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ORIGINAL RESEARCH

Impact of Pressure Recovery on Echocardiographic Assessment of Asymptomatic Aortic Stenosis: A SEAS Substudy

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OBJECTIVES The aim of this analysis was to assess the diagnostic importance of pressure recovery in evaluation of aortic stenosis (AS) severity.

BACKGROUND Although pressure recovery has previously been demonstrated to be particularly important in assessment of AS severity in groups of patients with moderate AS or small aortic roots, it has never been evaluated in a large clinical patient cohort.

METHODS Data from 1,563 patients in the SEAS (Simvastatin and Ezetimibe in Aortic Stenosis) study was used. Inner aortic diameter was measured at annulus, sinus, sinotubular junction, and supracoronary level. Aortic valve area index (AVAI) was calculated by continuity equation and pressure recovery and pressure recovery adjusted AVAI (energy loss index [ELI]), by validated equations. Primarily, sinotubular junction diameter was used to calculate pressure recovery and ELI, but pressure recovery and ELI calculated at different aortic root levels were compared. Severe AS was identified as AVAI and ELI $\leq$0.6 cm^2/m^2. Patients were grouped into tertiles of peak transaortic velocity.

RESULTS Pressure recovery increased with increasing peak transaortic velocity. Overestimation of AS severity by unadjusted AVAI was largest in the lowest tertile and if pressure recovery was assessed at the sinotubular junction. In multivariate analysis, a larger difference between AVAI and ELI was associated with lower peak transaortic velocity (beta = 0.35) independent of higher left ventricular ejection fraction (beta = −0.049), male sex (beta = −0.075), younger age (beta = 0.093), and smaller aortic sinus diameter (beta = 0.233) (multiple $R^2 = 0.18$, $p < 0.001$). Overall, 47.5% of patients classified as having severe AS by AVAI were reclassified to nonsevere AS when pressure recovery was taken into account.

CONCLUSIONS For accurate assessment of AS severity, pressure recovery adjustment of AVA must be routinely performed. Estimation of pressure recovery at the sinotubular junction is suggested. (J Am Coll Cardiol Img 2010;3:555–62) © 2010 by the American College of Cardiology Foundation

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Downstream pressure recovery in the aorta affects transvalvular pressure gradient measurement and estimation of aortic valve area (AVA) by continuity equation in patients with aortic stenosis (AS) (1). Convergence of flow through the stenotic aortic valve to the vena contracta converts potential energy to kinetic energy with a resulting reduction in pressure at the vena contracta (2–4). As streamlines then diverge and slow again distal to the vena contracta, there is reconversion of some kinetic energy to potential energy with recovery of a proportion of the pressure lost from the left ventricular (LV) outflow tract to the vena contracta (5). Because Doppler-based methods detect peak flow velocity that occurs at the vena contracta, the transvalvular pressure drop estimated by Doppler will be greater than that calculated from simultaneous invasive pressure measurements in the LV outflow tract and in the aortic root distal to the vena contracta. The implication of pressure recovery is that AVA calculated by Doppler-based methods underestimates valve area calculated by catheter-based methods where aortic pressure is routinely measured in the aortic root several centimeters distal to the aortic valve (1,6–8). To overcome these limitations, Garcia et al. (9) derived an equation to calculate the pressure recovery adjusted effective valve area, named energy loss index (ELI). Although pressure recovery has previously been demonstrated to be particularly important in patients with moderate AS and small aortic roots (1,8,10), it has never been evaluated in a large, homogeneous cohort, and so its diagnostic importance has not been assessed. Thus, the aim of the present study was to evaluate the impact of pressure recovery on assessment of AS severity in asymptomatic patients recruited in the SEAS (Simvastatin and Ezetimibe in Aortic Stenosis) study (11).

