Letters to the Editor

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- Type with double-spacing
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Chronic ischemic mitral regurgitation: Types and subtypes

To the Editor:

We have read with great interest the three contributions concerning ischemic mitral regurgitation (IMR) recently published in the *Journal*.¹⁻³ All authors give the impression that a definition of subgroups of patients is urgently needed to permit uniform reporting, compare surgical results, and tailor the optimal surgical option for each patient. However, different classification schemes are suggested in each article. According to Steven Bolling's well-known statement that "IMR is a ventricular disease, not a valvular disease," we also believe that a more detailed analysis of the different aspects of postinfarction ventricular pathophysiology would be advisable. The proposed classification of chronic IMR is described in Table 1.

It is often difficult to give a clear cutoff between "regional" and "global" dysfunction (types 1 and 2), but the distinction appears useful owing to the poorer natural history and less satisfactory surgical outcome in the presence of advanced ventricular remodeling. In this classification, the term "ventricular-papillary dysfunction," introduced by Grossi and coworkers,² is used in a broader sense. It refers not only to papillary muscle fibrosis, but also to papillary muscle displacement and tethering of mitral cusps (subtype A) and to annular dilation resulting from postinfarction ventricular remodeling (subtype B). In fact, both mechanisms frequently coexist (subtype C): annular dilation is very common in

global ventricular-papillary dysfunction, whereas regional dysfunction generally correlates with papillary muscle (usually posteromedial) atrophy or elongation. In this setting, varying degrees of each pathophysiologic mechanism should probably best be addressed by specific surgical repair.

Unlike others,² we did not include the term "isolated annular dilation" in the classification because, in our opinion, dilation of the mitral annulus appears correlated to left ventricular dysfunction and therefore does not represent the primary cause of regurgitation. Likewise, we only considered "functional regurgitation" subtypes and did not include insufficiency caused by structural damage (ruptured papillary muscle or chordal disinsertion).

This subdivision does not introduce new concepts; rather, it tries to classify all known pathophysiologic mechanisms of chronic functional IMR with the purpose to select patients for specific surgical correction and, most important, compare results of operations in similar subgroups of patients.

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TABLE 1. Chronic functional ischemic mitral regurgitation varieties

	Ventricular-papillary dysfunction	
	Regional (type 1)	Global (type 2)
Papillary muscle(s) atrophy, elongation, or displacement (subtype A)	1A	2A
Annular dilation (subtype B)	1B	2B
A and B (subtype C)	1C	2C

References

1. Miller DC. Ischemic mitral regurgitation redux—To repair or to replace? *J Thorac Cardiovasc Surg.* 2001;122:1059-62.
2. Grossi EA, Goldberg JD, LaPietra A, Ye X, Zakow P, Sussman M, et al. Ischemic mitral valve reconstruction and replacement: comparison of long-term survival and complications. *J Thorac Cardiovasc Surg.* 2001;122:1107-24.
3. Gillinov AM, Wierup PN, Blackstone EH, Bishay ES, Cosgrove DM, White J, et al. Is repair preferable to replacement for ischemic mitral regurgitation? *J Thorac Cardiovasc Surg.* 2001;122:1125-41.

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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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1. Miller DC. Ischemic mitral regurgitation redux—To repair or to replace? *J Thorac Cardiovasc Surg.* 2001;122:1059-62.
2. Grossi EA, Goldberg JD, LaPietra A, Ye X, Zakow P, Sussman M, et al. Ischemic mitral valve reconstruction and replacement: comparison of long-term survival and complications. *J Thorac Cardiovasc Surg.* 2001;122:1107-24.
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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-