Title: Classical mechanical dyssynchrony is rare in TAVI-induced left bundle branch block

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Abstract

Aims

Left bundle branch block (LBBB) is a frequent conduction abnormality after transcatheter aortic valve implantation (TAVI). We aimed to investigate how TAVI procedure related conduction abnormalities influence ventricular mechanics **and prognosis**, with particular focus on new-onset persistent LBBB.

Methods and results

A total of 140 consecutive patients with severe aortic stenosis (83 ± 8 years old, 49% women) undergoing TAVI in a single tertiary center were included in a repeated measures study. Changes in myocardial function and contraction patterns were investigated in relation to changes in electrical conduction and afterload by speckle tracking echocardiography. Whether patients with new-onset LBBB acquired classical dyssynchronous contractions was assessed by longitudinal strain in apical 4-chamber view. GLS improvement was seen in all patients (-15.1 ± 4.3 vs $-16.1\pm 3.9\%$, $p<0.01$, $n=140$), and all subgroups, regardless of pre-existing or procedure-acquired conduction abnormalities immediately after TAVI. New-onset LBBB fulfilling strict ECG criteria was observed in 28 patients (20%). The vast majority of new-onset LBBB patients ($n=26$, 93%) had homogenous contractions. Classical dyssynchronous LBBB contractions were only observed in 2 patients (7 %) with new-onset LBBB. Patients with new-onset LBBB and patients without acquired conduction disorders had similar mortality rates during 19 ± 9 months of follow-up (11.1 [95% CI 4.6-26.8] vs. 8.1 [95% CI 4.8-13.7] per 100 patients years, $p=0.53$).

Conclusion

Classical dyssynchronous LBBB contractions were absent in most patients with new-onset post-TAVI LBBB, even when applying strict ECG criteria. Patients with and without new-onset LBBB experienced similar prognosis with regards to mortality.

Keywords

TAVI, left bundle branch block, dyssynchrony

Introduction

New-onset left bundle branch block (LBBB) has been a concern after transcatheter aortic valve implantation (TAVI)¹. The importance of post TAVI new-onset left bundle branch block (LBBB) is still under debate due to the diverging results among studies regarding impact on LV function, mortality and functional class²⁻⁷. This stands in apparent contrast to heart failure populations where LBBB is considered an unfavourable prognostic marker⁸. All patients with LBBB do not, however, exhibit the same mechanical dyssynchrony^{9, 10}. The classical dyssynchronous contraction pattern of true LBBB activation associated with response to cardiac resynchronization therapy (CRT) is characterized by an opposing motion of the septal and lateral wall in combination with an early systolic septal wall contraction¹¹. Interestingly, a different contraction pattern has been described in a case report of five patients with new-onset strict LBBB after TAVI¹², raising the question of whether acquired LBBB in TAVI patients may be functionally and prognostically different from LBBB in heart failure.

Speckle tracking echocardiography allows accurate assessment of cardiac mechanics, and has previously demonstrated subtle but prognostically important dysfunction in patients with aortic stenosis (AS)^{13, 14}. The aim of this study was to assess the immediate effect of new-onset conduction abnormalities on myocardial mechanics by speckle tracking echocardiography, with particular focus on new-onset persistent LBBB. Furthermore, we aimed to compare mortality in patients with new-onset persistent LBBB and patients without TAVI induced conduction abnormalities.

