

A197

**TITLE:** THE RELATIONSHIP BETWEEN END-TIDAL AND ARTERIAL CARBON DIOXIDE IN NEUROINTENSIVE CARE PATIENTS  
**AUTHORS:** G.B. RUSSELL, M.D., J.M. GRAYBEAL, CRTT  
**AFFILIATION:** Division of Respiratory & Intensive Care, Department of Anesthesia, Penn State University College of Medicine, Hershey, PA 17033

**INTRODUCTION:** Capnography, by mass spectrometry or infra-red capnometry, is used in critical care units to monitor patients requiring mechanical ventilation. End-tidal carbon dioxide (ETCO<sub>2</sub>) is used clinically as a reflection of the arterial partial pressure of carbon dioxide (PaCO<sub>2</sub>), with the understanding that PaCO<sub>2</sub> exceeds ETCO<sub>2</sub> (the usual gradient, P(a-ET)CO<sub>2</sub>, is reported as 4.2 ± 4.4 mmHg) and this gradient is primarily a reflection of respiratory deadspace. If the gradient remains stable, this relationship can provide a valuable clinical monitor of ventilation, particularly in patients where respiratory acid-base status is not only maintained, but manipulations have therapeutic value. Mechanically ventilated neurointensive care (NICU) patients are an example of this. Hyperventilation is often used to therapeutically lower intracranial pressure while the increased PaCO<sub>2</sub> secondary to hypoventilation can cause intracranial hypertension and decreased cerebral perfusion. A high correlation between PaCO<sub>2</sub> and ETCO<sub>2</sub> has been reported during the initial resuscitation of head-injured patients (1). However, because of observed differences in PaCO<sub>2</sub> and ETCO<sub>2</sub> in other critical care patients we hypothesized that this correlation does not persist beyond the initial resuscitation of NICU patients.

**METHODS:** After approval of the institutional Clinical Investigations Committee, 12 male NICU patients (mean age 48 yrs; 7 post-craniotomy, 2 encephalopathies, 3 closed head injuries) were studied. Clinical care was not altered for the study. When clinically indicated arterial blood gases were measured, the ETCO<sub>2</sub> was determined from the capnograph (Hewlett Packard 78520A infrared capnometer). The P(a-ET)CO<sub>2</sub> was evaluated for possible effects of changes in the other monitored parameters, which included: heart rate (HR), systemic systolic, diastolic and mean blood pressure (SBP, DBP, and MAP, respectively), central venous pressure (CVP), ventilator rate (VR), total respiratory rate (RR), intracranial pressure (ICP), and inspired oxygen concentration (FiO<sub>2</sub>). Regression analysis was used to determine the significance of the relationship between PaCO<sub>2</sub> and ETCO<sub>2</sub> and the gradient and other assessed variables. Student t tests were used where applicable. A p value ≤ 0.05 determined significance.

**RESULTS:** A total of 135 comparisons, 11.3 ± 5.8 per patient (values = mean ± S.D.), of PaCO<sub>2</sub> and ETCO<sub>2</sub> were made. During the study period HR was 94 ± 16; MAP, 90 ± 15 mmHg; CVP, 9 ± 8 mmHg; VR, 11 ± 3 bpm; RR, 17 ± 7 bpm; ICP, 11 ± 8 mmHg; and FiO<sub>2</sub>, 0.50 ± 0.14. The P(a-ET)CO<sub>2</sub> was 6.9 ± 4.4 mmHg, (-11 to 21 mmHg), with PaCO<sub>2</sub> = 34 ± 6 mmHg and ETCO<sub>2</sub> = 27 ± 6 mmHg. There was a significant correlation between PaCO<sub>2</sub> and ETCO<sub>2</sub> for the total population studied (r = 0.72, p < 0.001). However, when the relationship between PaCO<sub>2</sub> and ETCO<sub>2</sub> for individual patients was analyzed only 7 of 12 patients had significant correlations (figure). The P(a-ET)CO<sub>2</sub> correlated positively with DBP (p < 0.001), MAP (p < 0.001), and to FiO<sub>2</sub> (p < 0.001).

