APPARENT PRE-EXCITATION IN TRICUSPID ATRESIA
Thomas M. Zellers, David J. Driscoll, Co-burn J. Porter, Mayo Clinic, Rochester, Minnesota.

146 It was our clinical impression that ECG evidence of pre-excitation (PE) is found more commonly in pts with tricuspid atresia (TA) than previously reported (0.29-1.3%). We attempted to determine a) the incidence of PE by surface ECG in pts with TA and b) if this represents the presence of a true accessory pathway.

Surface ECGs from 183 consecutive pts with TA evaluated at the Mayo Clinic between 1981-1986 were reviewed by 3 pediatric cardiologists for evidence of PE. The patients' ages ranged from 4/12 to 21 years. The M:F ratio was 5:4. PE was diagnosed if the PR interval (PR) was $\leq$.10 seconds, the QRS was $\geq$.10 seconds and a delta wave was present. Of the 183 pts, 22 (12%) had PR $\leq$.10 seconds and 9 pts (5%; 6F, 3M) fulfilled criteria for PE, 5 of which had a history of SVT. Four of the 5 pts had invasive electrophysiologic studies (EPS) performed. Only 1 pt had an accessory pathway, but 2 had enhanced AV node conduction and 1 had normal AV node conduction.

We conclude: 1) surface ECG evidence for PE in pts with TA is more common (5%) than previously reported. 2) Apparent PE can be due to an accessory pathway or enhanced AV node conduction (in combination with LVH). 3) Patients with TA being considered for intracardiac repair should have their ECG reviewed carefully for apparent PE; if found, especially in the presence of SVT, they should undergo EPS to determine the presence and location of an accessory pathway for surgical ablation.

EFFICACY OF ECHOCARDIOGRAPHY IN DIAGNOSING LSVC
Thomas M. Zellers, Donald J. Hagler, Paul R. Julsrud, Mayo Clinic, Rochester, Minnesota.

147 The diagnosis of a left superior vena cava (LSVC) is important in planning the repair of a congenital heart defect as it may be the source of a sizeable right-to-left shunt. The medical record of 65 consecutive pts with a LSVC evaluated at the Mayo Clinic between 1980-1986 were reviewed. All pts had cardiac catheterization (CATH) and two-dimensional echocardiography (2D-ECHO) performed. The M:F ratio was 5:4; the mean age was 8.3 years. Bilateral SVC were present in 59 (91%) and a unilateral SVC was present in 6 (9%), 3 with absent RSVC syndrome. Eighty-one percent of the LSVC entered the coronary sinus CS (11% entered an unroofed CS), 8% entered a pulmonary venous atrium, and 11% entered a common atrium. CATH was used successfully to identify a LSVC in 100% of pts. 2D-ECHO, utilizing contrast and color flow in some pts, was used successfully in 86% (grp1; p=0.008), and unsuccessfully in 14% (grp2). There was no significant difference between the two groups with regard to age, gender, diagnosis, type of SVC or place of drainage. In grp1, a dilated CS was identified in 61%, contrast was used to identify a LSVC in 23%, and 25% were identified as a venous structure entering an atrium. When present the CS was of normal size in grp2 pts. Of the 9 pts, 7 had contrast used and 3 had color flow performed. We conclude: 1) Using 2D-ECHO, a LSVC was successfully identified in only 86% of our pts. 2) The failure to identify 14% could not be attributed to patient age, gender, type of congenital heart disease, type of LSVC or place of drainage. 3) If a suspected LSVC is not identified by 2D-ECHO, other non-invasive means, such as MRI, should be employed.

ACETALDEHYDE INCREASES FREE IRON IN THE HEART: POSSIBLE MECHANISM IN ALCOHOL CARDIOMYOPATHY.
●148 Shu-Lun Zhang, Daniela Lax, Ying Li, Lee Williams, Nancy A. Staley, George R. Noren, Stanley Einzig, University of Minnesota, Dept. of Ped., Minneapolis.

