Universiteit Leiden
The Netherlands

Changes in computed-tomography-derived segmental left ventricular longitudinal strain after transcatheter aortic valve implantation

Singh, G.K.; Fortuni, F.; Kuneman, J.H.; Vollema, E.M.; Kley, F. van der; Marsan, N.A.; ... ; Bax, J.J.

Citation
Singh, G. K., Fortuni, F., Kuneman, J. H., Vollema, E. M., Kley, F. van der, Marsan, N. A., ... Bax, J. J. (2023). Changes in computed-tomography-derived segmental left ventricular longitudinal strain after transcatheter aortic valve implantation. *American Journal Of Cardiology, 198*, 95-100. doi:10.1016/j.amjcard.2023.04.021

Version:	Publisher's Version
License:	Creative Commons CC BY 4.0 license
Downloaded from:	https://hdl.handle.net/1887/3728725

Note: To cite this publication please use the final published version (if applicable).

Changes in Computed-Tomography-Derived Segmental Left Ventricular Longitudinal Strain After Transcatheter Aortic Valve Implantation

Gurpreet K. Singh, MD[a], Federico Fortuni, MD[a,b], Jurrien H. Kuneman, MD[a], E. Mara Vollema, MD[a], Frank van der Kley, MD[a], Nina Ajmone Marsan, MD, PhD[a], Victoria Delgado, MD, PhD[a,c], and Jeroen J. Bax, MD, PhD[a,d,*]

Patients with severe aortic stenosis (AS) may show left ventricular (LV) apical longitudinal strain sparing. Transcatheter aortic valve implantation (TAVI) improves LV systolic function in patients with severe AS. However, the changes in regional longitudinal strain after TAVI have not been extensively evaluated. This study aimed to characterize the effect of the pressure overload relief after TAVI on LV apical longitudinal strain sparing. A total of 156 patients (mean age 80 ± 7 years, 53% men) with severe AS who underwent computed tomography before and within 1 year after TAVI (mean time to follow-up 50 ± 30 days) were included. LV global and segmental longitudinal strain were assessed using feature tracking computed tomography. LV apical longitudinal strain sparing was evaluated as the ratio between the apical and midbasal longitudinal strain and was defined as an LV apical to midbasal longitudinal strain ratio >1. LV apical longitudinal strain remained stable after TAVI (from 19.5 ± 7.2% to 18.7 ± 7.7%, p = 0.20), whereas LV midbasal longitudinal strain showed a significant increase (from 12.9 ± 4.2% to 14.2 ± 4.0%, p ≤0.001). Before TAVI, 88% of the patients presented with LV apical strain ratio >1% and 19% presented with an LV apical strain ratio >2. After TAVI, these percentages significantly decreased to 77% and 5% (p = 0.009, p ≤0.001), respectively. In conclusion, LV apical sparing of strain is a relatively common finding in patients with severe AS who underwent TAVI and its prevalence decreases after the afterload relief after TAVI. **© 2023 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/)** **(Am J Cardiol 2023;198:95−100)**

In aortic stenosis (AS), the left ventricular (LV) myocardium remodels in response to the pressure overload, with an increase of the sarcomeres in parallel that leads to LV hypertrophy.[1] These structural changes reduce wall tension, increase LV contractility, and maintain systemic and myocardial perfusion. Nevertheless, LV hypertrophy is also accompanied by extracellular matrix deposition and fibrosis, which are the starting points of myocardial damage and eventually lead to overt LV diastolic and systolic dysfunction. When patients with severe AS become symptomatic or develop LV systolic dysfunction their prognosis is dismal with 50% mortality within 2 years.[2] Transcatheter aortic valve implantation (TAVI) or surgical aortic valve replacement are the only effective therapies to halt LV remodeling and restore LV systolic function in patients with severe AS.[3] Segmental and global longitudinal strain (GLS) are more sensitive markers of LV systolic function than LV ejection fraction in patients with severe AS. Particularly important is the presence of the LV apical sparing pattern of longitudinal strain (LS), which is characterized by a ratio between the apical and midbasal LS values >1.[4,5] This segmental pattern of LV LS has also been considered a potential marker of cardiac amyloidosis.[5] Little is known about the effect of pressure overload relief after aVR on LV apical sparing of LS. If the LV apical sparing pattern would be related to the presence of amyloid protein deposits, it could be hypothesized that the pattern would not change after aVR. Accordingly, the aim of the present study was to investigate the changes in LV global and segmental LS in patients with severe AS after TAVI using feature tracking computed tomography (CT).