**CONCLUSIONS:** ETCO<sub>2</sub> does not provide a stable reflection of PaCO<sub>2</sub> in all NICU patients. There is stable P(a-ET)CO<sub>2</sub> in approximately one half of patients evaluated. The individual factors that allow clinical dependence on the PaCO<sub>2</sub> to ETCO<sub>2</sub> relationship are not clarified at present. Arterial blood gases cannot be eliminated when monitoring ventilation in mechanically ventilated NICU patients.

**REFERENCE 1.** Mackersie, RC, Karagianes, TG; Use of the end tidal carbon dioxide tension for monitoring induced hypocapnia in head-injured patients. Crit Care Med. 1990;18;764.

A198

**TITLE:** BOTH SUFENTANIL AND FENTANYL INCREASE ICP SIGNIFICANTLY IN RESUSCITATED HEAD TRAUMA PATIENTS  
**AUTHORS:** PL Bailey, MD, RJ Sperry, MD, MV Reichman, MD, JC Peterson, MD, PB Petersen, MD, NL Pace, MD, TH Stanley, MD

**AFFILIATION:** Department of Anesthesiology, University of Utah Health Sciences Center, Salt Lake City, UT 84132

Controversy persists regarding the effects of opioids on intracranial pressure (ICP). The most common neurosurgical candidate at high risk for serious ICP problems is the head trauma victim. In this study we evaluated the acute impact of sufentanil and fentanyl on ICP. After institutional approval and informed consent from nearest of kin, nine severe head trauma patients received sufentanil (0.6 µg/kg) and fentanyl (3 µg/kg), intravenously each over one minute but 24 hr apart in a randomized, double-blind fashion. Prior to study, brain resuscitation included mechanical hyperventilation (PaCO<sub>2</sub> range 24-33 mmHg) and pharmacologic therapy with muscle relaxants, steroids, mannitol and sedation with midazolam and/or morphine. ICP, HR and blood pressure were continuously monitored. ICP was measured by a Camino® Catheter System, which utilizes an intracranial bolt and sterile miniature intracranial (intraparenchymal) pressure transducer. Data were analyzed by paired t tests, Wilcoxon or non-parametric t tests and Hotelling multivariate analysis of variance. Statistical significance was set at a P value of < 0.05.

Both sufentanil and fentanyl caused significant increases in ICP and decreases in BP (Table 1). Increases in ICP of 50% or more were common. Although no patient had a baseline ICP of 20 mmHg or more, 4 of 9 patients experienced increases to >20 mmHg. All peak changes occurred at 2 to 10 min after drug injection with most occurring 4 to 8 min after injection. Several patients required intervention with additional hyperventilation to attenuate ICP increases that were deemed clinically excessive.

These results indicate that modest doses of potent opioids can cause significant and potentially harmful increases in ICP in head trauma patients, even after full resuscitation has been instituted. These ICP increases, combined with even modest decreases in blood pressure, result in significant and potentially harmful decreases in cerebral perfusion pressure. While some animal investigations suggest that cerebrovasodilation may underlie opioid induced increases in ICP, the exact mechanism(s) resulting in the changes we found remains unclear. Caution and ICP monitoring should be exercised when applying these drugs in head trauma victims and perhaps other patients at risk for ICP problems.

FIGURE

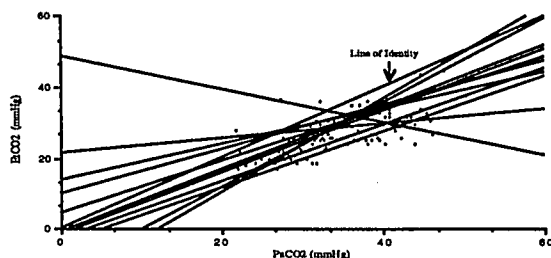


Table 1

	Sufentanil		Fentanyl	
	Baseline	Max/Min	Baseline	Max/Min
ICP	7.4 ± 2.0	*12.7 ± 3.0	9.3 ± 1.8	*18.6 ± 3.7
HR	85 ± 6	87 ± 4	99 ± 5	93 ± 4
MBP	93.4 ± 4.5	*82.3 ± 5.2	89.7 ± 4.9	*80.5 ± 5.5

Values expressed as mean ± SEM; \*P < 0.05