DO LONG TERM RESULTS JUSTIFY PEDIATRIC HEART RANSPLANTATION? <u>David Baum, Edward Stinson, Philip</u> Oyer, John Baldwin, Sharon Hunt, Vaughn Starnes, Norman Shumway. Depts. of Pediatrics and Cardiac Surgery, Stanford Univ. Medical Center, Stanford, CA 79

Heart transplantation (Tx) is gaining acceptance as a treat-ment for young patients with end stage cardiac disease, although the reported data are limited and follow-up brief. From 1/74 through 8/86, 30 patients with cardiomyopathy and 4 with congen-ital heart disease (ages 2.8-18.9 years; 24M/10F) received Tx at Stanford. Immunosuppression was accomplished with steroids, azathionrine articthymcyte albulin and after 1000 with such Stanford. Immunosuppression was accomplished with steroids, azathioprine, anti-thymocyte globulin and after 1980, with cyclo-sporine. Re-Tx was required for intractable rejection (3) and advanced coronary disease (1). Twenty three patients are alive 2 weeks-9.6 years post-Tx. Age at Tx did not affect survival. All are active (NYH Class I), and attend school or work. In the 11 fatalities, post-Tx survival ranged between 2 days and 11.4 years. Death was due to infection (5), rejection (3), graft failure (1), lymphoma (1) and coronary disease (1). Our 99.1 patient year experience yielded linearized infection and re-jection rates of 1.1 and 0.6 episodes/patient year, respectively. Of 6 patients with recognized coronary disease, 3 demonstrated the disorder as soon as 2 years post-Tx. Hypertension requiring therapy invariably developed in patients given cyclosporine. Based upon data from Stanford's 34 patients, probability for post-Tx survival is 78% at 2 years and 66% at 5 years. These long term observations suggest that Tx deserves consideration for selected young patients with end stage cardiac disease.

PROGNOSIS OF NEWBORNS AND INFANTS WITH PREMATURE VENTRICULAR CONTRACTIONS

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Between Sept 1982 and Oct 1984, we diagnosed premature ventricular contractions (PVCs) in 9 newborns and infants (3M/6F). CXRs were obtained in 6 patients(pts) and echocardiograms were performed in 8 pts. 7 pts were judged to have an otherwise normal heart while 1 pt was found to have a small VSD and another had mild peripheral PS. 24-hour Holter monitor recordings in 7 pts revealed frequent or infrequent uniform PVCs, infrequent multiform PVCs, uniform ventricular couplets, accelerated ventricular rhythm, and non-sustained uniform ventricular tachycardia. Frequent uniform PVCs were uniform ventricular tachycardia. Frequent uniform FVOS were present in 6 pts and were completely suppressed during sinus tachycardia in 5/6. 1 pt was treated with propanolol (PR) which decreased but did not completely eliminate the PVCs. None of the other pts has received any antiarrhythmic treatment. Follow-up in these pts has ranged from 1 to 25 mos with a mean follow-up of 10.2 mos. At each pt's most recent follow-up examination, PVCs were still present. All pts are alive and well and none have ever had any symptoms either at the time of initial diagnosis or during subsequent follow-up. We conclude that: 1)PVCs in newborns and infants with otherwise normal hearts are benign; & 2) antiarrhythmic treatment is unnecessary and does not influence an already excellent prognosis. We there-fore recommend that newborns and infants discovered to have PVCs and no (or trivial)underlying heart disease be followed but not treated with antiarrhythmic drugs.

> CONDUCTION ABNORMALITIES FOLLOWING REPAIR OF TETRALOGY OF FALLOT

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Ventricular dysrhythmias following intracardiac repair of tetralogy of Fallot have been previously demonstrated. Less attention has been directed to postoperative conduction abnor-malities in these patients. Between October, 1982 and March, 1984 we performed hemodynamic and electrophysiologic studies in 11 patients (8 males/3 females) who had previously under-gone repair of tetralogy of Fallot. A wide spectrum of conduction system abnormalities including sinus node dysfunction (5/11), atrioventricular node dysfunction (3/11), and His-Purkinje disease (7/11) were found. Confirmation that bifasicular disease cannot be reliably diagnosed from the surface electrocardiogram alone was obtained. Spontaneous and/or indu-cible ventricular dysrkythmias were documented in 5/11 patients. However, there was no consistent relationship between the presence or degree of residual right ventricular hypertension and the presence of ventricular dysrhythmias. We conclude that: 1) frequent conduction abnormalities as well as ventricular dysrhythmias may occur in patients following repair of tetralogy of Fallot; and 2) ventricular dysrhythmias may occur in these patients even when residual right ventricular hypertension is not present. We therefore recommend that all patients should undergo postoperative hemodynamic and electrophysiologic study following intracardiac repair of tetralogy of Fallot.

PERMANENT PACING INDICATIONS FOLLOWING MUSTARD OPERATION FOR TRANSPOSITION OF THE GREAT ARTERIES. Stanley D. Beder, Mark H. Cohen, and Thomas A. Riemenschneider. (Spon. by Jerome Liebman).

