Prevalence and prognostic value of right ventricular dysfunction in severe aortic stenosis

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Aims
Systolic pulmonary artery pressure (sPAP) is a well-known outcome predictor in patients with valvular heart disease. Limited data are available regarding the evaluation of right ventricular (RV) performance, particularly in patients with aortic stenosis (AS). The aim of this study was to evaluate the prevalence, determinants, and prognostic significance of RV dysfunction in severe AS independently from the strategy of treatment chosen.

Methods and results
Two hundred patients (mean age: 79.9 ± 8.8 years) with severe AS underwent two-dimensional and speckle tracking echocardiography for the evaluation of left ventricular (LV) and RV functions, aortic valve gradients, and sPAP. A tricuspid annular plane systolic excursion (TAPSE) ≤17 mm defined RV dysfunction. RV dysfunction was detected in 48 patients (24%). At multivariable regression analysis, LV global longitudinal strain (r = −0.29, P = 0.001), mean aortic gradient (r = 0.25, P = 0.002), and LV ejection fraction (r = 0.18, P = 0.02) were well correlated with TAPSE. After a median 16-month follow-up, cardiovascular death occurred in 17 patients. At multivariate Cox regression analysis, biventricular dysfunction (TAPSE ≤17 mm and LVEF ≤50%) emerged as the strongest predictor of prognosis (hazard ratio 4.08, 95% confidence interval 1.36–12.22; P = 0.012).

Conclusions
RV dysfunction is common in AS patients, and this finding can likely be accounted for by the RV–LV interdependence. Given that biventricular function impairment was a strong predictor of mortality in our population, we suggest that RV dysfunction should be systematically looked for in AS patients.

Keywords
Aortic stenosis ● Right ventricle ● Cardiovascular mortality

Introduction
Right ventricular (RV) dysfunction is a well-known predictor of both morbidity and mortality in various cardiovascular (CV) diseases, including heart failure (HF), myocardial infarction, and primary pulmonary hypertension (PPH). In patients with valvular heart disease, PH—rather than RV dysfunction—has been traditionally considered a predictor of poor prognosis, particularly in mitral regurgitation (MR). Current European recommendations specify, in fact, that a systolic pulmonary artery pressure (sPAP) >50 mmHg at rest and an exercise sPAP >60 mmHg should be an indication for surgery (Classes IIa and IIb, respectively) in asymptomatic patients with severe primary MR. Neglecting the evaluation of the RV as opposed to the left ventricle (LV) is due to that PH is considered the main determinant of RV dysfunction in left-sided heart diseases. Although several studies have addressed the evaluation of sPAP in aortic stenosis (AS) patients, along with its impact on patient survival, scarce data are available regarding the prevalence and prognostic significance of RV dysfunction. In spite of this, there is some evidence supporting a relationship between increased LV afterload and RV remodelling and dysfunction. Among several echocardiographic measures of RV function that have been proposed, tricuspid annular plane systolic excursion (TAPSE) is the one that has been more extensively studied. In patients with various CV diseases, a cut-off TAPSE of 17 mm was able to identify patients with RV dysfunction and to predict poor prognosis. The present study sought therefore to assess the prevalence and prognostic impact of RV dysfunction, as assessed by TAPSE, in AS patients.

Methods
Between January 2012 and June 2013, 200 severe AS (aortic surface <1 cm² or <0.6 cm²/m²) patients underwent standard transthoracic
echocardiography for the evaluation of aortic valve gradients, LV and RV functions, as well as concomitant valvulopathies. Two-dimensional (2D) speckle tracking echocardiography was performed to assess LV global longitudinal strain (LV-GLS). Patients with suboptimal acoustic windows, cardiomyopathies (e.g., dilative cardiomyopathy, alcoholic cardiomyopathy, cardiac amyloidosis etc.), and concomitant more than moderate valve disease were excluded from analysis. Clinical data including New York Heart Association (NYHA) class, age, gender, obesity, hypertension, hypercholesterolaemia, diabetes, coronary artery disease (CAD), and treatments were collected for each patient. The study protocol was approved by the local ethical committee, and all patients provided written consent prior to study participation.