Methods

A total of 156 patients who underwent TAVI for severe AS between November 2007 and September 2016 with CT data before TAVI and within 1 year of follow-up were included.

Patients with previous aVR or without CT data with image reconstructions at the 10% phase of the cardiac cycle were excluded. The severity of AS was quantified with echocardiography using the continuity equation[6], and severe AS was defined as an aortic valve area indexed to body surface area <0.6 cm^2/m^2.[3,6] Clinical data were collected from the electronic records of the Cardiology

[a]Department of Cardiology, Leiden University Medical Center, Leiden, The Netherlands; [b]Department of Cardiology, San Giovanni Battista Hospital, Foligno, Italy; [c]Heart Institute, Hospital University Germans Trias i Pujol, Badalona, Spain; and [d]Heart Center, Turku University Hospital, University of Turku, Turku, Finland. Manuscript received January 15, 2023; revised manuscript received and accepted April 13, 2023.

Drs. Singh and Fortuni contributed equally as first authorship.

Funding: none.

See page 99 for Declaration of Conflict of Interest.

*Corresponding author: Tel: +3175262020; fax: +31715266809.

E-mail address: j.j.bax@lumc.nl (J.J. Bax).

0002-9149/© 2023 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/)

https://doi.org/10.1016/j.amjcard.2023.04.021

Department (EPD Vision, version 12.3.5.0, Leiden University Medical Center, Leiden, The Netherlands) and included demographics, symptoms, cardiovascular risk factors, and medication use. Segmental and global LV and right ventricular (RV) LS were assessed, and the prevalence of LV apical sparing was evaluated. Furthermore, changes in LV and RV LS and LV apical sparing after TAVI were evaluated. The institutional review board approved this retrospective study and waived the need for patient written informed consent.

Multidetector row CT scans were performed using a 320-slice CT scanner (Aquilion ONE, Toshiba Medical Systems, Otawara, Japan) before (median 8, interquartile range [IQR] 3 to 18 days before the procedure) and after TAVI (median 39, IQR 32 to 58 days after the procedure) according to a dedicated cardiac CT protocol.[7] The entire cardiac cycle was imaged using prospective dose modulation, which was triggered to the electrocardiogram. The images were subsequently transferred to a remote work station, enabling offline analysis using a dedicated software (Medis Suite CT v3.1, Medis Medical Imaging Systems, Leiden, The Netherlands). LV GLS, RV GLS, LV ejection fraction, LV mass, LV end-diastolic volume, and LV end-systolic volume were assessed using the novel feature tracking tool of the software. The 2-, 3-, and 4-chamber LV views were manually reconstructed from the 3-dimensional multiplanar reconstructions of the CT images. LV GLS, LV ejection fraction, LV end-diastolic volume, and LV end-systolic volume were subsequently measured by tracing the LV endocardial borders manually in each view at end-diastole and end-systole, and the remaining cardiac phases were identified automatically by the software.[8] Adequate trace quality was assessed throughout the cardiac cycle and adjustments of the tracings were performed if the endocardial border was not properly traced by the software. The RV endocardial border was also manually traced in the apical 4-chamber view, including both the free wall and the septum, and the RV GLS was derived by the dedicated software. A bull's eye plot of the LV with regional LS values for the 17 segments of the myocardium was obtained with the software.[9] To characterize the myocardial deformation of the LV apex relative to the midbasal segments of the LV, the following parameters were identified: (1) the apical LS, which was calculated as the average value of LS of the 5 apical segments of the LV, (2) the midbasal LS, which represented the average LS of the 12 midbasal segments of the LV, and (3) the relative LV apical strain sparing, which was evaluated as the ratio between the apical and midbasal LS. The percentage of patients with values of apical to midbasal LV LS >1, and >2 were calculated to investigate the prevalence of LV apical strain sparing according to different definitions provided in previous studies.[5,10,11] Figure 1 shows the basal, mid-, and apical segments of the LV and illustrates how the relative LV apical sparing of strain was assessed. In addition, stroke volume was calculated by subtracting the LV end-systolic volume from the LV end-diastolic volume; and LV mass was measured by tracing the epicardial and endocardial borders at end-diastole.

