

**Results** Surfactant properties did not change in the control group. In the CPB group, PL content increased in TA 24 hours after CPB. LA concentration dropped 4 hours after CPB ( $P < 0.01$ ) but recovered within 24 hours. The PL:protein ratio of LA was decreased at 24 hours as compared with baseline ( $P < 0.01$ ). The relative amount of phosphatidylglycerol in LA-PL content dropped linearly over time. The relative content of the hydrophobic SP-B and SP-C

in LA increased almost threefold as compared with baseline. There were no significant changes in biophysical function of LA.

**Conclusions** CPB in children induces profound changes in the surfactant system, involving both PL and protein components. Biophysical function may be maintained by compensatory increases in SP-B and SP-C of LA.

## 7 Evaluation of a mathematical model for blood gases and acid-base status during extracorporeal circulation

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Critical Care 2003, 7 (Suppl 1):7 (DOI 10.1186/cc2153)

**Objectives** Blood gases and acid–base status are important parameters during extracorporeal circulation. They are controlled by the perfusionist, by varying arterial pump flow, gas flow over the oxygenator, inspiratory oxygen fraction, and carbon dioxide content in the inspiratory gas mix. To support the perfusionist with suggestions based on a control algorithm, a reliable system description is needed. Reliability of a complex model of human acid–base and blood gas status under extracorporeal circulation was evaluated using clinical documentation data.

**Methods** A mathematical model for blood gas and acid–base status under extracorporeal circulation was developed. This model consists of a multiple compartment model for the oxygenator, and models for arterial and venous  $PCO_2$ ,  $PO_2$ , (including temperature- and pH-dependent shift in oxygen-binding capacity of haemoglobin),  $SO_2$ , bicarbonate, base excess and pH. It was implemented in a Matlab/Simulink environment. Input parameters were oxygenator type, gas flow,  $FiO_2$ , arterial pump flow, temperature, haemoglobin concentration and haematocrit. As output parameters, venous and arterial  $SO_2$ ,  $PO_2$ ,  $PCO_2$  and pH were analyzed. The model was tested by using clinical monitoring data during extracorporeal cir-

ulation of patients undergoing aorto-coronary bypass grafting as input data, and comparing the model output with the results of conventional blood gas analyses (Rapidlab 288<sup>®</sup>) retrospectively.

**Results** Estimations of arterial  $PO_2$ ,  $PCO_2$  and  $SO_2$  were adequate. They followed the time course appropriately and remained within a narrow error band (Max. dev.:  $PO_2 < 17$  mmHg,  $PCO_2 < 7$  mmHg,  $SO_2 < 0.01\%$ ). Venous  $PO_2$  followed appropriately (Max. dev.:  $< 4$  mmHg), whereas  $PCO_2$  (Max. dev.:  $< 8$  mmHg) did not reproduce the time course. Simulations for arterial and venous pH overestimated continuously and were not acceptable (Max. dev.: arterial pH +0.14, venous pH +0.07). The best results were achieved for estimation of  $SO_2$ .

**Conclusions** Modelling the patients' acid–base status and blood gases will be important for further development of control algorithms used in extracorporeal circulation. The presented model shows good concordance with clinical data for blood gas estimation, but needs to be reviewed concerning acid–base status. Further validation and in controlled experimental setups will be required.

## 8 Transpulmonary vascular gradients of nitric oxide pathway metabolites and asymmetrical dimethyl-L-arginine in the flow - or pressure-overloaded pulmonary vasculature

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Critical Care 2003, 7 (Suppl 1):8 (DOI 10.1186/cc2154)

**Objectives** Alterations in pulmonary vascular nitric oxide (NO) production have been implicated in the regulation of pulmonary vascular tone and the development of pulmonary hypertension (PH). Asymmetrical dimethyl-L-arginine (ADMA), an endogenous inhibitor of NO synthesis, has been suggested to counteract endothelial NO production.

**Methods** Transpulmonary gradients of nitrite ( $NO_2$ ), nitrate ( $NO_3$ ) and ADMA were determined in patients with increased pulmonary flow (Qp) before (1) and after (2) interventional closure of atrial septal defect (ASD), and in patients with increased pulmonary vascular resistance (Rp) (3). Twenty patients with ASD: median age

6.1 years (range 3.5–17.1 years), median Qp/Qs 2.1, Rp/Rs  $< 0.12$ . Twenty patients with PH: median age 8.1 years (range 1.2–13.5 years), median Rp/Rs 1.1 (range 0.36–1.79).  $NO_2$ ,  $NO_3$  (chromatography mass spectrometry) and ADMA (high-performance liquid chromatography) were measured in plasma samples from the main pulmonary artery (PA) and femoral artery (SA).

**Results** (1) In ASD patients,  $NO_2$  showed a significant gradient with a median SA:PA ratio of 1.34 ( $P < 0.01$ ), but this was not so for ADMA (1.05) or  $NO_3$  (1.01). (2) After closure, SA:PA ratio of  $NO_2$  decreased to 0.89 ( $P < 0.05$ ), indicating a switch from  $NO_2$