Title: END-TIDAL CARBON DIOXIDE MEASUREMENT IN INFANTS AFTER CARDIAC SURGERY

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Introduction. Monitoring end-tidal carbon dioxide tension (ETCO2) during anesthesia has become widely advocated as an accurate predictor of arterial carbon dioxide tension (PaCO2) in adults. Its reliability in small infants is less well established, especially beyond the normal range of PaCO2. Recent evidence suggests that in the presence of significant pulmonary disease or in children with intracardiac R-L shunts ETCO2 does not provide a precise estimate of PaCO2[1]. Others have suggested that ETCO2 may vary with cardiac index (CI).

This study was designed to test the reliability of ETCO2 monitoring in small infants following repair of their congenital heart disease. Testing occurred over a wide range of PaCO2 while changes in CI and alveolar-arterial oxygen gradient were recorded.

Methods. Infants under the age of 2 years weighing less than 10 kg who had undergone repair of their congenital heart defect were chosen for study during the postoperative period while ventilation was controlled and the patient was still paralyzed with pancuronium and sedated with morphine. All patients had arterial and right atrial catheters placed at the time of surgery along with a pulmonary artery thermistor for measurement of cardiac index by thermodilution technique. A Hewlett Packard model 27210A capnometer was placed in line between the endotracheal tube and the ventilator. The inspired oxygen was maintained at 40% and stepwise changes in ETCO2 were induced by systematic changes in ventilatory rate to produce ETCO2 values of approximately 20, 30, 40 and 55mmHg, individualized for each patient to vary arterial pH between 7.30 and 7.65. After equilibration and stabilization at each ventilatory rate, ETCO2 was recorded and an arterial sample was obtained and cardiac output measured. Data were analyzed using analysis of variance with the Newman-Kewls' modification for multiple range testing and using correlation coefficients for the regression line comparing PaCO2 to ETCO2.

Results. Twenty-three infants with a mean body surface area of 0.32m2 were studied, providing 114 values of PaCO2 and ETCO2 for comparison. ETCO2 varied from 17-58mmHg and PCO2 from 17-67mmHg. Although the regression line and 95% confidence limits shown in Figure 1 for all data imply excellent correlation, spread of the data away from the line of identity at high PaCO2 was apparent. Separate analysis of data for ETCO2 greater than 50mmHg revealed an R value of 0.24 and deviations from PaCO2 of as much as 14mmHg. Underestimates of PaCO2 by ETCO2 could not be accounted for by decreases in CI since there was a small but significant increase in CI at high PaCO2 (3.1±0.2L/M/M2 vs 3.6±0.3:MEAN±SEM $p<.05$).

Discussion. We conclude that ETCO2 monitoring in small infants following repair of their congenital heart disease is a reliable predictor of PaCO2 at low and normal values but may significantly mispredict PaCO2 at higher values. This variance cannot be ascribed to changes in CI and underestimation may be deleterious in infants whose pulmonary vascular resistance is particularly sensitive to rises in PaCO2.

References.

1. Lindahl SGE, Yates AP, Hatch DJ: Relationship between invasive and noninvasive measurements of gas exchange in anestehtized infants and children. Anesthesiology 66:168-175, 1987.

Figure 1