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Reply to the Editor:

Fundarò and colleagues address the historically difficult task of creating a universally accepted classification system for ischemic mitral regurgitation (IMR). As they note, multiple systems are used for the description of IMR. In fact, the three articles that Fundarò and coworkers cite,¹⁻³ all of which appeared in the same issue of the *Journal*, contain slightly different categorizations for IMR. The most important point, however, is that these three classification systems are actually quite similar to one another. All are based on the mechanism(s) of IMR.

Recent observations have furthered our understanding of the pathophysiology of IMR. In some instances, the mechanisms of IMR are quite complicated; therefore, some precision may be sacrificed as each patient is placed in a single group that specifies the predominant mechanism. Nevertheless, such categorization will facilitate more meaningful communication of the nature of IMR.

The authors of the three articles agree that IMR may be caused by papillary muscle disease or by changes in ventricular and/or annular geometry, the latter resulting in functional IMR. Occasionally, more than one of these mechanisms is operative. Miller's classification system¹ is particularly useful. He suggests that the primary mechanism of IMR generally falls into one of the following categories:

1. Papillary muscle rupture
2. Papillary muscle infarction and elongation without rupture
3. Functional IMR from
 - a. Annular dilatation
 - b. Leaflet tethering
 - c. Annular dilatation and leaflet tethering

By means of echocardiography, the majority of patients with IMR can be placed into one of these groups.

Fundarò and colleagues offer a classification system that is similar to the others mentioned here. However, their distinction between regional and global left ventricular dysfunction does not speak directly to the mechanism of IMR. Rather, it introduces another type of classification system. In addition, subtype A (papillary muscle atrophy, elongation, or displacement) contains disparate entities with different mechanisms of IMR.

Classification systems have a long history of value in cardiac surgery and medicine in general. They are designed to reflect understanding of the nature of diseases (eg, Miller's classification of IMR), to facilitate guidelines and indications for treatment (eg, American Heart Association guidelines), to allow complex observations to be communicated effectively (eg, nomenclature for heart disease), and to reflect differences in prognosis (eg, cancer staging). Fundarò's classification attempts to reflect three different purposes of classification systems simultaneously: mechanism, indications for operation, and prognosis. This may be too ambitious, given the complex nature of IMR.

We, Miller, and Grossi and colleagues propose classification systems that address clarity in understanding the mechanisms of IMR. Choice of operation based on optimization of prognosis is a far more complex and multifactorial issue that, at present, is not clearly amenable to simple classification.

Given our improved understanding of the mechanisms of IMR, it is time that we adopted a common classification system. We recommend following Miller's lead; his mechanism-based description of IMR permits clear categorization of patients.

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12/8/126569

What are the best temperature, flow, and hematocrit levels for pediatric cardiopulmonary bypass?

To the Editor:

I read with great pleasure the letter by Durandy, Hulin, and Lecompte¹ on normothermic cardiopulmonary bypass in pediatric surgery, as well as the reply by Jonas, Newburger, and Bellinger.²

I spent 1 year with Durandy and Lecompte in Paris in 1995, at the beginning of their experience with normothermic cardiopulmonary bypass, and witnessed the extremely smooth and "physiologic" postoperative course of more than 300 consecutive children with congenital heart defects. Most of them had very complicated anatomy and intracardiac repair, and almost all of them were extubated very early, without the need for inotropic support. I repeatedly suggested that Durandy and Lecompte report their extraordinary experience. Their reason for refusing to report their data at that time was the need for more substantial background.

I then decided to follow their methods of cardiopulmonary bypass, and the initial experience has been already reported.^{3,4} Therefore, I viewed the publication of their letter with a great sense of relief, because the feasibility and the advantages of normothermic cardiopulmonary bypass have been now supported by their experience with 1600 congenital cases.

To further support the message given in the letter, I feel obliged to extend the information provided by Durandy, Hulin, and Lecompte. In their letter they discussed only the problem of temperature, although there are at least other three factors used in their practice of cardiopulmonary bypass that contributed to their good results: flow, hematocrit, and leukocyte depletion.