Continuous variables are presented as mean ± SD if normally distributed and as median and IQR if they did not follow a normal distribution. Categorical data are presented as frequencies and percentages. CT morphologic and functional cardiac parameters before and after TAVI were compared using the paired Student's *t* test for normally distributed continuous variables, the Wilcoxon signed rank test for non-normally distributed continuous variables, and the McNemar test for categorical variables. A 2-sided p <0.05 was considered statistically significant. Statistical analysis was performed using the SPSS software (version 25.0; IBM, Armonk, New York).

Results

Baseline clinical characteristics of the patient population are listed in Table 1. A total of 156 patients with severe AS

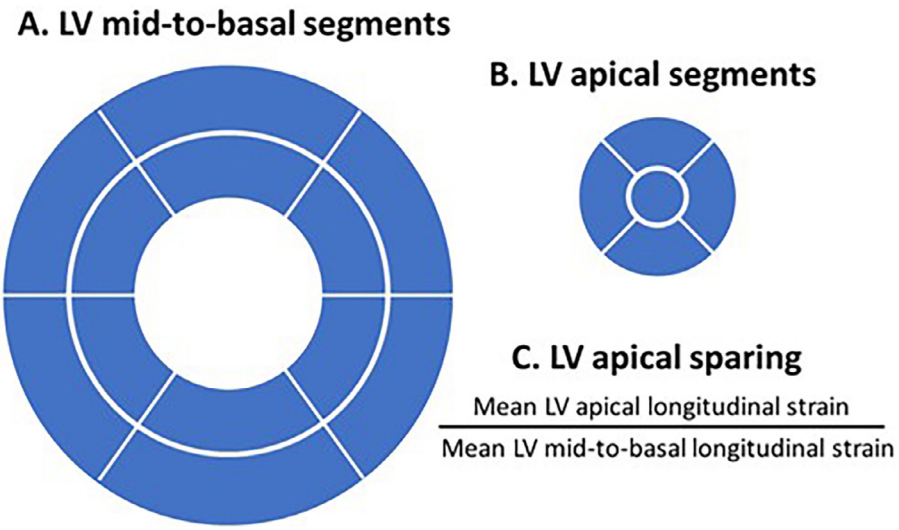

Figure 1. MDCT-derived segmental strain to evaluate LV apical sparing of strain. *Panel A* shows the 12 mid- and basal segments of the LV, whose longitudinal strain values were averaged to obtain an estimation of the mid-to-basal LV contraction. *Panel B* shows the 5 apical segments of the LV whose longitudinal strain values were averaged to evaluate the apical contraction. *Panel C* illustrates the formula that was used to assess the relative LV apical sparing of strain.

Table 1
Baseline characteristics

	Total population n=156
Age, years	80 ± 7
Male (%)	82 (53)
Body surface area, m^2	1.84 ± 0.19
Creatinine, mg/dL	0.98 (0.77-1.14)
Systolic blood pressure, mmHg	137 ± 23
Diastolic blood pressure, mmHg	69 ± 12
NYHA class (%)	
Class 1-2	58 (38)
Class 3-4	95 (62)
Cardiovascular risk factors (%)	
Diabetes mellitus	52 (33)
Hypertension	116 (74)
Hypercholesterolemia	123 (79)
Smoking	62 (40)
History of CAD	105 (67)
Medication (%)	
Beta-blockers	104 (67)
ACE or ARB	90 (58)
Statin	105 (68)
Diuretics	91 (59)
Aspirin	80 (53)
Oral anticoagulants	50 (33)

ACE = angiotensin converting enzyme; ARB = angiotensin receptor blocker; CAD = coronary artery disease; LV = left ventricular; NYHA = New York Heart Association.

Diabetes mellitus was defined as having a history of diabetes mellitus and medical therapy with insulin, oral glucose-lowering drugs or diet; Hypertension was defined as a documented history or prior use of antihypertensive medication; Hypercholesterolemia was defined as previous statin use and/or having a documented history of hypercholesterolemia.

who underwent TAVI (53% men, 80 ± 7 years) were included. The majority of patients (62%) presented with New York Heart Association class III to IV heart failure symptoms. There was a high prevalence of cardiovascular risk factors, including hypertension (74%), hypercholesterolemia (79%) and known coronary artery disease (67%). Most of the patients used cardiovascular medication, including angiotensin converting enzyme or angiotensin receptor blocker (58%), β blockers (67%), diuretics (59%), and statins (68%).