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Sick sinus syndrome is a frequent complication following Mustard operation. Between October 1971 and May 1985, we performed the Mustard operation on 78 patients with transposition of the great arteries. Sixty-six patients Were followed long-term for up to 10 years after surgery. We reviewed patient records and Holter monitor recordings in these latter 66 patients to determine if the degree of bradycardia predicted symptoms requiring permanent pacing. A total of 8/66 patients received pacemakers Indications for pacing included asymptomatic bradycardia during the first postoperative month, symptomatic brady-cardia (seizures and syncope) late following surgery, and use of antiarrhythmic drugs other than digoxin for the control of symptomatic atrial flutter or supraventri-cular tachycardia. The mean minimal heart rates documented by Holter monitor recordings did not differ significantly between the asymptomatic and symptomatic patients (p 0.15). We conclude that the degree of bradycardia is not a reliable predictor of symptoms or the need for pacing in postoperative Mustard patients during early and intermediate duration follow-up.

QUANTITATION OF LASER IRRADIATION BY-PRODUCTS Giora Ben-Shachar, Michael L. Spector, Dennis E. Morse, Merril E. Adams, Mark C. Sivakoff, Thoma A. Riemen-schneider (Spon. by J. Liebman), CWRU, Department of Cleveland, OH. 83

In-vivo laser irradiation can create hazardous by-products. The purpose of the study was to evaluate the nature of the de-bris, and quantitate them. Segments of fresh porcine myocardium and vascular wall were irradiated in sodium phosphate buffer, with Argon laser. The amounts of tissue vaporized and accumula-tion of tissue debris were measured, and analyzed with Scanning Electron Microscopy (SEM). Gasseous & thrombi by-products & Electron Microscopy (SEM). Gasseous & thrombi by-products & temperature changes were measured in an artificial circulation using a centrifugal pump (Bio-medicus 520D). Blood was hepar-inized (5000 u) and circulated @ 21/min. Continous laser irradi-ation of the blood was done @ 6 1/2 watts with 400 micron regular quartz or hot-tip fiber, for 10 minutes. Vascular walls and myo-cardial debris formed at a rate of 227 and 61.5 mg respectively per 1,000 mg tissue lased, showing charred fragmented tissue seg-ments on SEM with sizes ranging up to 3 mm. Fragments of cell wall, and intracellular membranes were also identified. 23.5 and 15.5 ml of gas were formed by both fiberoptic and hot-tip irradiation respectively. Temperature elevations were between and 15.5 ml of gas were formed by both fiberoptic and not-tip irradiation respectively. Temperature elevations were between 1 and 3.9°C in lasing chamber and 1 and 2°C in downstream cham-ber. 15x5 and 6x4 mm charred thrombi formed at catheter tip respectively, regardless of anticoagulant used. In conclusion: very significant amounts of gas, tissue debris and charred thrombi were formed during continuous lasing process to the extent that modifications in laser techniques and application must be devel-oped before laser therapy can be applied in clinical settings.

HEAD GROWTH IN INFANTS WITH CONGENITAL HEART

HEAD GROWTH IN INFANTS WITH CONGENITAL HEART DISEASE (CHD). <u>William Berman, Jr., Steven</u> 84 <u>M. Yabek, Raymond R. Fripp, and Rochelle</u> <u>Burstein.</u> UNM School of Medicine, UNM Hospital, Dept Ped, Albuquerque, NM We studied patterns of head and somatic growth (height-ht, weight-wt and head circumference-OFC) in 30 infants with CHD. Infants referred in the first 3 mos of life for CHD requiring chronic medical or sur-gical therapy were enrolled if they had no recognize-able syndrome, intrauterine infection or chromosomal anomaly. Measurements of ht, wt, OFC and were made at 3,6,12,15, and 24 mo of age. 8 initially were cyan-otic, 3 have died, and 22 have undergone surgery at a mean age of 15 mos. As a group, the infants developed normally but grew poorly, with wt (20th percentile-%-at 18 months) affected more than OFC and ht (30th % at 18 months). 11 of the 30 showed no growth failure; 2 of those 11 were cyanotic, 1 has died and 6 are post-18 months). 11 of the 30 showed no growth failure; 2 of those 11 were cyanotic, 1 has died and 6 are post-op. Of the 19 with growth failure, 10 were cyanotic, 2 have died and 16 are post-op. Therapy made no im-pact on growth patterns of 12 of the 19; but 7 of the growth failure group exhibited catch-up growth (wt> ht>OFC) following surgery at a mean age of 8 months. Mean OFC increased from the 10th to the 25th % follow-ing surgery, even though OFC fell below the 5th % in 4 subjects. Catch-up head growth can accompany catch-up growth in ht and wt following successful surgery in some children with CHD; in others, normal or delayed growth patterns are unaffected by therapy.