Left ventricular global work index and prediction of cardiovascular mortality after transcatheter aortic valve implantation

Anwer, Shehab ; Nussbaum, Sinuhe ; Winkler, Neria E ; Benz, Dominik C ; Zuercher, Dominik ; G Donati, Thierry ; Tsiourantani, Glykeria ; Wilzeck, Verena ; Michel, Jonathan M ; Kasel, Albert M ; Tanner, Felix C

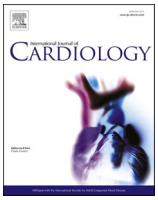
DOI: https://doi.org/10.1016/j.ijcard.2023.131660

International Journal of Cardiology 399 (2024) 131660

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard



Left ventricular global work index and prediction of cardiovascular mortality after transcatheter aortic valve implantation

Shehab Anwer [a,1], Sinuhe Nussbaum [a,1], Neria E. Winkler [a], Dominik C. Benz [a], Dominik Zuercher [a], Thierry G. Donati [a], Glykeria Tsiourantani [a], Verena Wilzeck [a], Jonathan M. Michel [a], Albert M. Kasel [a], Felix C. Tanner [a,*]

[a] Department of Cardiology, University Heart Center, University Hospital Zurich and University of Zurich, Zurich, Switzerland

ARTICLE INFO

Keywords:
Myocardial work
Strain
Aortic stenosis
Transcatheter aortic valve implantation

ABSTRACT

Introduction: Echocardiography is used for assessment of patients after transcatheter aortic valve implantation (TAVI). Global work index (GWI) integrates LV deformation throughout the cardiac cycle and LV afterload and may be advantageous for long-term follow-up.

Methods: We analysed 144 patients with severe aortic stenosis who underwent TAVI and echocardiography within two weeks afterwards. GE EchoPAC v2.6 was applied for determining LV ejection fraction, global longitudinal strain (GLS), stroke work (SW), cardiac power output (CPO), and GWI. The endpoint was cardiovascular mortality.

Results: During median follow-up of 625 [IQR: 511–770] days, 20 (14%) patients died. Clinical baseline characteristics were comparable between non-survivors and survivors. GWI ($p = 0.003$) and LVEF ($p = 0.039$) were lower in non-survivors, while GLS, SW, and CPO were not different. In Kaplan-Meier analysis patients with GWI ≤1234 mmHg% exhibited a lower survival probability ($P = 0.006$). In univariable Cox regression, a significant mortality association was identified for GWI ($P = 0.004$), weaker for LVEF ($P = 0.014$), but not for the other parameters. In multivariable Cox regression, GWI independently improved an LV systolic function model including LVEF and GLS. Similarly, GWI but not LVEF independently improved outcome association of different clinical models.

Conclusions: GWI was lower in non-survivors than survivors, differentiated non-survivors from survivors, was associated with mortality independent of clinical or LV parameters, and improved the fitness of clinical or LV prediction models. In contrast, GLS, SW, and CPO did not show any of these properties. GWI provides added value for follow-up after TAVI possibly by integrating LV deformation throughout the cardiac cycle.

1. Introduction

Aortic stenosis (AS) is the most common valvular heart disease in the Western society and has a significant impact on morbidity and mortality. Its natural course is slowly progressive leading to adaptive cardiac remodelling. Hence, assessment of cardiac morphology and function bears clinical relevance in patients with AS undergoing transcatheter aortic valve implantation (TAVI) or surgical aortic valve replacement (SAVR). [1–5]

Echocardiography is an integral modality for assessing LV function. As LV ejection fraction (LVEF) exhibits rather low sensitivity for detecting early changes in LV systolic function, there has been increasing interest in global longitudinal strain (GLS). A reduced GLS is associated with worse outcome in patients with AS even before LVEF is impaired; hence, GLS is a useful parameter for outcome prediction in patients with AS. However, one of the drawbacks of strain imaging is its load dependency. [5–17]

Myocardial work analysis (MW) was introduced as an indicator of LV work. It is based on integration of LV GLS from echocardiographic data with non-invasively estimated LV pressure derived from bedside arterial blood pressure to produce estimated LV pressure strain loops (PSL). Such loops describe LV deformation throughout the cardiac cycle and correct for afterload. While a normal heart demonstrates high work efficiency, functional impairment in one or several segments can add a mechanical

* Department of Cardiology, University Heart Center, University Hospital Zurich and University of Zurich, Rämistrasse 100, 8091 Zurich, Switzerland
E-mail addresses: shehab.anwer@usz.ch, shehab.anwer@gmail.com (S. Anwer), felix.tanner@usz.ch (F. C. Tanner).

[1] SA and SN have contributed equally and share the first author position.

https://doi.org/10.1016/j.ijcard.2023.131660
Received 9 September 2023; Received in revised form 20 November 2023; Accepted 18 December 2023
Available online 30 December 2023

burden to the remaining myocardium possibly contributing to adverse remodelling and worse outcome. Recent studies identified MW as a superior prognostic factor in different cardiac conditions such as arterial hypertension, chronic heart failure, acute coronary syndrome, cardiac resynchronization therapy, and various cardiomyopathies. Hence, LV MW indices may provide additional information on myocardial remodelling as compared to GLS serving as better follow-up parameters after aortic valve replacement. Such an effect, however, may be related to integration of ventricular deformation throughout the cardiac cycle or correction for afterload or both. [17–24]

This study aims to a) determine different indicators of LV systolic function and LV work shortly after TAVI including EF, GLS, stroke work, power output, and MW PSL, and b) understand outcome association of these indicators to delineate their prognostic value during long-term follow-up.

