Anesthesiology
V 75, No 3A, Sep 1991

A965

TITLE: METABOLIC EFFECTS OF CORRECTIVE OPERATIONS IN INFANTS AND CHILDREN WITH CONGENITAL HEART DEFECTS

AUTHORS: K. Puhakka, MD, J. Räsänen, MD, M. Leijala, MD, K. Peltola, MD

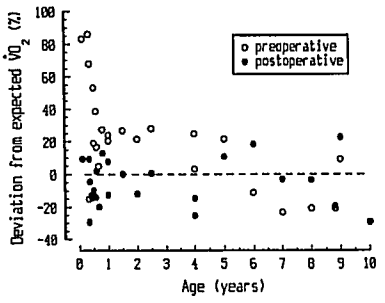
AFFILIATION: Departments of Anesthesiology, Oulu University, Finland and University of South Florida, Tampa, Florida; Departments of Anesthesiology and Surgery, University Children's Hospital, Helsinki, Finland

Changes in cardiopulmonary function secondary to repair of cardiac malformations and the stress of the operation may markedly affect total body oxygen requirements in the immediate postoperative period. To quantify the effect of surgical correction of congenital heart defects on metabolic rate, we measured oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) in infants and in children up to ten years of age immediately before and after the operation.

The study protocol was approved by the Helsinki University Children's Hospital Review Board, and informed consent was obtained from each patient's family prior to the study. Heart rate, respiratory rate, body temperature, oxyhemoglobin saturation, hemoglobin concentration, $\dot{V}O_2$, and $\dot{V}CO_2$ were measured on the morning of the operation after premedication, and the measurements were repeated in the Intensive Care Unit as soon as the patient was able to breathe room air spontaneously. A pediatric indirect calorimeter (Deltatrac, Datex Inc., Helsinki, Finland) equipped with a canopy for collecting expiratory gases was used for measuring $\dot{V}O_2$ and $\dot{V}CO_2$. The measured values of $\dot{V}O_2$ and $\dot{V}CO_2$ were compared with values expected for normal children of the same weight using regression equations published by Lindahl.[1] The data were analyzed with repeated-measures analysis of variance and linear regression.

Nine of the 25 patients studied had a cyanotic cardiac malformation, 15 had a left-to-right intracardiac shunt, and one had isolated aortic stenosis. Surgical correction of the cardiac defect effected a 15±22% decrease in $\dot{V}O_2$ ($p<0.001$). The change in $\dot{V}O_2$ correlated best with the patient's age ($r=-0.72$). The largest relative decline in $\dot{V}O_2$, up to 43% was observed in infants, who also frequently had preoperative oxygen consumption values well above those expected (Figure). A large decrease in $\dot{V}O_2$ was observed in patients with heart failure prior to the operation (23±18%) while a slight increase in average $\dot{V}O_2$ was seen in those with normal preoperative cardiac performance (-4±24%; $p<0.05$). The observed changes in $\dot{V}CO_2$ paralleled those of $\dot{V}O_2$.

Correction of cardiac defects has previously been shown to have an unloading effect on the cardiopulmonary system with decreased resting energy expenditure and improved excercise tolerance. The results of this study reveal an immediate decrease in resting $\dot{V}O_2$ resulting from the operation, particularly in infants and in patients with preoperative congestive heart failure.



1. Br J Anaesth 62:70-76,1989

Supported by grants from the Alma and K.A. Snellman Foundation, Oulu, Finland and The Finnish Academy of Science.

A966

Title: EFFECT OF A BRAIN REPERFUSION STRATEGY ON CEREBRAL BLOOD FLOW AND METABOLISM AFTER TOTAL CIRCULATORY ARREST IN CHILDREN

Authors: WJ Greeley, MD, J Mault, MD, B Baldwin, CRNA, J Stockwell, MD, JG Reves, MD, RM Ungerleider, MD

Affiliation: Departments of Anesthesiology, Surgery and Pediatrics, Duke Heart Center, Durham, NC 27710

INTRODUCTION: Recent studies examing cerebral blood flow (CBF) and cerebral metabolism ($CMRO_2$) following periods of total circulatory arrest (TCA) used during the repair of congenital heart defects (CHD) in neonates and infants have demonstrated cerebral hypoperfusion and reduced metabolism after TCA. [1,2] This study was designed to examine the effect on CBF, $CMRO_2$, oxygen extraction ($A\text{-}VO_2$) and oxygen delivery (DO_2) by increasing pump flow rate (PFR) during rewarming on cardiopulmonary bypass (CPB) after TCA.

