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Lung injury and mechanical ventilation in cardiac surgery: a review

Lesão pulmonar e ventilação mecânica em cirurgia cardíaca: revisão

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ABSTRACT

Respiratory failure after cardiopulmonary bypass heart surgery can result from many pre-, intra- or postoperative respiratory system-related factors.

This review was aimed to discuss some factors related to acute lung injury observed during the postoperative period of cardiac surgery and the mechanical ventilation modalities which should be considered to prevent hypoxemia.

Keywords: Cardiopulmonary bypass/therapeutic use; Cardiac surgical procedures/adverse effects; Lung injury/etiology; Lung injury/physiopathology; Respiratory distress syndrome, adult/etiology; Anoxia; Respiration, artificial; Postoperative period

INTRODUCTION

Countless factors may direct and/or indirectly influence postoperative pulmonary injury after cardiopulmonary bypass (CPB) heart surgery. Considering that these patients need early weaning and extubation, the literature discusses ventilatory techniques and modalities aimed to prevent and correct the frequently observed hypoxemia. However, there is no consensus on the best ventilatory modality to be used both intra- and postoperatively in cardiac surgery patients.

Therefore, this review objective was to discuss the etiology and pathophysiology of lung injury after CBP cardiac surgery, as well as the ventilatory modalities and strategies proposed for these patients.

METHODS

This literature review was conducted on experimental and clinical research databases. Were included the databases: Pubmed, MedLine, Scielo, Lilacs, Scopus, Excerpta Medica, Biological Abstracts, Chemical Abstracts and Index Medicus. Articles published within the last 20 years, both in English and Portuguese, were searched using the terms: cardiac surgery, cardiopulmonary bypass, mechanical ventilation, lung injury and respiratory distress syndrome. Randomized clinical trials, experimental trials and literature reviews were included in the analysis. Letters to the Editor and Case Reports were excluded. The articles sample consisted of six experimental trials, 26 prospective clinical trials, seven retrospective clinical studies and five literature reviews.

Incidence of postoperative respiratory failure

Respiratory failure after cardiac surgery is an important postoperative morbidity issue.[1] Weiss et al.[2] identified acute respiratory distress syndrome (ARDS) in 1.32% of their patients. This low incidence of acute lung injury could be partially ascribed to the intervention for reperfusion myocardial ischemia prevention with Allopurinol both during and after CBP. Another trial by Kaul et al.[3] found in their sample an ARDS incidence of 2.5%.

ARDS was identified in 12 (0.5%) out of 2,464 patients included in the Asimakopoulos et al.[4] trial, and eleven (91.6%) of these patients eventually died; in all of them, severe respiratory failure was part of multiple organ failure syndrome. The only surviving patient developed ARDS with no other organ failures.

Milot et al.[5] studied 3,278 CBP cardiac surgery patients and identified ARDS in 0.4% (13 patients), with a 15% mortality rate (2 out 13 patients). One of the two deceased patients had multiple organ failure.

Cardiac surgery postoperative period respiratory failure

Lung function and oxygenation are impaired in 20 to 90% of CBP cardiac surgery patients.[6] Postoperative lung injury remains an important morbidity cause, and its genesis is often related to anesthesia, CBP[4,6,7] and surgical trauma.[1,3] Significantly, previous cardiac surgery, postoperative circulatory shock and the number of transfusions during surgery are considered acute lung injury and ARDS triggering factors.[5]

Canver and Chanda,[8] in a study including 8,802 patients undergoing coronary artery bypass grafting, identified respiratory failure in patients who required longer than 72 hours mechanical ventilation after surgery. Out of these, 491 (5.6%) developed respiratory failure associated with other significantly contributing for increased risk postoperative complications as sepsis, endocarditis, gastrointestinal bleeding, renal failure, mediastinitis, need of reoperation within 24 hours, and severe bleeding. The CBP time was the only intraoperative factor that significantly contributed to increase the risk of postoperative respiratory failure. The authors concluded that respiratory failure following coronary artery bypass grafting is influenced by postoperative extra-cardiac organs impairment, or systemic complications.[8]

In a cardiac surgery patients' study by Messent et al.[9], were observed to be predictive of ARDS development: prolonged CBP time, intra-aortic balloon or ventricular assistance need, and postoperative dialysis need. However, Weiss et al.[2] reported no correlation between intra-aortic balloon use and low PaO_2/FiO_2 ratio, as only 17 out their 466 patients required the device.[2]

