Ross II operation, because the left ventricular end of the graft can be used as a margin for suturing to the mitral annulus. A sewing cuff around the graft is unnecessary. However, stronger lengthwise reinforcement is essential to maintain the shape of the autograft. It seems to me that the pulmonary autograft flattened out in Dr Tireli's patient, and that the consequent left ventricular inflow obstruction caused pulmonary congestion because the lengthwise reinforcement of the pulmonary autograft failed. A semi-flexible Dacron graft is probably an inappropriate choice of material for the external cylinder of the pulmonary autograft in pediatric patients. A semi-rigid graft designed to resist elongation both lengthwise and crosswise is more suitable for reinforcing the pulmonary autograft. At present, I believe that a non-stretch-type polytetrafluoroethylene graft (Gore-Tex; W. L. Gore & Associates, Inc, Flagstaff, Ariz) is the best material. Because the left ventricular end (the pulmonary commissure end) of the graft is anastomosed to the mitral annulus in the top hat method, varus deformity of the pulmonary commissures and subsequent narrowing of the orifice are prevented. If the left atrial end of the graft (the pulmonary annular end) is anastomosed to the mitral annulus, left ventricular outflow obstruction is infallible, because the implanted graft protrudes into the left ventricle. In addition, varus deformity of the pulmonary commissures because of the left ventricular inner pressure is infallible in the absence of a rigid commissural stent.

A great advantage of our technique,² in which a sewing cuff made of a polytetrafluoroethylene felt is anastomosed to the middle portion of the polytetrafluoroethylene (Gore-Tex) graft, is that neither end of the graft protrudes into the left atrium or the left ventricle. Furthermore, because of the sewing cuff, the polytetrafluoroethylene (Gore-Tex) graft is fairly close to the left ventricular end (the pulmonary commissure end) of the graft and is tightly anastomosed to the mitral annulus. Therefore, varus deformity of the pulmonary commissure is prevented in the absence of a rigid stent. The polytetrafluoroethylene (Gore-Tex) external cylinder ensures an adequate neo-mitral orifice.

In conclusion, although there are some technical difficulties, I believe that the Ross II operation is a useful surgical option

for pediatric patients with a narrow mitral annulus. With our technique, the Ross II operation is suitable for infants with a narrow left atrium or atrioventricular septal defect and left ventricular outflow obstruction.

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Intraoperative hematocrit and cardiopulmonary bypass

To the Editor:

I read with interest the article by Habib and associates¹ in the June issue of the *Journal* concerning the role of the lowest hematocrit as the predictor variable on early and late postoperative outcomes. It is important to maintain the hematocrit within the normal range and avoid extremes. The mere effect of such a strong association begs for a prospective randomized study, as stated by the authors. However, their discussion and conclusion based on the large number of retrospective patients are incomplete. In the field of cardiovascular epidemiology, anemia occurs in 15% to 55% of patients with chronic heart failure.² However, it is unknown whether a strong association of anemia and heart failure has any temporal and causal relation, that is, whether anemia precedes and leads to heart failure.

Nonetheless, the authors do not mention the characteristics and prevalence of heart failure in the baseline data. An argument for expanded plasma volume occurring in acute heart failure, seen in decompensated heart failure, might contribute to low hemoglobin concentrations. Such a feature is not uncommon in patients undergoing non-percutaneous transluminal coronary angioplasty in our era.

It would seem intuitive to deduct that patients who undergo off-pump cardiac bypass surgery with minimal blood loss have less activation of the inflammatory cascade from the bypass circuit and do better. However, given the already available data from randomized trials, no difference exists between the 2.³ This suggests a minimal role for the intraoperative spectrum of hematocrit in off-pump surgery. Another important question that was not answered was whether aprotinin was given consistently, because this drug can also modify patients' outcomes.⁴

Habib and colleagues¹ gave no information about the mean number of grafts per patient, perioperative blood loss, blood-saving techniques, and "transfusion trigger." These issues are important because there is a dose-dependent association between blood transfusion and the development of severe postoperative infection and death in patients undergoing cardiac surgery.⁵

Despite this omission, the overall in-hospital mortality rate is slightly higher than that reported for a similar series of 2,569 patients undergoing coronary artery bypass grafting in whom the "transfusion trigger" during cardiopulmonary bypass (CPB) was a packed-cell volume of less than 20% and mortality was defined as death during the hospital stay or within 30 days of surgery (3.6% vs 2.79%).⁵

Was the pump primed with blood? Administration of blood during CPB may begin a cascade of events that contributes to postoperative. Thus, whenever possible, preoperative pharmacologic therapy, but not transfusional, treatment of anemia should be used.

Also, the authors probe the data to determine any association of anemia and late mortality. It is interesting that the authors do not report the reason for patient death. Because the case mix of death is important, if cardiovascular death was the cause, did any of the patients who received more blood or had the lowest hematocrit have a lower graft patency in the short and long term?