The baseline and follow-up CT data are listed in Table 2. The time interval between TAVI and the follow-up CT was 39 days (IQR 32 to 58). According to the CT performed after TAVI, there was a significant increase in stroke volume from 43 ± 12 to 48 ± 14 ml/m^2 ($p \leq 0.001$) and a significant decrease in LV mass from 93 ± 22 to 83 ± 19 g/m^2 ($p \leq 0.001$). LV ejection fraction improved during follow-up, whereas LV GLS (15.1 ± 5.5 to 15.5 ± 5.8, p = 0.45) and RV GLS (19.7 ± 8.5 to 20.0 ± 8.2, p = 0.79) remained unchanged. Before TAVI, the majority of the patients presented with an LV apical strain sparing ratio >1 (88%), whereas only 19% presented with a ratio >2. Interestingly, after TAVI, these percentages significantly decreased to 77% and 5% (p = 0.009, $p \leq 0.001$), respectively. The segmental regional LV LS values followed different trends after the procedure. Although there was no difference in the average apical LS (from 19.5 ± 7.2% before to 18.7 ± 7.7% after TAVI, p = 0.20), the midbasal LS showed a significant increase (from 12.9 ± 4.2% before to 14.2 ± 3.9% after TAVI, $p \leq 0.001$) (Figure 2 and 3).

Table 2
MDCT characteristics at baseline and follow-up

	CT-pre TAVI n=156	CT-post TAVI n=156	p-Value
Tricuspid morphology (%)	148 (97)	-	
LV end-diastolic volume, ml	148 (122-181)	163 (133 - 195)	<0.001
LV end-systolic volume, ml	64 (48 - 97)	64 (47 - 99)	0.93
Stroke volume indexed, ml/m^2	43 ± 12	48 ± 14	<0.001
LV mass, g/m^2	93 ± 22	83 ± 19	<0.001
LV ejection fraction (%)	51 ± 14	53 ± 16	0.04
LV global longitudinal strain (%)	15.1 ± 5.5	15.5 ± 5.8	0.45
RV global longitudinal strain (%)	19.7 ± 8.5	20.0 ± 8.2	0.79
Apical longitudinal strain (%)	19.5 ± 7.2	18.7 ± 7.7	0.20
Mid-basal longitudinal strain (%)	12.9 ± 4.2	14.2 ± 3.9	<0.001
LV apical sparing	1.6 ± 0.5	1.3 ± 0.4	<0.001
LV apical sparing > 1	137 (88%)	120 (77%)	0.009
LV apical sparing > 2	30 (19%)	8 (5%)	<0.001

CT = computed tomography; LV = left ventricular; RV = right ventricular.

Discussion

This study shows that LV apical strain sparing is a common finding in patients with severe AS who underwent TAVI. However, after TAVI, the LS of the LV mid- and basal segments improve, whereas the LS of the apex remains stable, leading to a significant decrease in the frequency of LV apical strain sparing after the procedure.

In the present cohort of patients with severe AS who underwent TAVI, 88% presented with LV apical sparing of strain before TAVI. This apical strain sparing pattern has been associated with the presence of cardiac amyloidosis[5,11] but can also be simply related to LV remodeling and chronic pressure overload induced by severe AS.[12,13] Transthyretin (TTR) cardiac amyloidosis is an underestimated condition, which recently appeared to be relatively frequent in the older patients with a higher prevalence in certain diseases, such as heart failure with preserved ejection fraction and severe AS.[5,14] In cardiac amyloidosis because of TTR, the misfolded TTR agglomerates tend to deposit more at the basal segments of the LV than the apical segments, causing an impairment of the systolic function of the LV base relative to the apex, which is known as LV apical strain sparing. Nevertheless, the situation in patients with concomitant severe AS is more complex, and the LV apical sparing of strain may also be because of the chronic LV pressure overload and LV hypertrophic response to increase LV contractility and maintain systemic perfusion.[15,16] According to the Laplace law, LV wall tension because of chronic pressure overload is directly proportional to the LV dimensions, which are larger at the base than the apex; therefore, LV wall stress is more pronounced in the basal segments.[12] In severe AS, increased wall stress, LV hypertrophy, and mismatch between oxygen demand and perfusion could all contribute to the reduction in LS of the basal LV segments at a larger magnitude than in the apical segments, which leads to relative LV apical strain sparing. In the present study, it was

