duction was assessed in 8 pts (7 f, 1 m, 52 ± 11 yrs) with common type AVN reentrant tachycardia (AVNRT) before and after radiofrequency current (RFC) ablation of the slow pathway (SP). AVN conduction curves and refractoriness (ref) were analyzed during baseline and following i.v. administration of propranolol (0.2 mg/kg) and atropine (0.04 mg/kg) before (day 1) and after RFC ablation (day 2). During pre-ablation baseline state, curves consistent with two antegrade AVN conduction pathways were observed in 4 pts and with three pathways in 3 pts. Autonomic blockade (AB) produced new dual patterns in 1 pt and annulled them in 2/7 pts, preventing from inducibility of AVNRT observed during baseline in 1; AVN ref was either unaffected (5 pts), markedly prolonged (2 pts) or shortened (1 pt), with nonpredictable changes of atrium-to-hissian (A-H) conduction interval curves. After RFC ablation, AVNRT inducibility was abolished in all pts and dual AVN physiology in all but 1 pt during baseline; ref of the fast pathway (FP) was reduced in 4/7 pts. AB after RFC exposed dual AVN physiology in another pt in whom also changes in AVN ref and AH interval curves were produced.

Conclusions: Data from this study suggest that the electrophysiologic substrate of AVNRT is more complex than the one defined by a dual pathway. Pharmacologic AB may expose otherwise unrecognized dual AVN physiology without consistent ability of inducing AVNRT after RFC ablation. AVN ref and A-H conduction do not present predictable behavioural patterns in response to either AB and RFC applied to the posterior-inferior approach to the AV node.

976-14 Immediate Heart Rate Response to Orthostatic Stress During β-blocker Therapy for Vasodepressor Syncope

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Although β-blockers are preferred agents for therapy of vasodepressor syncope (VDS), they are not uniformly effective and their mechanism of action is incompletely understood. Since we have previously shown a differential therapeutic response to β-blocker therapy between pts with isoproterenol-independent [iso(−)] and isoproterenol-dependent [iso(+)] VDS during tilt table testing we sought to determine whether this was due to a differential heart rate (HR) response to orthostasis during β-blockade. We therefore examined immediate HR and blood pressure responses to upright tilt before and after initiation of therapy with atenolol (12.5–50 mg daily) in 62 pts with VDS and positive tilt tests. The protocol comprised upright tilt (60°) for up to 60 min followed by repeat tilt for 15 min during isoproterenol (iso) infusion. Supine HR, mean arterial pressure (MAP) and pulse pressure (PP) were determined as the mean of 3 consecutive 1-min samples during supine rest; orthostatic HR, MAP, and PP were the mean of the samples recorded in the first 3 min after upright tilt (before infusion of iso). Response to atenolol required completion of tilt with and without infusion of iso. There were 15 iso(−) pts and 47 iso(+) pts. The groups did not differ significantly in blood pressure response (MAP, PP) to orthostasis. Supine HR fell and the ΔHR in response to orthostasis was blunted during therapy in both groups:

	Baseline (Mean ± SD)			Rx (Mean ± SD)		
	Iso(+)	Iso(−)	p	Iso(+)	Iso(−)	p
Supine HR	69 ± 13	68 ± 9	NS	57 ± 9	58 ± 8	NS
Orthostatic ΔHR	8 ± 7	12 ± 9	NS	3 ± 5	3 ± 4	NS

11 iso(−) pts (73%) had a therapeutic response to β-blockade compared with 46 iso(+) pts (98%, p = 0.01); the orthostatic ΔHR in the iso(−) pts who failed β-blocker therapy was no different from the response in the patients with a therapeutic response. *Conclusions:* The HR response to orthostasis is comparably blunted after β-blockade in pts with iso(−) and iso(+) VDS, indicating that failure to respond is not due to inadequate β-blockade and suggests that in some pts iso-independent VDS may be independent of a cardiac β_1 receptor mediated mechanism.

