are shown in Figure 2. There is considerable uncertainty about all the centers' true ranks, which naturally arises from the high degree of overlap of the confidence intervals in Figure 1. We can only state with confidence that center E is in the top half (despite being ranked fourth) and center B is in the bottom half; any further attempt at detailed ranking is spurious. Table 1 presents the probabilities that centers near the top or bottom of the league table truly are the best or worst centers. No center receives more than 30% chance of being either the winner or loser, although center P turns out most likely to be the worst by a small margin.

Such an analysis illustrates the grave dangers of institutional ranking unless there is clear heterogeneity among centers. It also explains why there are generally such radical changes in rankings from year to year when profiling institutions. Presentations that do not emphasize rankings, such as the funnel plots of Stark and colleagues,³ are thus to be preferred.

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Reply to the Editor:

We thank Shuhaiber and Spiegelhalter for their important questions regarding our article. Both questions relate to whether the differences in institutional outcomes for mortality after congenital heart surgery demonstrated in our article represent true differences in performance or were an artifact of our methodology, the Risk Adjustment in Congenital Heart Disease (RACHS-1) method.

Shuhaiber questions whether differences might have been mitigated had a more comprehensive method of risk adjustment been used. We agree that improved methods of risk adjustment would have increased our ability to compare outcomes accurately. However, methods including physiologic variables would have required validation in a population with complex congenital heart problems and would require extensive data collection. The RACHS-1 method was derived from a formal, consensus-based process and has been validated with two diverse data sets, with favorable performance characteristics. The consensus committee that developed RACHS-1 specifically sought to create a method of risk adjustment useful to understand group outcomes using data elements that are frequently available. To clarify, risk categories were incorporated into the risk adjustment model as binary covariates, which do not impose a linear or exponential relationship among categories. We agree that although most centers in the analysis had similar relative ranks across risk categories or worse performance for higher risk procedures, in 5 centers a surprising pattern of worse performance for higher risk performance was observed. Explorations by centers of why these patterns emerged should include a search for unmeasured risk factors but should also evaluate more programmatic possibilities, such as surgical referral patterns, location of postoperative care, and so on.

Spiegelhalter questions whether ranking institutions is an appropriate way to judge relative performance. Although we agree in general about the imprecision inherent in using ranks, especially when numbers of cases are small, we are attempting to guide quality improvement efforts in a field where considerable variability in institutional surgical mortality has been demonstrated by many investigators but annual caseloads are small and are unlikely to increase substantially. Although there may be uncertainty about a center's exact rank or about how large a difference in ranks is clinically important, program directors trying to guide improvement efforts should find it more useful to know their observed rank than to be informed that their center's relative performance did not reach statistical significance.

We would like to emphasize that we would never suggest "profiling" an institu-

tion on the basis of any single analysis, especially one derived from administrative data in a single calendar year. However, these analyses may prove useful to illuminate potential quality problems that need to be explored further, preferably by the institution itself.

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Symmetry aortic connector system To the Editor:

I read with interest the article by Donsky and associates¹ in which they outlined several misadventures with the Symmetry aortic connector system (St Jude Medical, Inc, St Paul, Minn). In our practice, we have an extensive series of off-pump coronary artery bypass operations in which the Symmetry connector has been used. Although the manufacturer has not recommended any anticoagulation regimen postoperatively, my colleagues and I routinely administer clopidogrel postoperatively for 6 weeks.

We justified this therapy after we demonstrated, at least by thrombelastography, a relative state of hypercoagulation after off-pump operations when compared with conventional cardiopulmonary bypass.² Furthermore, after deployment of an intracoronary stent, it is standard to prescribe a postprocedure course of clopidogrel therapy (ie, CLASSICS trial³). Since some of these stents are also composed of nitinol (ie, Scimed Radius stent, Boston Scientific, Boston, Mass), the management of a patient with an aortic connector should be no different from the documented protocol well described in the cardiology literature.

