Systemic effects of carbon dioxide insufflation technique for de-airing in left-sided cardiac surgery

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Objective: Systemic effects of carbon dioxide (CO_2) insufflation during left-sided cardiac surgery were evaluated in a prospective randomized study, with regard to acid–base status, gas exchange, cerebral hemodynamics, and red blood cell morphology.

Methods: Twenty patients undergoing elective left-sided cardiac surgery were randomized to de-airing procedure either by CO_2 insufflation technique (CO_2 group, n = 10) or by Lund technique without CO_2 insufflation (Lund group, n = 10). Groups underwent assessment of acid–base status by intermittent arterial blood gases and in-line blood gas monitoring. Capnography was used to determine volume of CO_2 produced. Cerebral hemodynamics was measured by transcranial Doppler sonography and near-infrared spectroscopy. Red cell morphology from cardiotomy suction and vent tubing was studied by scanning electron microscopy.

Results: Patients in the CO_2 group consequently developed significantly higher levels of hypercapnia with a concomitant increase in the volume of CO_2 produced despite significantly higher oxygenator gas flows compared with the Lund group. Effects on cerebral hemodynamics were observed in the CO_2 group with significantly higher blood flow velocities in the middle cerebral artery and higher regional cerebral saturation. Red blood cell damage was observed in the CO_2 group by scanning electron microscopy (97% in CO_2 group vs 18% in Lund group).

Conclusions: Insufflation of CO_2 into the cardiothoracic wound cavity during left-sided cardiac surgery can induce hypercapnic acidosis and increased cerebral blood flow and local blood cell damage. These systemic effects should be monitored by in-line capnography and acid–base measurements for early and effective correction by increase in gas flows to the oxygenator. (J Thorac Cardiovasc Surg 2014;147:295-300)

ACD

Insufflation of carbon dioxide (CO_2) into the pericardial cavity during left-sided cardiac surgery can reduce residual intracardiac air and reduce the risk of air embolism.[1-3] However, reports have described development of hypercapnic acidosis from CO_2 insufflation.[2,4,5] Furthermore, hypercapnia by its vasodilator effect is known to increase cerebral blood flow,[6] which could contribute to increased risk of cerebral embolism. Our group has previously described a technique for de-airing (Lund technique) of the left heart that does not utilize CO_2.[7-9] In a prior prospective randomized study,[9] we compared the Lund technique to the CO_2 insufflation technique, and observed significantly higher rates of air emboli recorded by transesophageal echocardiography and microembolic signals by transcranial Doppler sonography (TCD) associated with CO_2 insufflation.[9] In this study we evaluated systemic effects of CO_2 insufflation with regard to acid–base balance, gas exchange, cerebral hemodynamics, and red blood cell morphology in the previously described prospective sample.[9]

MATERIALS AND METHODS

Study Population

Patients scheduled for elective open aortic surgery were considered for study inclusion as previously described.[9] Preoperative exclusion criteria were a history of carotid artery disease, chronic obstructive pulmonary disease and emphysema, previous thoracic surgery, and patients who were being considered for left internal thoracic artery harvesting. Intraoperative exclusion criteria were accidental opening of the pleura during sternotomy in the CO_2 group, failure to wean from the cardiopulmonary bypass (CPB), and failure to obtain adequate Doppler signals from the middle cerebral arteries. Twenty consecutive patients were randomized to either the Lund de-airing technique (Lund group, n = 10) or the CO_2 insufflation technique (CO_2 group, n = 10) during induction of anaesthesia by opening envelopes indicating de-airing technique created from a computer generated randomization list. The study was approved by the ethics committee of Lund University, Sweden, and all included patients provided written informed consent. The study protocol was registered with ClinicalTrials.gov (identifier No. NCT00934596).

Operative Management

Patients were anaesthetized and monitored during surgery using standardized procedures, including intraoperative transesophageal echocardiography. Patients were ventilated using a SERVO-i ventilator (Maquet Inc, Solna, Sweden) equipped with a module for calculation of CO_2 minute production (Capnostat, Respironics Novametrix Inc, City, Conn). Surgery was

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Abbreviations and Acronyms

CO_2 = carbon dioxide
CPB = cardiopulmonary bypass
LV = left ventricle
rSO_2 = regional cerebral oxygen saturation
SEM = scanning electron microscopy
TCD = transcranial Doppler sonography

performed using standard surgical technique. CPB was established using a membrane oxygenator (Compact Flow EVO Phiso; Sorin Group, Mirandola, Italy), an arterial filter (Cobe Century; Sorin Group, Mirandola, Italy), and polyvinylchloride tubing (silicone tubing in the pump heads). Roller pumps (Stockert S3; Sorin Group GmbH, Munich, Germany) and a heat exchanger (T3; Sorin Group, Mirandola, Italy) were used in all patients. CPB was maintained with a nonpulsatile blood flow rate of approximately 2.5 L/minute/m^2 at normothermia. During CPB, patients were cooled to 25°C-28°C as measured in the urinary bladder or tympanic membrane depending on whether the aortic arch was replaced or not, respectively. Antegrade cold blood cardioplegia was used for myocardial protection and the left ventricle (LV) was vented through the apex in all patients using a 15F Polystan LV drainage catheter (Maquet Inc, Solna, Sweden).

