LOW ENERGY DIRECT CURRENT ABLATION IN PATIENTS WITH SUPRAVENTRICULAR TACHYCARDIA.

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We evaluated a new system (NS) that maximizes energy delivery while reducing barotrauma. This NS consists of 1) a low energy direct current power source (Cardiac Recorders, UK) with a short time constant capacitive discharge, and 2) a 7F ablation catheter with a contoured distal electrode (Bard). The NS was used in 14 pts (5 males, 9 females) with disabling supraventricular tachycardias (SVT) refractory to antiarrhythmic drugs (AAD). Mean age was 52 years (range 20-73). SVT includes atrial fibrillation (AF) in 7 pts, A. flutter in 1, AV nodal tachycardia (AVNT) in 2, Wolff-Parkinson-White (WPW) syndrome in 4 (manifest accessory pathway in 3 and concealed in 1).

Anodal shocks of 5 to 40 joules (j) were given, during 1 to 3 sessions, without complications. The mean cumulative energy (j), voltage (volts) and current (amps) were respectively, 187±150 j, 13,868±10,429 volts and 107±72 amps (p=NS between AF pts and other SVT). All 7 AF pts were in complete heart block with a junctional escape rhythm (1447±114 msec) and received a VVIR pacemaker. The other 7 pts remained in sinus rhythm with 1:1 AV conduction. After a mean follow-up of 2.2 months (range .1-4), all AF pts remain asymptomatic. SVT recurred in 1 pt with AVNT and in 1 pt with WPW. Both are asymptomatic after AAD treatment. Delta waves are either absent or modified in 3/3 WPW.

Our initial experience with this NS has a success rate of 86%. Effective ablation of SVT, using less than 200 j but more than 10,000 volts of mean cumulative energy per pt, suggests that voltage, rather than barotrauma, may be responsible for tissue injury.

LOW ENERGY RADIOFREQUENCY CURRENT FOR AV NODAL ABLATION

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Endocardial catheter ablation of the AV node has been achieved by using a relatively large number of applications of radiofrequency current (RFC). We investigated the usefulness of a 6F tip deflectable catheter with a 4 mm distal electrode for localization of the proximal His bundle and delivery of RFC. With the pt mildly sedated, RFC (500 KHz) was delivered for a maximum of 60 secs between the distal electrode and a plate applied to the pt's back.

Of 11 symptomatic pts (6 men, 5 women), aged 39-76 yrs (62±12), 7 had chronic or paroxysmal atrial fibrillation, 2 ectopic atrial tachycardia and 2 AV nodal reentrant tachycardia. The preset power delivered was 14 watts in 1 pt, 20 W in 9 and 30 W in 1. Voltage ranged from 40-60 V (45±6). The number of RFC applications required to achieve AV block ranged from 1 to 5 (median 2). Total energy delivered ranged from 240-934 joules (575±244). Complete AV block occurred in 9 pts with immediate junctional escape rate 46±12 per min. 2 initially conducted with high degree AV block and developed complete AV block 2 days later.

All pts received a rate responsive pacemaker. They were all symptom free at follow-up in 2 to 6 months.

Conclusion: AV nodal ablation with low RFC can be achieved with high success rate using a modified catheter technique.

CATHETER ABLATION OF ACCESSORY PATHWAYS: CLINICAL RESULTS WITH LOW ENERGY NON-ARCING SHOCKS.

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Catheter ablation remains an experimental treatment for accessory pathways because of the risk of rupture of the coronary sinus (CS)and cardiac tamponade. Rupture is a consequence of high pressures secondary to arcing and gas expansion at the catheter tip. We have devised a system that delivers shocks of low energy and high peak voltages without arcing. This system comprises a short time constant high voltage capacitor and modified electrode.

The method used employs the delivery of a test shock of 5-15 Joules through this system to the site of earliest atrial activation during reentrant tachycardia. If transient loss of preexcitation or retrograde conduction is produced, repeated higher energy shocks of 15-25 Joules are delivered to this site. If there is no effect, further mapping is performed and further test shocks are delivered to other sites.

This method has been used in 10 patients of mean age 35 (range 20-78). The site of the accessory pathway was posteroseptal (PS) in 5, left free wall (LF) in 3 and right free wall (RF) in 2. Test shocks had no effect on AP conduction in 4 Pts (2 PS 2 RF) and catheter ablation was abandoned. Further shocks were delivered to the remaining 6 Pts. Shocks were delivered outside the CS os in 3 (PS), inside the CS in 2 and from the left ventricle in 1. There were no episodes of CS rupture, tamponade or coronary artery spasm. The only complications seen were transient episodes of AF and 2° AV block. There were no rises in cardiac enzymes. Results at a follow up of mean 8 months show no pathway conduction in 4, modified conduction in 2. 5 Pts remain free of tachycardia on no medication and one is controlled on previously ineffective medication. Successful ablation was produced with a mean of 5 shocks and mean cumulative energy of 95 Joules. Analysis of voltage and current during shock delivery confirmed the shocks were non-arcing.

Conclusion Repeated non arcing shocks are effective for the ablation of accessory pathways at low energies and without barotrauma. Initial clinical experience suggests that this method may widen the application of catheter ablation for accessory pathways.

Monday, March 19, 1990
2:00PM–3:30PM, Room 36
Endothelium-Dependent Vasodilation

PULMONARY VASODILATION BY POTASSIUM CHANNEL ACTIVATION

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The purpose of the present study was to study the nature of potassium channel (PC) activation in the pulmonary vascular bed of the intact, spontaneously-breathing cat. Since pulmonary blood flow and left atrial pressure were kept constant, changes in lobar arterial pressure directly reflected changes in lobar vascular resistance. Under resting (low) pulmonary vasomotor tone (PVT), intralobar bolus injections of PC activators, pinacidil (10-100ug) and cromokalim (3-30ug) decreased systemic arterial pressure in a dose-dependent manner with little effect on baseline lobar arterial pressure. When PVT was actively increased by intralobar infusion of U46619, a thromboxane A_2 mimic, intralobar bolus injections of pinacidil and cromokalim at similar doses decreased lobar arterial and systemic arterial pressures in a dose-related fashion. The marked pulmonary vasodilator response to pinacidil and cromokalim was abolished by the glibenclamide, 5mg/kg i.a. given over 15 min. In contrast, this dose of glibenclamide did not alter pulmonary vasodilator responses to acetylcholine, bradykinin, isoproterenol, prostacyclin and calcitonin gene-related peptide. Results of the present experiments provide the first reported data demonstrating the presence and blockade of PC which mediate vasodilation in the lung. Moreover, this newly-described vasodilator mechanism may play an important role in regulating the pulmonary vascular bed and in the pathogenesis of pulmonary hypertensive disorders, such as cor pulmonale and primary hypertension.