LETTERS TO THE EDITOR

Aortic stiffness in aortic valve stenosis: reply

We appreciate the interest of Nemes and Forster in our article and we would like to warmly thank them. The main purpose of our study was to assess the relationship between aortic stiffness, left ventricular (LV) function, and BNP release in a series of patients with severe aortic stenosis (AS) and preserved LV ejection fraction. A normal control group would have been useful for the comparison of arterial mechanical properties, but matching on age and cardiovascular risk factors may remain challenging. Antonini-Canterin et al. showed in a previous study that carotid stiffness, assessed by e-tracking, is similar in patients with calcific AS and in control patients of similar age, sex, and cardiovascular risk factors. However, at the level of the ascending aorta the results may differ, and aortic stiffness in patients with AS could be higher than in matched control subjects, considering the possible damage to the endothelium due to turbulent flow transmitted from the stenotic aortic valve. This could also explain the improvement of aortic stiffness after aortic valve replacement in the study of Nemes et al. Whether this improvement in aortic stiffness could contribute to the reverse remodelling of LV after aortic valve replacement has to be demonstrated.

In our study, aortic beta index was used to assess arterial mechanical properties. It is less affected by arterial pressure changes since it is a parameter adjusted for the logarithmic relationship between stiffness indices and pressure. Young’s incremental elastic modulus, which takes into account the thickness of the arterial wall, estimates the elastic properties of the arterial wall material and could provide important additional information on the intrinsic arterial mechanical properties. Unfortunately, the current available transthoracic echocardiographic methods do not allow the assessment of this parameter at the level of the ascending aorta. To improve the assessment of aortic mechanical properties, future development of high sensitive e-tracking systems is needed.

The first echocardiographic study showing alterations in aortic stiffness in AS patients with normal epicardial coronary arteries was published in 2004. Transoesophageal echocardiography-derived aortic elastic modulus \(E(p)\) and Young’s circumferential static elastic modulus \(E(s)\) were found to be similarly significantly increased in AS patients with normal epicardial coronary arteries and cases with left anterior descending coronary artery stenosis, when compared with controls. In a subgroup analysis, AS patients with type 2 diabetes mellitus were demonstrated to have similar aortic distensibility compared with non-diabetic patients with AS.

Moreover, it was examined whether surgical solutions for AS could also have any effects on aortic function. Barbetseas et al. found that 1 week after aortic valve replacement (AVR) with a mechanical valve, a further increase in already elevated ASI could be demonstrated. The results of this study showed ameliorated aortic function (reduction in ASI) at 6-month follow-up postoperatively. In a longer 1-year follow-up study, a progressive improvement in ASI to levels comparable with age-, sex-, and risk factor-matched controls was found. Similar findings could be demonstrated with a transient immediate increase after aortic full root replacement (FRR) followed by a progressive decrease in ASI.

Due to the above-mentioned changes in aortic function in AS following surgery, further studies are warranted to examine the relationship between aortic stiffness and LV function after AVR/FRR.

Conflict of interest: none declared.

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References
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Relationship between aortic valve stenosis, its replacement, and aortic function

The paper of Rosca et al. was read with great interest entitled ‘Impact of aortic stiffness on left ventricular function and B-type natriuretic peptide release in severe aortic stenosis’. The major finding of this study was that in patients with severe aortic valve stenosis (AS) and preserved left ventricular (LV) ejection fraction, independently of the valvular load, an increase in aortic rigidity, as assessed by aortic stiffness index (ASI), is independently correlated with reduced LV longitudinal function, increased LV filling pressures, and BNP levels. The results are impressive, but we feel that a few additional comments are necessary.

References

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References

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Echocardiographic evaluation of systolic and mean pulmonary artery pressure in patients with pulmonary hypertension: reply

We would like to thank Chemla et al.1 for their interest in our recently published work. Their letter, however, raises some critical concerns that should be appropriately addressed.

In fact, as expected, in our patient population the mean bias of the modified formula is nearly zero, due to the property of the least squares regression optimization algorithm. Therefore, the standard deviation is comparable between the two formulas. In the validating sample, comparable absolute mean bias is observed between the ‘classical’ and modified formulas, with comparable standard deviations for systolic pulmonary artery pressure (sPAP) and a much smaller standard deviation for mean pulmonary artery pressure (mPAP) parameter in the modified formula.

As we underline also in our article, using the modified formulas, increasing values for Lin and decreasing values for Bland–Altman coefficients were found both in the initial and in the validating sample, indicating that the ‘advantages’ of using the modified formulas are mainly in increasing the grade of independence between bias and mean and not particularly on reducing the mean bias. Thus, we concluded that a larger population and further investigation regarding the correlation between echocardiographic and invasive evaluation are needed in order to improve the precision of the Bernoulli-derived formulas, and not simply identify corrections of the existing ones, as we did in this work.

Furthermore, as we remark in the end of the section ‘Results’, sensitivity, specificity, positive predictive value and negative predictive value were all calculated for both the equations, considering a 25 and a 40 mmHg cut-off, respectively, for the echocardiographically estimated mPAP and sPAP, as proposed by the current ESC Guidelines.2 Always present in our manuscript, we already acknowledged the limitation regarding possible bias imposed by poor quality in early echocardiography machines before 1995.

The conventional mPAP equation (i.e. mPAP = 0.61 × sPAP + 2) is—by definition—derived by the sPAP estimation with the simplified Bernoulli equation (4vTR + right atrial pressure).

Finally, the same reference appearing in our paper regarding the conventional mPAP formula3 has been used in the ESC Official Guidelines, co-authored by Drs Humbert and Simonneau, that also co-authored Chemla’s past work.4

Further studies in larger PH populations are necessary to address the utility of our two formulas.

References

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<th>Bias = [real-estimated] = residual</th>
<th>mPAPdiff_classical</th>
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<th>sPAPdiff_modified</th>
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