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ABBREVIATIONS AND ACRONYMS

AS = aortic stenosis
AVA = aortic valve area
AVAI = aortic valve area index
ELI = energy loss index
Junctional = sinotubular junction
LV = left ventricle

METHODS

Study population. The present substudy was prospectively planned within the SEAS study that enrolled 1,873 patients with asymptomatic AS, defined by echocardiography at local study centers as aortic valve thickening and peak transaortic Doppler velocity ≥2.5 and ≤4.0 m/s. Patients were randomized from January 2001 to February 2004 in 173 European study centers to ≥4-year placebo-controlled combined treatment with ezetimibe 10 mg/day and simvastatin 40 mg/day. Patients with coronary heart disease, heart failure, diabetes, history of stroke or peripheral vascular disease, clinically significant mitral valve disease, severe or predominant aortic regurgitation, rheumatic valvular disease, aortic valve prosthesis, or renal insufficiency, and patients already on lipid-lowering therapy were not included in the SEAS study. The SEAS study protocol, design, and patient recruitment was recently published (11).

The present study population comprises the 1,563 (83.4%) of the total 1,873 patients recruited in the SEAS study, in whom AVA and aortic diameter at the sinotubular junction (junctional) could be measured on the baseline echocardiogram. Compared with the 311 ineligible patients, the present study population did not differ in age, sex, or body mass index. The SEAS study was approved by regional ethics committees in all participating countries. All patients gave written informed consent to participate in the SEAS study.

Echocardiography. Baseline echocardiograms were obtained using echocardiographs with second harmonic imaging and following a standardized protocol in all participating hospitals (12). All echocardiograms were sent for expert interpretation at the SEAS echocardiography core laboratory at Haukeland University Hospital, Bergen, Norway.

Quantitative echocardiography was performed following the joint European Association and American Society of Echocardiography guidelines (13). End-diastolic diameter of the aortic root was measured at annulus, sinus, junctional, and supracoronary (1 cm distal to junction) aortic level in 2-dimensional parasternal long-axis view by an inner-edge-to-inner-edge method. LV stroke volume and ejection fraction were measured by biplane Simpson method (13). LV stroke volume <22 ml/m$^{2.04}$ was defined as low, which corresponds to the previous cutoff of 35 ml when correcting for body surface area (13). Subaortic and transaortic blood velocities and gradients were derived from velocity time integrals, measured by pulsed-wave Doppler in the LV outflow tract and by continuous-wave Doppler from different windows by imaging and nonimaging transducers, respectively. The highest transaortic velocity was used for tracing of the time-velocity integral. The effective AVA was

Table 1. Characteristics of the Total Study Population

Variable	n = 1,563
Age, yrs	67 ± 10
Women, n (%)	607 (39)
Systolic blood pressure, mm Hg	144 ± 20
Diastolic blood pressure, mm Hg	82 ± 10
Heart rate, beats/min	66 ± 12
History of hypertension, n (%)	803 (51)
Height, cm	170 ± 9
Weight, kg	78 ± 15
Body surface area, m^2	1.89 ± 0.20
Body mass index, kg/m^2	26.9 ± 4.4

All p = NS between tertiles of peak transaortic velocity.

calculated using the continuity equation with the velocity time integrals and indexed for body surface area (AVAI). Pressure recovery (mm Hg) was calculated as $4v^2 \times 2AVA/Aa[1 - (AVA/Aa)]$, where v is the maximum Doppler transvalvular velocity (1,6), AVA is calculated by the continuity equation, Aa is the aortic area, and pressure recovery adjusted AVAI (i.e., the ELI $[cm^2/m^2]$ as: $AVA \times Aa/(Aa - AVA)/m^2$) (1,9). Severe AS was defined conventionally as AVAI ≤ 0.6 cm^2/m^2 and adjusted for pressure recovery as ELI ≤ 0.6 cm^2/m^2 (14–16).