Other features are related to cardiac surgery patients' respiratory failure, such as atelectasis, increased shunt, pulmonary mechanics and chest wall changes, capillary bed and pulmonary parenchyma changes secondary to left ventricular dysfunction, or pulmonary endothelial injury.[2,6,10] Three hundred and six postoperative cardiac surgery patients were studied in the Hospital das Clínicas da Universidade Estadual de Campinas; 30 patients required Swan-Ganz catheter monitoring showing increased pulmonary shunt in 19 of them.[11]

Respiratory pattern changes, muscle incoordination, and reduced pulmonary complacency due to pulmonary and chest wall mechanical properties changes are common during the postoperative period.[12,13]

Reduced urinary output-related creatinine level increases were described by Weiss et al.[2] as a significant risk factor for hypoxemia between one and 12 hours after cardiac surgery.

Most of cardiac surgery patients are extubated early.[14]

Innovative anesthesia techniques and surgical technical advances aim patients' extubation between four and six hours after surgery. Given that, fast-track weaning protocols have been increasingly used in anesthesia recovery and intensive care units. Usually patients not compliant with this protocol's inclusion criteria are those with respiratory dysfunction (represented by increased alveolar-arterial oxygen gradient) or hemodynamical instability from post CBP cardiac dysfunction.[15]

For Nozawa et al.,[10] CBP time above 120 minutes influences mechanical ventilation weaning, and is one of the increased surgical risk factors.

Figueiredo et al.[16] have shown that mechanical ventilation weaning in non-complicated immediate postoperative period of cardiac elective surgery is completed about seven hours after intensive car unit (ICU) admission.[16]

Why extracorporeal circulation leads to respiratory failure?

Pulmonary function is affected by CBP-triggered inflammatory cascade effects. In this process, are released inflammatory mediators, free radicals, proteases, leukotrienes, aracdonic acid byproducts and others.[1] Increased mediators release, produced during CBP, leads to increased pulmonary permeability, with interstitial inflammatory cells and water plus proteins accumulation, leading to micro-atelectasis, increased pulmonary shunt, reduced surfactant production, reduced complacency and increased pulmonary resistance. All together, these factors increase the postoperative respiratory load.[1,8,17]

Intraoperative inflammatory mediators filtration is not able to reduce inflammatory mediators levels, and also does not change the postoperative organ dysfunction rate in CBP grafted patients.[18]

Cox et al.[1] studied 52 good ventricular function patients (ejection fraction > 30%) with no pulmonary antecedents undergoing non-CBP coronary artery bypass grafting. The authors identified that, irrespective the CBP-mediated inflammatory mechanisms, the postoperative alveolar-arterial gradient was increased in both groups. This suggest that CBP is not the only pulmonary dysfunction associated factor, but it is also related to the surgical trauma and anesthesia.[1]

Asimakopoulos et al.[4] evaluated ARDS incidence in 2,464 patients undergoing CBP cardiac surgery; the statistical analysis revealed that this syndrome was associated with ventricular dysfunction (ejection fraction < 30%), heart failure (NYHA classes III and IV), in addition to emergency cardiac surgery. Another finding included systemic inflammatory response syndrome (SIRS) and hypotension in postoperative ARDS patients.[4]

Differently, in a swine experimental trial, Magnusson et al.[19] evaluated the pulmonary function following hypothermia and CBP. Atelectasis and intrapulmonary shunt were increased in the CBP group. Possibly, this difference may be explained for their pulmonary function evaluation 45 minutes after the CBP completion, while in the Cox et al.[1] study the first evaluation was conducted only upon ICU arrival.

Other post-CBP pulmonary dysfunction risk factors are excessive hypervolemia and hemodilution, as stated by Boldt et al.[13] These authors concluded that extravascular edema is associated with impaired pulmonary gas exchange in post-CBP positive fluid balance patients, more frequent in older than 65 years patients.[6]

Systemic inflammatory response syndrome, acute lung injury and acute respiratory distress syndrome in cardiac surgery patients

Cardiovascular bypass (CBP) cardiac surgery causes systemic inflammatory response syndrome (SIRS). The patient's blood contents contact with the CBP circuit surface, the ischemia and reperfusion injury, the heparin-protamine complex reaction, the transfusion related acute lung injury (TRALI), the ventilation induced lung injury (VILI) and surgical trauma are possible SIRS causes. This inflammatory response may contribute to the development of postoperative complications including myocardial dysfunction, respiratory failure, renal and neurological dysfunction, liver function impairment and multiple organ failure.[20]

