REVIEW ARTICLE

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Clinical Strategies to Prevent Pulmonary Complications in Cardiac Surgery: An Overview

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POSTOPERATIVE PULMONARY COMPLICATIONS (PPC) occur frequently in cardiac surgery (different PPC summarized in Table 1). The incidences vary from 10% to 25%,[1–3] and at least 2% to 5% of all patients undergoing cardiac surgery are at risk to develop a severe postoperative lung dysfunction, such as an acute respiratory distress syndrome. Postoperative pulmonary complications lead to prolonged hospital stays and increased costs.[4] Data from autopsy reports suggest that 5% to 8% of cardiac surgery-associated mortality could be related to respiratory complications.[5–7]

One hypothesis suggests that the pathogenesis of PPC has a multifactorial origin. Factors contributing to the pathogenesis of PPC are as follows: (1) Cardiopulmonary bypass-(CPB) associated complications: An inflammatory reaction due to the use of a cardiopulmonary bypass; (2) Lung injury with endothelial dysfunction due to inflammation and ischemia; (3) Ventilation-associated complication: Atelectasis, chest wall instability, and nosocomial infections; (4) Exacerbation of preexisting conditions: Pulmonary comorbidities, such as chronic obstructive pulmonary disease and smoking.

Prevention of PPC after cardiac surgery has been the subject of extensive research.[8] Because of the multifactorial pathogenesis, a variety of prevention strategies and perioperative interventions have been investigated during the past decades.

Most of these preventive and interventional actions focused on single factors that contribute to PPC (risk factors are summarized in Table 2). In general, few studies have delved into multimodal concepts that combine prevention and intervention at various stages of the perioperative process. The purpose of this review article is to give an up-to-date overview of dedicated strategies that can be used to prevent the occurrence of PPC in cardiac surgical patients. For practical reasons, this review focuses on clinical interventions that could be applied easily without need for any experimental setting and is based on a comprehensive literature search that was performed in several databases (MEDLINE, Cochrane Controlled Trials, BIOSYS). The literature search was completed in December, 2013; however, recently published articles regarding this subject were added, and the following search terms were used: *Cardiac surgery, pulmonary complications, prevention,* and *perioperative.* When a specific subject was found to be contributory, this subject (eg, steroids, fast-track, ventilation) was added to identify more literature regarding this specific topic.

CARDIOPULMONARY BYPASS

The inflammatory response due to the use of CPB is one of the underlying mechanisms in PPC after cardiac surgery. A number of different mechanisms activate the inflammatory response during CPB. Direct blood contact with artificial surfaces of the CPB system activates leukocyte and cytokine release.[9] Nonpulsatility, ischemia, and reperfusion can activate this response as well. This inflammatory response leads to increased pulmonary vascular permeability and, thus, pulmonary edema with respiratory failure. The activation of coagulation during CPB leads to the formation of microemboli in the pulmonary circulation and results in increased deadspace. During conventional CPB, perfusion of the lung is reduced significantly. The activation of inflammatory cascades during this low-flow ischemia has been shown in a number of studies.[10–12] Pulmonary perfusion during CPB could reduce this inflammatory response in animals.[10] In patients, pulmonary indices (alveolo-arterial oxygen delivery, PaO_2/F_IO_2 ratio, and lung compliance) were significantly better preserved when treated with pulsatile pulmonary perfusion during CPB.[13] Postoperative bronchoalveolar lavage specimens showed a lower absolute count of white blood cells and a lower percentage of neutrophils in patients with pulsatile pulmonary perfusion, suggesting that a lower alveolar inflammatory response was present. However, circulating cytokine levels were not significantly different compared with subjects in a control group.[14] Alveolar macrophages, not circulating monocytes, activate this inflammatory response.[14] Pulsatile pulmonary perfusion did not show a benefit in terms of mortality. Whether pulsatile perfusion improves the outcome in high-risk patients is not yet clear and needs to be studied further.