1. **Flow.** The flow generally used for cardiopulmonary bypass is 2.0 to 2.4 L · m⁻² · min⁻¹ or 100 to 120 mL · kg⁻¹ · min⁻¹. Even if this flow rate is accepted as the gold standard for ad-

equate systemic perfusion, and it is called "full flow," it is far from the physiologic value of 3.5 to $5.0 \text{ L} \cdot \text{m}^{-2} \cdot \text{min}^{-1}$. In most hospitals the so-called "full flow" is reduced during the central part of the procedure to a low flow or to circulatory arrest. The types of damage induced by flow reduction (metabolic derangement; endothelial lesions; vascular, myocardial, neurologic, hematologic, and respiratory impairment) are not much different from those induced by temperature reduction.^{3,4} In fact, I learned from the Paris group to perfuse the children with a flow of 3.0 to $3.5 \text{ L} \cdot \text{m}^{-2} \cdot \text{min}^{-1}$, arbitrarily called "high flow."

2. **Hematocrit.** The negative consequences of a systemic perfusion with low hematocrit, widely known for a long time,^{3,4} are now acknowledged even by Jonas. After years suggesting that perfusion with high hemodilution (hematocrit value around 20%) should be used, his group⁵ is now providing evidence of better neurologic protection with a hematocrit value of 30%. From the Paris group I learned to maintain the hematocrit value at least at 30% during cardiopulmonary bypass.

3. **Leukocyte depletion.** The role of leukocyte activation as the main cause of the postoperative inflammatory syndrome, frequently observed in the pediatric population, is generally well known, even if leukocyte depletion is not yet used globally.

In summary, cardiopulmonary bypass as used by the Paris group is not only normothermic, but also associated with high flow, high hematocrit, and leukocyte depletion.

In their reply to the letter, Jonas, Newburger, and Bellinger² suggested two main objections: a small margin of safety in the event of equipment failure and inadequate surgical exposure.

1. **Small margin of safety.** The incidence of "equipment failure" on cardiopulmonary bypass reported in the literature is between 1 every 1,000 or

10,000 procedures. Even if we consider these figures as an underestimation of the reality, this percentage is much lower than the percentage of children with low cardiac output, need for extensive inotropic and respiratory support, neurologic complications, and the related mortality and morbidity reported even in the best centers after "conventional" cardiopulmonary bypass.

2. **Inadequate surgical exposure.** The good results of both the experience of the Paris group¹ with neonates and infants with cyanotic congenital heart defects (tetralogy of Fallot, transposition of the great arteries) and our more limited personal experience including infants with total anomalous pulmonary venous connection^{3,4} demonstrate the feasibility of the technique. Of course, it is necessary to have adequate venous drainage, perfect surgical exposure, and a surgeon ready to accept some discomfort for himself (or herself) rather than for the patients.

In conclusion, Durandy, Hulin, and Lecompte are to be gratefully acknowledged for teaching all of those involved in the care of children with congenital heart defects that surgery for congenital heart disease can and should be performed with a perfusion much closer to the physiologic condition.

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Reply to the Editor:

We thank Corno for his comments on our recent letter. Obviously, normothermic cardiopulmonary bypass associated with warm blood cardioplegia is only part of the perfusion protocol. The flow and the hemoglobin level must be adapted to the systemic oxygen consumption. The quality of the donor blood product must be optimal, but we must also stress the importance of a low volume of priming fluid. We use 180 mL for neonates and infants with a body surface area up to 0.27 m^2 (4.5-5 kg) and 225 mL for infants up to 0.42 m^2 (8-8.5 kg). We increase this volume progressively to obtain a 600-mL prime volume for children from 0.85 to 1.6 m^2 (20-60 kg). With this protocol we never use ultrafiltration during or after bypass.

It is true that most of the surgeons who visited us have been convinced by the advantages of this protocol and have changed their minds about normothermic perfusion. Several surgical units are now using this method. We are reluctant to perform a prospective randomized trial comparing hypothermic and normothermic perfusion. We hope that, in the not too distant future, we will have enough data to publish a single-center or a multicenter experience about normothermic cardiopulmonary bypass in pediatric surgery.

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