Methods

Study population

This was a repeated measures study of acute changes in myocardial function and ventricular mechanics in TAVI patients with normal ECG and conduction abnormalities performed in a single tertiary center. All patients with an uncomplicated, primary TAVI procedure performed at Oslo University Hospital, Rikshospitalet, Oslo, Norway, between January 1, 2015 and June 15, 2017 were screened for inclusion in the study. Exclusion criteria were ventricular paced rhythm, paroxysmal arrhythmia or bigeminal ventricular ectopy, infusion of vasoactive drugs, and insufficient image quality during recording of the baseline or postoperative echocardiogram. Logistic Euroscore and New York Heart Association (NYHA) functional class were determined based on medical history and cardiac symptoms obtained from medical records. Coronary artery disease (CAD) was defined as previous myocardial infarction or evidence of significant stenosis on an epicardial coronary artery or bypass graft ($\geq 50\%$ diameter stenosis in the absence of collateral perfusion) on coronary angiogram performed in all patients at baseline. Twelve-lead electrocardiogram (ECG) and blood pressure were recorded at baseline and at post-procedural echocardiography. QRS duration < 120 ms was classified as normal. Strict LBBB was defined as QRS duration ≥ 140 ms (men) or ≥ 130 ms (women) and QS or rS in leads V1 and V2, and mid-QRS slurring or notching in ≥ 2 contiguous leads of V1, V2, V5, V6, I, and aVL¹⁵. Right bundle branch block (RBBB) was defined as QRS duration ≥ 120 ms with positive terminal deflection in V1. Nonspecific intraventricular conduction delay was defined by the presence of QRS duration ≥ 120 ms not fulfilling the criteria of LBBB or RBBB. Conduction abnormalities were confirmed at discharge by ECG in all patients. The study complied with the Declaration of Helsinki. The study was approved by the Regional Committee for Medical Research Ethics.

Echocardiography

Echocardiography was performed using the Vivid E9 ultrasound system (GE Healthcare, Horten, Norway) at baseline, on day 1 or 2 after TAVI in 96 % of the patients and before discharge from hospital in the remaining 4 %. Offline data analyses were performed on EchoPac version 201 (GE Healthcare). Left ventricular (LV) ejection fraction (EF) was calculated by the modified Simpson's method, and LV dimensions were obtained in M-mode or B-mode. LV mass was calculated by 2D linear model using the Devereux formula¹⁶ and indexed for body surface area (BSA). We recorded e' by tissue Doppler imaging in the septal annulus, and E wave from mitral blood flow. Left atrial (LA) volume was calculated by the biplane area-length method and indexed for BSA. The tricuspid annular plane systolic excursion (TAPSE) was obtained from RV focused apical view. Valvulo-arterial impedance calculated as the sum of the systolic arterial pressure and the mean aortic transvalvular pressure gradient divided by stroke volume index was used as a measure of global afterload¹⁷. Low flow-low gradient AS was defined as an AS with stroke volume index ≤ 35 ml/m² combined with mean aortic valve gradient ≤ 40 mmHg¹⁸. Global longitudinal strain (GLS) was assessed by speckle tracking, and defined as the average peak longitudinal strain in a 16 segment LV model and LV mechanical dispersion was defined as the standard deviation of time from Q/R on ECG to peak negative strain in 16 LV segments¹⁹.

LV contraction in LBBB patients was examined according to the 2D longitudinal strain pattern obtained in apical 4-chamber view as previously described²⁰. A classical dyssynchronous LBBB contraction pattern had to meet all 3 of the following criteria:

1) early shortening of a basal or mid-ventricular septal segment combined with early stretching of a basal or mid-ventricular lateral wall segment; 2) early peak shortening (within the first 70% of the ejection phase) in a septal segment; and 3) peak lateral wall shortening after aortic valve closure (AVC). All other LBBB contraction patterns were defined as non-classical. The

contraction patterns by strain were reviewed by two independent observers (L.G.K. and Ø.H.L), and consensus was attained in conference among the observers in cases of disagreement (n=2). We determined the onset of contraction, peak contraction and intraventricular differences in timing intervals by strain analyses of the basal and mid septal and lateral wall segments in apical 4 chamber view (Figure 2). Onset of contraction and maximal pre-stretch was defined as the point of the segmental strain curve with highest strain value after onset of QRS followed by negative strain rates. Opposing wall motion was defined as the absolute difference of septal and lateral wall strains at the time of maximal lateral wall pre-stretch. Peak contraction was defined as the point with lowest strain value with subsequent positive strain rate.