The Lund De-Airing Technique

The Lund de-airing technique has been described by us previously.[7-9] It comprises induced collapse of both lungs by opening the pleura bilaterally and disconnection of the ventilator before opening the heart to prevent air from entering the pulmonary veins, and a gradually staged cardiac filling and lung ventilation before CPB weaning, to expel air trapped in the pulmonary veins.

The CO_2 Insufflation De-Airing Technique

In this group the pleural cavities were left intact. During CPB, patients were ventilated with 1 L/minute, 5 breaths/minute, and positive end expiratory pressure 5 cm water. Before cannulation for CPB, CO_2 insufflation was initiated with a gas diffuser placed in the cardiothoracic cavity (Cardia Innovation AB, Stockholm, Sweden) according to the manufacturer's instructions as follows.[10] CO_2 flow was set at 10 L/minute and continued until 10 minutes post-CPB. Following completion of the surgical procedure and closure of the heart, the heart and lungs were passively filled, the heart was massaged, and the left heart de-aired continuously through the LV vent. Full ventilation was then resumed, and the aortic root was de-aired by active suctioning with the LV vent clamped. The aortic crossclamp was released and the LV vent opened again. After good cardiac contractions, the LV preload was gradually increased by reducing the venous return to the CPB circuit, and the de-airing continued through the vent in the LV apex under transesophageal echocardiography monitoring. When no air emboli were observed in the left atrium, LV, and aortic root, the drainage in the LV was reduced and the heart was allowed to eject. De-airing was continued, and when no air emboli were observed in the left side of the heart, the patients were weaned from CPB and the LV vent was clamped in situ.

Monitoring of Arterial Blood Gases and Gas Dynamics

Arterial blood samples were drawn intermittently every 15 minutes for analysis of blood gases (ABL800 FLEX; Radiometer, Copenhagen, Denmark). Temperature-corrected blood gases were used to facilitate alpha-stat pH management. In addition, pH and CO_2 partial pressure were continuously monitored from the arterial line using an in-line blood gas monitor (CDI Blood Parameter Monitoring System 500; Terumo Cardiovascular System, Ann Arbor, Mich). CO_2 partial pressure was targeted to 5.5 to 6.5 kPa for both groups and the gas flow in the oxygenator readjusted when CO_2 partial pressure diverged from this interval. CO_2 concentration at the gas outlet on the oxygenator was measured using a capnograph (IRMA CO_2; Phasein AB, Danderyd, Sweden). CO_2 minute production (VCO_2 mL/min) from the oxygenator was calculated as gas flow in the oxygenator multiplied by the concentration of CO_2 at the oxygenator gas outlet. Total volume of CO_2 in the CO_2 group was thus the sum of CO_2 from dead space ventilation measured in the ventilator and CO_2 measured at the oxygenator gas outlet. In the Lund group CO_2 minute production was measured at the oxygenator gas outlet only as the ventilator was disconnected during CPB. For clinical comparison an upper cut-off value at 60 minutes of CPB time was chosen for 2 reasons: (1) to retain adequate number of observations in both groups for statistical comparison, and (2) to permit comparison between the groups when all patients are in cooling or early rewarming phase of surgery.

Arterial and mixed venous oxygen content in blood was calculated according to the following formula:

$$\text{Oxygen content mL/L} = \text{Haemoglobin (g/L)} \times 1.36 \times \text{saturation (\%)}/100 + 0.0031 \times \text{oxygen tension (kPa)}$$

Oxygen consumption (VO_2) was calculated according to the following formula:

$$VO_2 \text{ mL/min} = \text{arteriovenous oxygen content difference} \times \text{pump flow (L/min)}/100$$

Respiratory quotient (RQ) was calculated according to the formula:

$$RQ = \text{volume of } CO_2 \text{ (mL/min)}/\ VO_2 \text{ (mL/min)}$$

TCD Monitoring

Middle cerebral arteries of both hemispheres were monitored continuously for mean blood flow velocities using multifrequency TCD sonography (Doppler box; DWL, Singen, Germany) beginning at the start of surgery and continuing up to 10 minutes after weaning of CPB.[11] The bilateral probes were fixed transtemporally by head brace. The insonation and reference gate depths were between 50 and 60 mm, sample volume 10 mm, power 180 mW, and gain 10.

Scanning Electron Microscopy (SEM)

At the conclusion of CPB in patients 4 and 5 from the CO_2 group, clot formation was suspected in the cardiotomy suction tubing close to the pump head. However SEM imaging of the PVC tubings from these patients showed no clot formation but varying degrees of damaged red blood cells. Segments of the PVC cardiotomy suction and vent tubings immediately proximal to the respective pump heads were consequently sent for SEM study in the remaining 10 patients from both groups. Two 15-mm long sections from each tubing sample were fixed in 2% glutaraldehyde in Sorensen buffer at pH 7 for 2 hours. Each tubing segment was then cut in half along the long axis and dehydrated in a series of graded ethanol concentrations until a dry ethanol critical point drying was reached. Each section was mounted on stub, sputtercoated with 20 nm gold and examined under a Philips 515 scanning electron microscope by 1 expert who was blinded to intervention groups. All images were recorded at the same magnification. Four representative images from each individual, 20 from each group, were thus available for the comparative study. Five images from each group were randomly selected for detailed calculation of the proportion of damaged red blood cells in each image.

Statistical Analysis

The randomization list was generated using the Plan procedure in SAS version 8.2 (SAS Institute, Cary, NC). Data were analysed using the stat