VENTRICULAR SEPTAL SUMMIT STIMULATION IN ATRIO-VENTRICULAR NODE REENTRY TACHYCARDIA

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Programmed premature ventricular complexes (PVC) during supraventricular tachycardia can assist in diagnosing AV nodal reentry tachycardia (AVNRT). We compared the effect of pacing from the RV apex (RA) 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 (Aa) 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, RVS advanced the preceding atrial complex (A) in 7 patients, while RVA advanced the A in 5 of 14 patients (70% vs 36%, 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 (78% of TCL vs 88%) and the maximal Aa 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 ≤15 msec 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 ventricles as well LRSA at 69 to 83% 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) RVS compared to RVA 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 ANTIARRHYTHMIC 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 AFL (217±21 days) atrial flutter (AFL) received intravenous (IV) placebo (P) followed by sequential doses of ibutilide at 5, 10 and 20 μg/kg IV over 10 min each as 3-min intervals. The 6 male and 4 female patients were 64±18 years of age; 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±21 to 254±33 msec immediately prior to termination. AFL was prolonged to 462±246 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.