Statistical analysis. The study population was divided into tertiles of peak transaortic velocity (Group #1: <2.79 m/s; Group #2: 2.79 to 3.33 m/s; Group #3: >3.33 m/s). Continuous variables are presented as mean ± SD and categorical variables as percentages. Comparisons of groups were performed by analysis of variance and Sidak post hoc test. Pressure recovery and ELI at different aortic levels were compared by parametric statistics. Univariate correlations were assessed by Pearson correlation coefficients. Multivariate linear regression analysis was used to identify independent covariates of pressure recovery. Results are given as standardized beta coefficients for individual variables and as multiple R^2 for the model. To demonstrate the association of pressure recovery with aortic root dimension, the study population was also divided into tertiles of junctional aortic diameter. Interaction between AVAI and pressure recovery in predicting AS was tested in a logistic regression model. A p value ≤ 0.05 was considered statistically significant in both univariate and multivariate analyses.

Table 2. Echocardiographic Findings in the Total Study Population and When Divided Into Tertiles of Peak Transaortic Jet Velocity

Variables	<2.79 m/s (Tertile #1)	2.79–3.33 m/s (Tertile #2)	>3.33 m/s (Tertile #3)	Total
Annular diameter, cm	2.18 ± 0.27	2.19 ± 0.24	2.20 ± 0.28	2.19 ± 0.26
Sinus diameter, cm	3.05 ± 0.43	3.10 ± 0.45	3.10 ± 0.45	3.08 ± 0.44
Junctional diameter, cm	2.79 ± 0.40	2.82 ± 0.43	2.84 ± 0.45	2.82 ± 0.43
Supracoronary diameter, cm	3.07 ± 0.44	3.14 ± 0.49	3.13 ± 0.48	3.11 ± 0.47
LV end-diastolic diameter, cm	5.04 ± 0.64	5.03 ± 0.63	5.07 ± 0.62	5.05 ± 0.63
Septal wall thickness, cm	1.12 ± 0.26*	1.15 ± 0.27*	1.21 ± 0.29	1.16 ± 0.28
Posterior wall thickness, cm	0.87 ± 0.19*	0.88 ± 0.18*	0.92 ± 0.19	0.89 ± 0.19
LV mass, g	187 ± 67*	191 ± 63*	207 ± 69	195 ± 67
LV mass index, g/m^2	98 ± 31*	100 ± 29*	108 ± 32	102 ± 31
Stroke volume, $ml/m^{2.04}$	22 ± 5	23 ± 7	25 ± 9	24 ± 8
Ejection fraction, %	66 ± 7	66 ± 7	67 ± 7	66 ± 7
Peak transaortic velocity, m/s	2.49 ± 0.21*†	3.04 ± 0.16*	3.71 ± 0.29	3.08 ± 0.54
Peak transaortic gradient, mm Hg	25 ± 4*†	37 ± 4*	55 ± 9	39 ± 14
Mean transaortic gradient, mm Hg	14 ± 3*†	21 ± 3*	32 ± 6	23 ± 9
AVA, cm^2	1.51 ± 0.50*†	1.25 ± 0.40*	1.06 ± 0.35	1.27 ± 0.46
AVAI, cm^2/m^2	0.80 ± 0.24*†	0.66 ± 0.20*	0.56 ± 0.18	0.67 ± 0.23
Pressure recovery, mm Hg	4 ± 1*†	6 ± 2*	8 ± 2	6 ± 2
Pressure recovery/peak transaortic gradient, %	17 ± 4*†	15 ± 4*	14 ± 4	16 ± 4
ELI, cm^2/m^2	1.14 ± 0.56*†	0.86 ± 0.35*	0.69 ± 0.26	0.89 ± 0.45
AVAI – ELI, cm	−0.35 ± 0.38*†	−0.20 ± 0.17*	−0.13 ± 0.10	−0.23 ± 0.26
AVAI – ELI, %	−39 ± 34*†	−28 ± 16*	−22 ± 11	−30 ± 24
Pressure recovery/peak transaortic gradient ratio >20%, n (%)	(131) 25.5*†	(85) 15.7*	(47) 9.2	(263) 16.8

*p < 0.001 versus Tertile #3; †p < 0.001 versus Tertile #2.
AVA = aortic valve area; AVAI = aortic valve area index; ELI = energy loss index; LV = left ventricle.

