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EARLY DETECTION OF CONDUCTION SYSTEM ABNORMALITIES (CSA) AFTER MUSTARD'S OPERATION FOR TRANSPOSITION*

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The association of symptomatic and fatal dysrhythmias after Mustard's operation requires early detection and characterization of CSA to identify susceptible children and suggest surgical modifications. We performed bedside electrophysiologic studies (EPS) on 21 infants with single transposition on days 1, 3 and 8 following Mustard repair using epicardial atrial and ventricular temporary pacing wires. Pre-operative studies were performed in 12 subjects. Median age at study was 8 mos (range 3-15 mos); 11 of 21 had prior Bialock-Hanlon septectomy (BHS). Sinus node dysfunction (SND) was documented in 2/11 pre-operatively (both had BHS), and 15/21 postoperatively; 9/17 (6 with BHS) had SND persisting to day 8. AV node dysfunction (AVND) developed postoperatively in 4/21. Changes in retrograde VA conduction were either transient (7/21) or persistent (14/21). 4 subjects had inducible supraventricular tachydysrhythmias (STD) with three subsequently developing spontaneous STD requiring chronic therapy. No child without inducible STD has yet had spontaneous STD. We conclude that early bedside EPS will document CSA and identify those at risk for later symptomatic dysrhythmias. SND is prevalent in this series, and AVND is surprisingly common. The finding of inducible STD appears to be particularly predictive of subsequent course.

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RESPONSE TO EXERCISE AFTER FONTAN PROCEDURE. Geordie

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Previous studies demonstrating maldistribution of pulmonary blood flow in patients who had undergone Fontan procedures (FP) predict high physiologic dead space during exercise. We used non-invasive exercise testing to assess gas exchange in 5 patients age 10-19, compared to 11 age and sex matched controls. Oxygen saturation was measured by ear oximetry at rest and after exercise. The ventilatory equivalents for oxygen ($\dot{V}_E/\dot{V}O_2$) and carbon dioxide ($\dot{V}_E/\dot{V}CO_2$) were measured during progressive exercise and mixed expired pCO_2 (P_{eCO_2}) and end-tidal pCO_2 (P_{tCO_2}) were measured during steady state exercise on a cycle ergometer. We obtained the following results:

	FP	Controls	Significance
$\dot{V}_E/\dot{V}O_2$	41 ± 11	24 ± 2	$p < 0.001$
$\dot{V}_E/\dot{V}CO_2$	47 ± 14	27 ± 2	$p < 0.001$
P_{eCO_2} , torr	17.8 ± 2.5	26.9 ± 2.8	$p < 0.001$
P_{tCO_2} , torr	28.5 ± 5.2	39.1 ± 2.0	$p < 0.001$

In addition, the patients had a mean oxygen saturation of $89 \pm 6\%$ at rest and desaturated further during exercise ($81 \pm 11, p < 0.05$). These data show high ventilation for O_2 consumption and CO_2 production, low expired CO_2 concentrations and oxygen desaturation during exercise. The results strongly indicate elevated physiologic dead space and ventilation perfusion mismatch consistent with maldistribution of pulmonary blood flow.

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THE EFFECT OF RIGHT TO LEFT INTRACARDIAC SHUNTING ON

ARTERIAL LIDOCAINE LEVELS IN A CANINE MODEL

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Because lidocaine uptake occurs in the lung, a reduction in effective pulmonary blood flow (Qep) would be expected to raise serum concentrations. To test this hypothesis, a right to left atrial shunt (40-50% of the basal cardiac output) was developed in each of 7 adult mongrel dogs by connecting cannulae inserted into the right and left atrial appendages to a Sarns rotary pump. Eight additional dogs served as controls. Following a 4 mg/kg intravenous bolus injection of lidocaine, central venous and aortic pressures, cardiac output, and an arterial blood sample were obtained at 0, 3, 5, 8, 10, 15, 20, 30, 40, 50, and 60 minutes. Serum lidocaine concentrations (L), were determined using a homogeneous enzyme assay (EMIT). Cardiac output, pressures, and acid-base status were not significantly different for the 2 groups. Lidocaine concentrations are tabulated.

L (ug/ml)	0"	3"	5"	8"	10"	15"	20"	30"	40"	50"	60"
Control (n=8)	0	4.5	3.4	2.4	2.2	1.5	1.3	1.1	1.0	0.9	0.8
Shunt (n=7)	0	5.9	4.3	3.0	2.6	2.0	1.8	1.4	1.3	1.2	1.1
% increase		31	26	25	18	33	38	27	30	33	38
p		.03	.11	.05	.24	.07	.14	.13	.08	.04	.005

These results indicate that with an acute 40-50% reduction in Qep, arterial lidocaine levels are elevated from 18-38%. Therefore, patients with right to left intracardiac shunting may be at increased risk for developing lidocaine toxicity, and an initial dosage reduction in those patients is appropriate.