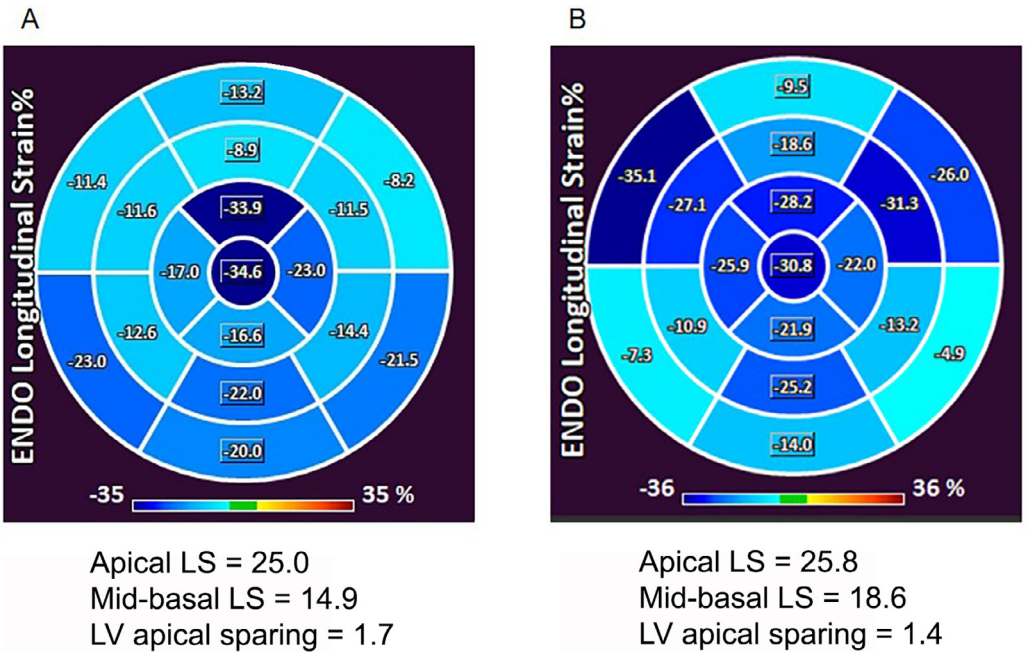

Figure 2. Illustrative case of MDCT-derived segmental strain before and after TAVI. *Panel A* shows the segmental longitudinal strain values before TAVI, with the mean apical longitudinal strain, mean mid-to-basal longitudinal strain and the relative LV sparing. *Panel B* shows the segmental longitudinal strain values after TAVI, with the mean apical longitudinal strain, mean mid-to-basal longitudinal strain and the relative LV sparing. It can be noticed that after TAVI. although the mean apical longitudinal strain remained the same, the midbasal longitudinal strain increased and this resulted in significant decrease in LV apical sparing of strain.

shown that the prevalence of CT-derived LV apical strain sparing dramatically decreases after TAVI, potentially indicating an important role of pressure overload in determining this condition that is reversible after LV pressure relief with TAVI.

Previously published reports showed that LV apical strain sparing is a relatively common finding in severe AS and should trigger additional evaluation to identify concomitant cardiac amyloidosis. In a study by Phelan et al,[11] LV apical strain sparing was shown to have a very high diagnostic accuracy to differentiate between LV hypertrophy because of AS compared with LV hypertrophy related to cardiac amyloidosis. Nonetheless, when cardiac amyloidosis and severe AS coexist, the diagnosis can become challenging, and a limitation of the study by Phelan et al is the lack of patients with concomitant AS and cardiac amyloidosis to validate their hypothesis.[11] More recently, in a population of 151 patients with severe AS systematically screened for TTR cardiac amyloidosis, Castano et al[17] demonstrated no difference in the LV apical strain sparing as assessed with speckle-tracking echocardiography between patients with lone severe AS and those with severe AS and concomitant TTR cardiac amyloidosis. In contrast to the previous studies,[11,17] in our study, we analyzed LV segmental LS with CT before and shortly after TAVI, demonstrating an important decrease in the prevalence of LV apical strain sparing. The relatively early decrease in LV apical strain sparing after TAVI may explain the low diagnostic accuracy of this marker in identifying cardiac amyloidosis in patients with severe AS because LV apical strain sparing may be simply related to the pressure overload. Also, 88% of the patients with severe AS presented with a LV apical strain sparing ratio >1 before TAVI, suggesting that this cut-off value could be too sensitive to distinguish the presence of concomitant cardiac amyloidosis from the expected LV apical sparing seen in patients with severe AS.

