derived in their report, and, despite the simplicity, we believe, from a mathematical and engineering viewpoint, is incorrect, according to previous reports.^{4,5}

$$BP_{d-pre} = F_d \cdot SVR_{pre} \quad (1)$$

$$BP_{s-pre} = (F_s + F_d + F_{r-pre}) \cdot SVR_{pre} \quad (2)$$

$$BP_{d-post} = F_d \cdot SVR_{post} \quad (3)$$

$$BP_{s-post} = (F_s + F_d + F_{r-post}) \cdot SVR_{post} \quad (4)$$

where BP_s and BP_d is the systolic and diastolic blood pressure, respectively; F_s , F_d , and F_r is the systolic, diastolic, and regurgitant flow, respectively; PP, pulse pressure; SVR, systemic vascular resistance; and subscripts $_{-pre}$ and $_{-post}$ indicate pre- and post-transcatheter aortic valve implantation, respectively.

Subtracting Equation 3 from 4 and 1 from 2, assuming $SVR_{pre} = SVR_{post}$, yields

$$PP_{pre} = F_s \cdot SVR_{pre} \quad (5)$$

$$PP_{post} = (F_s + F_r) \cdot SVR_{post} \quad (6)$$

Dividing Equation 6 by 5 and rearranging yields

$$F_r = (PP_{post}/PP_{pre} - 1) \cdot F_s \quad (7)$$

Rearranging the formula for RAI in their report would yield a different formula:

$$RAI = [(BP_{d-post}/BP_{s-post}) - (BP_{d-pre}/BP_{s-pre})] \cdot 100 \quad (8)$$

However, despite a reasonably large series, multiple logistic regression analysis was not possible owing to issues with a lack of statistical

power. Echocardiographic assessment of PVL severity can be difficult owing to interpretation and blood pressure management during general anesthesia. RAI is also dependent on anesthetic management and vasoconstriction administration.

We thank Heinz and colleagues¹ for their work; however, we must caution against the adoption of RAI until these issues have been addressed.

Michael Poullis, BSc(Hons), MBBS,
MD, FRCS(CTh)
Department of Cardiothoracic
Surgery
Liverpool Heart and Chest Hospital
Liverpool, United Kingdom

Michael Poullis, BSc(Hons), MBBS,
MD, FRCS(CTh)
Department of Cardiothoracic
Surgery
Liverpool Heart and Chest Hospital
Liverpool, United Kingdom

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<http://dx.doi.org/10.1016/j.jtcvs.2014.02.020>

Reply to the Editor:

We thank Dr Poullis for his kind interest in our article and for his

comments. Despite acknowledging the practicability of an index that is based on hemodynamic criteria, he questions the value of the relative amplitude index (RAI) presented in our article. Although the issues raised are of great interest, he has probably misunderstood the main aim of introducing the RAI to assess the impact of paravalvular regurgitation (PAR) after transcatheter valve implantation.

Dr Poullis implies that the RAI has a lower value than echocardiography for assessing PAR. At this point, we would like to take the chance and underline that a comparison of established assessment methods for PAR with RAI was far beyond the aim of the study. It was not our intention to challenge echocardiography for assessment of valve function and PAR postimplantation. We do consider echocardiography to be the criterion standard for anatomic and functional evaluation of the implanted valve, and we regularly use it in all cases for intraprocedural and postprocedural assessment. Although the data depicting the relevance of PAR on survival are unquestionable, little is known regarding the prognostic capacity of detected PAR with respect to outcome. The RAI was therefore designed to distinguish between patients with moderate PAR and positive outcome from those with moderate PAR and negative outcome.¹ Several factors, such as preexisting aortic regurgitation, may influence the impact of PAR on outcome. In this study we were able to show that increased RAI was associated with both a relevant PAR and mortality in the perioperative period and at follow-up. This association is based on the finding that the degree of aortic regurgitation did not correlate with RAI, whereas relevant PAR did. The difference between preoperative and postoperative regurgitation seems to play the major role, however, and the standardized calculation of this

difference is the major contribution of RAI.