The acute respiratory distress syndrome (ARDS) is defined by the Brazilian Consensus on Mechanical Ventilation as an acute onset respiratory failure syndrome characterized by bilateral chest radiography infiltrate, severe hypoxemia (defined as PaO_2/FiO_2 ratio < 200), pulmonary capillary wedge pressure < 18 mmHg, or lack of left atrial hypertension clinical and echography signs (presence of a lung injury risk factor). The term acute lung injury (ALI) is equally defined as ARDS, only differing for a less marked hypoxemia, PaO_2/Fio_2 < 300.[20]

Most ARDS studies use the Murray et al.[21] severity score. It involves quantification of hypoxemia level, static respiratory complacency, involved pulmonary quadrants, and end-expiratory pressure (PEEP) level. An end score equal or above 2.5 is considered ARDS.[21]

ARDS pathophysiology is characterized by increased alveolar-capillary permeability, with protein transudation associated with systemic and local inflammation.[5] It is considered an extreme ALI form, leading to a mortality rate between 36% and 60%,[20] and reported by some authors as above 50%.[4]

SIRS and ARDS have been described after cardiorespiratory bypass cardiac surgery, and in this context, has a significant impact on patients' survival rate.[5]

Mechanical ventilation is known to cause VILI, which is indistinguishable from ARDS.[22] From this injury, a series of inflammatory reactions is triggered with mediators (as interleukin) release, changing the alveolar-capillary membrane permeability, easing protein transudation and diffused interstitial edema. Protective ventilatory strategies may reduce ARDS patients' mortality, however how this happens remains to be fully understood.[23,24]

Anesthesia derived pulmonary changes

Cardiac surgery requires general anesthesia, orotracheal intubation and controlled mechanical ventilation. Intraoperative hypoxemia is ascribed to poor gas distribution due to changed pulmonary volumes and respiratory system mechanical properties, and ventilatory control.[25] According to Ramos et al.[26] general anesthesia causes several respiratory physiological effects as: atelectasis formation, residual functional capacity (RFC) reduction, ventilation-perfusion ratio change, and mucociliary function impairment.[26] Anesthesia may also promote respiratory system complacency reduction and increased gases flow airway resistance, from reduced pulmonary volume. These findings indicate that postoperative pulmonary complications may onset even during anesthesia.[25]

Positive pressure mechanical ventilation hemodynamical changes

Positive pressure mechanical ventilation increases intrathoracic pressure, therefore reducing venous return to the right ventricle (RV) and subsequently to left ventricle (LV), and may lead to reduced cardiac output. Hypovolemic patients may have hemodynamical instability upon positive mechanical ventilation start. On the other hand, heart failure patients benefit from positive pressure, as it reduces left ventricle pre- and afterload.

Mechanical ventilation positive pressure increases pulmonary vascular resistance and right ventricle afterload. This should be considered in right ventricle failure patients, and ventilation pressures adjusted as low as possible.

Intrathoracic positive pressure reduces left ventricle afterload, because reduces the intrathoracic pressure versus aortal pressure gradient. Special attention should be given by weaning and extubation in borderline cardiac function patients. Post-extubation intrathoracic pressure drop leads to increased RV preload and concomitant LV afterload increase. These changes may reflect in secondary to heart failure acute respiratory insufficiency.[27,28] Non-invasive mechanical ventilation may prevent reintubation in these patients.

Cardiac surgery mechanical ventilation and ventilatory modalities and strategies

Mechanical ventilation highly contributed to increase survival in several clinical conditions, however, when inappropriately used, may increase morbidity and mortality rates.[22]

Although several trials have compared ventilatory modalities, there data are not sufficient to say if volume-controlled or pressure-controlled ventilation are different regarding their effects on ARDS patients' morbidity and mortality. From a physiologic stand point, just a ventilation modality change without changing tidal volume, respiratory rate, PEEP and plateau pressure, have little impact on patients' prognosis.[20] However, the III Consensus on Mechanical Ventilation states that, irrespective the modality chosen, when the ventilatory parameters are set, high tidal volumes and plateau pressures should be avoided.