**Doppler echocardiography**

All patients underwent standard transthoracic echocardiography using a Vivid 7 or Vivid 9 ultrasound system (GE Vingmed, Milwaukee, WI, USA) equipped with a M5S 3.5-MHz transducer. M-mode, 2D, color Doppler, pulsed-wave and continuous-wave Doppler data were stored onto a dedicated workstation for offline analysis (EchoPAC, GE Healthcare, Horten, Norway). Severity of AS was assessed according to the current guidelines. 

Continuous-wave Doppler was used to measure aortic transvalvular velocities. Peak aortic gradient and mean aortic gradient (MAG) were measured using the modified Bernoulli equation. The aortic valve area (AVA) was calculated by means of the standard continuity equation. LV dimensions and mass were measured in accordance with the joined European Association of Echocardiography and American Society of Echocardiography guidelines. LV function was obtained using the Simpson biplane method of discs from the apical four- and two-chamber views. 

Tissue Doppler imaging was used to detect lateral and septal mitral annulus velocities, which were averaged to obtain $e\prime_m$ velocity as a measure of global LV longitudinal function. 

The ratio of early transmirtal flow velocity to tissue Doppler imaging (TDI) annular velocity ($E/e\prime$) was considered an index of mean LV filling pressure. 

RV function was estimated by TAPSE, by placing an M-mode cursor through the lateral tricuspid annulus in a four-chamber view. The peak excursion of the tricuspid annulus was measured and the average value over 3–5 heart beats was taken into account. 

The maximal lateral tricuspid annulus velocity measured at TDI ($c\prime_m$) was also used to evaluate RV performance, and $c\prime_m \leq 9\, \text{cm/s}$ defined RV dysfunction as recommended. 

The sPAP was determined from the tricuspid regurgitation (TR) jet velocity using the modified Bernoulli equation, and this value was combined with an estimated (MAG) were measured using the modified Bernoulli equation. 


dependent from the tricuspid regurgitation (TR) jet velocity using the modified Bernoulli equation, and this value was combined with an estimated right atrial pressure by means of the diameter and collapsibility of the inferior vena cava. To calculate LV-GLS, 2D greyscale images were acquired in the standard apical four-, three-, and two-chamber views at a frame rate of at least 80 frames/s. During offline analysis, a line was traced along the endocardium’s inner border in each of the three apical views on an end-systolic frame, and a region of interest was automatically defined between the endocardial and epicardial borders, with GLS then automatically calculated from the strain in the three apical views.

**Follow-up**

Follow-up data were obtained by means of phone contacts with patients, their general practitioners, or their cardiologists. Particular care was taken to collect data pertaining to HF occurrences, hospitalization causes, and patient deaths. The clinical management of patients was determined independently by their personal physicians. The study’s primary endpoint was CV mortality, and its secondary endpoint was major adverse cardiovascular events (MACEs). MACEs were defined as the first event occurring among HF, cardiac-related hospitalization, and overall mortality.

**Statistical analysis**

Data were expressed as mean $\pm$ standard deviation for continuous variables and as percentages for categorical variables. Between-group comparisons were performed using Student’s t-tests or $\chi^2$ test, as appropriate. To identify physiological correlates of RV function, univariate linear regression analysis was carried out. After excluding variables showing collinearity (Pearson’s coefficient $>0.6$), all the variables that were significant at univariate analysis were entered into a stepwise multivariate regression analysis.