The limitations of the present study are inherent to its single-center retrospective design. The patients were not screened for cardiac amyloidosis; therefore, the association between this condition and LV segmental strain changes after TAVI could not be investigated. Although this study suggests a role of severe AS in determining a reversible impairment of myocardial LS of LV basal segments, the pathophysiology of this condition should be further clarified with other imaging techniques (e.g., perfusion cardiac magnetic resonance [CMR] or positron emission tomography). In this study, myocardial deformation was assessed based on multidetector row CT scans. Nevertheless, myocardial strain can be assessed with different imaging modalities using different software and technologies: speckle tracking for echocardiography and feature tracking for CT and CMR. Compared with echocardiography, CT and CMR are more complex and have less availability. Nevertheless, they are less operator-dependent and do not depend on the quality of the acoustic window, which could limit the feasibility and decrease the reliability of echocardiographic measurements in some patients. Although feature tracking and speckle-tracking measurements have different cut-off values for normality and cannot be used interchangeably, they have demonstrated to be closely related.[18,19] Finally, further prospective studies would be needed to investigate the prognostic implications of LV segmental strain changes, and changes in apical strain sparing after TAVI.

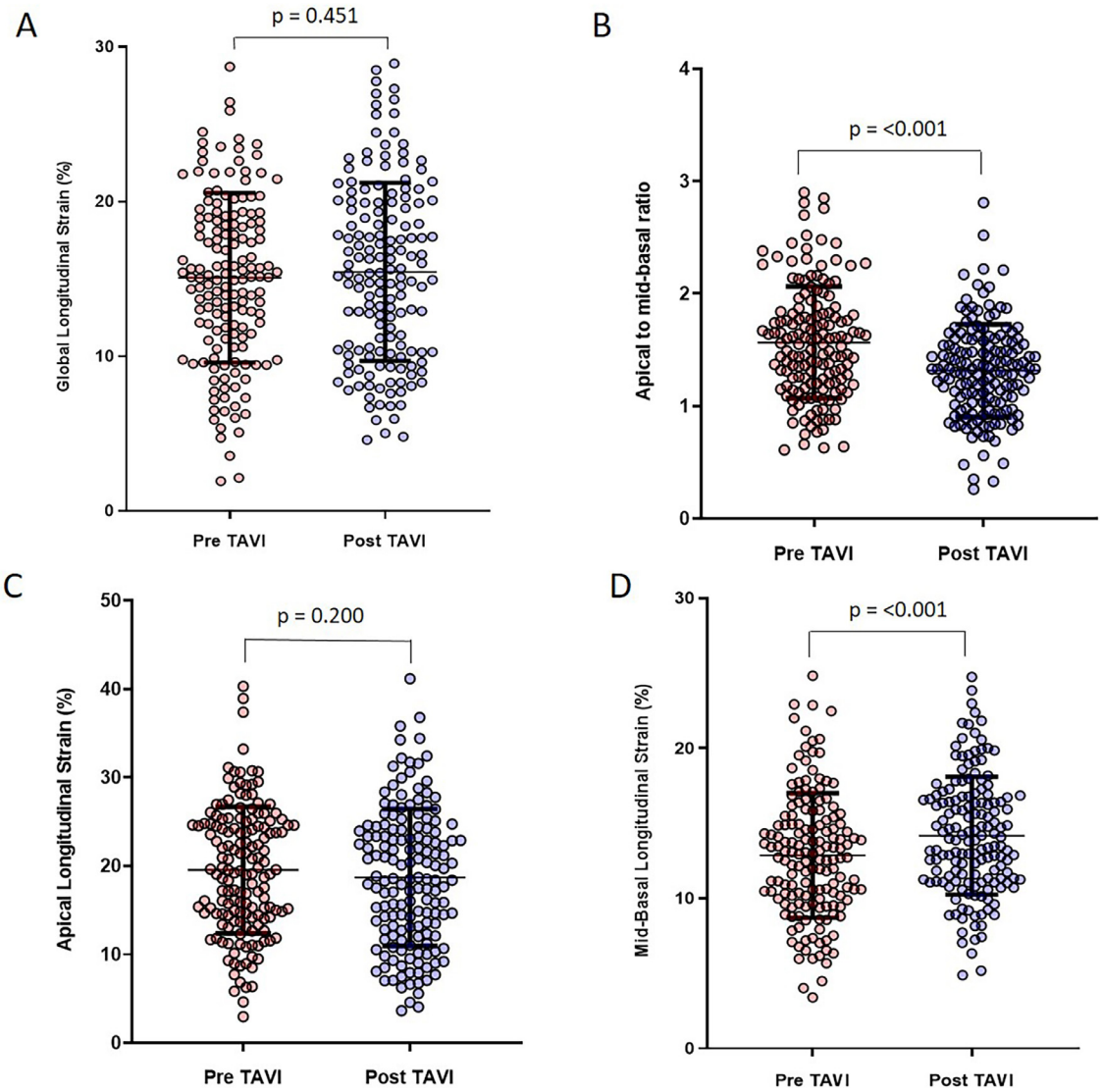

Figure 3. Changes in MDCT-derived global and segmental strain before and after TAVI**.** *Panel A* shows the LV GLS before and after TAVI. *Panel B* shows the relative LV sparing before and after TAVI. *Panel C* shows the apical longitudinal strain before and after TAVI. *Panel D* shows the longitudinal strain before and after TAVI.

