Reply
To the Editor:

We appreciate the interest and thoughtful comments of our colleagues [1] regarding our study on acute kidney injury (AKI) [2], and we are glad to provide more details and thoughts. AKI definition followed the indications of the Acute Kidney Injury Network [3], where a postoperative twofold creatinine increase or a urine output less than $0.5 \text{ mL/kg/12 h}$ means kidney injury. Moreover, inasmuch as our patients were somehow low-risk (92.5% elective procedures, 6.6% repeated procedures, average serum creatinine 1.06 mg/dL), only a very few (13/3219, 0.4%) required intraoperative hemofiltration. Moreover, it is well known that perioperative anemia is a major predictor of unfavorable outcomes and that blood transfusion is needed in these patients [4]. We were unable to assess the role of preoperative hemoglobin level on the occurrence of AKI and on the need for blood transfusions in our patients because this variable had more than 4% of missing data and was for this reason excluded from analysis, inasmuch as logistic regression requires complete patient data for each case. However, the fact that all multivariate models including intraoperative variables retained the number of units of red blood cells that were transfused during surgery as an independent predictor of AKI is an indirect demonstration that even in our patient population preoperative anemia is detrimental for patients. Finally, we chose not to study off-pump patients for the following reasons: (1) they were limited in number (330 patients, less than 10% of our coronary bypass patient population), and this would have precluded obtaining robust AKI predictors at multivariable models; (2) one of the major aims of our study was to assess the role of cardiopulmonary bypass and of cardiopulmonary bypass–related variables on AKI occurrence, and patients undergoing operation off pump could not, obviously, be included. Given that the role of on-pump and off-pump coronary bypass surgical procedures in perioperative AKI occurrence is still uncertain [5, 6], further studies are eagerly waited to clarify this point.

Alessandro Parolari, MD, PhD
Unit for Clinical Research in Atherothrombosis
Centro Cardiologico Monzino IRCCS
Department of Cardiovascular Sciences
University of Milan, Milan, Italy
e-mail: alessandro.parolari@cardiologicomonzino.it

References

Dexamethasone and Myocardial Protection in Neonatal Arterial Switch Operation
To the Editor:

We read the recent article by Heying and coworkers [1] with great interest and have some brief comments. The authors reported that 1 mg/kg of dexamethasone was given 4 hours before cardiopulmonary bypass (CPB) to provide antiinflammatory and myocardial protection in a neonatal arterial switch operation. Preoperative administration of glucocorticosteroids attenuates the release of some proinflammatory cytokines and also increases the production of an antiinflammatory cytokine, interleukin-10, in pediatric and adult cardiac surgical procedures [2, 3]. The other result of the study was lower myocardial CPB. The cTnT levels were related mainly to aortic cross-clamp duration. The authors reported that there was a difference in the cTnT levels only at the first hour after CPB, and at postoperative days 1, 2, 3, 5, and 10. However, there was no difference between two groups with respect to cTnT levels at 10 minutes after the beginning of CPB, the end of CPB, 4 hours after CPB, and at postoperative days 1, 2, 3, 5, and 10. We were not

Davide Pacini, MD
Department of Cardiac Surgery
5 Orsola-Malpighi Hospital
University of Bologna
Bologna, Italy

Francesco Alamanni, MD
Unit for Clinical Research in Atherothrombosis
Centro Cardiologico Monzino IRCCS
Department of Cardiovascular Sciences
University of Milan, Milan, Italy

References
given enough information about the number of patients with a small conal branch division. The division affects more myocardial damage and causes more troponin release than the aforementioned factors listed above.

In conclusion, the antiinflammatory effects of dexamethasone are clearly demonstrated, with significant results. However, the increase in cTnT level was different in only one period (cTnT measured at the first hour after CPB) among eight periods between the two groups, which consisted of very limited numbers of patients. Many factors can affect the cTnT levels in arterial switch operations. Therefore, we think it is inaccurate to state that dexamethasone leads to less myocardial damage and is effective in myocardial protection in the arterial switch operation.

Rıza Turkoz, MD
Emre Özker, MD
Ayda Turkoz, MD

Departments of Cardiovascular Surgery and Anesthesia
Baskent University
Istanbul Teaching and Medical Research Center
Althusize
Istanbul 34662, Turkey
e-mail: rturkoz@yahoo.com

References

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In their comment, Turkoz and colleagues [1] disagreed with the conclusions of our study that myocardial protection was provided by dexamethasone administration [2]. They suggest that lower cTnT levels were likely associated with the shorter period of myocardial ischemia in treated patients.

Since it might appear evident to relate postoperative myocardial cell damage only to the duration of myocardial ischemia during cardiopulmonary bypass (CPB), previous studies gave evidence of the deleterious role of proinflammatory cytokines on the heart [3], cytokines known to be upregulated by myocardial ischemia and reperfusion [4]. Our previous series conducted in a larger number of a similar patient population of neonates undergoing arterial switch operation showed that postoperative myocardial dysfunction associated with higher cTnT release was in turn related to the amount of interleukin (IL) 6 released during cardiac surgery [5]. The multivariate analysis of several independent risk factors for the occurrence of myocardial dysfunction (including aortic clamping time) revealed that postoperative IL-6 levels were the predictive ones.

Our present results show that pretreated neonates displayed less intramyocardial expression of proinflammatory cytokines harmful to the myocardium already before connection to CPB and myocardial ischemia. A shift of the cytokine balance toward the antiinflammatory response was associated with lower circulating levels of cTnT in the early postoperative period. This association has been shown by others to allow discrimination among adults with or without subclinical myocardial cell damage [6], and also with lower catecholamine requirement in the postoperative period [5]. Interestingly, in the present series, the known relationship between duration of myocardial ischemia and postoperative cTnT levels shown in the control group was abolished in the treated group, suggesting protection of myocardial tissue against operative stress.

Thus, based on current knowledge, our results confirm intramyocardial and systemic antiinflammatory shift of the perioperative cytokine balance in neonates treated with dexamethasone that is associated with a certain degree of myocardial protection.

As pointed out by Pouard and Bojan in their invited commentary [7], the important questions that need to be answered are:
1. Is the dose of dexamethasone (1 mg/kg) optimal?
2. Is the time point of dexamethasone administration (4 h before connection to CPB) optimal?

Marie-Christine Seghaye, MD, PhD
Department of Pediatrics
Pediatric Cardiology
University Liège
Rue de Gaillarmont 600
4032 Liège, Belgium
e-mail: mcssegaye@chu.ulg.ac.be

References

The Safety of Selective Cerebral Perfusion With Normothermia in Pediatric Patients

To the Editor:

I read with great interest the article by Ly and colleagues [1] on their retrospective study of antegrade selective cerebral perfusion (SCP) with hypothermia (≤28°C) versus normothermia (≥34°C) in pediatric patients undergoing arch repairs. They reported that the time to extubation, stay in the intensive care unit, and early mortality were similar between the hypothermic...