2. Methods

2.1. Study population

All the patients with severe AS undergoing TAVI at the University Hospital Zürich ($N = 1099$) are included in a prospective cohort study after their informed consent had been obtained. All the patients were evaluated by an interdisciplinary Heart Team and underwent treatment according to current guidelines. The study was approved by the institutional ethical review board of the University of Zürich.

The 144 patients included in the current study had an echocardiographic examination performed within two weeks after TAVI on a GE unit with cine loops stored in raw data format and exhibiting image quality suitable for complete longitudinal strain analysis and with bedside blood pressure available before the time of the echocardiography study. 573 patients were excluded because of one or more clinical reasons: no informed consent available, no echocardiographic examination within 3 months prior to procedure, any moderate or severe valvular regurgitation, mitral valve stenosis or repair, atrial fibrillation or flutter, or atrial ablations or devices. 382 patients were excluded because of one or more echocardiographic reasons: study not performed on a GE unit, poor ultrasound window, heart rate variability >10 bpm, cineloop frame rates <40/s, less than two consecutive cardiac cycles available, or more than two myocardial segments invisible.

2.2. Echocardiography

All echocardiographic studies were performed using commercially available units from GE Healthcare (Vivid 7 or E9 or E95, GE Healthcare, Amersham, Buckinghamshire, UK). The echocardiographic measurements were taken by experienced certified personnel according to current recommendations using GE EchoPAC v.203 (GE Healthcare, Amersham, Buckinghamshire, UK). Stroke work was determined as the product of stroke volume and mean arterial blood pressure, and power output was calculated as the product of stroke work and heart rate. Myocardial work was analysed using GE EchoPAC automated function imaging (AFI) module. LV strain measurements were carried out according to current recommendations using a 17-segment model. The tracking region of interest (ROI) covered the area from the LV endocardium to the subepicardial border. The tracking line was carefully observed throughout the cardiac cycle and manually adjusted when required. End-diastole was defined as the last frame before mitral valve closure and end-systole was defined by the closure of the prosthetic aortic valve. Once GLS was analysed, bed-side arterial blood pressure was entered to estimate LV systolic pressure and generate pressure-strain loops (PSL). The following MW parameters were reported in this study: the ratio between global constructive work (GCW) and the sum of GCW plus global wasted work (GWW) representing global work efficiency (GWE); and global work index (GWI) describing the area of the PSL. (Fig. 1).

2.3. Follow-up

Outcomes were reported according to the updated standardized endpoint definitions of the Valve Academic Research Consortium VARC-3 consensus document. (27) Follow-up was performed by phone call and/or assessment of all available hospital records to update mortality status of the patients. Cardiovascular (CV) mortality was defined as the

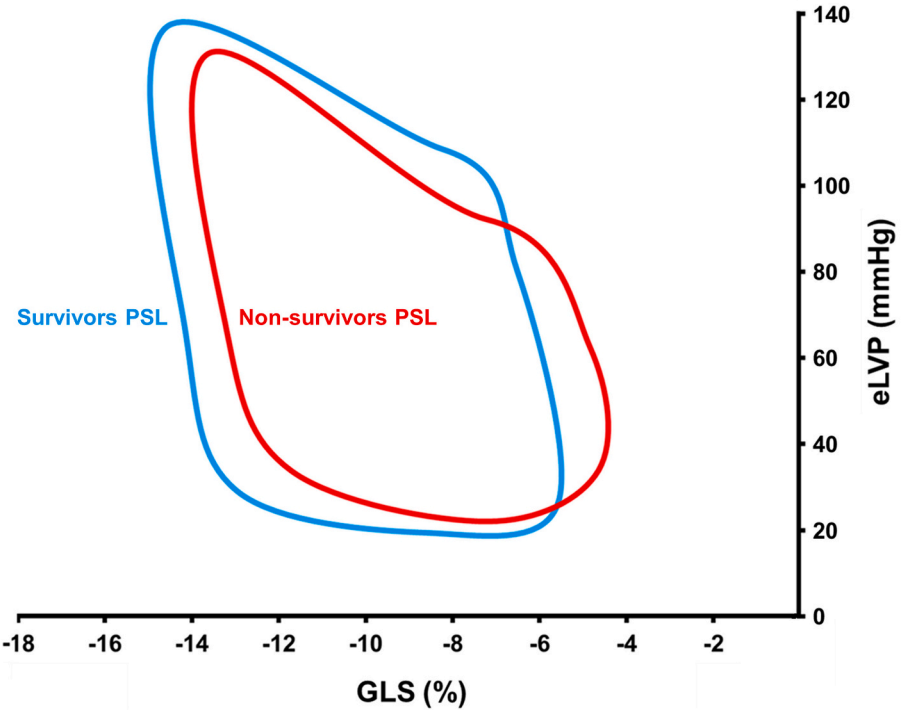


Fig. 1.. Pressure-strain loop of survivors (blue) and non-survivors (red)

primary endpoint according to the VARC-3 criteria (27). The study population was divided into those who survived during follow-up (survivors) and those who died at any time point after TAVI due to CV causes (non-survivors).