In conclusion, LV apical sparing can be identified with feature tracking CT and is a relatively common finding in patients with severe AS who underwent TAVI. However, the prevalence of LV apical sparing decreases shortly after pressure relief with TAVI, suggesting a reversible pathophysiology.

Declaration of Competing Interest

The Department of Cardiology of the Leiden University Medical Center received research grants from Abbott Vascular, Bayer, Bioventrix, Biotronik, Boston Scientific, Edwards Lifesciences, GE Healthcare, and Medtronic. Dr. Delgado received speaking fees from Abbott Vascular, Edward Lifesciences, GE Healthcare, MSD, and Medtronic. Dr. Marsan received speaking fees from Abbott Vascular and GE Healthcare. Dr. Bax received speaking fees from Abbott Vascular. The remaining authors have no conflicts of interest to declare.

1. Carabello BA, Paulus WJ. Aortic stenosis. *Lancet* 2009;373:956–966.
2. Varadarajan P, Kapoor N, Bansal RC, Pai RG. Clinical profile and natural history of 453 nonsurgically managed patients with severe aortic stenosis. *Ann Thorac Surg* 2006;82:2111–2115.
3. Baumgartner H, Falk V, Bax JJ, De Bonis M, Hamm C, Holm PJ, Iung B, Lancellotti P, Lansac E, Rodriguez Muñoz D, Rosenhek R, Sjögren J, Tornos Mas P, Vahanian A, Walther T, Wendler O, Windecker S, Zamorano JL, ESC Scientific Document Group. 2017 ESC/EACTS Guidelines for the management of valvular heart disease. *Eur Heart J* 2017;38:2739–2791.
4. Saito M, Imai M, Wake D, Higaki R, Nakao Y, Morioka H, Sumimoto T, Inoue K. Prognostic assessment of relative apical sparing pattern of longitudinal strain for severe aortic valve stenosis. *Int J Cardiol Heart Vasc* 2020;29:100551.
5. Ternacle J, Krapf L, Mohty D, Magne J, Nguyen A, Galat A, Gallet R, Teiger E, Côté N, Clavel MA, Tournoux F, Pibarot P, Damy T. Aortic stenosis and cardiac amyloidosis: JACC review topic of the week. *J Am Coll Cardiol* 2019;74:2638–2651.

