POSTOPERATIVE NONINVASIVE VENTILATION IN PATIENTS UNDERGOING CORONARY ARTERY BYPASS GRAFTING: A SMALL STEP WITH GREAT REPERCUSSIONS To the Editor:

Commonly, anesthesia, postoperative pain, and surgery induce respiratory modifications (hypoxemia, pulmonary volume decrease, and atelectasis) associated with a restrictive syndrome.¹ In some cases, postextubation acute respiratory failure is a serious complication after coronary artery bypass grafting. The role of noninvasive ventilation (NIV) in the postoperative period is growing as an alternative therapy to avoid reintubation and associated complications¹; however, pulmonary complications and NIV interactions still show a complex interaction with preoperative or intraoperative procedures.

In their recent article, Al Jaaly and colleagues² compared 2 strategies in the postoperative period: bilevel positive airway pressure (NIV) and usual care with oxygen therapy. Major findings of this study were that there were no significant differences in intensive care stay, postoperative stay, mean percentage of predicted forced expiratory volume in 1 second on days 1 and 3, and rate of pulmonary complications (except atelectasis).

We read this original article with interest; however, there are some interesting aspects that were not taken into account and could influence the

TABLE 1. Aspects related to atelectasis rate in postoperative surgery

Muscle paralysis
Intravenous or inhalational anesthetics
Body mass index*
Induction of anesthesia (use of a gas mixture)
Intraoperative fraction of inspired oxygen†
Surgery times
Sedation

*Although obese patients were not included in this analysis, there is no independent analysis of this factor between the groups. †This is an important element because it is associated with a greater reduction in the proportion of nitrogen and development of atelectasis. This aspect could explain the worse response in the group with standard oxygen versus noninvasive ventilation with respect to a different fraction of inspired oxygen; the intraoperative aspect was not evaluated in the study.⁵

interpretation of the outcome. (1) Small numbers of patients were enrolled in both arms of the study, and current smokers should have been grouped separately. Al Jaaly and colleagues² did not describe the preoperative baseline situation, and that is a crucial aspect.³ For assessment of postoperative acute respiratory failure, it would be relevant to know the baseline oxygenation index and fraction of inspired oxygen before and after extubation. (2) We do not have information regarding the use of any intraoperative recruitment maneuvers that have shown to have implications for the perioperative period.⁴ (3) Atelectasis rates were clearly different between groups. Atelectasis should be assessed by the radiologist, not the senior clinical team. The atelectasis was diagnosed on clinical basis, and bias may have been a factor. Chest radiography is not sensitive for detection of postoperative atelectasis. There are some factors in the preoperative and postoperative periods that need clarification because they may have influence the results observed⁵; we have summarized them in Table 1. Finally, limited training of the cardiac intensive care unit staff may have influenced the benefit of NIV. NIV was applied intermittently, rather than continuously for few hours.

We consider that further prospective randomized studies will encourage evaluating specific NIV strategies and how intra-operative factors are implicated in postoperative pulmonary complications, especially atelectasis.

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Reply to the Editor:

We thank Esquinas and colleagues for their interest in our article and for their comments. They make 3 points. (1) Our trial was small and did not include any subgroup analyses, in

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particular, current smokers versus former smokers and lifetime non-smokers. (2) Information on intraoperative recruitment maneuvers was not reported. Such maneuvers may have implications during the perioperative period. (3) Atelectasis was diagnosed clinically and not by a radiologist, and possible influences of preoperative and postoperative factors were not taken into account. We have responses to these points.

First, our trial was randomized. This created intervention and control groups that were balanced with regard to demographic, anthropometric, and respiratory factors. Randomization does not rule out effect modification (point 1); however, it does ensure that an overall treatment effect is not biased (point 3).

Second, subgroup analyses are controversial. International guidelines focus on prespecified subgroup analyses to avoid selective reporting.¹ Our trial was not powered to detect subgroup effects, and we did not specify any in advance other than preoperative chronic obstructive pulmonary disease (we originally aimed to stratify by chronic obstructive pulmonary disease, but there were insufficient patients with this comorbidity). If we were to carry out subgroup analyses, the findings would be very difficult to interpret. Failure to demonstrate statistical significance might well be due to low power, whereas observation of statistical significance might well be due to chance. For example, Esquinas and colleagues mention the potential influence of body weight: 17 patients with body mass index greater than 30 kg/m² had bilevel positive airway pressure, whereas 17 had usual care.

Third, our trial was designed pragmatically to estimate the effect of bilevel positive airway pressure in a real-life setting. Noninvasive ventilation was applied for a median of 11 hours. In the trial, as in usual care, "lung recruitment" (point 2) typically constituted lung reexpansion

and the application of positive end-expiratory pressure. Assessment of atelectasis by the senior clinical team was also pragmatic (point 3). From a specialist respiratory perspective, such assessment might be considered insensitive; however, it reflected accurately the information on which management decisions are made in practice. Moreover, we did show a substantial difference in atelectasis frequency; better sensitivity would be expected to increase, not decrease, the observed difference.

Finally, we would like to point out that their summary of the findings of our trial ignores our primary outcome (prespecified in our analysis plan) and our explanation of the reason for failing to observe differences between groups in predicted forced expiratory volume in 1 second and other outcomes that one would expect to differ. As we acknowledged and tried to explain in the article, we had expected the respiratory outcomes to parallel the clinical ones. Because this expectation was not met, we recommended a larger multicenter trial at the end of our Discussion section. This is one point on which we agree. Just as one swallow doesn't make a summer, one trial by itself very rarely brings about (or warrants) a change in practice. We recommended that a larger multicenter, randomized trial of bilevel positive airway pressure be carried out to decide whether the promise suggested in our trial can be substantiated.

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MITRAL VALVE REPLACEMENT IN THE PRESENCE OF MASSIVE CALCIFICATION

To the Editor:

I was pleasantly surprised to read the novel technique reported by Husain and colleagues.¹ I reported on the technique used at the All India Institute of Medical sciences way back in 1988,² which my group used when confronted with a heavily calcified mitral annulus. We used a piece of autologous pericardium to cover the raw surface of the brittle and sandy remnant after limited débridement of calcium and to prevent calcific emboli. This patient was followed up and seen again in 2001, at which time I reported the favorable result of 13 years free of embolism.³ He was last seen in 2009 and was well. Others⁴ have also reported the successful use of this technique.