and in 28% of ST↑ episodes (n = 60) not preceded by ST↑ in the previous 30 min ($p < 0.01$). Finally, we analyzed 12 clusters of ischemic episodes occurring in periods of 30 min and showing VA in at least one episode. The number of premature ventricular beats per min was strikingly higher during the first episode compared with the last one (27 ± 9 vs 2.7 ± 3.6, $p < 0.01$); the first and the last episode of the clusters did not differ in severity (1.9 ± 0.9 vs 2.0 ± 1.6 mm) and duration of ST↑ (4.0 ± 3.8 vs 6.5 ± 13 min). Thus, our data indicate that transient ischemia confers a significant protection from ischemia-induced VA in pts with variant angina, unrelated to a reduction in severity or duration of ischemia.

797 Atrial Fibrillation: Pacing, Defibrillation, and Clinical Outcome

Wednesday, March 27, 1996, 2:00 p.m.–3:30 p.m.
Orange County Convention Center, Room 224G

2:00

797-1 Increased Mortality, Stroke and Medical Costs Imposed by Atrial Fibrillation

Philip A. Wolf[1], William B. Kannel[1], Colin S. Baker[1], Ralph B. D'Agostino[1], Janet B. Mitchell[1]. [1] *Boston University School of Medicine, Boston, MA; Health Economics Research, Inc., Waltham, MA*

Epidemiologic data now indicate that atrial fibrillation (AF) is a common and serious condition associated with a 4–5 fold increased risk of stroke and a doubling of mortality. Data on the added cost imposed by AF are not available.

Methods: One year mortality, stroke rates, in-hospital and out-patient costs were determined in a sample of hospitalized patients, aged 65–84 years, based on US Medicare data. Logistic and multiple linear regression analyses were done to take associated cardiovascular diseases (CVD) and length of follow-up into account in 12,625 patients with recent onset AF, compared to 13,809 patients without AF matched for age, sex and associated CVD.

Results: 1-yr. mortality was increased by 25–70% and stroke occurrence from 1.65 to 2.26 fold in those with AF compared to comparable persons with CVD without AF. Total 1-year medical costs were increased by $2,500 (15.4%) in men with no age differential. In women, the cost was greater in those under age 75 (21.5% added cost) than above age 75. The added costs imposed by AF were almost entirely attributable to the *in-hospital* costs.

		AF/Non-AF Odds Ratio		Excess Total Medical Costs with AF ($)
		Mortality	Stroke	
Men	65–74	1.25*	1.85	2,784
	75–84	1.44	1.77	2,454
Women	65–74	1.70	2.26	3,580
	75–84	1.36	1.81	1,711

*Not significant (All other AF–non-AF differences significant at $p < 0.05$ level.)

Conclusions: AF is clearly not benign exacting a substantial toll in mortality, morbidity and medical costs above that of the associated medical conditions.

2:15

797-2 Effect of Polarity on the Success of Transthoracic Shocks in Patients With Atrial Fibrillation

Keith H. Newby, Robert Waugh, Michael Hardee, Joe Mertz, Andrea Natale. *Duke University/VA Medical Center, Durham, NC*

We evaluated the effect of polarity on transthoracic monophasic shocks in patients with atrial fibrillation requiring elective DC cardioversion. 102 consecutive patients (84 M, 18 F, mean age 63 ± 11) undergoing elective cardioversion were randomized to either standard or reverse polarity shocks using an external defibrillator. Electrode patches (surface area 93 cm^2) were placed in an anterior-posterior position on the chest wall with standard polarity defined as anterior patch (–), and reverse polarity as anterior patch (+). Cardioversion was performed using a step-up protocol beginning at 100 J, and increasing the energy to 150, 200, 300, and 360J as needed until sinus rhythm was achieved. Results were as follows:

	Standard	Reverse	P value
Patients	51	51	
DFT (J)	193 ± 93	140 ± 68	<0.001
Impedance (ohms)	67.9 ± 17	72.9 ± 17	NS
EF (%)	44.4 ± 14	46.5 ± 14	NS
LA size (cm)	4.58 ± 0.63	4.9 ± 0.68	<0.01
Number of Shocks	2.7 ± 1.6	1.7 ± 1.08	<0.0007

We conclude: 1) Despite the larger left atrial size in the reverse polarity group, the mean energy requirements for successful cardioversion of atrial fibrillation proved significantly lower using reverse polarity transthoracic shocks as opposed to standard polarity shocks.

