Doppler echocardiography markedly underestimates left ventricular stroke work loss in severe aortic valve stenosis

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Received 7 April 2006; received in revised form 3 June 2006; accepted 15 June 2006
Available online 24 July 2006

KEYWORDS
Aortic valve stenosis; Stroke work loss; Doppler echocardiography

Abstract

Aims: Left ventricular (LV) stroke work loss (SWL) is an index of aortic stenosis (AS) severity based on the energy loss concept. Traditionally known as an invasive measurement, SWL has also been determined noninvasively using Doppler echocardiography. The present work was done to compare the echocardiographic estimate against the standard invasive SWL.

Methods and results: We determined SWL in 113 adult patients with AS at cardiac catheterization (mean gradient as percentage of the LV mean systolic pressure) and by Doppler echocardiography (mean gradient as percentage of the sum of systolic cuff blood pressure and mean gradient). SWL averaged 26.5 ± 0.6% by echocardiography vs 30.9 ± 0.8% by catheterization (p < 0.000001). The underestimation by echocardiography was the worse the higher the invasive SWL was (p < 0.00001). Echocardiographic SWL exceeding 25% (previously suggested cutoff) had a sensitivity of 64% and specificity of 74% to identify severe AS (Gorlin valve area < 0.5 cm²/m²). Several patients with severe AS by valve area had echocardiographic SWL suggesting only mild AS.

Conclusions: In AS, echocardiography gives a biased estimate of LV SWL with marked underestimation in severe disease. Echocardiographic SWL may confuse rather than improve the assessment of AS in clinical practice.

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Introduction

Left ventricular (LV) stroke work loss (SWL), the percentage of mean aortic valve pressure gradient over the mean LV systolic pressure, was originally introduced by Tobin et al.\(^1\) as a flow-independent invasive index of aortic stenosis (AS). The utility of SWL has later been supported by several clinical and experimental invasive studies.\(^2\)–\(^4\) More recently, Doppler echocardiography has been used to determine SWL noninvasively as percentage of the mean aortic valve gradient over the sum of systolic blood pressure and mean gradient.\(^5\)–\(^8\) Bermejo et al.\(^7\) followed a series of more than 300 patients and reported that echocardiographic SWL outperformed all other echocardiographic indices in predicting the severity and long-term outcome of AS. The authors went on to suggest that the echocardiographic SWL should be used to guide management decisions in AS.\(^7\) The present work was done to directly compare Doppler echocardiography with cardiac catheterization in the assessment of LV SWL in patients with AS.

Methods

Patients

We considered for the present work adult patients undergoing preoperative evaluation for AS between August 2000 and January 2003 at our institution. Totally 137 patients (65 men) underwent clinical examination, 6-min walk test, echocardiography and a complete cardiac catheterization.\(^9\) Of them, 24 individuals could not be included in the present work due to either failed retrograde LV catheterization (\(n = 16\)) or missing data on brachial artery cuff blood pressure (\(n = 8\)). Table 1 summarizes the characteristics of the remaining 113 patients constituting our final study population. The majority of them had severe AS (valve area < 0.5 cm\(^2\)/m\(^2\); \(n = 87\)) and one-third had heart failure. Of the 113 patients, 104 ultimately underwent aortic valve replacement.

Echocardiography

The echocardiographic studies were done with an Acuson Sequoia scanner (Siemens Medical Solutions, CA, USA). The mean aortic valve pressure gradient was determined by continuous wave Doppler and the modified Bernoulli equation.\(^10\) In the presence of atrial fibrillation, the gradients were taken from strokes that followed R–R intervals representative of the average heart rate at the examination. SWL (%) was calculated as \(100 \times \frac{\text{mean gradient}}{	ext{cuff brachial artery systolic blood pressure + mean gradient}}\).\(^5\)–\(^8\); blood pressure was measured by a research nurse 0–3 h prior to the echocardiographic study. LV mass and ejection fraction were determined from M-mode and apical 2-dimensional views, respectively, as reported elsewhere.\(^9\) The reproducibility of our echocardiographic LV and aortic valve measurements has been validated earlier.\(^11\),\(^12\)

Cardiac catheterization

Cardiac catheterizations were made via the femoral route. Pressures in the right heart and pulmonary artery were measured using a Swan–Ganz catheter. Table 1 shows the characteristics of the patients with aortic stenosis. The data are presented as mean ± standard deviation.