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RESULTS

Aortic dimension. Clinical and hemodynamic characteristics for the total study population are shown in Table 1. Age, sex, systolic and diastolic blood pressure, height, weight, body surface area, body mass index, heart rate, and history of hypertension did not differ between tertile groups. Aortic dimensions did not differ between tertile groups (Table 2).

Pressure recovery and AS severity. Pressure recovery increased with severity of AS both in women and men (Fig. 1). As expected, women had smaller aortic roots and thus greater pressure recovery than men (2.57 ± 0.35 cm vs. 2.97 ± 0.41 cm, $p < 0.001$ and 6.1 ± 2.3 mm Hg vs. 5.8 ± 2.3 mm Hg, $p < 0.01$). Peak transaortic velocity did not differ between sexes, but pressure recovery as a percentage of total peak transvalvular gradient was significantly higher in women (16.3 ± 4.3% vs. 15.1 ± 4.4%, $p < 0.001$). However, pressure recovery as a proportion of total peak transvalvular gradient decreased with higher peak transaortic velocity from 17% in the lowest to 14% in the highest tertile (Table 2, Fig. 2). In multiple regression analysis, including pressure recovery as a percentage of total peak transvalvular gradient as the dependent variable and including peak transaortic velocity, aortic sinus diameter, left ventricular mass, and heart rate as independent covariates based on significant univariate associations, higher pressure recovery/peak transvalvular gradient ratio was associated with lower peak transaortic velocity (beta = −0.334) and aortic sinus diameter (beta = −0.437), and higher left ventricular mass (beta = 0.119) and heart rate (beta = 0.082, all $p < 0.001$) (multiple $R^2 = 0.30$, $p < 0.001$).

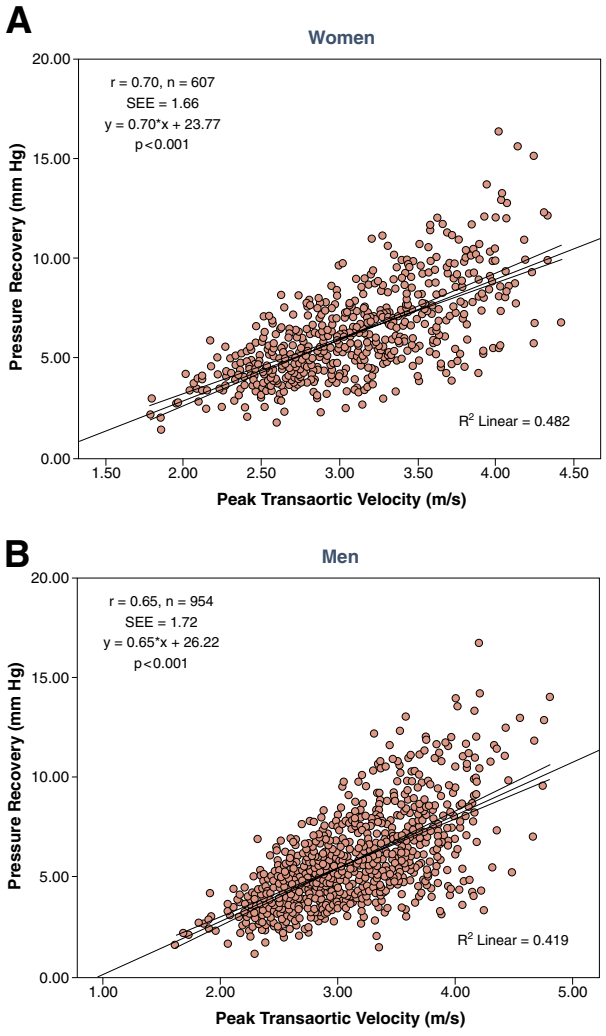

Figure 1. Pressure Recovery in Relation to AS Severity and Gender

(A) The relation between peak transaortic velocity and pressure recovery estimated at the sinotubular junction in women (Pearson correlation coefficient r = 0.70, $p < 0.001$) and **(B)** men (Pearson correlation coefficient r = 0.65, $p < 0.001$). Inserted **lines** represent regression line and SEE. AS = aortic stenosis; SEE = standard error of estimate.