Finally, according to the range of absolute differences in the perioperative hematocrit shown in Table 1 of the article (difference between preoperative and postoperative CPB hematocrits; range 12%-15.2%), it would seem more reasonable to use this parameter than individual values if

the conclusion of increased hemodilution severity is CPB based. If the data are reanalyzed on the basis of the difference between preoperative and postoperative CPB hematocrits, it may be more important that a decrease in hematocrit of more than approximately 10% during bypass is associated with a worse prognosis than discrete values.

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Reply to the Editor:

We thank Dr Shuhaiber for his interest in our recent article in the *Journal*.¹ We are encouraged that he concurs with us that low extremes of hematocrit during cardiopulmonary bypass (CPB) should be avoided and that the strong associations we reported between excessive hemodilutional anemia during bypass and adverse patient outcomes argue for a prospective study. He also requested methodologic clarifications and presented several comments that are worthy of addressing.

Our 5000-patient CPB retrospective series¹ (1994–2000) reflects a population in whom asanguinous rather than blood prime fluid was used. In a very small fraction of patients (anemic patients with low red blood cell volume), blood may have been added to the prime at the onset of CPB. These are included as intraoperative transfusions. Aprotinin was used relatively infrequently (<7%), and we believe it had little bearing, if any, on the reported findings. Standard intraoperative cell-saving methods were used routinely, and saved cells were returned to patients during or immediately after CPB. Hemoconcentration during CPB was used in 16% of patients.

Perioperative blood-loss amounts were not prospectively documented in our database and were not reported. Alternatively, excessive bleeding requiring re-exploration (Figure 1 and Table 4 of reference 1) and transfusions (Figure 3 and Table 4 of reference 1) were reported.¹ The “transfusion trigger” was not rigidly controlled during the 7-year period of this retrospective series, and this threshold may have varied slightly among surgeons and depended on the patient age and clinical status. We believe, however, that these variations do not diminish the clear and obviously strong relationship between nadir CPB hematocrit and transfusion rates (Figure 3 of reference 1).

As we reported,¹ low pre-CPB hematocrit is an important predictor of increased hemodilution during standard CPB (Table 2 of reference 1), but a history of congestive heart failure (documented in ~9% of patients in this series) is not. Notably, patients with acute heart failure that is associated with anemia as the result of compensatory plasma volume expansion almost never undergo operation in our practice. Such patient conditions are usually medically reversed before surgery. Alternatively, intraoperative fluid infusions by anesthesia and preoperative blood donation may lead to substantial plasma volume expansion that can contribute to the extent of hemodilution during CPB and should be carefully considered.

Dr Shuhaiber refers to the randomized prospective study by Puskas and colleagues,² which compares off-pump and on-pump bypass operations in 200 patients and states that the role of intraoperative hematocrit in off-pump CABG is minimal

because little or no differences in outcomes are reported. In responding to this point, it is important to note that off-pump and on-pump CABG procedures are different in many ways, one of which is hemodilution as the result of mixing the patient's blood with prime fluid. Although it is true that Puskas and colleagues reported generally similar in-hospital and 30-day mortalities, their data partially support several of our findings.¹ Indeed, they reported less hemodilution, less transfusion requirements, less coagulopathy, earlier extubation, decreased hospital stays, and less myocardial injury (cardiac enzyme levels) for their patients undergoing off-pump CABG versus on-pump CABG. These results are all in concert with our findings. The fact that no significant differences in other important outcome measures (including mortality) were found may be partially attributable to the size of the series.

For the 3,800 isolated patients undergoing CABG in this series, the mean number of grafts per patient was 3.1 ± 1.0 . Moreover, as we report in *Results*¹ on page 1441, operative mortality (defined as in hospital or out of hospital within 30 days of surgery) for isolated CABG was 2.4% (90/3800), a rate slightly lower (rather than higher) than that cited by Dr Shuhaiber in the CABG series.³ The 3.6% operative mortality that he reports reflects the overall rate for all 5,000 patients, including patients undergoing valve and combined CABG and valve operations.

As we stated in our article,¹ late mortality follow-up was based on queries of the Social Security Death Index Database, in which the cause of death is not available; thus we could not report the relationship between hematocrit on CPB and late cardiovascular death. Nonetheless, Dr Shuhaiber's questions about whether graft failure in CABG is linked to the extent of hemodilutional anemia or transfusions is clearly important and worthy of separate future investigation that is well outside the scope of our study.

Intuitively, Dr Shuhaiber's proposition that the decrease in hematocrit should be used as the independent (or predictor) variable rather than the actual low hematocrit is appealing. However, the extent of hemodilution on CPB is multifactorial, as we reported in Table 2.¹ Also, our¹ and DeFoe's⁴ data clearly demonstrated that a decrease of 10% or 15% in hematocrit is