Receiver operator characteristic (ROC) curves considering multiple dichotomies of TAPSE (14, 15, 16, 17, and 18 mm) and $c\prime_m$ (8, 9, 10, 11, and 12 cm/s) were obtained. The relative areas under the curves (AUCs) were then compared to verify that our cut-off was appropriate for mortality outcomes. The cut-off having the higher AUC was then retained for survival analysis. Long-term cardiovascular survival rates were calculated by means of the Kaplan–Meier method. The log-rank test and Breslow’s test were used to compare event rates. Univariate Cox regression analysis was applied to assess the prognostic value of various clinical and echocardiographic parameters with respect to the predefined endpoints. Multivariate Cox regression analysis was then performed (forward stepwise method, entry and removal value 0.05–0.10), providing that covariates were not too highly correlated (Spearman’s coefficient $<0.6$). Hazard ratios (HRs) were estimated after adjusting for other outcome predictors, and covariates were required to have a P-value of $<0.05$ to enter in the final model. Statistical analysis was performed using SPSS Version 20.0 (Chicago, IL, USA).

**Results**

**Patient population**

All clinical and echocardiographic characteristics of our population are summarized in Table 1.

**RV function evaluation**

RV dysfunction according to a TAPSE of $\leq 17\, \text{mm}$ was observed in 48 patients (24%). RV dysfunction patients displayed a significant reduction in all LV function parameters, including LVEF, $e\prime_m$, and LV-GLS, and exhibited more elevated sPAP values. No differences in AS severity were found between the two groups, but a low-flow/low-gradient AS pattern was more common in RV dysfunction patients (Table 1).

RV function correlates at univariate regression analysis are given in Table 2.

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RV function correlates at univariate regression analysis are given in Table 2.

LV-GLS ($r = -0.40, P < 0.0001$), LVEF ($r = 0.35, P < 0.0001$), $e\prime_m$ ($r = 0.27, P < 0.0001$), and indexed left atrial volume (LAV) ($r = -0.30, P = 0.001$) displayed the strongest correlation with TAPSE (Figure 1). Of note is that the correlation of sPAP with TAPSE was weak ($r = -0.27, P = 0.01$) when compared with other parameters (Table 2, upper panel). Two multivariate regression models were then performed, using alternatively LVEF and GLS as a measure of LV performance (Table 2, lower panels). In Model A, LVEF ($r = 0.18, P = 0.02$), MAG ($r = 0.25, P = 0.002$), and LAV ($r = -0.17, P = 0.04$) were significantly correlated with TAPSE, whereas sPAP was not. In Model B, when LV-GLS was used to estimate LV function, sPAP remained associated with TAPSE ($r = -0.20, P = 0.03$), whereas MAG ($r = 0.23, P = 0.01$) and LV-GLS ($r = -0.29, P = 0.001$) exhibited the strongest correlation.
Follow-up

Follow-up data were available for all patients, median follow-up lasting 16 (range: 1–20.7) months. During follow-up, CV death occurred in 17 patients (8.5%) and MACEs were observed in 53 patients (22.8%). Our cohort included 50 patients (25.4%) presenting HF symptoms and 43 patients (21.5%) died within the first year of follow-up. The development of HF symptoms was associated with a higher rate of MACEs (36%, p = 0.01). Additionally, the presence of HF symptoms at baseline was predictive of HF development during follow-up (odds ratio 6.9, 95% CI 2.1–22.2, p = 0.001).

All data are expressed as mean ± standard deviation or numbers and percentages.

ACE, angiotensin-converting enzyme; AVA, aortic valve area; IVSd, end-diastolic interventricular septum thickness; LFLG, low-flow/low-gradient aortic stenosis; LFNG, low-flow/normal-gradient aortic stenosis; LV, left ventricle; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic diameter; LV-GLS, left ventricular global longitudinal strain; MAG, mean aortic gradient; NFLG, normal-flow/low-gradient aortic stenosis; NFNG, normal-flow/normal-gradient aortic stenosis; NYHA, New York Heart Association functional class; sPAP, systolic pulmonary artery pressure; TAPSE, tricuspid annular plane systolic excursion; MR, mitral regurgitation.
other tested values [AUC 0.60, confidence interval (CI) 0.42–0.72 and AUC 0.58, CI 0.42–0.72, respectively], and were therefore retained for survival analysis.