2.4. Statistics

All statistical methods were performed using MedCalc ® for Windows (Version 20.1, MedCalc Software, Ostend, Belgium). Statistical significance was considered at a two-sided *p*-value <0.05. The Shapiro Wilk test was used for analysing the distribution of values. The majority of parameters showed a non-uniform distribution. Continuous variables are reported as median ± interquartile range, while categorial variables are presented as number and percentage. Comparing continuous variables was tested using Mann-Whitney test. Analysis of categorial variables was performed by Fischer's exact test. Receiver-operating-characteristics (ROC) curve analysis was used for identification of the optimal cut-off value to differentiate non-survivors from survivors. These cut-off values were used for performing Kaplan-Meier survival curve analyses. The log-rank test was applied for comparison of survival curves. Univariable Cox regression was used for testing the time-dependent association of target parameters with cardiovascular mortality, and multivariable models were generated and tested for model improvement based on Cox regression analysis of variance (Cox-ANOVA). For all the models the validity of the proportional hazard assumption was assessed by scaled Schoenfeld residuals.

3. Results

3.1. Baseline characteristics

During a median follow-up duration of 625 [511–770] days, 26 patients (18%) died (non-survivor group), among which 20 (14%) suffered a CV death. The remaining 124 patients (86%) constituted the survivor group.

Clinical and echocardiographic baseline characteristics were similar between the study groups as detailed in Table 1 except for a significantly lower mean blood pressure (MBP) among non-survivors (41.0 [30.0–52.0]) compared to survivors (50.0 [39.0–62.0], $P = 0.023$).

3.2. LV systolic function after TAVI

The majority of the study population ($n = 112$, 78%) exhibited a preserved LVEF (>50%) after TAVI. LVEF was significantly lower among non-survivors (51.0 [45.0–63.0] %) compared to survivors (59.0 [50.0–65.0] %, $P = 0.039$, Table 2). Both stroke work and power output showed comparable values among non-survivors and survivors without any significant differences between the groups (Table 2). LV GLS was slightly worse among non-survivors compared to the survivors without reaching statistical significance (Table 2). GWE was significantly lower among non-survivors (89.0 [82.0–93.0] %) than survivors (92.0 [88.0–95.0] %, $P = 0.047$, Table 2). Finally, GWI was significantly lower among non-survivors (1029.0 [641.0–1382.0) than survivors (1389 [1070.0–1653.0], $P = 0.003$, Fig. 2 and Table 2).

3.3. Survival after TAVI

A GWI value ≤1234 mmHg (AUC 71%; $P = 0.002$) best differentiated non-survivors from survivors. When the population was dichotomised accordingly, Kaplan-Meier curves revealed a lower survival probability for patients with GWI below the threshold ($\chi^2 = 7.43$, $P = 0.006$, Fig. 3).

3.4. Association with mortality after TAVI

Univariable Cox regression (Table 3) revealed a significant association with an increased risk of death for LVEF (HR 0.95, $P = 0.014$), GWE (HR 0.94, $P = 0.018$), and GWI (HR 0.95, $P = 0.004$). However, GWI showed a higher model fit ($\chi^2 = 8.71$) than LVEF or GWE ($\chi^2 = 5.40$ and 5.64 respectively). In contrast, no significant association was observed with SW, CPO, and GLS.

Multivariable Cox regression (Table 4) confirmed a significant association of GWI with the outcome. This association was independent of LVEF and GLS (Table 4) as well as various clinical parameters as

Table 1.
Clinical baseline characteristics

Parameter	All cohort (*n* = 144)	Survivors (*n* = 124)	Non-survivors (*n* = 20)	P
Age, years	82.0 [78.0–86.0]	80.0 [77.0–85.0]	82.0 [78.0–86.0]	0.545
Male, n (%)	73 (51)	62 (50)	11 (55)	0.679
BSA, m^2	1.8 [1.7–2.0]	1.8 [1.6–1.9]	1.84 [1.7–2.0]	0.275
BMI, kg/m^2	26.1 [24.2–29.2]	25.4 [23.1–27.5]	26.2 [24.6–29.6]	0.091
Creatinine clearance, mL/min	74 [69–112]	75 [67–121]	71 [66–119]	0.751
Heart rate, bpm	71.0 [63.0–79.0]	71.0 [66.0–76.0]	71.0 [62.0–79.0]	0.824
SBP, mmHg	129.0 [113.0–140.0]	119 [109.0–135.0]	130.0 [116.0–140.0]	0.193
DBP, mmHg	65.0 [55.0–71.0]	68.0 [59–79.0]	63.5 [55.0–70.0]	0.077
MBP, mmHg	43.0 [31.0–55.0]	50.0 [39.0–62.0]	41 [30.0–52.0]	0.023*
Hypertension, n (%)	114 (79)	97 (78)	17 (85)	0.135
Diabetes mellitus, n (%)	38 (26)	33 (27)	5 (25)	0.980
Smoking, n (%)	6 (4)	6 (5)	0 (0)	0.134
Dyslipidaemia, n (%)	93 (65)	80 (65)	13 (65)	0.967
COPD, n (%)	19 (13)	17 (14)	2 (1)	0.704
Clinically relevant CAD, n (%)	72 (50)	62 (50)	10 (50)	0.857
Renal replacement or dialysis, n (%)	3 (2)	3 (2)	0 (0)	0.500
Euro-Score II	2.9 [1.9–5.0]	2.7 [1.9–4.5]	3.6 [1.8–10.4]	0.188
STS-Score	3.2 [2.3–5.1]	3.2 [2.3–4.7]	3.3 [2.5–6.5]	0.444