6. Baumgartner H, Hung J, Bermejo J, Chambers JB, Edvardsen T, Goldstein S, Lancellotti P, LeFevre M, Miller F Jr, Otto CM. Recommendations on the echocardiographic assessment of aortic valve stenosis: a focused update from the European Association of Cardiovascular Imaging and the American Society of Echocardiography. *J Am Soc Echocardiogr* 2017;30:372–392.
7. van Rosendael PJ, Kamperidis V, Kong WK, van Rosendael AR, Marsan NA, Bax JJ, Delgado V. Comparison of quantity of calcific deposits by multidetector computed tomography in the aortic valve and coronary arteries. *Am J Cardiol* 2016;118:1533–1538.
8. Gegenava T, van der Bijl P, Vollema EM, van der Kley F, de Weger A, Hautemann D, Reiber JHC, Ajmone Marsan N, Bax JJ, Delgado V. Prognostic influence of feature tracking multidetector row computed tomography-derived left ventricular global longitudinal strain in patients with aortic stenosis treated with transcatheter aortic valve implantation. *Am J Cardiol* 2020;125:948–955.
9. Cerqueira MD, Weissman NJ, Dilsizian V, Jacobs AK, Kaul S, Laskey WK, Pennell DJ, Rumberger JA, Ryan T, Verani MS, American Heart Association Writing Group on Myocardial Segmentation and Registration for Cardiac Imaging. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart. A statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation* 2002;105:539–542.
10. Liu D, Hu K, Niemann M, Herrmann S, Cikes M, Störk S, Gaudron PD, Knop S, Ertl G, Bijnens B, Weidemann F. Effect of combined systolic and diastolic functional parameter assessment for differentiation of cardiac amyloidosis from other causes of concentric left ventricular hypertrophy. *Circ Cardiovasc Imaging* 2013;6:1066–1072.
11. Phelan D, Collier P, Thavendiranathan P, Popović ZB, Hanna M, Plana JC, Marwick TH, Thomas JD. Relative apical sparing of longitudinal strain using two-dimensional speckle-tracking echocardiography is both sensitive and specific for the diagnosis of cardiac amyloidosis. *Heart* 2012;98:1442–1448.
12. Balzer P, Furber A, Delépine S, Rouleau F, Lethimonnier F, Morel O, Tadéi A, Jallet P, Geslin P, le Jeune JJ. Regional assessment of wall curvature and wall stress in left ventricle with magnetic resonance imaging. *Am J Physiol* 1999;277:H901–H910.
13. Heng MK, Janz RF, Jobin J. Estimation of regional stress in the left ventricular septum and free wall: an echocardiographic study suggesting a mechanism for asymmetric septal hypertrophy. *Am Heart J* 1985;110:84–90.
14. Dorbala S, Cuddy S, Falk RH. How to image cardiac amyloidosis: a practical approach. *JACC Cardiovasc Imaging* 2020;13:1368–1383.
15. Fortuni F, Bax JJ, Delgado V. Changing the paradigm in the management of valvular heart disease: in addition to left ventricular ejection fraction, focus on the myocardium. *Circulation* 2021; 143:209–211.
16. Rassi AN, Pibarot P, Elmariah S. Left ventricular remodelling in aortic stenosis. *Can J Cardiol* 2014;30:1004–1011.
17. Castaño A, Narotsky DL, Hamid N, Khalique OK, Morgenstern R, DeLuca A, Rubin J, Chiuzan C, Nazif T, Vahl T, George I, Kodali S, Leon MB, Hahn R, Bokhari S, Maurer MS. Unveiling transthyretin cardiac amyloidosis and its predictors among elderly patients with severe aortic stenosis undergoing transcatheter aortic valve replacement. *Eur Heart J* 2017;38:2879–2887.
18. Obokata M, Nagata Y, Wu VC, Kado Y, Kurabayashi M, Otsuji Y, Takeuchi M. Direct comparison of cardiac magnetic resonance feature tracking and 2D/3D echocardiography speckle tracking for evaluation of global left ventricular strain. *Eur Heart J Cardiovasc Imaging* 2016;17:525–532.
19. Miskinyte E, Bucius P, Erley J, Zamani SM, Tanacli R, Stehning C, Schneeweis C, Lapinskas T, Pieske B, Falk V, Gebker R, Pedrizzetti G, Solowjowa N, Kelle S. Assessment of global longitudinal and circumferential strain using computed tomography feature tracking: intra-individual comparison with CMR feature tracking and myocardial tagging in patients with severe aortic stenosis. *J Clin Med* 2019;8:1423.