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One disease, two ventricular responses: rethinking left ventricular thresholds in severe aortic regurgitation

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Introduction: Current guidelines for severe aortic regurgitation (AR) incorporate symptoms, left ventricular (LV) dimensions and ejection fraction (LVEF) when determining the timing of intervention. However, conventional LV thresholds may not fully reflect differences in ventricular remodeling related to age, sex and previous hemodynamic loading conditions[1-3]. We present two contrasting cases illustrating the importance of interpreting LV dimensions and function within the broader clinical context.

TABLE 1. Comparative echocardiographic assessment of aortic regurgitation severity and left ventricular remodelling in two patients.

	Patient 1	Patient 2
Qualitative parameters:		
1. Doppler jet width	large, eccentric	large, eccentric
2. flow convergence zone	large	large
3. diastolic flow reversal in descending aorta	holodiastolic	holodiastolic
Semi-quantitative parameters:		
1. vena contracta width	0.6 cm	/
2. jet width/LVOT width	45%	42%
3. PHT	365 ms	300 ms
Quantitative parameters:		
1. EROA	0.5 cm^2	/
2. regurgitant volume	72mL	/
3. regurgitant fraction	46%	/
Haemodynamic consequences:		
1. LVEF	57 %	65%
2. LVEDD/LVESD	57/41 mm	47/33 mm
3. LVESDi	21 mm/m^2	19 mm/m^2
4. LVESVi	33 ml/m^2	21 ml/m^2
5. diastolic function:	/	7
E/e'	0,07 m/s	/
e' septal	0,07 m/s	0,13 m/s
e' lateral	trivial	1,67 m/s
TR Velocity	35 ml/m^2	21 ml/m^2
LAVI		

LVOT, left ventricular outflow tract; PHT, pressure half-time; EROA, effective regurgitant orifice area; LVEF, left ventricular ejection fraction; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LVESDi, left ventricular end-systolic diameter indexed to body surface area; LVESVi, left ventricular end-systolic volume indexed to body surface area; TR, tricuspid regurgitation; LAVI, left atrial volume index

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Case report: 46-year-old man (Patient 1) with a bicuspid aortic valve (AV) and no significant comorbidities presented with severe native AR and minimal symptoms. Echocardiography demonstrated LV dilatation (LV end-diastolic diameter [LVEDD] 57mm; LV end-systolic diameter [LVESD] 41mm; indexed LVESD 21 mm/m²), with LVEF of 57%. Indexed LV end-diastolic and end-systolic volumes were 77 mL/m² and 33 mL/m², respectively. In contrast, a 66-year-old woman (Patient 2) with previous mechanical AV replacement for severe aortic stenosis presented with minimal-exertion dyspnea. Echocardiography revealed severe paravalvular regurgitation with suspected partial prosthetic dehiscence. Despite significant symptoms, LV dimensions remained within normal limits (LVEDD 47mm; LVESD 33mm; indexed LVESD 19 mm/m²), with LVEF of 65%. Indexed LV end-diastolic and end-systolic volumes were 59 ml/m² and 21 mL/m², respectively. In Patient 1, septal and lateral e' velocities were both 7 cm/s, with a borderline left atrial volume index (LAVI) of 35 mL/m². Interference from the AR jet prevented reliable assessment of transmitral inflow, leaving LV filling pressures indeterminate. In Patient 2, the available echocardiographic indices were consistent with normal resting LV filling pressures (**Table 1**). However, her history of dyspnea on minimal exertion raised the possibility of an exercise-induce increase in filling pressures, which was not assessed directly. Previous pressure overload may have influenced LV geometry and compliance in the second patient, potentially modifying the ventricular response to regurgitant volume. Both patients underwent multidisciplinary Heart Team evaluation and were accepted for cardiac surgery.

Conclusion: These cases illustrate contrasting patterns of LV remodeling in severe AR. While the younger patient exhibited LV dilatation, the older patient developed significant symptoms without marked ventricular enlargement. Differences in age, sex and previous hemodynamic loading may contribute to these findings. Contemporary studies suggest that adverse outcomes can occur below traditional LV dimension thresholds and that indexed LV volumes may provide additional prognostic information[2-4]. Preserved LVEF and the absence of substantial LV dilatation should therefore not be interpreted as sufficient evidence of favorable ventricular adaptation. An integrated assessment of regurgitation severity, LV geometry, ventricular function and clinical status remains essential when determining the timing of intervention.

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