TAVI procedure

TAVI procedure was conducted under mild sedation through femoral access. Implanted bioprotheses were the balloon-expanded Edwards-Sapien 3 (Edwards Lifesciences Ltd. Orange County, Ca, USA; 121 patients) and Edwards-Sapien XT (Edwards Lifesciences Ltd. Orange County, Ca, USA; 1 patient) valves and the self-expanding CoreValve Evolute R (Medtronic, Minneapolis, MN, USA; 8 patients) and Symetis Acurate TF (Symetis SA, Ecublens, Switzerland; 10 patients) valves. Balloon-expandable valves were deployed under rapid right ventricular pacing at 180/min. Self-expandable valves were usually deployed without pacing or only under slow-rapid right ventricular pacing at 120/min. Percutaneous coronary revascularization (PCI) was performed in 25 patients after baseline echocardiography prior to TAVI in patients with significant coronary artery stenosis. **Twenty-eight patients were excluded due to ventricular pacing at the time of post-procedural echocardiography. Of these, 19 received a permanent pacemaker due to atrio-ventricular block.**

Mortality

Data regarding all-cause mortality after discharge from hospital until March 7, 2018 were obtained from electronic hospital records synchronized with the Norwegian National Registry. Mean follow-up was 19 ± 9 month.

Statistics

Data were presented as mean $\pm$ standard deviation, frequencies with percentages or median with interquartile range, and compared by Student's t-test, paired Student's t-test, χ^2 , Fischer's exact test or by Mann-Whitney U test as appropriate (SPSS statistics 22.0, SPSS Inc., Chicago, IL, USA). Mortality rates were compared by Fischer's exact test (Stata version 15.0, Stata Corp., College Station, TX). Two-sided P-values < 0.05 were considered statistically significant.

Results

Baseline patient characteristics, ECG and echocardiography

A total of 140 consecutive patients treated with uncomplicated TAVI between January 1, 2015 and June 15, 2017 were included (83 ± 8 years old, 49 % women, NYHA functional class of 2.5 ± 0.6 and logistic Euroscore of 16.2 ± 10.2) (Table 1). Seventy-three patients (52 %) had a history of coronary artery disease. CABG surgery had previously been performed in 22 patients (16 %). ECG showed that 108 patients (77 %) had sinus rhythm and 32 atrial fibrillation (23 %). Patients were stratified according to QRS morphology as follows: Normal QRS duration (n=113) or pre-existing strict LBBB (n=10), RBBB (n=9) or non-specific conduction defects (n=8).

All patients had severe AS (Table 2) with average LV end-diastolic diameter of 50 ± 8 mm and interventricular septum diastolic wall thickness of 12 ± 2 mm and mildly increased LV mass [111 ± 24 g/m² (men) vs 102 ± 22 g/m² (women)]. LVEF was in general well preserved (52 ± 11 %), but GLS was abnormal in the total population (-15.1 ± 4.3 %). Patients had signs of diastolic dysfunction with elevated E/e' (21.2 ± 11.2), left atrial enlargement (57 ± 18 ml/m²), and mild pulmonary hypertension (tricuspid gradient 36 ± 13 mmHg). Study participants with low flow-low gradient AS (n=17) had an average LVEF of 38 ± 13 % and GLS -10.1 ± 4.8 % before TAVI.

Post-TAVI ECG and echocardiography

Out of the 113 patients with normal QRS duration at baseline, 81 patients maintained normal QRS duration after TAVI, while 28 (25 %) patients acquired persistent strict LBBB. New post-TAVI RBBB was observed in two patients with normal baseline QRS duration, and two patients with new post-TAVI conduction abnormalities and QRS > 120 ms were classified as non-specific conduction abnormalities. GLS improved immediately after TAVI in all groups of patients, regardless of pre-existing conduction abnormalities or an acquired LBBB (Table 2). Patients with low flow low gradient AS (n=17) had a particularly marked improvement in GLS (-10.1 ± 4.8 % vs -12.0 ± 4.2 %, $p=0.019$). Excluding this group, the remaining 123 patients had a GLS change of -15.8 ± 3.8 % vs -16.8 ± 3.4 %, $p<0.001$.

