as an improvement in the ejection fraction (from 62 ± 14% to 67 ± 10%; $P = .01$), which was detected in the majority of the patients with dysfunction prior to implantation. There was also an improvement in the functional class and quality of life questionnaires.

However, during follow-up we have not observed a significant reduction in paravalvular regurgitations (moderate in 23.4%, mild or trivial in 39.7% and absent in 36.9% on the third day after implantation versus moderate in 18.9%, mild or trivial in 46.8% and absent in 34.2% in the sixth month), with good agreement (κ=0.724).

These data are similar to those published in other series,[2,3] and we should point out the fact that in no case did the regurgitation affect hemolysis and that, in our series, it was not related to functional class or medium-term mortality. Its presence in trivial or mild cases could be considered to be of no greater clinical importance than the so-called "physiological" regurgitations observed in mechanical prostheses. In contrast, in a recently published multicenter study,[4] moderate or higher grade regurgitation has been found to be a predictor of mortality between 30 days and 1 year after the procedure.

These paravalvular regurgitations could develop because of a poor choice of the prosthesis size, insufficient expansion of the prosthesis, too low a placement site or perhaps a nonuniform distribution of the valve calcium when, upon expansion of the prosthesis, it remains pressed between the device and the aortic wall. If the late reduction in aortic regurgitation observed in the series of León et al., can be attributed to the adaptability and self-expandability of the prosthesis, it should also be detected in the other series. These differences could be due to the bias associated with the selection of a small cohort or to other mechanisms related to patient characteristics or to postimplantation treatment. The formation of a periprosthetic thrombus or intimal proliferation may have sealed small periprosthetic leaks in the series of León et al., and the difference with respect to other series could lie in the postimplantation treatment. It will be interesting to follow the course of these patients in case a hypothetical leak-sealing intimal proliferation should lead to the development of pannus and an increase in the transprosthetic gradient.

On the other hand, assessment of the changes in left ventricle following implantation in our patients revealed an improvement in the ejection fraction, especially in cases of nonischemic ventricular dysfunction, but there was no evidence of a significant reduction in hypertrophy during follow-up (the interventricular septal thickness decreased from 13.2 ± 2 mm to 12.4 ± 2 mm, $P = .3$; and that of posterior wall from 12.5 ± 2 mm to 12.2 ± 2 mm, $P = .78$). These findings coincide with those reported by De Jaegere et al.,[3] who detected no differences in left ventricular mass or diastolic function 30 days after implantation, and are in accordance with the fact that these are cases of nonphysiological hypertrophy (in contrast to that observed in athletes), secondary to a chronic pressure overload (aortic stenosis, sometimes accompanied by hypertension) with varying degrees of fibrosis and, thus, with slow and limited reversibility. This circumstance, however, does not appear to impede the short-term and medium-term improvement in functional class in these patients.

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Available online 25 May 2011

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doi:10.1016/j.rec.2011.03.005

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Paravalvular Regurgitations and Percutaneous Prosthetic Aortic Valve. Response

Regurgitaciones paravalvulares y prótesis aórticas percutáneas. Respuesta

To the Editor,

We appreciate the interest shown by Rodriguez-Bailón et al. in our articlew published in *Revista Española de Cardiología*,[1] and we would like to make a few points regarding their comments. Two recently published reviews of several different studies[2,3] regarding treatment using the CoreValve percutaneous prosthetic aortic valve and one large study[4] have shown that frequency and/or severity of paravalvular leaks tend to decrease. To our understanding, the difference in results with those from the Rodriguez-Bailón et al. study is based on methodology, with different criteria used to "quantify" these leaks. Some controversy exists among cardiac sonographers whether or not to evaluate paravalvular and central regurgitations the same, or if these should be classified into 4 or 3 grades as recommended in the most recent guidelines for prosthesis evaluations. On the other hand, the concept of "reducing" the leak also differs between studies, and some define a significant decrease as a reduction by more than 1 degree, but in other studies, as in ours, a decrease is deemed significant when it is at least 1 degree. Lastly, Rodriguez-Bailón et al. observed a decrease in the frequency of moderate regurgitations and an increase in mild regurgitations during the follow-up period, which was interpreted as an absence of changes due to high