Chronic alcohol(ETOH) exposure in man and turkeys (TK) leads to structural and functional changes in the heart; similar cardiac pathology has been found in some infants with the fetal alcohol syndrome. The mechanism of ETOH induced cardiac damage is unknown but may, in part, involve lipid peroxidation (LPO) of heart membranes. Acetaldehyde(ALD), the first oxidation product of ETOH, could be the main mediator of ETOH injury. The direct effect of ALD on heart tissue LPO has not been reported and this, therefore, was investigated in our study. Normal TK and Sprague Dawley rats were sacrificed by decapitation. Left ventricular(LV) homogenates were analyzed for LPO using the thiobarbituric acid reaction(malondialdehyde(MDA) production; mean±SE in nmolMDA/100mg prot.) before and after in vitro addition of 1%ALD. LPO was increased in rat LV(n=5) following the addition of ALD (250±9vs486±24,p<0.02). A similar increase was obtained using TK LV(n=5, see below), suggesting that the increase was not species specific. The cause of ALD induced increased LPO is unknown; one possible mechanism could involve delocalization of tissue iron (Fe) leading to enhanced LPO via an Fe catalyzed(e.g.Haber-Weiss) reaction. We found that in vitro addition of 1%ALD also resulted in a significant 2-fold increase in free Fe levels of TK(n=6) and rat(n=5)LV(TK:48±12 to 105±6;rat:99±5 to 174±10nmolFe/100mg prot.). The addition of Fe chelators deferoxamine(DEF) and dihydrobenzoic acid(DBA) at 0.02M protected against ALD induced increase in TK LV LPO(CON:321±66;1%ALD:567±96;DEF:196±24;DBA:343±36,p<0.03vs1%ALD). Thus, ALD increases free Fe levels and LPO in TK and rat heart homogenates. Should similar changes occur in vivo, these results suggest a mechanism of ETOH induced cardiomyopathy and a possible mode of treatment.

CRITICAL CARE

SIMULTANEOUS CHEST COMPRESSION AND VENTILATION DOES NOT ENHANCE CEREBRAL AND MYOCARDIAL PERFUSION IN AN INFANT MODEL OF CARDIOPULMONARY RESUSCITATION,
▲149 Ivor D. Berkowitz, Teerachai Chantarojanasiri, Raymond C. Koehler, Charles L. Schleien, J. Michael Dean, John R. Michael, Mark C. Rogers, Richard J. Traystman
Johns Hopkins Medical Institutions, Department of Anesthesiology/Critical Care Medicine, Baltimore, Maryland 21205

We evaluated whether simultaneous compression ventilation CPR (SCV-CPR) with high airway pressure improved cerebral (CBF) and myocardial (MBF) blood flow compared to conventional CPR (CON-CPR) in an infant model of CPR. CPR was performed on pentobarbital anesthetized piglets (3.5 - 6 kg) with epinephrine infusion. CON-CPR was performed (n=8), at 100 compressions/min, 60% duty cycle, and a 1:5 breath to compression ratio and 25 mmHg peak airway pressure. SCV-CPR was performed (n=8) at 60 compressions/min, duty cycle 60%, with 60 mmHg peak airway pressure. Sternal displacement was 20% in each group. There was no significant difference between CPR groups as regards systolic, mean, diastolic aortic and right atrial pressures, intracranial, cerebral perfusion (CPP), or myocardial perfusion (MPP) pressures, CBF and MBF, (microspheres). There was a progressive decrease in aortic, CPP and MPP in both groups with time. CBF ($ml.min^{-1}.100\ gm^{-1}$) decreased from 15±3 at 5 min to 5±1 at 50 min of (CON-CPR) and from 13±2 to 5±2 of SCV-CPR. MBF decreased from 38±7 at 5 min to 1±1 at 50 min and from 40±7 to 9±4 of SCV-CPR. This study demonstrates that SCV-CPR with peak airway pressures of 60 mmHg neither improves the perfusion pressure over those generated by CON-CPR nor prevents the decrease of CBF and MBF with prolonged CPR in this infant model. Supported by NS20020.