Despite fulfilling strict LBBB ECG criteria, only 2 of the 28 patients (7 %) with new strict LBBB had classical dyssynchronous contractions. The majority of patients with a new-onset LBBB (n=26, 93 %) had a non-classical, more homogenous contraction pattern with peak septal strain occurring late in the ejection phase close to AVC, and a late lateral wall contraction with peak strain after AVC (Figure 1, right panel). Comparing contraction patterns in patients with new-

onset LBBB to those who maintained a normal QRS, some aspects of the altered LV mechanics were in accordance with the expected mechanics after an induced LBBB, including shorter time to onset of contraction in septal segments, delayed onset of contraction in lateral wall segments and greater opposing wall motion. There were, however, no significant differences in time to peak contraction or strain values in basal and mid septal and lateral segments when comparing the two groups (Table 3). Patients with new-onset LBBB had higher prevalence of CAD (68 % vs 44 %, $p=0.05$) and slightly more pronounced mechanical dispersion after TAVI (62 ± 19 ms vs 49 ± 16 ms, $p<0.01$) than patients who maintained normal ventricular conduction. Severe paravalvular regurgitation was not observed in any patients after the procedure (Table 1).

Mortality

After 19 ± 9 months follow-up mortality rates were similar in patients who maintained normal QRS duration and patients with post-TAVI new onset LBBB (8.1 [95 % CI 4.8-13.7] per 100 patients years vs. 11.1 [95 % CI 4.6-26.8], $p=0.53$). No patients were lost to follow-up.

Discussion

This study explored immediate changes in myocardial function related to new-onset conduction abnormalities after TAVI. Although all cases of new LBBB fulfilled strict ECG criteria, very few patients acquired classical mechanical dyssynchrony. Patients with new-onset LBBB and patients who maintained narrow QRS duration had similar prognosis during follow-up. GLS improved immediately after the procedure following the reduction of afterload, regardless of pre-existing conduction abnormalities or an acquired LBBB.

Discordant echocardiography and ECG

A classical dyssynchronous mechanical deformation pattern caused by true LBBB activation delay is frequently found in heart failure patients with LBBB in their ECGs. This contraction pattern is characterized by an early systolic septal contraction with a rebound stretch, giving rise to a typical septal flash, and an early systolic lengthening of the lateral wall with a late peak contraction resulting in an opposing motion of the septal and lateral wall.

It has been shown that applying strict LBBB ECG criteria further increases the sensitivity for classical dyssynchrony associated with CRT response⁹. In this study, we found a high incidence (88 %) of LBBB fulfilling strict ECG criteria among patients with new post TAVI conduction disorders. Based on ECG, one would expect most of these patients to have dyssynchronous ventricular contractions. However, only 2 out of 28 with new strict LBBBs had classical ventricular dyssynchrony. Interestingly, the majority of patients with new-onset strict LBBB (93 %) had a homogenous contraction pattern with peak septal strain occurring late in ejection phase close to AVC (Figure 1). **The low prevalence of early peak septal contraction needed to fulfill classical dyssynchronous criteria is in accordance with the findings of Klein and coworkers¹². Our patients, however, had relatively sparse lateral wall pre-stretch and more synchronous timed peak contractions.**

LBBB contraction patterns resembling what we found in this post-TAVI setting have been described in heart failure, where a pseudo-normalized pattern was identified in patients less likely to respond to cardiac resynchronization therapy²¹. Similar strain patterns have been demonstrated in a CircAdapt model simulating profound LV lateral wall hypocontractility²². The AS patients in the current study, however, had preserved LV function. A previous case report

identified a pseudo-normalized strain pattern in new-onset LBBB after TAVI, but there have been no systematic studies of contraction patterns in TAVI cohorts previously.

While strict LBBB in ECG may be a sensitive marker of significant ventricular dyssynchrony in heart failure patients, the sensitivity for detecting echocardiographic dyssynchrony in this cohort of post-TAVI LBBB patients using strict ECG criteria was only 7 %. This emphasizes that LV function must be evaluated with echocardiography even when applying strict ECG criteria for LBBB in order to diagnose mechanical dyssynchrony. **One should, however, take into account that other LBBB ECG criteria may be more appropriate in AS populations due to structural differences from heart failure patients.** The results from our study raises the question whether the combination of improved global longitudinal function and the non-classical, homogenous contraction pattern of the post TAVI LBBB alleviates the mechanical dysfunction caused by the induced electrical delay, and may explain why TAVI induced LBBB seem to have a different prognostic impact ²⁻⁷ than the LBBB in other cardiac conditions. **We therefore suggest an evaluation of mechanical dyssynchrony in patients with persistent LBBB in conjunction with routine echocardiographic follow-up after TAVI.**

While earlier electrophysiological mapping studies have identified different electrical activation patterns in LBBB^{10, 23}, it is difficult to say whether ventricular conduction aspects, regional myocardial properties or procedure related factors make TAVI patients less prone to acquire classical mechanical dyssynchrony. There is a knowledge gap regarding the role of resynchronization therapy in patients with reduced LV function and new-onset LBBB after TAVI²⁴. Our findings suggest that a majority of patients with TAVI induced LBBB will not be CRT responders.