concordance (κ =0.724). In our opinion, the kappa coefficient is not the most appropriate instrument to use for evaluating the changes over time of an ordinal quantitative variable, such as the degree of regurgitation. Probably, a nonparametric test, such as the Wilcoxon test for paired samples, would be a better option. In a recent analysis performed using the data from our study (92 cases by January 2011), we obtained similar results after 1 month (significant improvement in leaks, $P < .001$), and with no changes after 1 year ($P = .09$), thus eliminating the bias introduced by the reduced number of patients. We insist on the self-expandability of the prosthesis as the probable cause of this decrease, as do other authors, basing our conclusions on the echocardiographic observation of this phenomenon within the first days following the procedure. We have not found more cases of periprosthetic thrombosis than in other series, as we have followed the antithrombotic protocols recommended by the manufacturers.

We have also confirmed an early decrease in ventricular hypertrophy ($P < .05$) using our most recent data, which has also been described recently by other authors,[5] and so we reiterate that differences in methodology could be the cause of the differences observed between studies.

In any case, we share the sentiment expressed by Rodriguez-Bailón et al. for the need for larger studies to further elucidate these "discrepancies," but perhaps our primary objective should be to solidify the criteria used for study methods and for the definition of variables and objectives.

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Available online 1 June 2011

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doi:10.1016/j.rec.2011.04.001

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Effect of Opening a New Catheterization Laboratory on Myocardial Infarction Patients

Impacto en pacientes con infarto agudo de miocardio de la apertura de un nuevo laboratorio de hemodinámica

To the Editor,

We have read with great interest the overall results of the study of Bosch et al.,[1] from the REGICOR group, concerning the impact of opening a new catheterization laboratory in a given geographical area, recently published in the *Revista Española de Cardiología*.

This work reports relevant findings in a small sample of patients, relating them to those of other similar studies. These previous works present divergent results, as expressed by the authors in the discussion section of the articles,[2,3] concerning the benefits of coronary angiography and eventual revascularization (mainly percutaneous) in patients being treated for acute myocardial infarction. Nevertheless, in the study we comment on, the myocardial infarction patients treated after a catheterization laboratory had been opened within the REGICOR framework, in which on-site revascularization procedures were not performed, had a better 30-day survival rate.

In previous reports, as the authors acknowledge, the benefits of a greater availability of catheterization laboratories appears to be explained in terms of the wider use of evidence-based medical therapies,[3] such as beta blockers and statins, which are strongly associated with short-term survival. Another important clinical variable that could explain the 30-day mortality rate would be the delay in the administration of fibrinolytic therapy. What was the influence of these variables on the multivariate model shown in Figure 1?

Finally, we would like to congratulate the authors for this highly interesting study which poses the debate as to the importance of increasing the availability of diagnostic procedures such as coronary angiography, which facilitate the optimal treatment of myocardial infarction patients, including coronary revascularization. The reason for these good results may be the utilization of this diagnostic tool, which leads to a greater number of revascularization procedures in patients at higher risk, precisely those who need it most. Previous registries in Spain, like the DESCARTES registry, revealed that these interventions were less frequently employed in the patients that most needed them, those at highest risk,[4] and dissociated the efficacy from the effectiveness of certain diagnostic and therapeutic interventions.[5] Studies like that of Bosch et al deliver an important message regarding the utility of diagnostic and therapeutic tools in patients with acute myocardial infarction.

FUNDING

Article partially subsidized by a research grant from the Cooperative Cardiovascular Disease Research Network, or RECAVA *(Red Temática de Investigación Cooperativa en Enfermedades Cardiovasculares),* of the *Instituto de Salud Carlos III,* Spain.

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Available online 25 May 2011