### Table 1 Characteristics of the patients with aortic stenosis (\(n = 113\))

<table>
<thead>
<tr>
<th>Characteristic or measurement</th>
<th>Mean value or number of patients</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>68</td>
<td>39–82</td>
</tr>
<tr>
<td>Sex, M/F</td>
<td>56/57</td>
<td></td>
</tr>
<tr>
<td>NYHA class, 1/2/3/4, (n)</td>
<td>7/67/42/1</td>
<td></td>
</tr>
<tr>
<td>Atrial fibrillation, (n) (%)</td>
<td>10 (9)</td>
<td></td>
</tr>
<tr>
<td>Heart failure, (n) (%)</td>
<td>37 (33)</td>
<td></td>
</tr>
<tr>
<td><strong>Invasive characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic valve area (cm(^2)/m(^2))</td>
<td>0.39 (87)</td>
<td>0.15–0.89</td>
</tr>
<tr>
<td>&lt;0.5 cm(^2)/m(^2), (n) (%)</td>
<td>144 (147–270)</td>
<td></td>
</tr>
<tr>
<td>LV peak systolic pressure (mmHg)</td>
<td>200</td>
<td></td>
</tr>
<tr>
<td>A0 systolic pressure (mmHg)</td>
<td>45</td>
<td>15–95</td>
</tr>
<tr>
<td>LV–A0 mean pressure gradient (mmHg)</td>
<td>13</td>
<td>4–36</td>
</tr>
<tr>
<td>Pulmonary wedge pressure (mmHg)</td>
<td>31 (27)</td>
<td></td>
</tr>
<tr>
<td>Coronary artery disease at angiography, (n) (%)(^b)</td>
<td>138 (98–200)</td>
<td></td>
</tr>
<tr>
<td><strong>Non-invasive characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brachial artery systolic blood pressure (mmHg)</td>
<td>49* (15–123)</td>
<td></td>
</tr>
<tr>
<td>LV–A0 mean pressure gradient (mmHg)</td>
<td>59 (21–79)</td>
<td></td>
</tr>
<tr>
<td>LV ejection fraction (%) &lt;50%, (n) (%)</td>
<td>17 (15)</td>
<td></td>
</tr>
<tr>
<td>LV mass index (g/m(^2))</td>
<td>153</td>
<td>77–269</td>
</tr>
<tr>
<td>LV hypertrophy, (n) (%)(^b)</td>
<td>80 (74)</td>
<td></td>
</tr>
</tbody>
</table>

AO, aortic; LV, left ventricular; and NYHA, New York Heart Association.

* \(p < 0.01\) compared with the invasive mean gradient.

\(^a\) Heart failure was diagnosed when the patient had resting pulmonary wedge pressure \(> 15\) mmHg at catheterization in association with a history of dyspnea or fatigue on ordinary effort.

\(^b\) See Methods for diagnostic criteria.
catheter (Edwards Lifesciences, CA, USA). The aortic valve was crossed retrogradely with left or right Amplatz coronary artery catheters (Boston Scientific, MA, USA) for pressure recording in the left ventricle and during catheter pullback into the aortic root. All the pressures were measured with the zero reference level set at the mid-axillary line. Cardiac output was determined by the Fick method. The aortic valve area was calculated by the Gorlin formula and indexed to body area. SWL (%) was determined as \( \frac{100 \times \text{mean pressure gradient}}{\text{mean LV systolic pressure}} \).\(^1\) The measurements were made by tracing superimposed LV and aortic pressure recordings on an \( x-y \) digitizing table. The areas under the respective pressure curves represented the mean systolic LV and aortic pressures and allowed the calculation of the mean gradient and SWL. In the presence of atrial fibrillation, we selected for analysis LV and aortic pressure tracings with identical preceding R–R intervals.

Ethics

The present work was designed and executed in accordance with the principles outlined in the Declaration of Helsinki. The ethical committee of our institution approved the study protocol and all participants signed an informed consent document.

