TITLE : TOTAL INTRAVENOUS ANESTHESIA WITH PROPOFOL AND WITHOUT INTUBATION FOR MAGNETIC RESONANCE IMAGING (MRI) IN PEDIATRIC PATIENTS

AUTHORS : M. VANGERVEN, M.D., P. WOUTERS,M.D., E. VANDERMEERSCH,M.D., H.VAN AKEN,M.D.,
AFFILIATION : Dept. Anest., K.U. Leuven 3000, Belgium

Anesthesia for patients undergoing MRI is complicated by many problems due to the static and dynamic magnetic field and the emitted radiofrequent signals. Several anesthetic techniques have been proposed but none seem to meet all the demands especially when intracranial pathology is present. Propofol can be administered by continuous infusion for the maintenance of anesthesia or sedation (1). This study was undertaken to evaluate a total intravenous anesthetic technique with propofol in spontaneously breathing children without intubation.

After obtaining institutional and informed patient consent (parents), 20 neuropediatric patients (age 1 month-12 years, mean 38 months) were studied. Premedication consisted of Atropine Syrup 0,02 mg/kg po. All children were induced with propofol 1 mg/kg IV, followed by a continuous infusion of propofol 8 mg/kg/h. The infusion rate was adjusted to obtain adequate immobilisation. When indicated, propofol boluses (1 mg/kg) were added. Oxygen (4 l/min) was given by face mask. The infusion was terminated near the end of MRI. In all patients heart rate, respiratory rate, blood pressure and temperature were recorded. Arterial oxygen saturation (SaO_2) was monitored by a pulse oximeter. End-tidal CO_2 ($PET\ CO_2$) was continuously measured in samples aspirated via a modified nasal cannula (2). In 10 patients capillary blood gases were obtained at 3 and 20 min after induction in order to evaluate the clinical use of PET CO_2 monitoring. Values are expressed as mean $\pm$ SEM.

$PET\ CO_2$ and SaO_2, measured at 3, 10, 20 and 30 min., are shown in table 1. SaO_2 never declined below 96 %. Capillary to end-tidal PCO_2 difference was within the physiologic range 4,25 $\pm$ 0,7 mm Hg. All other parameters remained well within acceptable limits. The mean time before the infusion rate could be reduced to 6 mg/kg/h was 6 min. In 7 patients additional propofol boluses were necessary. Only one sequence out of 49 had to be restarted because of motion artifacts.

Conclusion : The described technique of propofol sedation meets the demands of safety and has the advantage of simplicity, rapid induction and fast recovery. Continuous measurement of PET CO_2 via a nasal cannula is a reliable technique for monitoring of respiratory function in sedated children.

References : 1. Anesthesiology 71:260-277, 1989
2. J Clin Monit V5, 2:105-110, 1989

Table. 1: SaO2 and PET CO2 at 3, 10, 20 and 30 minutes following start of propofol infusion in children undergoing MRI.

n= 20	3'	10'	20'	30'
SaO2 (%)	99 ± 0.22	99 ± 0.18	99 ± 0.27	99 ± 0.25
PET CO2 (mmHg)	35 ± 1.5	36 ± 1.6	38 ± 1.7	41 ± 1.6

Mean ± SEM

TITLE: POSTDUCTAL HYPOXEMIA IS ASSOCIATED WITH PLASMA EICOSANOIDS IN NEONATES WITH CONGENITAL DIAPHRAGMATIC HERNIA (CDH).

AUTHORS: D.K. Nakayama MD, R. Evans PhD, and E.K. Motoyama MD.
AFFILIATION: Departments of Pediatric Surgery, Anesthesiology, and Pulmonology, the Children's Hospital of Pittsburgh, and the Departments of Surgery and Anesthesiology, the University of Pittsburgh, Pittsburgh PA 15213.

Despite improvements in neonatal anesthesia and intensive care, CDH continues to have a high mortality rate. CDH is often complicated by severe pulmonary hypertension, right-to-left shunting, and hypoxemia. A "honeymoon period" of adequate gas exchange often precedes episodes of hypoxemia, suggesting the involvement of vasoactive substances in its pathophysiology, in addition to pulmonary hypoplasia. Pulmonary vasodilator therapy has met with minimal success. Eicosanoids, specifically thromboxane A2 (TXA2, vasoconstrictor) and prostacyclin (PGI2, vasodilator), have been considered as possible causative factors of pulmonary vasoreactivity, but their relation to episodes of hypoxemia in CDH has not been elucidated.

Plasma samples were collected every 8 h from 11 newborns with CDH within 5 days of life following emergency surgical repair. Thromboxane B2 (TXB2) and 6-keto prostaglandin F1 alpha (6kPGF), stable metabolites of TXA2 and PGI2, respectively, were measured by double antibody radioimmunoassay. TXB2, 6KP, and TXB2/6KP were correlated with a-A ratio (postductal PaO2/(713-PaCO2)xFIO2) and postductal PaCO2. Measurements obtained while infants were on ECMO treatment were excluded from analysis.

Both postductal hypoxemia and hypercarbia were correlated significantly with plasma levels of eicosanoids. The a-A ratio was inversely correlated with TXB2 ($r = -0.71$, $p < 0.005$; Figure 1), 6kPGF ($r = -0.65$, $p < 0.02$; Figure 2), and TXB2/6kPGF ($r = -0.50$, $p < 0.01$, Figure 3). Postductal PaCO2 was also correlated with TXB2 ($r = 0.70$, $p < 0.01$) and 6kPGF ($r = 0.85$, $p < 0.001$), but not with TXB2/6kPGF. The average plasma level of TXB2 among nonsurvivors ($n = 3$) was higher than that in survivors ($n = 8$; 120 $\pm$ 56.9 vs 107 $\pm$ 38.0 pg/ml, mean $\pm$ S.E.M.), but not significantly. The average 6kPGF among nonsurvivors was higher than that in survivors (355 $\pm$ 162.2 vs 117 $\pm$ 20.4 pg/ml), but this also failed to reach significance. Thus, eicosanoids may play a role in the development of pulmonary hypertension, respiratory failure and mortality in CDH.

Figure 1. TXB2 vs a-A ratio.

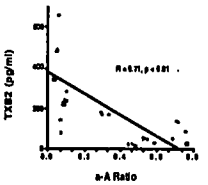

Fig. 2. 6kPGF vs aA ratio.

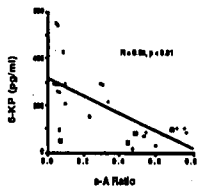

Fig. 3. TXB2/6kPGF vs aA ratio.