Gajic et al.[29] reported a 3,261 intensive care unit critically ill patients' analysis, being all of tem mechanically ventilated for several causes, however with no previous lung injury; 205 of them (6.2%) developed ARDS. The lung injury was ascribed to the use of high volumes and pressures. No PEEP differences were identified for ARDS developing or not developing patients. Low tidal volume (< 6 mL/kg) and plateau pressure (< 30 cmH_2O) are recommended.

Intraoperative mechanical ventilation

Mechanical ventilation is essential during the surgery, and may be prolonged during the postoperative period. Perioperative appropriate ventilatory assistance may minimize pulmonary functions changes, thus reducing postoperative complications.[7]

Although current anesthesia devices and ventilators have more effective low tidal volume offer, and have incorporated some ventilatory assistance tools such as PEEP and pressure controlled ventilation (PCV), these are still little used in anesthesia.[30] Overall, there is no consensus on intraoperative mechanical ventilation,[25] requiring additional investigation.

Traditional cardiac surgery ventilatory support recommends high tidal volumes (10-15 mL/kg) mechanical ventilation in order to minimize atelectasis, and minimal positive pressure to improve arterial oxygenation. However, post-cardiac surgery pulmonary injury patients' studies reported low that tidal volumes reduce systemic and pulmonary inflammatory response, and additionally increase survival.

Zupancich et al. studied 40 patients undergoing CBP coronary artery bypass surgery, and compared two groups: 1) 10-12 mL/kg volume and 2-3 cmH_2O PEEP; 2) low 8 mL/kg volume and 10 cmH_2O PEEP. Interleukins 6 and 8 were dosed on bronchoalveolar lavage fluid and plasma, collected at 3 times: before sternotomy, after CBP and after 6 hours mechanical ventilation. The study results showed interleukins 6 and 8 considerable increases after CBP in both groups. These values kept increasing after 6 hours ventilation only in high volumes and low PEEP patients. Therefore, the authors concluded that mechanical ventilation may be a factor influencing post-cardiac surgery inflammatory response.[31]

Pressure controlled versus volume controlled postoperative mechanical ventilation

Mechanical ventilation as a tool for respiratory failure management has satisfactory advanced, changing the outcomes, as in the ARDS patients.[32] PCV mode appears to be associated to earlier respiratory system mechanics' recovery as compared with volume controlled ventilation (VCV).[23] The III Consensus on Mechanical Ventilation recommends the use of controlled pressure, due to its protective ventilation concepts-appropriate working mechanism, with controlled inspired pressure.[20]

When VCV was compared to inversed relationship PCV, this last lead to reduced cardiac load and dead space.[31] When VCV mode is preferred, descending flow wave should be chosen, as provides better inspired air distribution leading to lower airway pressure.[20]

Castellana et al.[33] studied the mechanical ventilation effects on 61 coronary artery bypass grafting patients' oxygenation. Only $PaO_2/FiO_2 < 200$ mmHg PCV or VCV ventilated patients were randomized. The results showed increased PaO_2/FiO_2 ratio and reduced pulmonary shunt fraction with both modes, without significant differences. The authors also discuss the short hypoxemia time to analyze the best ventilatory mode. However, it was noticed that the hypoxemia degree was related to the immediate postoperative ventilation required time. Increased mechanical ventilation time is directly related to lung injury and respiratory infections incidence, as well as with ICU length of stay and hospital costs.[33]

Controlled pressure ventilation has a characteristic flow pattern, theoretically favorable to a more homogeneous with lower alveolar pressures pulmonary inflation, in addition to airway pressure limitation. There is physiological evidence suggesting that it may be more effective for CBP cardiac surgery postoperative period, as in these patients both ventilation and edema are heterogeneously distributed throughout the pulmonary parenchyma, determining different time constants for different pulmonary regions and alveolar inflation.[33]

In Hospital das Clínicas da Universidade de São Paulo, PCV is used as the ventilatory modality of choice for patients developing important postoperative hypoxemia after CBP coronary artery bypass grafting.[33] This guideline was based on this ventilation mode being associated to early respiratory system mechanics recovery versus VCV in ARDS.[32] However, we couldn't find in the literature studies showing PCV superiority for cardiac surgery postoperative period.[33]

PCV apparently reduces the ventilation induced lung injury risk, because allows more precise control of maximal airway pressures and provides more homogeneous alveolar gas distribution.[24] This is due to the flow valve control, maintaining constant airway pressure, the flow resulting from the controlled pressure set and the patient's respiratory mechanics. This ventilation modality most important care is related to strict tidal volume surveillance, as it changes according to airway complacency and resistance changes.[24] Castellana et al.[33] presented the theoretical PCV advantages: airway plateau pressure limitation (lower barotraumas incidence), consequently reducing ventilation induce lung injury (VILI) and more homogeneous gas distribution.

