LETTERS TO THE EDITOR

Leukocyte and platelet depletion with a blood cell separator

To the Editor:

Morioka and colleagues are to be congratulated on their elegant and sophisticated study in 42 adults of the hematologic and respiratory consequences of leukocyte and platelet depletion (LPD). On the basis of apparent differences in arterial oxygen tension (PaO_2), alveolar-arterial oxygen pressure difference (A-aDO_2), and respiratory index (RI) in the LPD group and control group, the authors conclude that "depletion of leukocytes and platelets reduced respiratory dysfunction."

This potentially important conclusion is, however, thrown into doubt by a number of significant limitations in their study:

1. Changes in respiratory function were studied only until 18 hours after the release of aortic occlusion. We have previously demonstrated that the peak respiratory injury after cardiopulmonary bypass occurs at 24 to 48 hours after the operation; the current study therefore misses the period of maximum respiratory injury.
2. Changes in respiratory function are assessed, in Table III, by "perioperative changes in PaO_2 and in RI and PEEP necessary to ensure an *adequate* PaO_2 " [italics added]. Although the authors presumably mean change in inspired oxygen fraction (and not PaO_2) to maintain an *adequate* PaO_2 , they do not define the latter, making the results difficult to interpret.
3. The authors initially state that PaO_2 was significantly better in the LPD group until 4 hours after release of aortic occlusion. We are then, confusingly, told that there was no significant difference in postoperative PaO_2 between the groups if the preoperative PaO_2 was greater than 520 mm Hg on an inspired oxygen concentration of 100%. In a subsequent subgroup analysis, the authors state that patients whose preoperative PaO_2 was less than 520 mm Hg on an inspired oxygen concentration of 100% are improved by LPD, but we are not informed of the number of patients in each subgroup. Similarly, an analysis of the duration of cardiopulmonary bypass states that LPD was of benefit only in patients whose bypass time exceeded 160 minutes. The authors do not state why such a seemingly arbitrary time point was chosen or how many patients were in each group.
4. The authors state that positive end-expiratory pressure (PEEP) requirements were "significantly higher" ($p < 0.01$) in the control group at 4, 6, 12, and 18 hours after release of aortic occlusion. Again, there is no definition of the criteria for use of PEEP, and Table III shows that the actual difference in PEEP requirements between the groups was 1 cm H_2O (2.2 ± 0.3 vs 3.3 ± 0.2), which is very unlikely to be of clinical significance.
5. It is stated that there were significant differences in A-aDO_2 between the groups, but no raw data are presented to support this.

Although the authors are to be commended on their investigation of a potentially important therapeutic maneuver for respiratory injury after cardiopulmonary bypass, the inadequacies and inconsistencies in their study cast considerable doubt on their conclusions. Their recommendation that LPD be used "for severely ill patients or patients in whom the duration of cardiopulmonary bypass is expected to be long" is premature.

David P. Taggart, MD(Hons)FRCS
Oxford Heart Centre
John Radcliffe Hospital
Headington
Oxford OX3 9DU, United Kingdom

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[A reply was requested from Morioka and he declined to respond.]

Arterial switch procedure without coronary relocation: A late complication

To the Editor:

In 1992 in this *Journal* we¹ reported on an operative technique used in two patients with complex coronary artery anatomy undergoing the arterial switch procedure. The operation involved the construction of an aortopulmonary fenestration with a bovine pericardial tunnel to allow perfusion of the coronary arteries from the neo-aorta. We report a late death after coronary occlusion in a subsequent child in whom this procedure was undertaken.

The patient was a female infant who was born with cyanosis, tachypnea, and a systolic murmur. Echocardiography revealed double-outlet right ventricle with transposition of the great arteries and a subpulmonary ventricular septal defect. She underwent cardiac catheterization and a balloon atrial septostomy at 3 days of age. The coronary arteries arose from a single origin. Congestive cardiac failure developed as a result of excessive pulmonary blood flow, and she underwent pulmonary artery banding at 29 days old. A pericardial effusion then developed, which was drained when she was 40 days of age.

Symptoms of congestive cardiac failure continued, and anatomic correction was planned. However, after median sternotomy, asystole occurred after broad complex brady-