Title: ARTERIAL (Pa CO2) TO END-TIDAL (Pet CO2) CARBON DIOXIDE TENSION DIFFERENCE DURING SINGLE (SLT) AND DOUBLE (DLT) LUNG TRANSPLANTATIONS

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Introduction: During controlled ventilation Pa CO2 to Pet CO2 gradient is usually small (1) but, in some patients or situations, gradient may be enlarged and variable (severity of lung disease, acute modification of cardiac output, manipulation of pulmonary vessels). This study was aimed at examining the relationship between Pa CO2 and Pet CO2 during lung transplantation.

Patients and Methods: 5 patients undergoing SLT and 5 undergoing DLT without cardio-pulmonary bypass were studied after institutional approval and informed consent. 6 patients suffered from COPD, 4 from fibrosis.

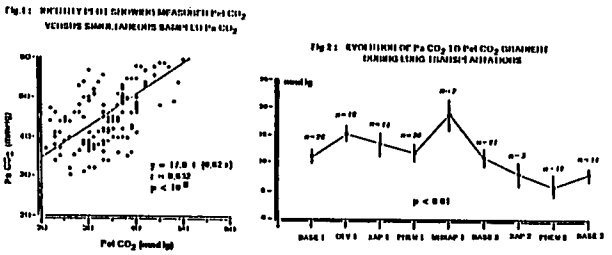
Age ranged from 36 to 55 years, body weight from 44 to 75 kg, BSA from 1.44 to 1.89 m^2. Anesthesia was achieved by intravenous drugs, intubation used a Carlens tube. All patients received controlled ventilation (FiO2 between 40 % and 100%) at a rate and volume appropriate to maintain Pa CO2 at preinduction value. Simultaneous recording of Pet CO2 (side-stream infrared analyzer Nellcor 1000) and arterial blood gas sampling were performed.

Results are expressed as mean ± SEM. Statistical analysis was performed using linear regression (Pa CO2 vs Pet CO2 for each patient and in the whole group) and one-way ANOVA for the comparison of values measured at remarkable surgical stages of SLT and DLT: (BASE1) during ventilation of both native lungs, (OLV1) during one-lung ventilation, (XAP1) during one-lung ventilation after cross-clamping of the pulmonary artery, (PNEU1) after pneumonectomy during transplantation of the first lung, (UNXAP1) after reperfusion of the first graft and before ventilation, (BASE2) during ventilation of the native and transplanted lungs. The following stages correspond to the second period of DLT: (XAP2) after clamping of the second native pulmonary artery, (PNEU2) after pneumonectomy and during transplantation of the second lung, (BASE3) during ventilation of the transplanted lungs.

Results: 145 pairs of data were collected. In 4 patients (2 fibrosis, 2 emphysema) no correlation was found. In the 6 other patients, correlation coefficient ranged from 0.67 to 0.76. Pa CO2 and Pet CO2 are related in the whole group (fig. 1). Mean values of Pa CO2 to Pet CO2 gradients differ according to the different stages of SLT and DLT (fig 2).

Conclusion: Variability of Pa CO2 to Pet CO2 gradient is important and is related to acute modifications of circulatory and ventilatory patterns. Pet CO2 measurements are misleading in the evaluation of ventilatory requirements.

References: 1. Nunn JF, Hill DW. J. Appl. Physiol., 15:383-389, 1960



TITLE: EFFECTS OF PROPOFOL SEDATION ON COMMON LABORATORY MEASUREMENTS

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Introduction. Sedation of patients in intensive care units (ICU) is often necessary. Propofol is an anesthetic agent that may be useful for ICU sedation. This study was designed to assess the impact of prolonged propofol sedation on renal function, hepatic cellular function and serum fat concentrations.

Methods. Following IRB approval and informed consent, 10 critically ill head injured patients were studied. Patients in the control group (N = 5) received morphine 2-10 mg./hr. and pentobarbital 2-10 mg./kg. as needed for sedation. Patients in the propofol group (N = 5) received a loading dose of up to 1 mg./kg. of propofol followed by constant infusion at 0.01 to 0.05 mg./kg./hr. Laboratory determinations were carried out prior to the onset of sedation therapy, 72 hrs. after onset of sedation, and 24 hrs. following completion of sedation. Sedation continued 7 days or until clinical improvement occurred.

Results. Patients in both groups were similar with respect to control laboratory determinations (unpaired t test). Tables I and II illustrate the changes noted during sedation.

Table I: Effects of Morphine Sedation (Mean ± SD)

	Before Sedation	During Sedation	After Sedation
BUN	12 ± 6.7	-------	14 ± 5.0
Creatinine	1.2 ± 0.2	-------	1.0 ± 0.2
LD	470 ± 160	494 ± 267	419 ± 154
AST	70.4 ± 30	41 ± 8	43 ± 23
Cholesterol	144 ± 46	132 ± 25	152 ± 38
Triglycerides	74 ± 20	103 ± 5.6	132 ± 50

No significant changes noted.

Table II: Effects of Propofol Sedation (mean ± SD)

	Before	During	After
BUN	13.2 ± 4.6	------	21 ± 4.7*
CR	1.3 ± 0.2	------	1 ± 0.3
LD	452 ± 173	576 ± 361	474 ± 165
AST	103 ± 80	49 ± 31	381 ± 74
Chol	97 ± 37	111 ± 25*	131 ± 35*
Trig.	56 ± 33	154 ± 47*	116 ± 54

* = $p < 0.05$ greater than control (paired t test)

Discussion. Patients receiving propofol sedation demonstrated mild changes in laboratory determinations that were not clinically significant, leading us to conclude that long-term sedation with propofol is not associated with major laboratory abnormalities.

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