976-15 Assessment of Pacing Maneuvers to Validate Anterograde Accessory Pathway Potentials

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Four pacing maneuvers have been proposed to validate anterograde accessory potentials (APP): 1) atrial pacing to induce complete block between the atrial electrogram (Egm) and the APP; 2) atrial pacing to induce complete block between the APP and the ventricular Egm; 3) ventricular pacing to advance the ventricular Egm without altering the timing of the APP; 4) ventricular pacing to advance the APP without altering the timing of the atrial Egm. The purpose of this study was to determine the specificity of these validation techniques by applying them to split Egm's known, based on the location of the recording site, to consist of two atrial components. In 23 patients undergoing an electrophysiology test, a double atrial Egm was recorded in the high, lateral right atrium, mid lateral right atrium, right atrial appendage or the right atrium-inferior vena cava junction. The two atrial Egm components were 54 ± 15 msec apart at baseline. In each patient, critically timed atrial or ventricular premature depolarizations resulted in complete block between the second atrial component and the ventricular Egm, advancement of the ventricular Egm without altering the timing of the second atrial component, and advancement of the second atrial component by 10–35 msec without altering the timing of the first component. In no case could complete block be induced between the two components of the atrial Egm. In conclusion, among the four criteria proposed to validate anterograde APP's, the only one which may be specific for an APP is the induction of complete block between the atrial Egm and the APP.

977 Electrophysiology — Basic and Supraventricular Arrhythmias

Tuesday, March 21, 1995, 3:00 p.m.–5:00 p.m.
Ernest N. Morial Convention Center, Hall E
Presentation Hour: 4:00 p.m.–5:00 p.m.

977-70 Spiral Wave of Excitation as a Mechanism of Functional Reentry in the Isolated Atrium. A New Model of Atrial Reentry

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Background: Functional reentrant excitation in atrial tissue (i.e., reentry with no central anatomical obstacle) was proposed to be caused by the "leading circle" mechanisms (Allessie's model). According to this concept, there is no excitable gap and the central core of functional conduction block (FCB) results from continuous centripetal invasion of wavefronts that prevent the cells in the core to recover. *Hypothesis:* The mechanism of functional reentry in atrial tissue is caused by a spiral wave with an excitable gap and a central core of FCB caused by the steep *curvature* of the spiral wave tip. *Methods:* Blocks of 3.0 by 4.0 cm of canine left and right atrial tissues (N = 3) were mounted in a tissue bath with endocardial surface up and reentrant activity initiated by premature stimulation and/or by rapid pacing and acetylcholine (10^{-6} to 10^{-5}M). Isochronal activation maps of the induced reentrant excitation were constructed using 512 bipolar electrodes with 1.6 mm interelectrode distance and were also displayed dynamically on the computer screen to visualize the pattern of activation. *Results:* Ten episodes of both nonsustained and sustained atrial reentry was induced and mapped. The induced reentrant activity was clearly a spiral wave with a rotation period of 184 ± 74 (110 to 350) msec and rotating in a counterclockwise direction in all. The area of FCB in the central core of the spiral wave was caused by the steep *curvature* of the tip of the spiral wave that was excitable during regular pacing but not excited during the spiral wave activity. Spiral wave activity could be terminated by electrical stimulation indicating the presence of an excitable gap. *Conclusion:* Functional reentry in normal atrial tissue is caused by a spiral wave of excitation with an excitable gap.

977-71 Conduction Patterns of the Sinoatrial Node and Perinodal Region Demonstrated by an Optical Mapping Technique

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Studies of the conduction patterns within the sinus node region have been limited to extracellular surface and intracellular microelectrode techniques. Even with multiple electrodes, data is limited to a finite number of discrete points within the tissue, thus pacemaker shifts, entrance block, and antegrade versus retrograde conduction differences have not been completely elucidated. To study the entire sinus node (SAN), functional perinodal boundary (FPB), and crista terminalis (CT) tissue preparation, we use a 144 element photodiode array recorded simultaneously with bipolar sensing and pacing electrograms. A wide margin of right atrial tissue including the interatrial septum (IAS), from an adult New Zealand white rabbit, was dissected and viability maintained by superfusion of 37°C, O_2 enriched Krebs. The tissue was stained with 2 mM di-4-ANEPPS, a voltage sensitive, optically active dye. The isochronal activation patterns are constructed by linear interpolation of times in the image space between photodiode elements (625 μm). Each isochronal band represents 1 ms. conduction time. Single decremental premature stimuli (560 ms) were delivered into a sensed sinus rhythm (900 ms) at the inferior CT (STIM).