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THE HEMODYNAMIC EFFECTS OF PROSTAGLANDIN E₁ IN COMPLETE TRANSPOSITION OF THE GREAT ARTERIES

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Quantitative assessment of hemodynamics in neonates with complete transposition (TGA) was performed using two-dimensional (2D) directed pulsed Doppler echocardiography. Both the aortic valve (RV) and pulmonary valve (LV) flow velocities were obtained in 5 neonates before and during a 1 hour infusion of prostaglandin E₁ (PGE) at 0.1 mcg/kg/min following balloon atrial septostomy. Ductus arteriosus (DA) size by 2D imaging increased in all and pulmonary to aortic shunting was excluded by Doppler sampling in DA. Stroke volume changes were estimated by the product of the Doppler time-velocity integral (TVI) and heart rate (cm/min). Doppler systolic time intervals (PEP/ET) were calculated.

n=8	HR	TVI x HR		PEP/ET		
		LV	RV	RV x HR	LV	
Off PGE	135	2122	1125	1.89	0.30	0.52
On PGE	148	1767	1466	1.21	0.35	0.42
p value	<0.05	<0.005	<0.005	<0.005	0.1	0.1

LV stroke volume decreased while RV stroke volume increased. The ratio of LV to RV stroke volume consistently decreased. An increase in LV and decrease in RV systolic time intervals did not reach a significant level.

Conclusions: PGE in neonates with TGA results in decreased LV stroke volume. Enhanced intraatrial mixing in TGA with PGE may be mediated, in part, by decreased LV filling.

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PULSED DOPPLER DIAGNOSIS OF ATRIOVENTRICULAR VALVE INSUFFICIENCY IN UTERO

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Atrioventricular valve insufficiency (AVVI) has been suggested as a cause for nonimmune hydrops in utero. Pulsed 2-D-directed Doppler echocardiography was utilized for the detection of AVVI in 41 fetuses ranging in gestational age from 16-39 weeks. AVVI was diagnosed by a systolic velocity in either atria of greater than 1 meter per second.

AVVI was present in 5/41 fetuses (12%) and confirmed after birth in 3/3. Of 22 fetuses at risk for congenital heart disease but found to be normal, one had AVVI (5%). Of 11 patients with fetal dysrhythmia, 2/2 with complete heart block (1 normal and 1 with AV canal) had intermittent AVVI, and 7 with premature atrial contractions and 2 with atrial tachycardia had none. Of 6 patients with abnormal cardiac findings, 2 had AVVI (1 with endocardial fibroelastosis and bilateral AVVI, and 1 with cerebral arteriovenous malformation) while there was no AVVI in 4 patients with VSD including 1 with hydrops. Two patients with a normal heart and hydrops had no AVVI.

Conclusions: (1) AVVI occurs in utero and may be diagnosed by pulsed Doppler echocardiography. (2) AVVI may be present in normals, in fetuses with congenital heart block with or without heart disease, and is not present in all forms of nonimmune hydrops fetalis.

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CALCIUM BLOCKERS AFTER B-BLOCKADE IN CONSCIOUS PUPPIES William Jackson, S. Clapp, B. Perry, Spons. by A. Gruskin, Wayne State University, Detroit, MI.

Following reports of cardiac arrest, acute conversion therapy with verapamil is contraindicated in children on chronic propranolol for recurrent SVT. The exact hemodynamic changes produced by verapamil after β -blockade have not been shown in children or awake, instrumented immature animals. We proposed to document these alterations and compare them to those produced by a similarly effective calcium-blocking agent, diltiazem, potentially of use in pediatric patients. Fourteen puppies were chronically instrumented for: aortic flow(CO); regional LV shortening(SF); pressures--carotid(MBP), pulmonary(PAP), and left atrial(LAP); heart rate(HR) and P-R interval. Measurements during infusions of each agent at 5-125 μ g/kg/min were repeated after a propranolol bolus (0.7-0.9mg/kg) lowered HR 10%, simulating P therapy. Results at maximal infusion (mean \pm S.D.; * p < 0.1):

	C.O.	MRP	H.R.	P-R
	mmHg	beats/min	sec.	
Control	100	96 \pm 6	144 \pm 18	.08
V	69 \pm 12	85 \pm 6	151 \pm 9	*.12
V + P	* 24 \pm 10	* 51 \pm 6	* 79 \pm 15	*.18
Control	100	94 \pm 10	167 \pm 13	.08
D	93 \pm 6	88 \pm 12	158 \pm 14	.11
D + P	66 \pm 8	74 \pm 10	*127 \pm 18	*.13

Verapamil's effects before β -blockade were similar to those documented in other studies. Diltiazem produced significantly less decrease in cardiac output ($p < 0.1$) both before and after β -blockade. Throughout, LAP rose ($p < 0.1$) when CO decreased significantly, while PAP did not change. SF did not show significant changes. 7/14 animals progressed to severe bradycardia after V+P responsive only to Ca⁺⁺ gluconate infusion with immediate improvement in CO ($p < 0.1$). We have documented verapamil's profound depressant effect in the presence of β -blockade. We also conclude that diltiazem, with similar antiarrhythmic properties, produces less negative inotropic effect than verapamil in unsedated immature animals.