All parameters are described as median [inter-quartile range] unless stated otherwise. * significanl difference between the survivors and non-survivors, P < 0.05

Table 2.
Echocardiographic baseline characteristics

Parameter	All cohort (n = 144)	Survivors (n = 124)	Non-survivors (n = 20)	P
LVEDVI, ml/m^2	52.0 [45.0–64.0]	52.0 [45.0–62.0]	54.0 [44.0–70.0]	0.355
LVEF, %	58 [52.0–64.0]	59.0 [50.0–65.0]	51.0 [45.0–63.0]	0.039*
SVI, $ml/min/m^2$	38.0 [35.0–54.0]	38.0 [30.0–45.0]	39 [35.0–48.0]	0.075
SW, Joules	1423.0 [1043.0–2022.0]	1389.0 [1051.0–1993.0]	1691.0 [969.0–2151.0]	0.640
CPO, Watts	245.0 [183.0–328.0]	244.0 [181.0–328.0]	252.0 [189.0–332.0]	0.751
GLS, %	−15.0 [−17.0 to −13.0]	−15.0 [−17.0 to −13.0]	−14 [−16.5 to −11.0]	0.118
GWE, %	91.0 [88.0–94.0]	92.0 [88.0–95.0]	89.0 [82.0–93.0]	0.047*
GWI, mmHg %	1335.0 [1016.0–1641.0]	1389.0 [1070.0–1653.0]	1029.0 [641.0–1382.0]	0.003*

All parameters are described as median [inter-quartile range] unless stated otherwise.

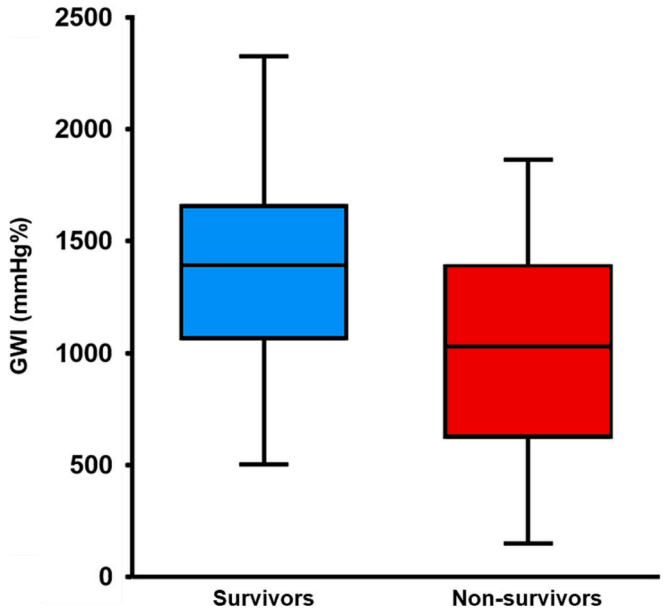


Fig. 2.. Global work index (GWI) was significantly lower among the non-survivors than the survivors ($P = 0.003$)

summarized in Table 5 (clinical model 1: age, gender, chronic kidney disease, and coronary artery disease; clinical model 2: BMI, creatinine clearance, and mean blood pressure). Inclusion of GWI to the aforementioned models (Tables 4 and 5) significantly improved model fit of all the multivariable models in comparison to the respective nested models (ANOVA X^2 for systolic function model: $P = 0.008$; for clinical model 1: $P = 0.030$; for clinical model 2: $P = 0.020$).

4. Discussion

This study determines the association of various parameters characterizing LV systolic function with long-term cardiovascular mortality after TAVI. GWI was lower among non-survivors than survivors, differentiated non-survivors from survivors, and showed a strong association with outcome, while GLS, SW, and CPO did not. Association of GWI with mortality was independent of age, gender, chronic kidney disease, and coronary artery disease and improved the fitness of this clinical model. Similar observations were made in a systolic function model including LVEF and GLS. Hence, GWI provides added value for follow-up after TAVI which may be related to both integration of LV afterload and measurement of LV deformation throughout the cardiac cycle.