A clinically relevant pressure recovery (i.e., pressure recovery >20% of the peak transaortic pressure gradient) was found in 16.8% of patients in the total study population. The prevalence was higher in groups of patients with lower peak transaortic velocity or smaller aortic root diameter (Tables 2 and 3).

Both AVAI and ELI, as well as the AVAI-ELI difference decreased with increasing peak transaortic velocity, but the overestimation of AS severity by unadjusted AVAI was greatest in patients in the lower tertile (Tables 2 and 3, Fig. 3). In the total study population, the difference between AVAI and ELI was −30 ± 24%. The difference increased with lower peak transaortic velocity or smaller aortic root dimension (Tables 2 and 3). In multivariate regression analysis, a larger difference between AVAI and ELI was associated with lower peak transaortic velocity (beta = 0.353) independent of higher LV ejection fraction (beta = −0.049), male sex (beta = −0.075), younger age (beta = 0.093), and smaller AS diameter (beta = 0.233) (multiple $R^2 = 0.18$, $p < 0.001$) (Table 4). No independent association was found with LV mass when added to the model.

Percentages of patients classified as severe AS by AVAI and ELI are presented in Figure 4. Overall, 45.7% of patients classified as having severe AS by unadjusted AVAI were reclassified as having non-

severe AS when pressure recovery was taken into account. The proportional reductions in prevalence of severe AS when pressure recovery was taken into account was 68.2% in the lower tertile and 46.4% and 38.2% in the middle and higher tertiles (all $p < 0.001$) (Fig. 4). The prevalence of severe AS by ELI with low stroke volume did not differ between tertile groups ($p = 0.671$).

In logistic regression including severe AS by ELI as dependent variable and AVAI, pressure recovery and the product of the 2 as independent variables, no interaction between the 2 in prediction of AS was found.

Pressure recovery at different aortic levels. Pressure recovery and ELI calculated at the junctional aortic level were significantly higher compared with calculations made at sinus or supracoronary levels, respectively (Table 5). Consequently, fewer patients were diagnosed with severe AS by ELI using junctional diameter, as compared with the calculation of ELI using sinus or supracoronary diameter, respectively ($p < 0.001$) (Table 5). Compared with the use of junctional diameter, 49 patients were reclassified from nonsevere to severe AS by using sinus diameter and 51 patients by using supracoronary diameter.

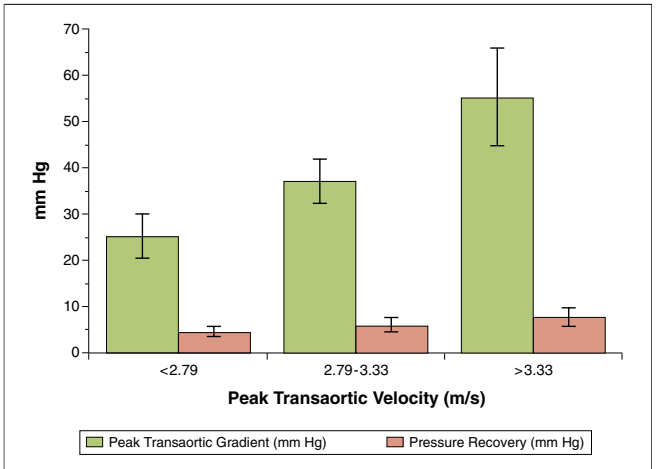

Figure 2. Pressure Recovery in Relation to AS Severity

Peak transaortic gradient **(green bars)** and pressure recovery **(pink bars)** in the study population grouped into tertiles of peak transaortic velocity. Vertical lines indicate 1 SD within groups. Abbreviation as in Figure 1.

