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THROMBORESISTANT CARDIOPULMONARY BYPASS CIRCUITS: ROOM FOR IMPROVEMENT?

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Keywords

Cardiopulmonary bypass; thromboresistant circuits; heparin-coated circuits

Circulating blood contact with the surfaces of non-thromboresistant cardiopulmonary bypass (CPB) circuits induces several harmful inflammatory and hematological effects. Hussaini and colleagues in an effort to develop a more biocompatible CPB circuit that reduces inflammation, thrombogenesis and end-organ injury tested a thromboresistant CPB circuit using a heparin-bonded circuit (HBC) (1). The use of HBC is controversial as prior human studies using HBC have reported modest reductions at best in complement activation (2) as well as postoperative length of stay and end-organ injury (3). However, these studies have been criticized for a possible selection bias. Nonetheless, the use of these circuits is associated with reduced circulating complement (4,5), interleukin-6 (5) and activated granulocytes (4,6). The authors of this current study hypothesized that the outcomes using HBC may be improved by: 1) maintenance of a higher hematocrit (Hct) > 25%; 2) use of a closed perfusion system to eliminate the blood-gas interface (another site of foreign surface exposure); and 3) systemic normothermia. To study this, the authors compared HBC to conventional CPB (non-heparin-bonded circuits; NHB) in a porcine model (1).

Anticoagulation as measured by ACT was achieved in both groups, although supratherapeutic levels were often achieved in the NHB group during CPB. Intraoperative hemodynamics were similar, and blood loss was higher in the NHB group as might be expected given the higher ACT (347.8 ± 79.3 ml vs. 214.1 ± 34.5 ml). In addition, thrombin anti-thrombin complex (TATIII) formation occurred more rapidly and remained elevated in the NHB group compared to the HBC group. Alkaline phosphatase, aspartate aminotransferase, alanine aminotransferase, creatine phosphokinase and gamma-glutamyl transferase levels were also higher in the NHB group, and postoperative leukocyte and platelet counts were significantly lower. As might be expected, animals in the HBC group required 51% less heparin and 61% less protamine than in the NHB group. Microscopically, the arterial filters of the NHB circuits were clogged by activated leukocytes, red blood cells and platelets while the filters from HBC circuits were free of debris.

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Like all studies, there are several unanswered questions. First, the modified perfusion strategy changed several variables. This begs the question: which changes from conventional CPB are responsible for the observed effects—the increased hematocrit, normothermia, the HBC? The use of normothermia may be especially concerning to some surgeons since hypoperfusion may still occur while on CPB and hypothermia offers some end-organ protection. Finally, no cytokine levels were measured to further quantify the pro-inflammatory effects of these perfusion strategies. Nonetheless, these data suggest modification of conventional perfusion strategies may reduce CPB-associated inflammatory, immunologic and hematologic derangements. Further study is needed to identify the mechanisms that lead to authors' observed results. Hopefully further mechanistic insight will lead to new perfusion strategies and circuits for patients.

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