To date we have not experienced any complications with the aforementioned aortic connector and agree with the authors that the 2 cases that they described

involved patients with complex disease and other mitigating factors. We therefore continue not only to use the connector but to promote its use to our colleagues.

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doi:10.1067/mtc.2003.282

Reply to the Editor:

Since submission of our report, we too have begun to administer clopidogrel routinely, 75 mg by mouth every morning starting on the first postoperative morning and continuing through the second postoperative month. We have detected no new problems.

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Symmetry aortic connector system

To the Editor:

The article by Donsky and colleagues¹ regarding thrombotic occlusion of vein grafts after use of the Symmetry aortic connector system (St Jude Medical, Inc, St Paul, Minn) aroused my interest because of my experience with this device. Similar to the authors' experience, I have had occlusion of the aortic orifice at the connector site within a few months of surgery in 3 patients. All patients were obese, diabetic, and hypertensive, as in the patients refer-

enced in the article. It has been my experience, however, that these occlusions occur when a small (gray) connector is used, but not when a large (blue or purple) connector can be used. The authors do not mention the size of the connectors used in their case report. I would like to ask their opinion regarding my observation.

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doi:10.1067/mtc.2003.284

Reply to the Editor:

Size of the connector was not recorded in the permanent record.

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Does normothermic cardiopulmonary bypass influence clinical outcomes, cytokine production, and in vitro platelet function?

To the Editor:

We read with great interest the article by Gaudino and associates¹ in the June 2002 issue of this *Journal*. They evaluated prospectively the clinical outcomes of patients and some inflammatory and fibrinolytic markers such as C-reactive protein, interleukin (IL) 6, prothrombin time, and platelets. They concluded that normothermic systemic perfusion did not influence the clinical course or the extent of inflammatory and hemostatic activation in patients undergoing primary isolated coronary artery bypass. The markers were determined before surgical intervention; 21, 48, and 72 hours thereafter; and at hospital discharge.

We² previously investigated platelet activation and aggregation up to 24 hours after operation by examining the serial changes of platelet count and small particle formation in patients undergoing coronary

artery bypass with normothermic cardiopulmonary bypass (CPB). Platelet counts decreased during CPB, and 24 hours after CPB they had increased to approximately half the pre-CPB levels. Small particle formation was the main type of platelet aggregation observed before surgery. Medium particle formation was also recognized, but no patient had large particle formation. After systemic heparinization, small and medium particle formation occurred. One hour after the initiation of CPB, only small particles were seen; 2 hours after the end of CPB, no small particles were observed. Small particle formation was the main platelet aggregation type observed 24 hours after CPB.

We³ also evaluated cytokine production and levels of thrombomodulin and soluble endothelium-derived adhesion molecules in patients undergoing coronary artery bypass under normothermic CPB. The study was scheduled also up to 24 hours after the operation. IL-6 values were elevated minimally after 30 minutes of CPB, and they showed a surge at the end of CPB or 2 hours after CPB in some patients. Other patients showed stable levels. The IL-6 values were reduced after 2 hours, but 24 hours after CPB they were still higher than the initial levels. There was a huge difference in IL-6 changes among patients. A surge of IL-8 occurred 2 hours after CPB, and the values returned to the initial levels 24 hours after CPB. Thrombomodulin levels were reduced 30 minutes after the initiation of CPB; however, they began to recover during CPB. The levels returned to the initial levels 2 hours after CPB and exceeded them 24 hours after CPB. Levels of soluble endothelium-derived adhesion molecules were reduced after 30 minutes of CPB; they returned to the initial levels 2 hours after CPB and exceeded them 24 hours after CPB.

Our studies may endorse the conclusions of Gaudino and associates. CPB for less than 3 hours does not influence clinical outcomes, and in vitro studies, such as platelet function and cytokine production, did not differ under normothermic and moderate hypothermic conditions.

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