To elucidate further on the differences in outcome, we would like to mention the following observations: as shown in the Results section, 5 of 7 patients with a RAI of at least 14 were dead at follow-up. The mortality among patients with PAR was lower (7/11). A closer look at the dead patients revealed that all patients with both RAI of at least 14 and PAR of at least 2+ were dead, whereas mortality was higher among patients with RAI of at least 14 and no significant PAR than among patients with PAR of at least 2+ and normal RAI. It seems to us that RAI can discriminate between prognostically relevant PAR independent of the severity of the regurgitation. Moreover, there are still some patients without significant PAR but with a "pathologic" RAI, suggesting that other factors influencing hemodynamics may play a role in mortality after transcatheter valve implantation. This supported by the fact that 3 of 5 patients with RAI of at least 14 who died had no PAR. As stated in the Discussion section, severe atherosclerosis with increased arterial stiffness and subsequent low diastolic and mean arterial pressures may lead to end-organ hypoperfusion and failure in these patients.

Addressing the question on the correlation between RAI and degree of PAR, we could not calculate a correlation coefficient because the degree of PAR cannot be used as a continuous variable. With respect to the variables included for the risk factor analysis, in our opinion it would not be correct to add anatomic or radiologic covariates that are proven risk factors for PAR in the univariate analysis for 30-day mortality. The addition of these factors in the analysis would have increased confounder bias and for this reason was omitted. Furthermore, the possible association of RAI with perioperative complications, including renal and respiratory

failure as well as myocardial infarction and stroke, are summarized in the original article's Table 1.

Dr Poulis correctly addresses the topic of respiratory failure in patients undergoing transcatheter valve implantation. In our hands, the vast majority of postoperative respiratory failure cases were associated with pneumonia and infectious complications and not with preexisting pulmonary disease. This finding is not new and was expected in this high-risk and old group of patients.

We agree that RAI could also be dependent on blood pressure (BP) management and use of inotropic agents, as well as cardiac and rhythm instability. We tried to increase calculation precision by adopting a uniform protocol of standardized measurements. As stated in the text, preimplantation BP measurements were the average of 3 different time points between skin incision and balloon valvuloplasty under stable hemodynamic and rhythm conditions. Similarly, postimplantation data were averaged by considering 3 different measurements between valve implantation and skin suturing. To minimize the risk of error, we performed additional calculations of the RAI index from noninvasive measurements. Those results were also based on preoperative measurements at admission and the average of 5 postoperative measurements after extubation. Both the invasive and noninvasive measurements yielded equivalent results, with sensitivity and specificity of 71.4% and 95.1%, respectively, which we consider to represent a positive proof of concept for the proposed index.

With regard to the formula with which the index was calculated, we disagree with the use of the mere diastolic BP (DBP) and systolic BP (SBP) without taking into consideration the BP amplitude (pulse pressure).² A closer look at the original article's Figure E2, which shows that DBP alone does not correlate

with relevant PAR, confirms this assumption. Because we intended to calculate the influence of BP amplitude before and after valve implantation, both the SBP and the DBP were noted at those different time points (1 and 3 in equation). To yield reproducible results independently of the existing BP situation of the individual patient, a ratio of amplitude (1 and 3 in equation) divided by the current SBP (2 in equation) was calculated. Because many patients have a preexisting difference between SBP and DBP (high BP amplitude), the preimplantation ratio (subtrahend) was subtracted from the post implantation ratio (minuend), and the result (difference) was multiplied by 100% to get a common index.

$$RAI = \left(\frac{\text{Post-TAVI BP Amplitude (1)}}{\text{Post-TAVI SBP (2)}} - \frac{\text{Pre-TAVI BP Amplitude (3)}}{\text{Pre-TAVI SBP (2)}} \right) \times 100\%$$

Nikolaos Bonaros, MD, PhD

Anneliese Heinz, MD

Department of Cardiac Surgery

Innsbruck Medical University

Innsbruck, Austria

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<http://dx.doi.org/10.1016/j.jtcvs.2014.02.086>

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