At Kaplan-Meier analysis, RV dysfunction described either by a TAPSE of $\leq 17$ mm or a $s'_t$ of $\leq 9$ cm/s was associated with a slightly elevated mortality (Figure 2A and B). When considering LV and RV functions, patients with LVEF $>50\%$ had a longer survival regardless of normal or reduced TAPSE values ($24.3 \pm 0.4$ and $23.5 \pm 1.1$ months, respectively, $P = \text{NS}$), whereas the combination of RV and LV dysfunctions (LVEF $\leq 50\%$ and TAPSE $\leq 17$ mm) was associated with a dramatic decrease in survival time ($19.7 \pm 1.7$ months, $P = 0.02$; Figure 3). Predictors of CV mortality and MACEs at univariate Cox regression analysis are given in Table 3. After correcting for CAD, AVA, and sPAP, the combination of either a TAPSE of $\leq 17$ mm and an LVEF of $\leq 50\%$ or of a $s'_t$ of $\leq 9$ cm/s and an LVEF

### Table 2 Uni- and multivariate predictors of TAPSE at linear regression analysis

<table>
<thead>
<tr>
<th>Predictors</th>
<th>$r$</th>
<th>$P$-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>IVSd</td>
<td>-0.046</td>
<td>0.53</td>
</tr>
<tr>
<td>LVEDD*</td>
<td>0.066</td>
<td>0.36</td>
</tr>
<tr>
<td>LVESD*</td>
<td>-0.19</td>
<td>0.007</td>
</tr>
<tr>
<td>LV mass index</td>
<td>0.079</td>
<td>0.27</td>
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<tr>
<td>Relative wall thickness</td>
<td>0.034</td>
<td>0.65</td>
</tr>
<tr>
<td>Left atrial volume index</td>
<td>-0.30</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Aortic valve area</td>
<td>0.17</td>
<td>0.01</td>
</tr>
<tr>
<td>Peak aortic velocity*</td>
<td>0.24</td>
<td>0.001</td>
</tr>
<tr>
<td>Mean aortic gradient</td>
<td>0.19</td>
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</tr>
<tr>
<td>sPAP</td>
<td>-0.27</td>
<td>0.01</td>
</tr>
<tr>
<td>Mean mitral E/e'</td>
<td>-0.057</td>
<td>0.45</td>
</tr>
<tr>
<td>Mean $S'$ mitral annulus*</td>
<td>0.27</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LVEF</td>
<td>0.35</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LV-GLS</td>
<td>-0.40</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
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Multivariate regression analysis—Model A

- Left atrial volume index: $-0.17$, $p = 0.04$
- Aortic valve area: $0.14$, $p = 0.08$
- Mean aortic gradient: $0.25$, $p = 0.002$
- sPAP: $-0.12$, $p = 0.12$
- LVEF: $0.18$, $p = 0.02$

Multivariable regression analysis—Model B

- Left atrial volume index: $-0.14$, $p = 0.11$
- Aortic valve area: $0.08$, $p = 0.52$
- Mean aortic gradient: $0.23$, $p = 0.01$
- sPAP: $-0.20$, $p = 0.03$
- LV-GLS: $-0.29$, $p = 0.001$

*Variables not included in the multivariate modelling because highly correlated with another variable.
of ≤50% resulted the strongest predictor of CV death (HR 4.08, 95% CI: 1.36–12.22, \(P = 0.012\) and HR 3.1, 95% CI: 0.96–10.07, \(P = 0.05\), respectively). At multivariable Cox regression analysis, CAD (HR 3.2, 95% CI: 1.36–12.22, \(P = 0.01\)), moderate MR (HR 7.25, 95% CI: 2.96–17.76, \(P = 0.0001\)), and LV-GLS (HR 1.18, 95% CI: 1.04–1.34, \(P = 0.01\)) were predictors of MACEs.

