Title: PULMONARY AND SYSTEMIC HEMODYNAMIC EFFECTS OF HYPERVENTILATION IN INFANTS AFTER REPAIR OF CONGENITAL HEART DISEASE

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Introduction. Hyperventilation is commonly employed to reduce elevated pulmonary vascular resistance (PVRI) in neonates with persistent pulmonary hypertension, but its use is increasingly controversial and its applicability to infants following repair of congenital heart disease has not been defined. Release of vasoactive substances in response to changes in ventilatory pattern or PaCO2 may complicate and further modulate PVRI changes induced by hyperventilation. The purpose of this study was to investigate the hemodynamic effects of hyperventilation and changes in PaCO2 in infants following repair of their congenital heart disease. We also measured transpulmonary gradients of thromboxane B2 after changes in ventilation.

Methods. Infants under 1 year of age who had undergone repair of a ventricular septal defect (including atrioventricular canal defects) on cardiopulmonary bypass were chosen for study during the postoperative period while ventilation was controlled and the patient was still paralyzed with pancuronium and sedated with morphine. Informed consent was obtained from the parents and the study was approved by the institutional committee on clinical investigation. Patients were hemodynamically stable on no inotropic support at the time of the study and had no evidence of a residual left-to-right shunt. All patients had arterial and right and left atrial catheters placed during surgery, along with a pulmonary artery catheter and thermistor for measurement of cardiac index by thermodilution technique. The inspired oxygen was maintained at 40% and stepwise changes in PaCO2 were induced by systematic changes in ventilatory rate to produce PaCO2 values of approximately 20, 30, 40 and 55mmHg, individualized for each patient in order to vary arterial pH between 7.30 and 7.65. After stabilization at each ventilatory rate, an arterial sample was obtained, cardiac index measured and all hemodynamic and airway variables recorded. Blood samples from the pulmonary artery and left atrial catheters were obtained simultaneously for measurement of thromboxane B2.

Results. Among the 17 patients studied (Table 1), all had preoperative evidence of large left-to-right shunts and pulmonary artery hypertension. The mean weight and body surface area were 5.3 ± 0.4kg and 0.29 ± 0.01M respectively. The mean age was 7 ± 1 month. Hyperventilation (PaCO2<45) lowered PVR in 15/17 patients despite increases in mean airway pressure, but more extreme hyperventilation did not produce further pulmonary vasodilation in the group as a whole. Significant increases in pulmonary artery pressure and PVRI occurred at elevated PCO2 in 16/17 patients and approached or exceeded systemic pressure in 3 patients. These changes occurred independently of thromboxane levels. Systemic vascular resistance was unaffected by ventilation whereas cardiac index increased without changes in heart rate when the PaCO2 was elevated.

Conclusions. Despite increases in mean airway pressure, moderate hyperventilation decreases PVRI in the postoperative cardiac infant. More extreme hyperventilation is not reliably useful in lowering PVR further, even in patients who have persistant pulmonary hypertension postoperatively. Moderate hypercarbia raises pulmonary vascular resistance and pressure. This may profoundly alter hemodynamic stability in selected patients. The net effect on vascular tone induced by changes in pH and PaCO2 as a result of changing minute ventilation are significantly more pronounced in the pulmonary compared to systemic vascular bed. Thromboxane does not appear to mediate these effects on PVRI in a simple fashion.

TABLE 1

PCO_2 (mmHg)	21 ± 1	33 ± 1*	45 ± 1*	56 ± 1*
pH	7.64 ± .01	7.50 ± .01*	7.39 ± .01*	7.29 ± .01*
AP (mmHg)	65 ± 3	75 ± 2*	72 ± 3	78 ± 4*
PAP (mmHg)	21 ± 2	22 ± 2	25 ± 2*	37 ± 4*
CI ($l/m/m^2$)	3.2 ± 0.2	3.3 ± 0.2	3.5 ± 0.2	3.9 ± 0.3*
HR (b/m)	142 ± 5	136 ± 5	130 ± 5*	145 ± 6
SVRI (W.u.)	18 ± 1	21 ± 1	19 ± 1	19 ± 1
PVRI (W.u.)	4.0 ± 0.6	4.1 ± 0.7	5.1 ± 0.9*	8.4 ± 1.6*

* $p < .01$ compared to first column