The authors Vedin et al. [1] present in this issue their results from a prospective clinical study investigating the effects of cardiopulmonary bypass (CPB) on lung function parameters. To that end, 50 low-risk patients suffering from coronary artery disease were randomised into two groups: the first group \( (n=25) \) underwent surgery with the use of CPB (on-pump), the other group \( (n=25) \) without CPB support (off-pump). Contrary to expectations, there was no difference in lung function parameters between the groups. However, gas exchange was impaired in both groups during the 20 h post-op observation period. The observation that lung function parameters deteriorate in the off-pump group is surprising, since the occurrence of organ dysfunction after heart operations has so far been assumed to be associated with use of CPB. Such organ dysfunction may affect the kidneys, myocardium and most particularly, the lungs [2–5]. The question remains, what is the cause, or which pathomechanism is responsible for their findings, when lung function impairment occurs equally in patients undergoing on- or off-pump surgery? To answer this question it is necessary to thoroughly understand some important aspects of the clinical and pathophysiological basics involved during the use of CPB.

The use of cardiopulmonary bypass during heart operations is associated with an unspecific inflammatory reaction that correlates with the occurrence of organ dysfunction and sometimes even serious organ failure. Pulmonary dysfunction is a frequent complication during the postoperative course after cardiac surgery using CPB [6–10], clinically manifested as interstitial pulmonary oedema and the formation of excessive bronchial secretions [11,12]. In particularly serious cases pulmonary dysfunction leads to an acute respiratory distress syndrome (ARDS), with an accompanying mortality rate of over 50% [14,15].

One major cause for organ dysfunction after cardiac surgery using CPB is believed to be an unspecific inflammatory reaction caused by the contact of blood cells with the foreign surface of the heart lung machine. Once an inflammatory reaction is initiated, it is maintained by a series of humoral and cellular factors (complement system, cytokines, endothelial interaction, the endogenous NO-system, prostaglandins and coagulation and/or fibrinolysis). The clinical phenomena (pulmonary oedema, secretions, impairment of gas exchange) associated with this inflammatory reaction are observed in the majority of patients after cardiac surgery using CPB [16]. The duration of CPB correlates directly with the severity of clinical symptoms [17]. Despite the apparent transparency of these findings, no correlation has been demonstrated between the concentration of inflammatory mediators and the severity of the resulting systemic inflammatory reaction. It is entirely conceivable that a rise in inflammatory factors in one patient leads to a minor inflammatory reaction, while in another patient, the same rise causes a pronounced inflammatory reaction. However, the complexity of these processes is enormous, and it is possible that these assumed correlations cannot yet be proved using the current measurement methods and techniques. It is therefore necessary to consider alternative explanations, such as those independent of potentially inflammatory processes.

There are indirect indications in the literature that pulmonary dysfunction might be related to ischemic injury of the lung during CPB [18,19]. Sievers' group [20] and others [7,21] showed in clinical trials that intermittent perfusion of the lung with cold blood during CPB improves the gas exchange during the immediate post-op period. However, the fundamental pathomechanism leading to functional improvement was not examined in those studies.

During total CPB blood flow to the lungs is limited to flow through the bronchial arteries. We were able to prove in an experimental study that bronchial artery blood circulation is substantially reduced during CPB [22,23]. The reduction in flow was associated with metabolic and ultrastructural changes of lung tissue suggesting the presence of ischemia of the lungs during CPB. All of these changes could be ameliorated by controlled perfusion of the pulmonary artery with cold blood during conventional CPB. Furthermore, we were able to demonstrate that indicators for an inflammatory response of the lungs obtained from bronchialalveolar lavage fluids could also be reversed by controlled perfusion of the lung. This was an unexpected observation since the accumulation of inflammatory mediators is thought to be caused by the cardiopulmonary bypass circuit. If this is the case, pulmonary perfusion while on bypass should not have been able to affect these parameters. This observation may suggest a different pathomechanism for CPB associated inflammatory changes, at least in lungs. Vedin et al.’s results allow us to come to the same conclusion, namely that other mechanisms play key roles in impairing lung function after cardiac surgery. However, the significance of this study is limited by the following factors:
1. This is a small study with few patients. We observe that the study’s design lacks a power analysis. 2. The impairment of lung function was rather minimal. It is conceivable that, had this study been carried out on patients at high risk for lung dysfunction, one would observe less injury of the lung in the off-pump patients. In this case the authors’ conclusion would not be valid. We therefore believe that the trial should be carried out on a larger cohort of patients with high risk for pulmonary complications.

In summary, the present study by Vedin et al. is another very important contribution in the search for perioperative strategies to reduce the pulmonary injury after cardiac operations.

References


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