

223 EVALUATION OF PULSE OXIMETRY IN PEDIATRIC PATIENTS. Hugh G. Welch, William G. Perkins, Jeffery Jennings and Sachchida Sinha. Department of Pediatrics. The University of Tennessee Memorial Research Center and Hospital, Knoxville.

We investigated a new oximeter suitable for pediatric patients (Nellcor) by comparing its values for oxygen saturation with those obtained by manometric analysis of arterial samples drawn simultaneously. Oximeter readings were obtained for patients in the Intensive Care Unit (wt < 1300 g). The arterial blood samples were analyzed with the micro version of the Van Slyke apparatus. The remainder of the sample was then either saturated with oxygen and reanalyzed for oxygen capacity--or saved for measurement of hemoglobin concentration. In the latter case, oxygen capacity was calculated with the binding constant. In our hands, the oxygen saturation values obtained with the oximeter compared favorably with those from manometric analysis at high saturation, but less well at lower saturation. At saturations of >85%, the oximeter values were 92±4% ($\bar{X} \pm SD$) while the manometric values were 90±4%, the difference not being significant ($p > 0.05$). At saturations below 85%, the oximeter values were 84±9%, the manometric values 68±9% ($p < 0.05$). We conclude that pulse oximetry may be a useful adjunct to monitoring pediatric patients, but that some caution must be used, especially at saturation levels lower than about 80%.

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224 SURVIVAL OF CARDIAC ARREST IN RELATION TO LENGTH OF RESUSCITATION AND POOR PATIENT PROGNOSIS. Jean A. Wright, Peter A. Ahmann, Jan Barfield, (Spon. by James F. Schwartz.) Emory University School of Medicine, Department of Pediatrics, Atlanta, GA.

An ongoing prospective study of children sustaining cardiac arrest (CA) was begun to assess outcome and relate this to length of resuscitation and whether there was written documentation of poor prognosis (DPP) prior to the arrest. Patients entered the study at the time of CA (chest compressions). CA data was recorded, and charts reviewed for DPP. Outcome was defined as surviving arrest, (SA) or survival to discharge (STD). Of 43 patients, 12 died immediately, 28 SA, but later died, and 13 STD. The relationship of length of resuscitation and DPP is shown below.

LENGTH OF RESUSCITATION	ALL PATIENTS		SURVIVORS (STD)	
	DPP	NO DPP	DPP	NO DPP
0 - 10 MIN.	8	14	1	9
10 - 20 MIN.	2	4	0	2
20 - 30 MIN.	1	4	0	1
MORE THAN 30 MIN.	1	9	0	0

Patients with DPP are less likely to survive in all categories of arrest duration, and less likely to be resuscitated for longer periods. There are no survivors after 24 months in this study. Of the 31 with no DPP's, 22 died. This suggests that many could have had a poor prognosis, but it was never written. Careful attention to documentation of patient prognosis when known will allow wise decision making by physicians when confronted with CA.

225 THE NATURAL HISTORY OF PEDIATRIC CARDIAC ARREST. Jean A. Wright, Peter A. Ahmann, Virginia Balfour, Barbara Goncalves, (Spon. by James F. Schwartz.) Emory University School of Medicine, Department of Pediatrics, Atlanta.

The natural history of outcome in children sustaining a cardiac arrest (CA) has not been adequately described. The purpose of this ongoing prospective study is to describe the outcome of CA in a pediatric tertiary care hospital and to define the clinical setting around the arrest in order to relate these events to outcome.

Patients entered the study at the time of CA (defined as the need for cardiac compressions). CA data such as blood gases, blood pressure, length of resuscitation, and drugs given, etc., were recorded. Serial coma exams through the first 72 hours post-arrest were performed. Survivors received ongoing neurologic examinations.

TOTAL PATIENT POPULATION (TPP)	43		
SURVIVING THE ARREST (SA)	28	SA-28	43-TPP
SURVIVAL TO DISCHARGE (STD)	13	STD-13	15-DIED
SEVERE NEURO. DEFICIT (SND)	2	SND-2	11-IS
INTACT SURVIVAL (IS)	11		

A data base such as the above is necessary before new CA techniques can be assessed. Presently, there are 11 intact survivors. However, 5 of these survivors are less than 3 months of age, and further re-evaluation will be necessary to determine their eventual outcome. Resuscitative efforts beyond 24 minutes in this study were unsuccessful. Further base line data is required before experimental pharmacologic methods of "brain resuscitation" can be assessed.

226 HEMODYNAMIC EFFECTS OF PROLONGED SEIZURES IN NEONATAL PIGS. Richard S. K. Young, Raymond R. Fripp, John C. Werner, Susan K. Yagel, Glenn McGrath, H Gregg Schuler. (Spon. by Nicholas M. Nelson). The Pennsylvania State University, The Milton S. Hershey Medical Center, Department of Pediatrics, Hershey, PA.