Statistical analysis

Student’s paired \( t \)-test was used to compare the measurements of SWL made by the two methods. Univariate associations between continuous data were analyzed by linear regression. The data are given as mean \( \pm \) SE unless indicated otherwise. All the analyses were conducted using commercially available statistical software (SYSTAT Version 9.1, Systat Inc., CA, USA).

Results

SWL by echocardiography vs by cardiac catheterization

SWL averaged 26.5 \( \pm \) 0.6% by echocardiography vs 30.9 \( \pm \) 0.8% by catheterization (\( p < 0.0000001 \)) (Fig. 1). When patients only with sinus rhythm were analyzed (\( n = 103 \)), the respective means were 26.1 \( \pm \) 0.6% vs 30.5 \( \pm \) 0.8% (\( p < 0.0000001 \)). Fig. 2 shows a Bland–Altman analysis of the (dis)agreement between the echocardiographic and invasive SWLs. The graph demonstrates that echocardiography markedly underestimated high values of SWL while tending to overestimate very low values of SWL.

Echocardiographic SWL in relation to AVA

Fig. 3 is a plot of the echocardiographic SWL against the AVA measured at cardiac catheterization. Of
note, several patients with critically severe AS by the invasive AVA (<0.5 cm²/m²) had echocardio-
graphic SWL < 20%. A value of SWL exceeding 25%, which is the suggested cutoff for severe AS, had a sensitivity of only 64%, and a specificity of 74%, to identify severe AS by the above AVA criterion.

Discussion

Our work showed that Doppler echocardiography gives a strongly biased estimate of SWL in patients with AS. The method underestimates invasive SWL the more the higher this is. Individual patients may have low SWL at echocardiography (<20%) despite critically reduced AVA and a clear benefit from subsequent surgery. Based on these data we caution not to use echocardiographic SWL to guide management decisions in AS.

LV SWL was initially described by Tobin et al. as a simple and flow-independent invasive measure of AS severity. The authors reported that SWL was inversely related to AVA and that a value of >30% was highly predictive of severe AS (AVA < 0.6 cm²/m²). Sprigings et al. later confirmed an inverse exponential relation between SWL and AVA. Bermejo et al. recently analyzed echocardiographic data from a total of 307 patients and reported that echocardiographic SWL outperformed all other indices, including peak jet velocity, mean pressure gradient, AVA, and aortic valve resistance, in predicting death or valve replacement in AS. SWL values > 25% appeared to best discriminate the clinical endpoints. The strength of the work is that clinical outcomes were used to define the severity of AS and that the different echocardiographic indices were compared in terms of their predictive values. Still, we feel that the results of Bermejo et al. should be taken cautiously together with their view of echocardiographic SWL as a useful tool for clinical decision-making.

It is worthy of note that the mean aortic valve systolic gradient (the nominator in the SWL formula) was statistically significantly higher by echocardiography than by cardiac catheterization (see Table 1) and that the underestimation of SWL thus resulted solely from the overestimation of the LV mean systolic pressure (the denominator) non-invasively. Thus the overall bias was less due to a weakness of echocardiography per se than to the seriously flawed idea that the LV mean systolic pressure could be reliably estimated from the valve gradient and cuff blood pressure.

A limitation of our work is that the echocardiographic SWL was not based on strictly simultaneous data on aortic valve gradient and cuff blood pressure since some of the latter measurements were taken 0–3 h prior to echocardiography. However, in this respect our study is comparable to the work of Bermejo et al. where SWL was also calculated from non-simultaneous measurements.

We conclude that Doppler echocardiography gives a biased estimate of SWL in AS. The method underestimates SWL in severe AS in particular. Echocardiographic SWL should not be used to guide management decisions in AS.

Acknowledgements

We thank Liisa Blubaum, R.N., and Sini Piirilä, R.N., for skilful assistance during the course of this work. This study was supported by grants from (1) Sigrid Juselius Foundation, Helsinki, Finland; (2) Research Foundation (EVO grant) of Helsinki University Central Hospital, Helsinki, Finland; and (3) Finnish Foundation for Cardiovascular Research, Helsinki, Finland.

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