**Discussion**

To the best of our knowledge, no study has so far specifically addressed the evaluation of RV function in AS patients. In this study involving 200 severe AS outpatients, a TAPSE of ≤17 mm was used to define RV dysfunction. According to this cut-off, the prevalence of RV impairment in our population was 24%. LVEF, LV-GLS, and MAG had a good correlation with TAPSE, whereas the correlation between TAPSE and sPAP was weaker or absent. These results suggest that the ventricular interdependence might have a fundamental role to play in the pathophysiology of RV dysfunction in AS. Biventricular impairment was associated with increased CV mortality as well. Our results emphasize that, in AS, prognosis depends not only on stenosis severity and LVEF, but rather on the concomitant development of RV dysfunction. This supports the necessity of a routine RV assessment in AS patients.

**Prevalence of RV dysfunction in severe AS**

Numerous surveys have shown the negative prognostic impact of PH in MR patients. On the contrary, only few studies have been published, which show the negative prognostic impact of PH in AS. Following aortic valve replacement, a temporary reduction in sPAP has been observed, yet sPAP values were shown rise again 12 months after the intervention, which suggests that irreversible alterations of RV function or pulmonary vascular bed might have occurred. Our study is the first to show that RV dysfunction is rather common in AS, as it was detected in one quarter of the enrolled patients. RV performance was evaluated using 2D echocardiography, which is the referral technique for assessing the aetiology and type of valve disease, as well as global heart performance. Among the various methods used to assess RV function, we selected TAPSE owing to its feasibility, high reproducibility, and robust predictive power. TAPSE values have been shown to correlated with radionuclide angiography and have been validated against biplane Simpson RV ejection fraction and RV fractional area shortening. A \(s'_\text{tr} \leq 9\) cm/s was used as a further index of RV dysfunction, and a reduced \(s'_\text{tr}\) confirmed our data on the negative prognostic impact of RV dysfunction in AS. With respect to TAPSE, however, less normative data are available for \(s'_\text{tr}\) and this is particularly true in the elderly as in the case of degenerative AS.

**Pathophysiological significance of RV dysfunction in AS**

According to a traditional model, in the context of left-sided heart disease, the progressive LV remodelling results in an increased LV end-diastolic pressure that can be transmitted backwards, leading to left atrial dilatation and increased pulmonary post-capillary...
...ventricular interdependence might be partially mirror what already observed by Le Tourneau et al. in severe MR, thereby supporting that ventricular interdependence might be the main explanation of RV dysfunction also in the case of increased LV afterload. The RV and LV are intimately attached through the interventricular septum, sharing common fibres that encircle both cardiac chambers. In AS, the chronic elevation in LV afterload causes LV hypertrophy and fibrosis, leading to a progressive deterioration in LV longitudinal function and finally to a decrease in LVEF. The alteration in LV function might therefore be transmitted to the RV through the interventricular septum, as already observed in some experimental models of AS as well as in HF with reduced or preserved LVEF. Interestingly, the positive effect of LV contraction on RV function was enhanced in left-sided pressure overload cases, which explains the positive relationship between MAG and TAPSE observed in our survey, along with the higher prevalence of RV dysfunction among low-flow/low-gradient AS patients. The molecular mechanisms and the proximity effects of growth-stimulating signals like angiotensin I and catecholamines produced in the hypertrophied LV might conceivably account for RV remodelling and functional deterioration in AS. This hypothesis is supported by the evidence that a moderate increase in LV afterload, as observed in essential hypertension, was shown to be associated with RV wall thickening and concentric remodelling detected at cardiac MRI and with RV longitudinal function impairment.

Clinical significance of RV function evaluation in AS

PH has been traditionally described as a crucial determinant of outcome in numerous heart diseases. In our study, RV dysfunction alone was responsible for a slight increase in CV mortality, whereas the combination of RV and LV dysfunction turned out to be a significant predictor of CV mortality, which has interesting clinical implications. At present, the management of AS patients is primarily based on the assessment of AS severity, LVEF, and symptom onset. Nevertheless, as previously stated by Pibarot and Dumesnil, AS should not be considered a disease entity limited to the aortic valve, but rather a systemic disease that is characterized by a concomitant...
deep alteration in the whole heart structure and function. Our study emphasizes the crucial role of biventricular function evaluation in AS patients. The primary structural abnormalities at the LV level might, in fact, spread to the RV along the anatomical and functional ventricular continuum, thereby influencing the prognosis of AS patients.