Alveolar recruitment maneuvers: CPAP and PEEP

Pulmonary recruitment is an inspiratory maneuver aimed to re-open collapsed alveolar units, and is different from PEEP that just prevents alveolar collapse. The maneuver effectiveness may be measured not only by increased end expiratory volume, but also improved oxygenation.[6]

Pulmonary recruitment maneuver, associated with continued positive airway pressure (CPAP) has shown significantly improved postoperative gas exchange in CBP cardiac surgery patients.[34] However, the inspiratory pressure peak alarm set should be careful, as its fundamental limiting this pressure to prevent or reduce potential barotraumas. Additionally, attention to the patient's hemodynamics is also recommended. Nielson et al. reported that a 10 to 20 seconds 40 cmH_2O CPAP pulmonary recruitment maneuver, in cardiac surgery patients, lead to significant (even reaching critical values) cardiac output drop.[35]

Several studies have been conducted in the last decades aimed to evaluate mechanical ventilation's role during CBP.[36] Berry et al.[37] identified that a 5 cmH_2O CPAP, both with 0.21 and 1.0 FiO_2 during CBP in cardiac surgery patients, reduced the alveolar-arterial oxygen gradient after 30 minutes, but not after four and eight hours after CBP, as compared with no-CPAP conventional CBP ventilation. The authors demonstrated that CPAP trended to improve the CBP-impaired pulmonary function. However, they reported that inflated lungs difficult the surgical access.

Another 5 cmH_2O CPAP during CBP study was conducted by Cogliati et al.[38] Elective non-CBP cardiac surgery patients were included. These were allocated to three different groups: no CPAP during CBP, 5 cmH_2O CPAP and 1.0 FiO_2 and a third group with 5 cmH_2O CPAP and 0.21 FiO_2. All three groups had worsened respiratory mechanics, but in the 5 cmH_2O CPAP and 0.21 FiO_2 group, the worsening was less evident.

Lamarche et al.[39] studied 75 patients allocated into five groups: high frequency ventilation with 0.21 or 1.0 FiO_2; 5 cmH_2O CPAP with 0.21 or 1.0 FiO_2; and disconnected from the respirator, showing no respiratory mechanics and gas exchange after sternal closure differences.[39]

Loekinger et al.[34] evaluated 14 patients undergoing elective cardiac surgery divided into two groups: "CPAP" group", 10 cmH_2O during CBP and "no CPAP" (control group). During the surgery, both groups were ventilated using the same parameters, i.e.: 7 mL/kg body weight tidal volume, respiratory rate 15 mpm, starting FiO_2 1.0, PEEP 5 cmH_2O. After the CBP end, alveolar recruitment maneuver (10 cmH_2O CPAP with limited inspiratory pressure) was performed in all patients, keeping 1.0 FiO_2. The group with 10 cmH_2O CPAP during CBP had better ventilation/perfusion distribution, and significantly reduced pulmonary shunt during the first four hours after CBP, as compared to the control group. Consequently, for the CPAP group the arterial partial oxygen pressure was higher and the alveolar-arterial gradient lower.

In this trial, all CPAP patients were extubated, and in the control group three patients had low output and respiratory dysfunction syndrome, one patient had isolated respiratory failure (requiring 20 hours non-invasive mechanical ventilation) and another developed multiple organ failure syndrome requiring additional 6 days mechanical ventilation. Therefore, static lung inflation (i.e., CPAP) during the CBP course could be a maneuver to reduce CBP adverse effects.[34]

In a recent randomized controlled trial, Figueiredo et al.[40] compared 10 cmH_2O CPAP versus open airway during CBP, and concluded that, although the PaO_2/FiO_2 improvement by 30 minutes post-CBP, this benefit was not durable on the postoperative gas exchange.

In a swine experimental trial, Magnusson et al.[41] used 5 cmH_2O CPAP versus open airway during CBP, and chest computed tomography after the procedure. These authors found no difference regarding atelectasis or intrapulmonary shunt reduction for the CPAP group.