Clinical findings do not necessarily support the hypothesis that off-pump cardiac surgery (OPCAB) instead of on-pump surgery reduces PPC. Staton et al reported their findings from 200 patients scheduled for coronary artery surgery who underwent either an off-pump or an on-pump procedure after being randomly allocated into a group assigned to 1 of the 2

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Table 1. Overview and Frequency of Postoperative Pulmonary Complications

Postoperative Pulmonary Complications	Incidence
Pleural effusion	27%-45%
Atelectasis	17%-88%
Phrenic nerve paralysis	30%-75%
Diaphragmatic dysfunction	2%-54%
Pneumonia	4%-20%
Pulmonary embolism	0.04%-3%
Aspiration	2%
Pneumothorax	1%
Acute respiratory distress syndrome	0.4%-2%

techniques. OPCAB resulted in an improved gas exchange and earlier postoperative extubation but did not have any impact on radiographic signs of PPC, spirometry, pulmonary edema, pneumonia, pleural effusion, or mortality.[15]

Additional strategies that have been discussed include reducing the impact of CPB on pulmonary dysfunction by applying ultrafiltration to scavenge proinflammatory mediators and minimizing the CPB circuit with more biocompatible components. Furthermore, leucocyte depletion and a less-intense use of the cardiotomy suction probably may help in preserving lung function.[9]

VENTILATION STRATEGY

Using an adequate ventilation strategy is suggested to be important in patients undergoing heart surgery. Whereas the use of lung-protective ventilation in intensive care patients at risk or with respiratory failure has been established for a decade, the situation for the cardiac surgical patient is less clear.

For abdominal surgery, a recent randomized controlled trial found that a combination of low tidal volumes (5-6 mL/kg BW), positive end-expiratory pressure, and recruitment maneuvers led to improved pulmonary function tests and, to some extent, fewer postoperative abnormal chest x-ray findings.[16] Restrictions must be made when extrapolating these results to the cardiac patient. Several investigations reported beneficial effects of a low-volume ventilation strategy on the inflammatory systemic and pulmonary response.[17,18] However, data regarding the clinical outcome of cardiac surgery patients treated with low-volume ventilation are sparse. One recent controlled trial on low-volume ventilation that included 149 cardiac surgery patients (6 mL/kg vs. 10 mL/kg tidal volume) could not find any differences in postoperative respiratory failure, mean ventilator times, or 28-day mortality. However, a higher percentage of patients were free of ventilation at 6 hours from intubation, and significantly fewer patients were reintubated in the low-tidal-volume group.[19] Moreover, whether lung-protective ventilation in cardiac surgery patients may lead to a less intense local inflammatory reaction in the lung is not clear. In 2 investigations on this topic, the results were inconclusive.[20,21]

Ventilation During CPB

During CPB, a variety of ventilation strategies, including recruitment maneuvers, have been tested. A recent meta-analysis confirmed that continuous positive airway pressure and recruitment maneuvers might improve gas exchange immediately after separation from CPB but do not have a sustainable effect on the postoperative course if performed only in the operating room during surgery.[22] Application of vital capacity maneuvers intraoperatively and postoperatively reduces atelectasis, and, therefore, may decrease the number of hypoxic episodes, but has no impact on time to extubation or the length of hospital stay.[23–25]

Optimal Positive End-Expiratory Pressure

Postoperative ventilation with positive end-expiratory pressure (PEEP) in the intensive care unit (ICU) is common practice in most centers. The optimal level of PEEP has been the subject of investigation in a study including 121 patients. In the intervention group (60 patients), during the first 4 postoperative hours, a PEEP level of 10 cm H_2O was applied, and then it was reduced to 5 cm H_2O until extubation. The control

Table 2. Risk Factors for Developing Postoperative Pulmonary Complications[8]

Preoperative	Chronic obstructive pulmonary disease
	Obesity
	Age
	Diabetes
	History of smoking
	Chronic heart failure
	Emergency surgery
	Previous cardiac surgery
	Immobility
Intraoperative	Respiratory depression
	Neurologic injury
	Lung deflation
	Cardiopulmonary bypass
	Topical cooling
	Internal mammary artery dissection
	Sternotomy incision
	Increased number of bypass grafts
	Increased duration of cardiopulmonary bypass
	Lower core temperature
Postoperative	Respiratory depression associated with prolonged anesthesia
	Phrenic nerve dysfunction
	Diaphragmatic dysfunction
	Pain
	Constant tidal volumes/short shallow respiration
	Reduced compliance
	Reduced vital capacity and functional residual capacity
	Ventilation-perfusion mismatch and physiologic shunt
	Fluid imbalance
	Immobility, position
	Chest tubes
	Nasogastric tubes
	Impaired mucocilliary clearance, ineffective cough
	Pleural effusion
	Atelectasis
	Pulmonary edema
	Aspiration