In the recent literature, MW parameters were useful for characterizing LV systolic function and LV adaptive remodelling in different cardiovascular conditions such as arterial hypertension, myocardial infarction, various cardiomyopathies, and response to cardiac resynchronization therapy. The method provided particularly promising results in characterizing ventricular function after cardiac resynchronization.

Table 3
Univariable Cox regression models for cardiovascular mortality

Variable	Cox Regression			Model Fit	
	HR	95% CI	P	χ^2	χ^2 P
LVEF, %	0.95	0.92–0.99	0.014*	5.40	0.020*
SW, Joule	1.00	0.98–1.01	0.578	0.31	0.582
CPO, Watt	1.00	0.99–1.01	0.898	0.12	0.899
GLS, %	1.10	0.94–1.28	0.231	1.11	0.235
GWE, %	0.94	0.89–0.98	0.006*	5.64	0.018*
GWI, mmHg%	0.96	0.94–0.99	0.004*	8.71	0.008*

* significant association with cardiovascular mortality P < 0.05 / Signifcant Model Fit test χ^2 P < 0.05

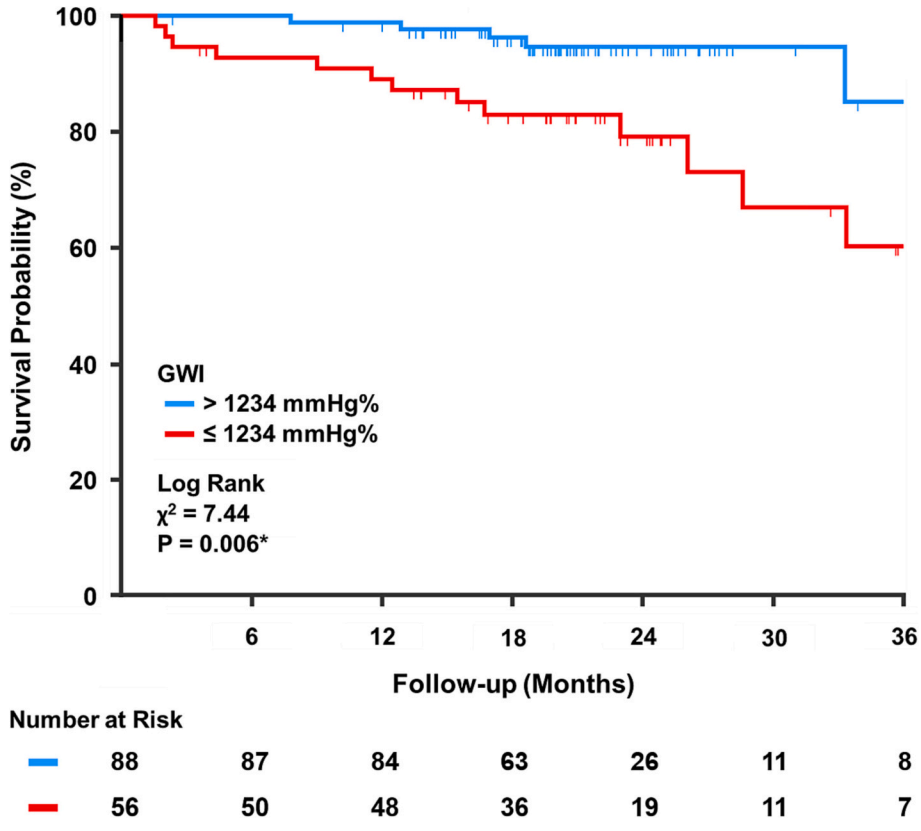


Fig. 3.. Comparison of survival probability according to global work index (GWI) for cardiovascular mortality.

Table 4
Multivariable Cox regression model of LVEF and GLS for cardiovascular mortality

Variables	Cox Regression			Model Fit	
	HR	95% CI	P	χ^2	χ^2 P
LVEF, %	0.95	0.92–0.99	0.014*	6.87	0.032*
GLS, %	1.10	0.94–1.29	0.224		
+ GWE	0.94	0.89–0.99	0.023*	10.88	0.012*
+ GWI	0.98	0.97–0.99	0.031*	12.71	0.008*

* significant association with cardiovascular mortality $P < 0.05$ / Signifcant Model Fit test χ^2 $P < 0.05$

Table 5
Multivariable Cox regression models of clinical characteristics (Clinical Model 1 & 2) for cardiovascular mortality

Variables	Cox Regression			Model Fit	
	HR	95% CI	P	χ^2	χ^2 P
Clinical Model 1					
Age, year	0.98	0.91–1.06	0.632		
Female gender	0.61	0.22–2.34	0.339	3.05	0.550
CAD	0.83	0.29–2.34	0.827		
Renal replacement or dialysis	1.06	0.01–9.37	0.957		
+ LVEF, %	0.95	0.91–0.99	0.040*	6.51	0.260
+ GWE, %	0.87	0.80–0.95	0.005*	10.29	0.031*
+ GWI, mmHg%	0.98	0.96–0.99	0.003*	12.39	0.030*
Clinical Model 2					
BMI, kg/m^2	0.96	0.86–1.08	0.504		
Creatinine clearance, mL/min	0.99	0.98–1.01	0.746	4.48	0.215
MBP, mmHg	1.03	0.99–1.06	0.067		
+ LVEF, %	0.95	0.91–0.99	0.022*	9.45	0.051
+ GWE, %	0.92	0.90–0.99	0.003*	10.65	0.031*
+ GWI, mmHg%	0.98	0.98–0.99	0.009*	12.65	0.020*

* significant association with cardiovascular mortality $P < 0.05$ / Signifcant Model Fit test χ^2 $P < 0.05$.

