

TUESDAY, MARCH 17, 1981

PM

PEDIATRIC CARDIOLOGY—CLINICAL STUDIES

4:00-5:30

COLLABORATIVE STUDY OF BLADE ATRIAL SEPTOSTOMY

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During the past 3 years, 5 institutions have collaborated in an evaluation of the efficacy of blade atrial septostomy (BS). Forty-three patients underwent BS, including 25 with transposition of the great arteries (TGA), 10 with mitral atresia complex (MAC), 3 with tricuspid atresia (TAT) and 5 with miscellaneous anomalies. Ages ranged from 1 day to 12 years. Twenty-nine (67%) were under 6 months of age, 6 between 7 and 12 months and 8 were older than 1 year at the time of BS. Clinical improvement occurred in 20 of 25 (80%) patients with TGA. In 2 patients a large atrial septal defect (ASD) was created by the procedure but adequate mixing did not occur and early Mustard operation was performed. Three other patients had borderline size ASD prior to BS and the blade did not engage the interatrial septum (IAS). Seven of 10 patients with MAC had a good result with BS. In 2 patients in this group the LA was too small to permit extension of the blade. Three patients with TAT and the 5 patients in the miscellaneous group had an excellent result. Immediate improvement occurred in 81% of the 43 cases. Four patients had intact IAS and BS was successfully performed by transseptal technique. Among 17 patients who have undergone follow-up evaluation of interatrial opening (IAO) re-stenosis of the IAS was observed in 3 patients 10 to 22 months after the BS. There was a single mortality, a result of laceration of the left atrial wall. Minor complications were observed in 3 patients. BS is an effective palliative procedure, even with thickened IAS or with an intact IAS.

HEMODYNAMIC EFFECTS OF HYDRALAZINE IN INFANTS WITH LARGE VENTRICULAR SEPTAL DEFECT.

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To evaluate the effects of acute afterload reduction, Hydralazine (HZ, .2 mg/kg) was administered at cardiac catheterization to 6 infants with large ventricular septal defect (VSD). Age ranged from 2.5 to 11 (mean 5.3) months. Prior to HZ and 5, 15, 25, and 35 minutes after HZ the following were measured: Ao, pulmonary capillary wedge (PcW), PA, RA, SVC pressures and saturations, heart rate (HR), and oxygen consumption (VO2). Hemodynamic effects were noted 5 minutes post-HZ but were most pronounced 35 minutes post-HZ. Pre-HZ baseline data were therefore compared to 35 minute post-HZ values. Pulmonary flow (Qp) did not change while systemic flow (Qs) increased significantly ( $4.6 \pm 0.3$  to  $6.6 \pm 0.5$  L/min/m<sup>2</sup>, p<.01, mean  $\pm$  SEM). The Qp/Qs ratio decreased ( $3.5 \pm 0.4$  to  $2.3 \pm 0.3$ , p<.01) as did the absolute left to right shunt ( $11.1 \pm 1.5$  to  $8.2 \pm 1.4$  L/min/m<sup>2</sup>, p<.05). As expected HZ caused a significant decrease in systemic resistance (Rs,  $13.5 \pm 0.7$  to  $9.6 \pm 0.8$  units, p<.001). Pulmonary resistance, PcW pressures, HR, and VO2 did not change following HZ. A small but significant decrease in RA pressure ( $4.2 \pm 0.7$  to  $2.3 \pm 0.7$  mmHg, p<.05) was observed. In conclusion, HZ caused a significant increase in Qs, and a significant decrease in both Qp/Qs ratio and absolute left to right shunt in 6 infants with large VSD. These effects appear to be related to the decrease in Rs which occurred with HZ. This is in contrast to our previous work documenting hemodynamic deterioration with nitroprusside in a similar group of infants with VSD. Thus, HZ may be beneficial in the management of infants with large VSD.

SAFETY AND EFFICACY OF SHORT AND LONG TERM VERAPAMIL THERAPY IN CHILDREN WITH TACHYCARDIA

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Seven children aged 3 months to 11 years have been treated with Verapamil (V) because of resistant, symptomatic supraventricular tachycardia (SVT), or because of complications of conventional therapy. Two of 3 patients with congenital heart disease were post-operative. The others had no gross cardiac anomalies. Three patients had SVT with WPW syndrome, 3 had ectopic SVT and 1 had atrial flutter (AF). Intravenous V was given in a dose of 0.15 to 0.25 mg/kg in 6 patients, 4 of whom converted successfully. One patient was placed on oral V only. Treatment failed in the patient with AF and one patient with WPW and a wide QRS tachycardia. Five patients were placed on continuing oral therapy. One patient died of post-operative complications while on V. Four patients have remained on oral V for 1 to 4 years at this time. The dose is 80 mg., 6 or 8 hourly depending upon response. Two of these have ectopic SVT which recurs when therapy is stopped. In the 2 with WPW and SVT, one patient has had no recurrences and in the other attacks have decreased from 1-2/week to 1-2/month. The duration of these attacks has also diminished. There have been no side effects of chronic drug administration. None of the 6 patients who received intravenous V showed any untoward effects during administration. Our experience indicates that V which so far has not been used extensively in children is an effective and safe anti-arrhythmic drug capable of controlling both ectopic and re-entrant SVT when conventional methods fail. The presence of underlying congenital heart disease should not be contraindication to its use.

CLINICAL AND ELECTROPHYSIOLOGIC PREDICTION OF NEED FOR PACEMAKER INSERTION IN CHILDREN WITH CONGENITAL COMPLETE ATRIOVENTRICULAR BLOCK

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The criteria for pacemaker insertion (PM) in patients with congenital complete AV block (CCAVB) have not been defined. In an attempt to define such criteria we evaluated 22 children with CCAVB, ages 1 day-14y (median 7y) with surface ECG, treadmill exercise testing (TM) noting change in resting heart rate (HR) and prevalence of ventricular ectopy (PVC) and intracardiac electrophysiologic studies (EPS) including identification of site of block, corrected pacemaker recovery time (CPRT), and His to ventricle interval (HV). Patients were followed 1-19y (median 6y), noting age of onset of any syncopal episodes (SX). Associated congenital heart defects (CHD) were found in 5/22 children. Site of block was localized to the AV node (AVN) in 17/22, His Bundle (HB) in 3/22, below HB (BHB) in 1/22, and was unknown in 1/22 patients. Data are as follows:

No.	BLOCK SITE			HR	CHD	SX	EPS CPRT-msec <2000>2000	No.	%ΔHR (mean)	PVC	
	AVN	HB	BHB								
PM 10	8	1	1	45	2/10	6/10	-	5/9	5/10	100	2/5
NO											
PM 11	9	2	-	60	3/11	1/11	2/9	2/9	6/11	130	1/6

The HV was normal in 20/20 patients. Evaluation of the 7 SX patients revealed AVN block in 6/7, 3 of whom had SX at 2y of age. The HR in all SX patients was <52BPM whereas no patient with HR>52BPM experienced syncope (p<0.01). CPRT estimation was performed in 4/7 SX patients; all were >2000msec. However, 3 asymptomatic patients also had CPRT>2000msec. The %Δ in HR during TM was not significantly different between sites of block. We conclude: 1. Localization of CCAVB to the AVN does not mitigate against syncope; 2. The best single predictor of SX either clinically or during EPS was a low HR; 3. EPS may be of little benefit as neither site of block nor CPRT are valid predictors of SX; 4. TM is of little benefit in predicting site of block or SX.