DISCUSSION

This is the first study to investigate the incidence and magnitude of pressure recovery in a large cohort of prospectively recruited patients with asymptomatic AS. The findings demonstrate that clinically important pressure recovery is present in a significant number of asymptomatic patients with AS and a transvalvular velocity of 2.5 to 4.0 m/s. As expected from experimental data (1,2,17) and confirmed in small clinical studies (18–20), the absolute magnitude of pressure recovery was greater in subjects with higher transvalvular velocities and more severe AS. However, the functional significance of pressure recovery was proportionately larger in patients with a lower degree of stenosis, confirming results in previous experimental and invasive studies (17,19).

Based on the present results, a substantial number of asymptomatic AS patients are likely to be misclassified as having severe AS if pressure recovery is not taken into account. Compared with previously published smaller studies (8,10,19), the percentage of reclassification to nonsevere stenosis was higher in our study, probably reflecting the relatively milder degree of AS in our study population. The higher incidence of reclassification in the lowest tertile reflects a greater proportional impact of pressure recovery in this group.

Continuous-wave Doppler echocardiography is widely used to derive estimates of valve area in patients with AS. Conventionally, an AVA of <1 cm^2 or an indexed AVA of ≤ 0.60 cm^2/m^2, respectively, is used to define severe stenosis (14), but these numbers are extrapolated from data acquired from invasive studies and may not be applicable to Doppler-derived data that will tend

Table 3. Absolute and Relative Differences Between AVAI and ELI and Proportion of Patients With Pressure Recovery/Peak Transaortic Gradient Ratio >20% in Tertiles of Aortic Junctional Diameter

Variables	<2.60 cm (Tertile #1)	2.60–2.97 cm (Tertile #2)	>2.98 cm (Tertile #3)	Total
AVAI − ELI, cm	−0.29 ± 0.33*†	−0.23 ± 0.27†	−0.17 ± 0.15	−0.23 ± 0.26
AVAI − ELI, %	−40 ± 34*†	−29 ± 17†	−21 ± 11	−30 ± 24
PR/peak transaortic gradient >20%, %	30.5*†	15.1†	5.5	16.8

*$p < 0.001$ versus Tertile #2; †$p < 0.001$ versus Tertile #3.
PR = pressure recovery; other abbreviations as in Table 2.

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Table 4. Independent Covariates of AVAI/ELI Difference Identified in Multiple Regression Analysis (Multiple $R^2 = 0.18$, $p < 0.001$)

Variables	B	SEE	Beta	p Value
Male sex	−0.041	0.016	−0.075	<0.05
Age, yrs	0.003	0.001	0.093	<0.001
Sinus diameter, cm	0.140	0.017	0.233	<0.001
Peak transaortic velocity, m/s	0.174	0.012	0.353	<0.001
Ejection fraction, %	−0.002	0.001	−0.049	<0.05

B = regression coefficient; other abbreviations as in Table 2.

to underestimate valve area compared with standard invasive methods where aortic pressure is measured in the aortic root several centimeters distal to the vena contracta (1,10,19). It is theoretically possible to perform invasive measures of pressure exactly at the vena contracta, but this is technically demanding and is not performed routinely in clinical practice. In physiological terms, it is the recovered pressure that reflects the load imposed on the LV by AS rather than the pressure drop at the vena contracta (21–23). Accurate estimation of the AS severity therefore should be performed by noninvasive area correction for the effect of energy loss (24,25).