METHODS: CBF, $CMRO_2$, $A\text{-}VO_2$ and DO_2 were measured in 41 infants undergoing repair of CHD using CPB. Standard anesthetic management was utilized. [1,2]. All patients were cooled to hypothermic conditions. Blood gas management during bypass maintained a pH of 7.35-7.40 and a PCO_2 of 35-40 mmHg, uncorrected for temperature.

Patients were grouped based on CPB rewarming conditions after TCA: 1) Control group (I), reperfusion after TCA at 100 ml/kg/min; 2) High flow group (II), reperfusion at 150 - 200 ml/kg/min. CBF was measured using xenon clearance methodology. [1,2] Cerebral venous oxygen content was directly measured by using a catheter placed in the right jugular venous bulb, and $CMRO_2$, $A\text{-}VO_2$ and DO_2 were determined before CPB (stage A); during stable hypothermic CPB, pre-TCA (stage B); at stable hypothermic CPB immediately after TCA (stage C); rewarmed on CPB (stage D); and after CPB (stage E). Intragroup data were analyzed using ANOVA for repeated measurements and intergroup data were analyzed using unpaired T-tests, with significance assumed at the $P < 0.05$ level.

RESULTS: There was no difference in $PaCO_2$, mean arterial pressure, temperature and hematocrit between groups during any of the study intervals. Both groups showed a significant decrease in CBF, $CMRO_2$ $A\text{-}VO_2$ and DO_2 during hypothermic bypass conditions at stages B & C compared to pre-bypass levels (A) ($p < 0.05$) (Table). In group I pts, CBF, $CMRO_2$ and DO_2 remained reduced during rewarming after TCA at stage D and even after being weaned from CPB at stage E. In group II pts, however, CBF and DO_2 returned to baseline during rewarming after TCA at stage D; but fell significantly below baseline after being weaned from CPB at stage E ($p < 0.05$). In both groups, CBF and $CMRO_2$ were significantly reduced after being weaned from bypass (stage E). There was a significant difference in CBF, $A\text{-}VO_2$ and DO_2 between the groups during rewarming at (stage D) ($p < 0.05$).

DISCUSSION: These data demonstrate: 1) CBF and $CMRO_2$ do not return to baseline levels after TCA; 2) It is possible to uncouple flow and metabolism during rewarming, favoring flow (supply) however, 3) Increasing PFR during rewarming does alter the degree of injury to cerebral metabolism following TCA. We conclude that cerebral protection during and after TCA is necessary, but cannot be accomplished simply by enchancing reperfusion. Increasing PFR during rewarming on CPB may increase oxygen delivery, but does not improve the abnormally low cerebral metabolism after CPB.

REFERENCES:

1. Greeley, et al: The effects of deep hypothermic cardiopulmonary bypass and total circulatory arrest on cerebral blood flow in infants and children. J. Thorac. Cardiovasc. Surg. 97:737-745, 1989.

2. Greeley WJ, et al: The effects of cardiopulmonary bypass on cerebral blood flow in neonates, infants and children. Circulation 80:209-215,1988.

CONTROL GROUP (I) (N=19)

	A	B	C	D	E
CBF	19.2±8.5	11.1 ± 4.4*	13.1±7.4*	14.4±6.9*	14.6±6.8*
$CMRO_2$	0.91±0.5	0.15±0.1*	0.26±0.19*	0.56±0.3*	0.63±0.4*
AVO_2	5.12±1.2	1.47±1.3*	2.60±1.8*	3.90±2.3	4.49±2.3
DO_2	234±98	103±47*	123±71*	167±74*	180±66*

HIGH FLOW GROUP (II) (N=22)

	A	B	C	D	E
CBF	19.0±9.6	13.6±9.9*	14.4±7.5*	20.5±5.5∞	11.8±5.5*
$CMRO_2$	0.96±0.6	0.18±0.1*	0.23±0.1*	0.60±0.2*	0.59±0.3*
$A\text{-}VO_2$	5.01±1.2	1.70±1.2*	1.81±0.9*	3.03±1.1*∞	4.9±2.0
DO_2	269±112	141±108*	151±87*	322±63∞	151±79*

MEAN VALUES ± S.D.

* $p < 0.05$, Stage B-E vs A (INTRAGROUP);

∞ $p < 0.05$, GRP II vs I (INTERGROUP) A= pre-CPB; B=CPB, cold, pre-TCA; C=CPB, cold, post-TCA D=CPB, warm; E=post-CBP