The integration of ventricular afterload may be one reason accounting for this finding. Furthermore, ventricular deformation is analysed throughout the cardiac cycle, which represents an major conceptual difference from GLS determining deformation at the time point of aortic valve closure only. The pressure strain loop resulting from cardiac cycle based analysis provides important additional information on ventricular function such as constructive and waisted work. Hence, this tool accounts for the coordination of ventricular contraction describing the efficiency of myocardial function. For these reasons, MW analysis may offer advantages over GLS and other parameters for follow-up of patients after aortic valve replacement. [12–24]

In patients with severe aortic stenosis, pre-TAVI MW was significantly lower in those with NYHA class III-IV compared to those with NYHA Class I-II. In another study the pre-TAVI MW values were higher than the respective post-TAVI parameters. To the best of our knowledge, MW parameters have not been studied in the context of long-term mortality after TAVI. The data of the current study demonstrate that GWI provided added value for follow-up of these patients. It is particularly interesting that the association of GWI with all-cause mortality was independent of various clinical and LV functional parameters including GLS. This finding may be attributable to the principal differences between GWI and GLS, while the lacking significance of the GLS outcome association in the current dataset may be due to the relatively low patient number. To study ventricular deformation against estimated ventricular pressure throughout the cardiac cycle may indeed provide a better picture of ventricular systole as compared to the information derived from deformation at the time point of aortic valve closure alone. However, the integration of afterload as it is achieved by determining SW or CPO seems to be much less useful for outcome prediction than GWI. This observation may again be related to the incomplete picture of ventricular systole resulting from inclusion of data not reflecting the full cardiac cycle. Hence, afterload correction seems to be a less relevant aspect of GWI than integration of LV deformation throughout the cardiac cycle, and the latter seems to determine the strong association of GWI with long-term mortality after TAVI. [18–27]

Limitations

This study is limited by its retrospective single-center design. A selection bias cannot be excluded completely because inclusion criteria involved echocardiographic quality criteria as well as several additional clinical parameters. LV functional parameters were used for Kaplan-Meier survival analyses based on an optimal threshold value which was determined by ROC analysis in the same study population. Additional studies and large-scale cohorts are necessary to validate the current findings.

5. Conclusions

In long-term follow-up of patients with severe aortic stenosis treated by TAVI, GWI was lower in non-survivors than survivors, differentiated non-survivors from survivors, was associated with mortality independent of age, gender, chronic kidney disease, coronary artery disease and LV functional parameters, and improved the fitness of the clinical as well as the LV functional model with regard to outcome association. In contrast, GLS, SW, and CPO did not show any of these properties in the current dataset. Hence, GWI provides added value for follow-up after TAVI, and this effect may be related to integration of LV deformation throughout the cardiac cycle, while an additional effect by inclusion of LV afterload cannot be excluded.

Financial support

This study was supported by the Swiss Heart Foundation (Research Grant FF21059).

CRediT authorship contribution statement

Shehab Anwer: Formal analysis, Methodology, Project administration, Software, Supervision, Validation, Writing – original draft, Writing – review & editing. **Sinuhe Nussbaum:** Data curation, Methodology, Software, Writing – review & editing. **Neria E. Winkler:** Project administration, Resources, Validation, Writing – review & editing. **Dominik C. Benz:** Writing – original draft, Writing – review & editing. **Dominik Zuercher:** Data curation, Investigation, Resources, Writing – original draft, Writing – review & editing. **Thierry G. Donati:** Data curation. **Glykeria Tsiourantani:** Data curation. **Verena Wilzeck:** Data curation. **Jonathan M. Michel:** Data curation, Investigation, Methodology. **Albert M. Kasel:** Data curation, Methodology. **Felix C. Tanner:** Conceptualization, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Validation, Writing – original draft, Writing – review & editing.

Declaration of competing interest

Nothing to Disclose.

Data availability

The data that support the findings of this study are available upon reasonable request from the authors.

References

[1] B. Iung, V. Delgado, R. Rosenhek, S. Price, B. Prendergast, O. Wendler, et al., Contemporary presentation and management of valvular heart disease: the