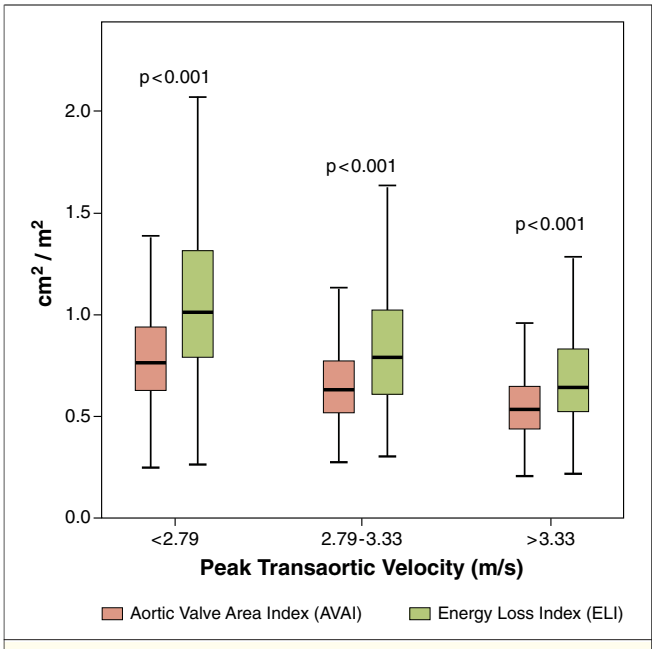

Figure 3. Difference Between AVAI and ELI in Relation to AS Severity

Unadjusted aortic valve area by continuity equation (**pink boxes**) and adjusted for pressure recovery (**green boxes**) in the study population grouped into tertiles of peak transaortic velocity. Boxes represent 25th to 75th percentiles within groups, and **horizontal** and **vertical lines** represent median and range, respectively. AVAI = aortic valve area index; ELI = energy loss index; other abbreviation as in Figure 1.

The present study investigated the concept of pressure recovery at 3 different levels of the aorta. Previous studies have assessed pressure recovery either at the sinus, the junctional, or supracoronary aortic level (1,7,8,19,20). Our data confirms that there are statistically significant differences in assessments made at these 3 sites and in the classification of AS based on these assessments. Given the fact that there is no gold standard and the present study did not include invasive validations or prognostic evaluation, the optimal assessment level could not be determined. It is argued that pressure recovery should be estimated at its greatest possible extent (1), which in our study was demonstrated to be at the sinotubular junction.

As demonstrated by the present results, ELI was on average 30% larger than AVAI, and clinically important pressure recovery (i.e., pressure recovery >20% of the peak transaortic gradient) was on average found in 16.8% of patients. Clinically important pressure recovery was more often present in patients with smaller aortic root dimensions or lower peak transaortic velocities, confirming previous findings in experimental studies (6) as well as invasive studies in small patient populations (1,18,19). The findings that the difference between AVAI and ELI diminished with increasing peak transaortic flow velocity are consistent with results in invasive studies reported in small patient populations (18,19).

Decisions on aortic valve replacement are driven primarily by symptoms and occasionally by concerns about LV function (14,26,27). It is often difficult, particularly in older patients with comorbidities, to be confident about the origin of symptoms, and a valve mistakenly classified as severely stenosed might lead to operation in a patient with a low chance of symptomatic improvement. Furthermore, echocardiographic grading of AS by peak transaortic velocities, gradients, or AVAI may yield inconsistent results regarding AS severity (28). In asymptomatic patients, echocardiographic assessment of AS does not rely solely on the estimation of valve area but also requires an assessment of valve morphology, the shape of the spectral Doppler waveform, and LV systolic function. Thus, a finding of a significant discrepancy between AVAI and ELI should prompt careful scrutiny of other parameters of severity, including exercise echocardiography (29). Few data exist on the prognostic value of Doppler measurements corrected for pressure