The cardiovascular effects of seizures in the neonate are not well defined. To determine the changes in systemic and pulmonary artery pressure, cardiac output, and left ventricular contractility during seizures, one week old pigs were intubated, paralyzed, mechanically ventilated, and catheterized with a Swan-ganz catheter. Seizures were induced with bicuculline (5 mg/kg/hr continuously i.v.) in 5 animals (6 controls received saline). Early changes consisted of significant systemic and pulmonary arterial hypertension. After 2 hours of seizures, the animals developed progressive systemic hypotension (saline, 93 ± 4 mmHg; bicuculline, 66 ± 8; Mean ± SE; $p < 0.01$; student t test) and decreased cardiac function (cardiac output: saline, 174 ± 27 ml/kg/min; bicuculline, 91 ± 15 ml/kg/min; $p < 0.01$). M-mode echocardiography disclosed a decrease in left ventricular contractility (Ejection Fraction: Saline, 0.70 ± 0.03; bicuculline, 0.49 ± 0.08; $p < 0.001$). Cardiac tissue frozen *in situ* showed significant increase in lactate (Saline, 13.3 ± 0.7 mmol/g/dry wt.; bicuculline, 102.8 ± 4.9; $p < 0.01$), and reductions in glucose, triglyceride, and ATP (Saline, 17.1 ± 0.1 mmol/g/dry wt.; bicuculline, 11.5 ± 0.3; $p < 0.01$). Prolonged seizures result in severe cardiac dysfunction which may be due to tissue acidosis or catecholamine release.

DEVELOPMENTAL BIOLOGY

227 ADAPTATION OF FETAL PULMONARY BLOOD FLOW TO LOCAL INFUSION OF TOLAZOLINE. Steven H. Abman, Frank J. Accurso, Robert M. Ward, and Randall B. Wilkening. (Spon. by Frederick C. Battaglia), Depts. of Pediatrics, University of Colorado School of Medicine, Denver, CO and MS Hershey Medical Center, Pennsylvania State University, Hershey, PA.

To study the direct effect of tolazoline (T) in a high resistance pulmonary vascular bed, we infused T in nine chronically prepared fetal sheep with and without calcium channel blockade. An electromagnetic flow transducer placed around the left pulmonary artery (LPA) measured blood flow to the left lung (Q_L). Catheters placed in the left atrium (LA), main pulmonary artery, aorta and amniotic cavity measured pressure. An infusion catheter was placed in the LPA. After obtaining baseline measurements for 30 minutes, T (4.5 mg/hour) was continuously infused into the LPA for 90 minutes. After an increase over the first 30 minutes, Q_L decreased toward the control value. Throughout the infusion, the concentration of T steadily increased in the LA (peak=0.53 ug/ml). When verapamil (V) was infused with T, the increase in Q_L was sustained throughout the 90 minute infusion period.

	Control	30 minutes	90 minutes	
T	60 ± 7	101 ± 13 #	65 ± 8 #	#p 0.05
T + V	61 ± 11	108 ± 12 ##	116 ± 9 ##	##NS

There were no significant changes in heart rate or pressures. V alone had no effect on Q_L . We conclude that T dilates the pulmonary vascular bed in the fetal sheep, but adaptation occurs despite increasing drug levels. The adaptive response to T may be a calcium dependent mechanism.

228 LONGITUDINAL TRENDS IN LEFT VENTRICULAR CARDIAC OUTPUT IN HEALTHY INFANTS OVER THE FIRST YEAR OF LIFE. D.C. Alverson, M. Aldrich, P. Angelus, C. Backstrom, S. Werner. University of New Mexico, School of Medicine, Department of Pediatrics, Albuquerque, New Mexico 87131

We studied left ventricular (LV) cardiac output longitudinally in 16 healthy infants from birth through the first year of life using noninvasive pulsed Doppler ultrasound technique. Ascending aorta blood flow velocity (V_{Ao}) was measured with a 5 MHz pulsed Doppler velocimeter. Ascending aortic cross sectional area (A_{Ao}) was determined using M-mode echography. Ascending aortic blood flow was calculated as $Q_{Ao} = V_{Ao} \times A_{Ao}$ and was corrected to body weight (BW) in kilograms or body surface area (BSA) in meters squared. Peak systolic aortic flow velocity averaged 53±2 cm/sec (±SD) over the one year time period. Mean ascending aortic flow velocity (V_{Ao}) remained relatively constant over the first year, averaging 20.5±.8cm/sec. Changes in A_{Ao} over time correlated with changes in BW and BSA ($r = .99$). Therefore LV output corrected for body weight or BSA remained relatively constant throughout the first year, ranging from 178-227ml/min/kg or 3.1 - 3.7 L_g/min/m² and averaging 204±15ml/min/kg or 3.5±.2 L_g/min/m². A similar strong correlation was seen between serial changes in LV stroke volume and changes in BW or BSA ($r = .99$). Unlike some other animal species, LV output with respect to BW or BSA changes little in humans over the first year of life.