Intraaortic Balloon Pumping During Cardioplegic Arrest Preserves Lung Function in Patients With Chronic Obstructive Pulmonary Disease

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Background. Linear flow during cardiopulmonary bypass is considered a potential mechanism of lung damage in patients with chronic obstructive pulmonary disease (COPD). We evaluated differences in lung function of patients with COPD undergoing preoperative intraaortic balloon pumping (IABP), between linear flow during cardiopulmonary bypass (IABP-off) and maintenance of pulsatile flow (IABP-on at automatic 80 bpm) during cardioplegic arrest.

Methods. Fifty patients with COPD undergoing preoperative IABP were randomized between January 2004 and July 2005 to receive nonpulsatile cardiopulmonary bypass with IABP discontinued during cardioplegic arrest (25 patients; group A), or IABP-induced pulsatile cardiopulmonary bypass (25 patients; group B). Hospital outcome, need for noninvasive ventilation, oxygenation (partial pressure of oxygen, arterial to fraction of inspired oxygen [Pao₂/Fio₂), respiratory system compliance, and scoring of chest radiographs were compared.

Results. There were no hospital deaths, no IABP-related complications, and no differences in postopera-

tive noninvasive ventilation (group A: 6 of 25, 24.0% vs group B: 5 of 25, 20%; p = not significant [NS]). One patient in both groups developed pneumonia (p = NS). Intensive care and hospital stay were comparable (p =NS). Group B showed lower intubation time (8.3 \pm 5.1 hours versus group A: 13.2 ± 6.0 ; p = 0.001), better Pao₂/Fio₂ at aortic declamping (369.5 ± 93.7 mm Hg vs 225.7 \pm 99.3; p = 0.001) at admission in intensive care $(321.3 \pm 96.9 \text{ vs } 246.2 \pm 109.7; p = 0.003)$, and at 24 hours $(349.8 \pm 100.4 \text{ vs } 240.8 \pm 77.3; p = 0.003)$. The respiratory system compliance was better in group B at the end of surgery (56.4 \pm 8.2 mL/cm H₂O vs 49.4 \pm 7.0; p = 0.004) and 8 hours postoperatively (76.4 \pm 8.2 vs 59.4 \pm 7.0; p =0.0001), as well as scoring of chest radiograph at intensive care admission (0.20 \pm 0.41 vs 0.38 \pm 0.56; p = 0.05) and on the first day (0.26 \pm 0.45 vs 0.50 \pm 0.67; p = 0.025).

Conclusions. Automatic 80 bpm IABP during cardioplegic arrest preserves lung function in patients with COPD.

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The use of cardiopulmonary bypass (CPB) during cardiac operations is associated with an unspecific inflammatory reaction that correlates with the occurrence of organ dysfunction and sometimes even serious organ failure [1, 2]. Pulmonary dysfunction is a frequent complication during the postoperative course of cardiac surgery using CPB [3–6]. The severity of such dysfunction varies from mild alterations in gas exchange, to various degrees of interstitial pulmonary edema with formation of excessive bronchial secretions, to the acute respiratory distress syndrome (ARDS) [3–5, 7, 8]. In consequence, up to 20% of patients undergoing operations with the use of CPB need prolonged ventilation for more than 48 hours postoperatively [7]. Moreover, clinically manifested ARDS is one of the most common causes of in-hospital

deaths and early and late complications after cardiac surgery [1–8].

Although the mechanisms behind CPB-induced lung

Although the mechanisms behind CPB-induced lung injury are complex (involving the inflammatory reaction caused by the contact of blood with foreign surfaces [5], the type of anesthesia and ventilation [5, 9], the switch from a pulsatile to a linear flow [10], and the induction of an ischemia-reperfusion state [5-8]), the observation that maintenance of a finite pulmonary artery blood flow during CPB attenuates the degree of the lung injury suggests that lung ischemia-reperfusion plays a significant role [11]. Moreover, it has to be kept in mind that during CPB the blood flow to the lungs is almost limited to the bronchial arteries, and it has been proven in some studies that bronchial artery blood circulation is substantially reduced during CPB [11, 12]. Several experimental models have shown that the decrease in bronchial artery blood flow during and after warm CPB is the main cause of the increased pulmonary vascular permeability with formation of tissue edema and cytokine production, and severe hypoxemia secondary to intrapulmonary shunt

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PVRI

RSC

SCR

SVRI

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Abbreviations and Acronyms AMI = acute myocardial infarction **CABG** = coronary artery bypass grafting CI = cardiac index **COPD** = chronic obstructive pulmonary disease **CPB** = cardiopulmonary bypass FEV₁ = forced expiratory volume in 1 second FEV_1/FVC = forced expiratory volume in 1 second/forced expiratory vital capacity **IABP** = intraaortic balloon pump ITU = intensive therapy unit MAP = mean arterial pressure **PCWP** = pulmonary capillary wedge pressure