CEREBRAL AND MYOCARDIAL BLOOD FLOW RESPONSE TO INCREASING DOSES OF EPINEPHRINE DURING CARDIOPULMONARY RESUSCITATION IN INFANT SWINE
●150 Ivor D. Berkowitz, Raymond C. Koehler, Charles L. Schleien, Kenneth Kubos, J. Michael Dean, John R. Michael, Mark C. Rogers, Richard J. Traystman
Johns Hopkins Medical Institutions, Department of Anesthesiology/Critical Care Medicine, Baltimore, Maryland 21205

We measured cerebral (CBF) and myocardial (MBF) blood flow during cardiopulmonary resuscitation (CPR) in two week old swine to determine whether 10 fold increments of infused epinephrine (EPI) would progressively enhance CBF and MBF. CPR was performed for 50 min on pentobarbital anesthetized, 3.5 - 6 kg swine with a pneumatic compressor at 100 compressions/min, 60% duty cycle and 1:5 breath to compression ratio. In groups 1, 2 and 3 (n=4), EPI was administered first as a bolus (0, 1, 10 ug/kg, respectively) and then continuously (0, 1, 10 ug/kg/min respectively). Plasma EPI (ng/ml) (HPLC) rose from pre-arrest levels of <1 to 38±25, and 92±17, and 1627±231 (±SE) at 10 min in groups 1, 2, and 3 respectively. At 10 min, CBF (ml/min/100gm) (microspheres) was 17±10 (±SE), 32±8 and 34±4 and cerebral O_2 uptake ($CMRO_2$) (ml/min/100gm) was 1.2±.7, 2.0±.2 and 2.1±.4 in groups 1, 2 and 3 respectively. MBF was 3±2, 20±13, and 48±7 in groups 1, 2 and 3 respectively (p <.05 between group 3 and 2). CBF, MBF, their respective perfusion pressures and $CMRO_2$ progressively decreased with prolonged CPR. We conclude that in this infant CPR model, infusing 1 ug/kg/min EPI improves MBF, CBF and $CMRO_2$. Increasing the dose to 10 ug/kg/min further elevates MBF, but has less incremental effect on CBF and $CMRO_2$. Supported by NS 20020.

EFFECTS OF INTRA-AORTIC BALLOON PUMPING DURING RIGHT VENTRICULAR INFLOW OBSTRUCTION IN THE NEWBORN LAMB.
151 Daniel Bernstein, Robert Shaddy, Michael Tyndall, Abraham M. Rudolph University of California, San Francisco. CVRI & Department of Pediatrics, San Francisco, CA.

The intra-aortic balloon pump (IABP), while effective in adults, could be limited in effectiveness in infants because of differences in coronary dynamics, central vascular impedence, heart rate, and the hemodynamic indications for its use. One potential indication is the decreased cardiac output (CO) secondary to right ventricular inflow obstruction (RVIO). In 6 young lambs, catheters were placed in the vena cava, left atrium, coronary sinus, aorta, and pulmonary artery. An IABP was advanced via the femoral artery to the thoracic aorta. RVIO was produced by inflating a Foley catheter in the right atrium, reducing CO by 50%. Studies were performed at rest, rest + IABP, RVIO, and RVIO + IABP. Regional blood flows were measured by the microsphere method. During resting studies, the IABP did not alter CO, heart rate, aortic pressures, systemic or myocardial O2 consumption (VO2) or O2 transport, or regional blood flows. During RVIO, aortic and left atrial pressures decreased, vena caval pressure doubled, and systemic and myocardial VO2 and O2 delivery decreased. Blood flow and O2 delivery to all organs decreased. The IABP had no significant effect on any of these parameters. In conclusion, although the IABP does not interfere with resting hemodynamics or regional blood flows, there is no evidence for its effectiveness in clinical situations where CO is decreased because of RVIO. The utility of the IABP may be affected by the etiology of the reduction in CO, or by the presence of a normal coronary vascular bed.