EURObservational research programme valvular heart disease II survey, Circulation. 140 (14) (2019 Oct) 1156–1169, https://doi.org/10.1161/CIRCULATIONAHA.119.041080. Epub 2019 Sep 12. PMID: 31510787.
[2] H. Baumgartner, V. Falk, J.J. Bax, M. De Bonis, C. Hamm, P.J. Holm, et al., 2017 ESC/EACTS guidelines for the management of valvular heart disease, Eur. Heart J. 38 (36) (2017 Sep 21) 2739–2791, https://doi.org/10.1093/eurheartj/ehx391 (PMID: 28886619).
[3] S. Hein, E. Arnon, S. Kostin, M. Schönburg, A. Elsässer, V. Polyakova, et al., Progression from compensated hypertrophy to failure in the pressure-overloaded human heart: structural deterioration and compensatory mechanisms, Circulation. 107 (7) (2003 Feb 25) 984–991, https://doi.org/10.1161/01.cir.0000051865.66123.b7 (PMID: 12600911).
[4] F. Weidemann, S. Herrmann, S. Störk, M. Niemann, S. Frantz, V. Lange, et al., Impact of myocardial fibrosis in patients with symptomatic severe aortic stenosis, Circulation. 120 (7) (2009 Aug 18) 577–584, https://doi.org/10.1161/CIRCULATIONAHA.108.847772. Epub 2009 Aug 3. PMID: 19652094.
[5] A.C.T. Ng, E.A. Prihadi, M.L. Antoni, M. Bertini, S.H. Ewe, N. Ajmone Marsan, et al., Left ventricular global longitudinal strain is predictive of all-cause mortality independent of aortic stenosis severity and ejection fraction, Eur. Heart J. Cardiovasc. Imaging 19 (8) (2018 Aug 1) 859–867, https://doi.org/10.1093/ehjci/jex189 (PMID: 28950306).
[6] T. Yingchoncharoen, C. Gibby, L.L. Rodriguez, R.A. Grimm, T.H. Marwick, Association of myocardial deformation with outcome in asymptomatic aortic stenosis with normal ejection fraction, Circ. Cardiovasc. Imaging 5 (6) (2012 Nov) 719–725, https://doi.org/10.1161/CIRCIMAGING.112.977348. Epub 2012 Sep 24. PMID: 23008423.
[7] L.G. Kearney, K. Lu, M. Ord, S.K. Patel, K. Profitis, G. Matalanis, et al., Global longitudinal strain is a strong independent predictor of all-cause mortality in patients with aortic stenosis, Eur. Heart J. Cardiovasc. Imaging 13 (10) (2012 Oct) 827–833, https://doi.org/10.1093/ehjci/jes115. Epub 2012 Jun 26. PMID: 22736713.
[8] R. Kafa, K. Kusunose, A.L. Goodman, L.G. Svensson, J.F. Sabik, B.P. Griffin, M. Y. Desai, Association of abnormal postoperative left ventricular global longitudinal strain with outcomes in severe aortic stenosis following aortic valve replacement, JAMA Cardiol. 1 (4) (2016 Jul 1) 494–496, https://doi.org/10.1001/jamacardio.2016.1132 (PMID: 27438331).
[9] C.P. Huded, K. Kusunose, F. Shahid, A.L. Goodman, A. Alashi, R.A. Grimm, et al., Novel echocardiographic parameters in patients with aortic stenosis and preserved left ventricular systolic function undergoing surgical aortic valve replacement, Am. J. Cardiol. 122 (2) (2018 Jul 15) 284–293, https://doi.org/10.1016/j.amjcard.2018.03.359. Epub 2018 Apr 12. PMID: 29751954.
[10] J.S. Dahl, J. Magne, P.A. Pellikka, E. Donal, T.H. Marwick, Assessment of subclinical left ventricular dysfunction in aortic stenosis, JACC Cardiovasc. Imaging 12 (1) (2019 Jan) 163–171, https://doi.org/10.1016/j.jcmg.2018.08.040 (PMID: 30621988).
[11] B. Fries, D. Liu, P. Gaudron, K. Hu, P. Nordbeck, G. Ertl, F. Weidemann, S. Herrmann, Role of global longitudinal strain in the prediction of outcome in patients with severe aortic valve stenosis, Am. J. Cardiol. 120 (4) (2017 Aug 15) 640–647, https://doi.org/10.1016/j.amjcard.2017.05.032. Epub 2017 May 30. PMID: 28648391.
[12] V. Reskovic Luksic, A. Postolache, C. Martinez, R. Dulgheru, F. Ilardi, J. Tridetti, et al., Global and regional myocardial function and outcomes after transcatheter aortic valve implantation for aortic stenosis and preserved ejection fraction, J. Cardiovasc. Med. (Hagerstown) 21 (3) (2020 Mar) 238–245, https://doi.org/10.2459/JCM.0000000000000918 (PMID: 31815853).
[13] F. Al-Rashid, M. Totzeck, N. Saur, R.A. Jánosi, A. Lind, A.A. Mahabadi, et al., Global longitudinal strain is associated with better outcomes in transcatheter aortic valve replacement, BMC Cardiovasc. Disord. 20 (1) (2020 Jun 3) 267, https://doi.org/10.1186/s12872-020-01556-4. PMID: 32493384; PMCID: PMC7268397.