2:30

797-3 The Safety of Atrial Defibrillation in the Presence of Bundle Branch Block: A Proposal for New Criteria

Edward T. Keelan, David Krum, John Hare, Kashef Mughal, Huagui Li, Masood Akhtar, Mohammad R. Jazayeri. *Sinai Samaritan/St. Luke's Medical Center, Milwaukee, WI*

Although we have shown recently that atrial defibrillation shocks (ADS) synchronized to normally conducted supraventricular beats do not cause ventricular fibrillation (VF), our data may not be valid when the QRS complex is wide. Ventricular vulnerability to ADS might be increased in the presence of bundle branch block because of delayed repolarization of the ipsilateral ventricular myocardium. Complete right or left bundle branch block (RBBB/LBBB) was created in 2 groups of 10 dogs by catheter ablation. ADS were delivered between decapolar catheters in the coronary sinus and lateral right atrium. After crushing the sinus node, atrial burst, single premature and short-long-short (S-L-S) pacing protocols were used to determine the safety of ADS preceeded by the shortest R-R intervals achievable. ADS were coupled to the last paced beat (R_2), starting 60 ms after the end of R_2 and scanning decrementally through R_2 until VF was induced.

Results: The intervals between ADS and local ventricular electrograms recorded in the right ventricle (V_2-ADS) were determined for ADS causing VF. In 7 dogs with RBBB and 4 with LBBB, VF was induced by ADS delivered within R_2 complex indicating encroachment of R_2 on the vulnerable period (VP) of the preceeding (R_1). The values for these 11 dogs are shown in the table:

	(Values in ms)	Burst	Premature	S-L-S
RBBB	R-R	225 ± 7	253 ± 20	303 ± 26
(n = 7)	V_2-ADS [Range]	10 ± 7 [5–15]	15 ± 12 [0–30	10 ± 11 [0–30]
LBBB	R-R	205 ± 7	205 ± 13	245 ± 21
(n = 4)	V_2-ADS [Range]	20	13 ± 15 [0–30]	20 ± 28 [0–40]

Conclusions: 1. In the presence of bundle branch block, R_2 may coincide with the VP of R_1. 2. In this study, a minimum R-R interval of 350 ms would have been required to avoid ADS-induced VF. 3. ADS delivered within the R_2 complex with V_2-ADS ≥ 50 ms did not cause VF regardless of the preceeding R-R intervals.

2:45

797-4 Vagal Stimulation Reduces Spatial Organization of Atrial Fibrillation and Increases Internal Atrial Defibrillation Threshold

Brett M. Baker, Gregory W. Botteron, H. Dieter Ambos, Joseph M. Smith. *Washington University, St. Louis, MO*

We used graded amounts of vagal stimulation in a canine model of atrial fibrillation (AF) to test the hyphothesis that 1) increased vagal tone decreases the spatial organization of atrial activation during AF and 2) increased vagal tone increases the internal atrial defibrillation threshold (aDFT). Sustained AF was induced in 12 dogs by burst atrial pacing performed during bilateral vagal stimulation at the lowest intensity of vagal stimulation that would sustain AF (mean = 5.83 ± 3.59 Hz) and at 20 Hz. Bipolar atrial electrogram sequences were recorded in the right atrium using a specially designed basket catheter with five decapolar arms. The spatial organization of atrial activation was quantified as the activation space constant (ASC), defined as the average distance over which activation sequences remained well correlated during AF. Internal atrial defibrillaton was performed across electrode catheters positioned at the right atrial appendage (cathode) and in the coronary sinus (anode). Atrial defibrillation threshold was defined as the lowest energy which terminated AF on at least 2 of 10 attempts.

A significant reduction in the spatial organization of atrial activaton was seen in nine of 12 animals, with the mean ASC decreasing from 3.49 ± 3.52 at low vagal stimulation to 1.05 ± 0.35 at 20 Hz vagal stimulaton ($p < 0.05$). This increase in vagal stimulation was also associated with a significant increase in aDFT in 11 of 12 animals (mean aDFT = 0.45 ± 0.29 vs. 0.98 ± 0.41 joules, $p < 0.01$). Thus increased vagal tone decreases the spatial organization of atrial activation during AF and increases internal atrial defibrillaton threshold in this canine model of atrial fibrillation.