Ventricular Septal Summit: Stimulation in Atrioventricular, Nodal, and Right Atrial Tachycardia

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Programmed premature ventricular complexes (PVC's) during supraventricular tachycardia can assist in diagnosing AV node reentry tachycardia (AVNRT). We compared the effect of pacing from the RV apex (RVA) vs RV septal summit (RVS) in 14 pts with typical stable AVNRT (tachycardia cycle length (TCL) variation < 5 msec). The percentage of TCL at which PVCs first advanced the succeeding atrial complex (A) as well as the amount of time the next A (A2) was advanced were noted. PVCs were introduced during tachycardia at 10 msec decrements from both RVA and RVS. RVS stimulation resulted in only direct stimulation of the low septal right atrium (LSRA) and RVS in four patients. In the remaining 10 patients, RVA advanced the succeeding atrial complex (A) in 7 patients, while RVS advanced the A in 5 of 14 patients (70% vs 56%, p<0.01). In the 3 patients when both RVA and RVS advanced the A, RVS was able to advance the A at a lesser degree of prematurity (75% of TCL vs 68%) and the maximal A2 was larger (65 vs 42 msec). In 4/10 patients, atrial tachycardia was excluded by RVS extrastimuli because of tachycardia termination without conduction to A (3 pts) or delay of < 15msec of the next A (1 pt) and only in 1/14 patient could atrial tachycardia be excluded by RVA because of the delay of the next A (40% vs 7%, p<0.01). In 7 patients, increased stimulus strength from RVS allowed for simultaneous capture of both ventricle as well LSRA at 69 to 63% of the TCL without affecting the tachycardia.

Conclusion: 1) RVS resulted in more pronounced effects on AVNRT compared with RVA because of proximity to the tachycardia circuit. 2) RVA compared to RVS stimulation allowed for exclusion of atrial tachycardia in significantly more pts. 3) Simultaneous activation of LSRA and RVS without affecting the tachycardia in 7 pts suggests that the tachycardia is confined to the region of the AV node without the involvement of contiguous atrial tissue.

CARDIOVERSION OF ATRIAL FLUTTER BY INTRAVENOUS IBUTILIDE: A NEW CLASS III ANTARRHYTHMIC AGENT

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Ibutilide is an investigational class III antiarrhythmic agent with a unique ionic mechanism. In an initial dose ranging study, 10 patients with sustained (2 to 21 days) atrial flutter (AFL) received intravenous (IV) placebo (P) by sequential doses of ibutilide at 5, 10 and 20 mg/kg IV over 10 min each at 5-min intervals. The 0 male and 4 female patients were 64±18 years old; 7 had significant structural heart disease with left atrial enlargement documented in 5 and a prior history of atrial arrhythmias in 6. Cumulative conversion efficacy is shown below.

No patients converted with P, but ibutilide terminated AFL in 9 of 10 patients within 30 min after the final dose. AFL cycle length increased from 217±26 to 245±33 msec immediately prior to termination. AFL was prolonged to 462±46 vs 426±41 msec following drug washout. Ibutilide had no effect on blood pressure. One patient with aortic stenosis and coronary artery disease developed polymorphic ventricular tachycardia requiring cardioversion 2 min after termination of AFL. No other adverse effects were noted. These preliminary data indicate that IV ibutilide administered in a monitored setting, rapidly and effectively terminated sustained AFL in a high proportion of patients.

ON THE MECHANISM OF PERPETUATION AND TERMINATION OF ATRIAL FIBRILLATION OF MAN

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To study the mechanism of perpetuation and termination of atrial fibrillation (AF), we analyzed the intra-atrial potentials during AF induced by programmed electrical stimulation with the concept of wavelength which represents the size of a microreentrant circuit. Thirty Pts with inducible AF were divided into two groups: 20 Pts with AF which terminated spontaneously (group 1) and 10 Pts with AF which did not terminate spontaneously (group 2). Wavelength is the product of refractory period (RF) and conduction velocity (CV). During AF, RF of the local atrial tissue correlates with the mean interval between each intra-atrial potential (mean ff) and CV of the impulses correlates inversely with the width of intra-atrial potentials (f width). Thus, the wavelength index (WLI) was defined as follows: WLI=(mean ff)/(mean f width). Ten intra-atrial potentials at the high right atrium were sampled for measurement. Group 1 had higher WLI just after the induction of AF than group 2 (1.33±0.31 vs 1.02±0.05, p<0.05). In group 1, WLI's were increased from the initial value to 1.49±0.36 just before the termination of AF (p< 0.001). In the three Pts of group 2, AF stopped after dipryamides was administered intravenously, while WLI's at the end of AF were higher than those before the drug administration (1.27±0.08 vs 1.16±0.04, p<0.05).

Conclusions: 1) Shorter wavelengths are necessary for the perpetuation of AF. 2) Prolongation of the wavelength appears to be a major determinant for the termination of AF.

ATRIAL FLUTTER CAUSES FALSE POSITIVE LATE POTENTIALS ON SIGNAL AVERAGED ELECTROCARDIOGRAM.

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We hypothesized that atrial flutter (AF) waves cause late potentials on signal averaged electrograms (SAECG). We performed an SAECG in 9 pts during AF and then after conversion to sinus rhythm. Time domain analysis of the QRS complex was performed using a band pass filter of 40-250 Hz. We analyzed unfiltered (Std QRS) and filtered (Fit QRS) QRS complex duration, duration of low amplitude signal <40 μV at the terminal portion of the QRS (LAS) and the root mean square of the voltage of the terminal 40 ms of the QRS complex (RMS). An SAECG was considered abnormal if 2 of the following criteria were satisfied: RMS < 25 μV, LAS < 35 ms, or Fit QRS > 120 ms. The mean values before and after conversion to sinus rhythm were as follows:

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<th>Std QRS</th>
<th>Fit QRS</th>
<th>LAS</th>
<th>RMS</th>
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<tr>
<td>AF</td>
<td>96±7</td>
<td>104±7</td>
<td>20±4</td>
<td>37±5</td>
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<tr>
<td>P value</td>
<td>&lt;0.03</td>
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During AF, 6/9 SAECG recordings were abnormal. During sinus rhythm, only 1/9SAECG recordings remained abnormal. Analysis of the surface 12 lead ECG during AF revealed AFL waves were present in the terminal portion of the filtered QRS complex in the 6 abnormal but not in the 3 normal SAECGs.

Conclusion: Atrial flutter waves which occur during the terminal portion of the QRS complex may cause false positive late potentials on signal averaged electrocardiogram. Therefore, signal averaged electrocardiograms obtained during atrial flutter which demonstrate late potentials should be repeated during sinus rhythm.