[14] S. Anwer, D. Oğuz, L. Galian-Gay, I. Peovska Mitevska, L. Baghdassarian, R. Dulgheru, et al., Echocardiographic findings on aortic stenosis: an observational, prospective, and multi-center registry, Perfusion. 36 (3) (2021 Apr) 269–276, https://doi.org/10.1177/0267659120924921. Epub 2020 Jul 10. PMID: 32650695.
[15] E. Donal, C. Bergerot, H. Thibault, L. Ernande, J. Loufoua, L. Augeul, et al., Influence of afterload on left ventricular radial and longitudinal systolic functions: a two-dimensional strain imaging study, Eur. J. Echocardiogr. 10 (8) (2009 Dec) 914–921, https://doi.org/10.1093/ejechocard/jep095. Epub 2009 Aug 7. PMID: 19666722.
[16] J.L. Robotham, M. Takata, M. Berman, Y. Harasawa, Ejection fraction revisited, Anesthesiology. 74 (1) (1991) 172–183.
[17] K.R. Walley, Left ventricular function: time-varying elastance and left ventricular aortic coupling, Crit. Care 20 (2016) 270.
[18] K. Russell, M. Eriksen, L. Aaberge, N. Wilhelmsen, H. Skulstad, E.W. Remme, et al., A novel clinical method for quantification of regional left ventricular pressure-strain loop area: a non-invasive index of myocardial work, Eur. Heart J. 33 (2012) 724–733.
[19] J. Chan, N.F.A. Edwards, B.K. Khandheria, K. Shiino, S. Sabapathy, B. Anderson, et al., A new approach to assess myocardial work by noninvasive left ventricular pressure-strain relations in hypertension and dilated cardiomyopathy, Eur. Heart J. Cardiovasc. Imaging 20 (2019) 31–39.
[20] R.P. Lustosa, F. Fortuni, P. van der Bijl, L. Goedemans, M. El Mahdiui, J. M. Montero-Cabezas, et al., Left ventricular myocardial work in the culprit vessel territory and impact on left ventricular remodelling in patients with ST-segment elevation myocardial infarction after primary percutaneous coronary intervention, Eur. Heart J. Cardiovasc. Imaging 22 (2021) 339–347.
[21] Y.L. Hiemstra, P. van der Bijl, M. El Mahdiui, J.J. Bax, V. Delgado, N.A. Marsan, Myocardial work in nonobstructive hypertrophic cardiomyopathy: implications for outcome, J. Am. Soc. Echocardiogr. 33 (2020) 1201–1208.
[22] E. Galli, C. Leclercq, A. Hubert, A. Bernard, O.A. Smiseth, P. Mabo, et al., Role of myocardial constructive work in the identification of responders to CRT, Eur. Heart J. Cardiovasc. Imaging 19 (2018) 1010–1018.
[23] P. van der Bijl, N.M. Vo, M.V. Kostyukevich, B. Mertens, N. Ajmone Marsan, V. Delgado, et al., Prognostic implications of global, left ventricular myocardial work efficiency before cardiac resynchronization therapy, Eur. Heart J. Cardiovasc. Imaging 20 (2019) 1388–1394.
[24] E. Boe, K. Russell, C. Eek, M. Eriksen, E.W. Remme, O.A. Smiseth, et al., Noninvasive myocardial work index identifies acute coronary occlusion in patients with non-ST-segment elevation-acute coronary syndrome, Eur. Heart J. Cardiovasc. Imaging 16 (2015) 1247–1255.
[25] J.U. Voigt, G. Pedrizzetti, P. Lysyansky, T.H. Marwick, H. Houle, R. Baumann, Pet al. Definitions for a common standard for 2D speckle tracking echocardiography: consensus document of the EACVI/ASE/industry task force to standardize deformation imaging, J. Am. Soc. Echocardiogr. 28 (2) (2015 Feb) 183–193, https://doi.org/10.1016/j.echo.2014.11.003 (PMID: 25623220).
[26] R.M. Lang, L.P. Badano, V. Mor-Avi, J. Afilalo, A. Armstrong, L. Ernande, et al., Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging, J. Am. Soc. Echocardiogr. 28 (1) (2015 Jan) 1–39, e14, https://doi.org/10.1016/j.echo.2014.10.003 (PMID: 25559473).
[27] VARC-3 WRITING COMMITTEE, P. Généreux, N. Piazza, M.C. Alu, T. Nazif, R. T. Hahn, P. Pibarot, et al., Valve academic research consortium 3: updated endpoint definitions for aortic valve clinical research, Eur. Heart J. 42 (19) (2021 May 14) 1825–1857, https://doi.org/10.1093/eurheartj/ehaa799 (PMID: 33871579).
[28] F. Fortuni, S.C. Butcher, F. van der Kley, R.P. Lustosa, I. Karalis, A. de Weger, et al., Left ventricular myocardial work in patients with severe aortic stenosis, J. Am. Soc. Echocardiogr. 34 (3) (2021 Mar) 257–266, https://doi.org/10.1016/j.echo.2020.10.014. Epub 2020 Nov 9. PMID: 33181281.
[29] R. Jain, T. Bajwa, S. Roemer, H. Huisheree, S.Q. Allaqaband, S. Kroboth, et al., Myocardial work assessment in severe aortic stenosis undergoing transcatheter aortic valve replacement, Eur. Heart J. Cardiovasc. Imaging 22 (6) (2021 May 10) 715–721, https://doi.org/10.1093/ehjci/jeaa257 (PMID: 33106854).