CHARACTERIZATION OF ATRIAL FLUTTER FOLLOWING THE MUSTARD PROCEDURE FOR TRANSPOSITION OF THE GREAT VESSELS

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A common long-term postoperative complication of the Mustard procedure has been the development of refractory atrial flutter (AFL). This arrhythmia can be life-threatening in these patients. Certain individuals exhibit a form of AFL with atypically slow atrial rates. This potentially could result in sudden death, which has been reported in these patients if the atrial cycle length exceeds the A-V nodal refractory period, resulting in 1:1 A-V conduction. This study was designed to characterize AFL resulting from a simulated Mustard procedure. To simulate the Mustard procedure in normal dogs (n = 10), a longitudinal atriotomy was performed; the atrial septum resected; and a continuous suture line placed in the usual baffle location without redirection of intra-atrial blood flow. Through ventriculotomies, right and left endocardial molds containing 252 bipolar electrode pairs were positioned to record activation time maps during induced AFL. All animals had induced sustained AFL with an average cycle length (CL) of 162 ± 21 ms. One reentrant circuit involved the tricuspid annulus (n = 5, CL = 167 ± 17 ms), where the wavefront was forced by a large posterior obstacle composed of the orifices of the superior vena cava, right atrial appendage, and inferior vena cava which were linked by the combination of functional block and the surgical incisions. Another pathway involved the orifices of the superior vena cava and right atrial appendage (n = 4, CL = 146 ± 11 ms); a third pathway involved the pulmonary vein orifices (n = 1, CL = 158 ms); and a fourth long pathway involved both atria (n = 1, CL = 207 ms). Thus, the suture lines necessary for performance of the Mustard procedure establish a uniquely arrhythmogenic anatomic substrate for the development of AFL postoperatively. This substrate is expressed as several different long reentrant pathways. In addition, most of the animals demonstrated regional slowing of the conduction velocity in association with the right atriotomy. Thus, the longer cycle lengths in these forms of AFL reflect both the longer reentrant path lengths and focal slow conduction. These data should lead to the modification of the surgical approach involved in the Mustard procedure to interrupt potential reentrant pathways at the time of corrective surgery.

HISTOLOGICAL SUBSTRATE FOR ARRHYTHMIA AFTER RIGHT VENTRICULAR OUTFLOW TRACT REPAIR

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Clinical electrophysiology studies have demonstrated inducible arrhythmias localized to areas of surgical scar following repair of tetralogy of Fallot. Although structural correlates of sustained ventricular arrhythmia have been identified in the border zone of myocardial infarction (MI), scarring following right ventricular outflow tract (RVOT) repair for congenital heart defects has not been similarly characterized.

Specimens were cut from the margin of the RVOT patch/RV free wall junction from 11 hearts obtained from autopsies between 1974 and 1989 at Texas Children's Hospital and prepared for light microscopy. The age of patients ranged from 5 months to 13 years and postoperative times from 2 days to 5.5 years with 8 specimens ≤ 25 days and 3 specimens ≥ 6 months.

Tissue damage with scattered hemorrhage, myocytolysis and loss of nuclei was noted in the immediate postoperative specimens (2-3 days). This evolved into well defined areas of cell death surrounded by myocytes with intact nuclei and cytoplasmic structure by 25 days. Specimens ≥ six months revealed areas with bundles of myocardial cells isolated by extensive fibrosis. These bundles formed a layer 3-7 cells deep in the subendocardium or surrounding the vessels of the intramyocardial vascular supply.

Viable myocardium in the late postoperative RVOT scars appeared to correlate with areas where greatest oxygenation was expected. This irregular pattern of viable myocardium and fibrosis structurally resembled the border zone of chronic MI where sustained ventricular arrhythmias have been correlated with the disordered conduction of the scarred tissue. This similarity supports the concept that the pattern of myocardial preservation of the RVOT scar forms a histological substrate for ventricular arrhythmia similar to MI.

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Poster Displayed: 2:00PM–5:00PM
Author Present: 4:00PM–5:00PM
Hall F, West Concourse
Coronary Artery Surgery

CORONARY ARTERY BYPASS SURGERY IN PATIENTS OVER 80 YEARS

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The outcome of coronary artery bypass surgery was studied in 158 patients over the age of 80 (mean age 82.0, range 80-100). 63% were male, 24% had class III angina and 59% had class IV angina, 6.3% had class III or IV congestive failure, 16% had diabetes, 48% had a prior myocardial infarction (MI), 22% had left main coronary disease, and the ejection fraction was 55 ± 14 (33% under 50). In hospital mortality was 9.6%, 2.6% suffered a Q wave infarction and 5.2% had a stroke. In hospital mortality was 6.5% without diabetes and 20.8% with diabetes (p=.02). In hospital mortality was 6.8% with an ejection fraction over 50 and 16.3% with an ejection fraction under 50 (p=.09). Five year survival was 66%, and MI free survival was 62%. The presence of diabetes was the prime correlate of survival. Two year survival without diabetes was 87% and 62% with diabetes (p=.03). Two year MI free survival without diabetes was 83% and 62% with diabetes (p=.08). Other factors including age, gender, prior MI, hypertension, number of vessels diseased, ejection fraction, number of grafts placed, angina class, and congestive failure class did not affect mid-term survival. Patients over 80 with diabetes are at increased risk of both in hospital and mid term mortality.

Conclusion: While coronary artery bypass surgery over age 80 has largely been used to treat severe angina pectoris rather than as a therapy to prolong life, selected patients have a favorable prognosis.

CORONARY SURGERY REDUCES MORTALITY IN SURVIVORS OF OUT-OF-HOSPITAL CARDIAC ARREST

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The effect of coronary surgery on subsequent cardiac arrest was determined in 265 patients from a cohort of 1079 consecutive patients discharged from hospital following out-of-hospital cardiac arrest between 1970 and 1988. Of the 1079 patients, 265 (25%) were known to have coronary artery disease and had a documented left ventricular ejection fraction (EF); 85 patients (32%) had a coronary artery bypass graft (CABG) performed after recovery from the arrest, and 180 patients (68%) were treated medically.

A multivariate Cox analysis was used to evaluate the effect of CABG on subsequent survival after adjusting for age, prior cardiac history, EF, year of initial cardiac arrest, myocardial infarction at the time of arrest, and a time dependent covariate for CABG. Although survival of patients treated by CABG was improved overall (RR = 0.56, 95% C.I.= 0.34-0.93, $p < 0.02$), the principal effect of CABG was to reduce the subsequent incidence of recurrent cardiac arrest. Patients treated by CABG had fewer episodes of cardiac arrest during follow-up (RR = 0.44, 95% C.I. = 0.22-0.87, $p < 0.01$). A second analysis using matching methodology confirmed both Cox findings.

Based on these retrospective observations, we conclude that the use of coronary surgery in selected patients who have been resuscitated from cardiac arrest may substantially reduce the incidence of recurrent cardiac arrest.