Limitations

This is a retrospective study conducted on consecutive AS patients meeting the eligibility criteria. CAD patients were included in this survey, because CAD is the most common comorbidity in AS. Patients with moderate MR were also included in our study. The prevalence of MR was slightly higher in cases of RV dysfunction, potentially influencing RV function deterioration.27 Despite this, moderate MR was associated with MACEs, but not with CV mortality at Cox analysis, thus supporting its influence on symptom onset and hospitalization more than on mortality. In our study, TAPSE and s′Lx were used to estimate RV function. Although these methods are easy to perform in routine clinical practice and have been validated in large patient cohorts, they derive the global RV performance from the simple displacement of the lateral tricuspid annulus during systole, such disregarding the contribution of the interventricular septum and RV outflow tract towards overall RV performance. Another limitation consisted in the short follow-up period, which probably accounted for the limited number of events recorded in this population. In our study, LV function, rather than sPAP, resulted the main determinants of RV performance. Although this finding may be explained by ventricular interdependence, our data refer to overall LV performance. Yet no relationship was found between LV hypertrophy and RV function, and no specific analysis was performed to describe regional LV function abnormalities (e.g., LV septal segments with respect to other ventricular segments). These limitations might also explain the low correlation coefficients we found between TAPSE and LV function parameters. Our results need to be confirmed in larger surveys, and the pathophysiological mechanisms we outlined should be corroborated by using more sensible methods of biventricular function estimation.

Conclusions

RV dysfunction according to TAPSE values is common in AS patients. The correlation between TAPSE and LV function parameters suggests that the RV–LV interdependence is a fundamental phenomenon in AS. Given that biventricular function impairment was a strong predictor of CV mortality in our population, we propose that RV function should be carefully looked for in AS patients. Further studies are needed to confirm the prognostic value of RV dysfunction in severe AS patients and investigate the RV reverse remodeling following AS treatment.

Conflict of interest: none declared.

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Benign primary cardiac neoplasm with intense FDG uptake

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A 48-year-old woman was referred to the cardiology division under suspicion of cardiac metastasis of melanoma. She had previously been diagnosed with orbital melanoma, and her right eye had been enucleated 1 year prior to her referral. Although there was no other metastatic lesion, a round-shaped mass with intense FDG uptake was found beside the right heart on positron emission tomography–computed tomography (PET–CT; Panels A and B). Echocardiography revealed a round-shaped mass at the right atrioventricular groove (Panel C, see Supplementary data online, Video S1). Cardiac magnetic resonance confirmed a well-demarcated, round mass with a size of 2.8 cm along the right atrioventricular groove. This mass showed intermediate signal intensity on both T1- and T2-weighted images without any delayed enhancement or fat suppression (Panels D and E). Due to her complaints of palpitation and paroxysmal elevations in blood pressure found in careful history-taking, a 24-h urine study was conducted and significant overproduction of catecholamine was proved.

Preoperative coronary angiography revealed a hypervascular cardiac mass supplied by multiple feeding vessels from the right coronary artery (Panel F, see Supplementary data online, Video S2). After a 2-week pretreatment with alpha- and beta-blockers, the mass was excised with meticulous ligation of the feeding vessels (Panel G). A histological examination revealed findings that were all compatible with pheochromocytoma (Panel H).

Cardiac pheochromocytoma is highly vascular and tends to involve the coronary arteries. It shows intense FDG uptake on PET–CT, although it is one of the benign tumours. Clinical suspicion followed by multimodality imaging is crucial for the diagnosis and treatment of cardiac pheochromocytoma.

Supplementary data are available at European Heart Journal – Cardiovascular Imaging online.

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