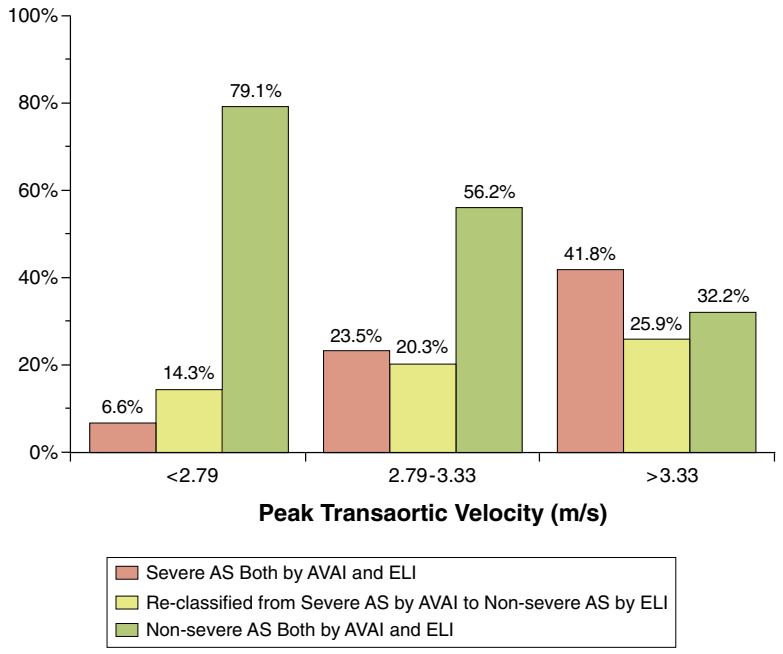

Figure 4. Reclassification From Severe to Nonsevere AS by Pressure Recovery Adjustment

Percentage of patients classified as having severe AS by both unadjusted aortic valve area by continuity equation and pressure recovery adjusted valve area increased **(pink bars)**, while percentage of patients having nonsevere AS by both ELI and AVAI **(green bars)** decreased with increasing peak transaortic velocity. Percentage of patients reclassified from severe AS by AVAI to nonsevere AS by ELI **(yellow bars)** increased with increasing peak transaortic velocity. Abbreviations as in Figures 1, 2, and 3.

recovery. Results by Garcia et al. (9) showed that ELI was superior to unadjusted AVAI in predicting adverse clinical outcomes in patients with moderate to severe AS. Whether AVAI or ELI is a better predictor of outcome in asymptomatic AS patients needs to be clarified in further analyses.

Study limitations. Routine catheterization was not performed in this study, and so we cannot confirm our estimates of pressure recovery by invasive measurements. However, in clinical practice, echocardiography is often the sole method used to assess the severity of AS. Current recommendations reserve transvalvular catheterization only in those instances where there is significant doubt concerning the true severity of stenosis. However, it should also be kept in mind that it is technically difficult to measure invasive pressure recovery accurately during routine cardiac catheterization.

Ultimately, proving the value of calculating pressure recovery and pressure recovery adjusted AVA, known as ELI, will require the demonstration of energy loss as a better prognosticator than AVA in predicting clinical outcome. However, the present study is the first to demonstrate the prevalence, correlates, and clinical relevance of pressure recovery and ELI in a large population of AS patients. It demonstrates that pressure recovery adjustment of AVA frequently leads to reclassification of AS severity.

Table 5. Estimation of Pressure Recovery and ELI at Different Levels of the Aorta

Variables	Sinus	Junctional	Supracoronary
Aortic diameter, cm	3.08 ± 0.44*†	2.82 ± 0.42†	3.11 ± 0.47
Pressure recovery, mm Hg	5.14 ± 1.97*	5.88 ± 2.27†	5.07 ± 1.98
ELI, cm^2/m^2	0.84 ± 0.38*	0.89 ± 0.45†	0.84 ± 0.38
Severe AS, %	19.7*	17.2†	20.0

*$p < 0.001$ versus junctional level; †$p \leq 0.001$ versus supracoronary level.
AS = aortic stenosis; other abbreviations as in Table 2.

CONCLUSIONS

The severity of AS is frequently overestimated if correction for pressure recovery is not performed. For accurate assessment of AS severity, pressure recovery adjustment of AVA must be routinely performed.

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Key Words: aortic stenosis ■ left ventricle ■ aortic valve area ■ energy loss ■ sinotubular junction.