during random periods of single-blinded, ventricular or atrial-based pacing at rates of 80 and 120 bpm. The scale had good reliability and sensitivity to change. By repeated measures, 2-way ANOVA, intensity of palp was higher with ventricular pacing (p = 0.025) or rate of 120 bpm (p < 0.001), with no evidence of interaction between the 2 factors (figure). Clinical determinants of palp severity were explored during ventricular pacing at 120 bpm. There was a trend toward more severe palp with larger LV end-diastolic volume (r = 0.48; p = 0.07), and in pts with VA dissociation (p = 0.09). Age, gender, and body mass index had no significant effects.



Conclusions: palp can be quantified clinically. Its intensity is determined mainly by the heart rate and the chamber of origin of the impulse. Clinical variables are less important in its pathogenesis. This technique holds promise for the study of receptors and neural pathways responsible for the perception of palp. It could be used to assess of interventions aimed at decreasing palp severity.

964-104 An Algorithm for Rate Adaptive AV Delay in Patients With Heart Failure

Eric Grubman, Evan Loh, Dusan Z. Kocovic. *Hospital of the University of Pennsylvania, Philadelphia, PA*

Modern pacemakers have the ability to adapt the PR interval, based on the sensed or paced heart rate. However, there is limited information about circadian changes of PR interval in normals and there is no information concerning the circadian changes in PR interval in patients with heart failure (HF). In order to determine an algorithm for optimal paced rate adaptive AV delay in patients with HF, we compared 24 hour Holter monitor recordings in 10 patients with HF (LVEF < 35%, NYHA class II-III) and 13 age and sex matched controls. 5 consecutive PR and R-R intervals were measured every 15 minutes, utilizing 10 sec rhythm strips (96 measurements/patient) in order to calculate mean heart rate (HR), PR interval, the PR change for a given change in heart rate (PR index), and the range of heart rate and PR interval over 24 hours.

	Mean HR (bpm)	PR (ms)	PR index	PR Range (ms)	HR Range (bpm)
CHF (n = 10)	94±13*	152±19	0.8 ± 0.2*	37*	39*
Control (n = 13)	75±17*	167±35	1.2 ± 0.3*	73*	64*

(Mean ± S.D.) *p < 0.05 vs. control, T test

The higher mean heart rate, shorter mean PR interval and PR change for a given change in heart rate (PR index) in patients with HF may represent enhanced sympathetic activity effects in patients with HF, which limits the ability of the AV node to progressively shorten PR conduction as heart rate increases. In conclusion, the mean change in PR interval is less in patients with HF. As the majority of the current rate adaptation AV delay algorithms in permanent pacemakers do not reflect these PR interval variations, further studies to define the pathophysiology and physiologic role of modifying these algorithms for rate adaptive AV delay remains to be determined.