In another swine experimental trial, Magnusson et al.[42] used recruitment maneuver with 40 cmH_2O lung inflation for 15 seconds, shown to be effective for atelectasis prevention during general anesthesia and after CBP.[37]

Lamarche et al.,[39] in a swine experimental model used mechanic ventilation during CBP showing that endothelial dysfunction may be prevented by mechanical ventilation, resulting in an endothelial effect similar to the observed with nitric oxide inhalation.

It is not clear how necessary is PEEP to keep oxygenation and increase lung volume in mechanically ventilated CBP cardiac surgery patients after recruitment maneuver.[6] Dyhr et al.[6] conducted a study in 16 patients undergoing CBP cardiac surgery ventilated with 1.0 FiO_2 during the anesthesia recovery period. The patients were randomized to one of two groups, both with pulmonary recruitment (two 20 seconds 45 cmH_2O inflations). The "PEEP group" maintained 1 cmH_2O above the pressure-volume curve lower inflexion point (14 ± 3 cmH_2O) PEEP for 150 minutes after the recruitment. The "ZEEP group" had no PEEP after the recruitment maneuver. In this group, measures didn't change, however in the PEEP group, the end expiratory lung volume was significantly increased ($p<0.001$) as well as PaO_2 ($p<0.05$) after recruitment. This study demonstrated that for CBP cardiac surgery patients requiring high FiO_2 during anesthesia recovery, the recruitment maneuver associated with PEEP improved the lung volume and oxygenation, and that this procedure was well tolerated.[6]

After recruitment maneuver in no cardiopulmonary disease, however with anesthesia-associated alveolar collapse patients, the lungs remain open with no PEEP use when low O_2 fractions are used. High inspired oxygen fraction ventilation leads to oxygen absorption and increased alveolar collapse risk. However, with high FiO_2 values, PEEP is necessary to keep appropriate arterial oxygen saturation.[39]

Weiss et al.[2] reported intraoperative PEEP use, however not associated with recruitment, with no beneficial hypoxemia effects observed.[2] However, in another trial, PEEP USED in pleurectomy patients intra- or postoperative periods reduced the pulmonary shunt and improved postoperative oxygenation.[43]

According to the III Consensus on Mechanical Ventilation, recruitment maneuvers are rarely shown in ARDS patients.

Prone position

Should be considered for patients requiring high FiO_2 and PEEP to keep appropriate $SatO_2$ or severe ALI/ARDS patients (respiratory system static complacency < 40 cmH_2O). Posture change risks should be considered.[21]

Catheters and drain tubes may difficult prone positioning, and sores prevention measures are required.

Prone position (PP) PaO_2/FiO_2 effectiveness for ARDS following cardiac surgery was evaluated by Maillet et al.[44] Sixteen ARDS after cardiac surgery patients were evaluated after PP intervention; the maneuver aimed to improve oxygenation and therefore, gas exchange. Patients remained in PP for average 18 hours, with PaO_2/FiO_2 improvement shown in 87.5% of the patients' population. No serious complication was associated with the intervention; however 5 patients had sores and 2 sternal infection. The authors concluded that PP for the treatment of ARDS after cardiac surgery is safe and able to improve PaO_2/FiO_2 ratio. Studies have shown that PP in critically ill ARDS patients results in improved PaO_2/FiO_2 ratio, however with no impacts on mortality.[45]

CONCLUSION

Acute lung injury or ARDS respiratory failure is frequent in during cardiac surgery patients' postoperative period. There is no literature consensus on the best ventilatory modality to be used. Overall low volumes, limited pressure, PEEP, volume control, in addition to judicious blood transfusion, are recommended in order to minimize lung injury in cardiac surgery.

RESUMO

A insuficiência respiratória após a cirurgia cardíaca com utilização da circulação extracorpórea pode ser resultante de inúmeros fatores relacionados às condições do sistema respiratório no pré, intra e pós-operatório. A finalidade desta revisão é discutir alguns dos fatores relacionados à lesão pulmonar observada no período pós-operatório de cirurgia cardíaca e quais os recursos ventilatórios têm sido propostos para minimizar e/ou tratar a hipoxemia dos pacientes.

Descritores: Circulação extracorpórea/uso terapêutico; Procedimentos cirúrgicos cardíacos/efeitos adversos/complicações Le¬são pulmonar/etiologia; Lesão pulmonar/fisiopatologia; Síndrome do desconforto respiratório do adulto/etiologia; Anoxia; Respiração artificial; Período pós-operatório

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