= pulmonary vascular resistance index

= systemic vascular resistance index

= respiratory system compliance

= scoring of chest radiographs

= troponin I

[11, 12], which is further associated with metabolic and ultrastructural changes of the lung tissue, anticipating the worsening of CPB-related lung ischemia [7, 12]. Although the nonpulsatile blood flow obtained with standard CPB circuits is considered an acceptable, nonphysiologic compromise with few disadvantages (including the induction of the systemic inflammatory response), the theoretical benefits of pulsatile blood flow include the reduction of vasoconstrictive reflexes, the optimization of oxygen consumption, and the reduction of tissue acidosis, secondary to the improvement of organ perfusion [10, 13–16]. Furthermore, we previously demonstrated [17] that IABP-induced pulsatile flow during aortic cross-clamp time better preserves splanchnic function in patients without multiorgan comorbidities.

Finally, it is well-known that already damaged lungs, because of preoperative chronic obstructive pulmonary disease (COPD), are at high risk for CPB-induced lung dysfunction, so that COPD still represents one of the most common indications to off-pump surgery [10, 18, 19]. In fact, patients with COPD have reduced pulmonary function reserve and any injury on their lungs could have a demonstrable clinical effect after CPB [19, 20].

Therefore, it was the aim of our study to test the hypothesis that the maintenance of pulsatile perfusion during aortic cross-clamp time (ACC), with the aid of intraaortic balloon pump (IABP), may attenuate CPB-related lung damage by improving blood flow through the bronchial arteries in the subset of patients at high risk for this complication, such as those with COPD.

Material and Methods

Patients and Study Design

Between January 2004 and July 2005, we prospectively enrolled 50 patients with COPD undergoing isolated

primary CABG for severe left main stem disease. All patients considered at risk for preoperative ischemic events because of severe and diffuse coronary lesions (critical left main disease greater than 90%, or severe left main lesion greater than 70% with severe right coronary stenosis greater than 75% and unstable angina despite intravenous nitrates) underwent preoperative IABP insertion. The study protocol was approved by the Institution's Ethical Committee-Institutional Review Board (September 2003). Informed consent was obtained from each patient enrolled in the study.

The diagnosis of COPD was based on the Summit database definition: each patient required treatment for chronic pulmonary compromise or had a forced expiratory volume in 1 second (FEV₁) less than 75% of predicted value or a forced expiratory volume in 1 second to forced expiratory vital capacity (FEV₁/FVC) less than 0.7. Each patient underwent a spirometry test and was seen by a pneumonologist preoperatively [20].

On admission at our Institution, the patients were randomized by lottery, drawing preprepared sealed envelopes containing the group assignment. Twenty-five patients (group A) received a preoperative IABP treatment before induction of anesthesia, with IABP turned off during cardioplegic arrest, and restarted with a 1:1 IABP mode immediately after cross-clamp removal; the other 25 (group B) received standard preoperative treatment with IABP, which was switched to an automatic 80 bpm mode during cross-clamp time, and switched again to a 1:1 IABP mode after cross-clamp removal. In order to avoid misleading data, patients older than 75 years or with splanchnic organ comorbidities (renal or liver failure, abdominal aortic aneurysm, or severe autoimmune disease) were excluded from the study.

Anesthesia

All patients underwent Swan-Ganz catheter insertion through the right internal jugular vein for continuous hemodynamic monitoring before anesthetic induction. Postoperative chest roentgenogram confirmed its exact positioning.

Anesthetic technique was the same for all patients: induction of anesthesia consisted of intravenous propofol infusion at 3 mg/kg combined with fentanyl administration at 0.10 mg/kg. Neuromuscular blockade was achieved by 4 mg/hour pancuronium bromide, and lungs were ventilated to normocapnia with air and oxygen (45% to 50%). A positive end-expiratory pressure (PEEP) was set at 5 mm Hg. During CPB, ventilation was discontinued but the PEEP was maintained. Propofol infusion (150 to 200 μg/kg per minute) and isoflurane (0.5% inspired concentration) maintained anesthesia. Arterial and central venous catheters were the standard. Inotropes were started immediately after aortic cross-clamp removal to maintain adequate mean systemic pressure, always starting with enoximone at a dosage of 5 μg/kg per minute. The need for further increase in inotropes was recorded: inotropic support was defined as low-dose when enoximone was administered at a dosage lower than or equal to 5 μg/kg per minute; medium-dose when

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