Patients with severe AS typically have reduced longitudinal LV function with the most pronounced impairment in the basal segments of the LV, corresponding to predilection areas of fibrosis²⁵. Patients in this cohort differ from known LBBB patient models by having concentric left ventricular hypertrophy, relatively preserved global systolic function, and better lateral wall function as opposed to the more dyssynchronous CRT cohorts with dilated, thin walled hearts, and presumably higher scar burden and worse LV function²¹. The underlying mechanisms of septal flash and echocardiographic dyssynchrony in TAVI populations remain to be investigated.

Improvement of myocardial function

GLS has proven incremental prognostic value over traditional risk markers in AS²⁶, and is superior to LVEF in predicting myocardial recovery following TAVI²⁷. Most previous studies have explored GLS changes after intermediate- and long-term follow up^{28, 29}. None of these studies, however, have assessed the effect of afterload reduction on LV function in relation to pre-existing or acquired ventricular conduction disorders shortly after TAVI. Interestingly, we found that GLS improved immediately after TAVI also in patients who acquired LBBB after the procedure, indicating a limited effect of conduction abnormalities on LV longitudinal function.

LVEF has been reported to improve immediately after TAVI without further improvement over time²⁹. Furthermore, patients with impaired systolic function prior to intervention seem to achieve greater improvement in LVEF than patients with more preserved systolic function³⁰. Our study confirmed these results, and provided additional information regarding changes in subgroups of pre-existing and acquired conduction disorders frequently seen in TAVI populations. **Patients with new-onset LBBB had more pronounced mechanical dispersion and higher prevalence of CAD, suggesting that both ventricular conduction aspects and myocardial properties influence mechanical dispersion. The absolute value of mechanical**

dispersion, however, was lower than a previous study exploring LBBB patients and arrhythmic events¹⁹.

Study limitations

This was a repeated measures observational study performed in a single tertiary center with its traditional inherent limitations. Although we found very low incidence of classical LV dyssynchrony immediately after TAVI, a prospective study is needed to determine whether there is a progression towards classical dyssynchrony and LV dysfunction over time. The amount of LV wasted myocardial work could not be explored in this cohort as the algorithm for this non-invasive estimation is yet to be validated in AS and other diseases with pressure gradients between LV and aorta. Ideally, cardiac magnetic resonance imaging might be able to elucidate myocardial characteristics associated with LBBB development in AS, but was not performed in our patients. There was no significant difference in mortality rates between patients with normal QRS duration and the new-onset LBBB group, but larger sample size is needed to claim non-inferiority in the new-onset LBBB regarding adverse outcome.

Conclusion

In the current study even strict ECG criteria had a very low sensitivity for detecting ventricular dyssynchrony in TAVI-induced LBBB. Nearly all patients with TAVI-induced strict LBBB had homogeneous ventricular contractions rather than the classical mechanical dyssynchrony one could expect from the conduction delay, suggesting that CRT response rate would be low in this group. LV longitudinal function improved after TAVI in all patients, including those with new-onset LBBB. Patients with and without new-onset LBBB had similar

short-term mortality. These findings could have implications for ventricular remodelling, and may explain why post TAVI LBBB is not clearly associated with negative prognosis.

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Disclosures

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Figure legends

Figure 1: Echocardiographic 2D strain analysis obtained from the apical 4-chamber view of aortic stenosis patients with pre-existing (left) and post-TAVI new-onset strict LBBB (right) with corresponding ECGs below. Yellow arrows illustrate the opposing wall motion of early septal contraction and lateral wall stretch. White arrow illustrates the late peak contraction of the lateral wall occurring after aortic valve closure (AVC).

Figure 2: Illustration of timing intervals and strain values measured in basal and mid septal segments (yellow curve; corresponding to yellow boxed segments in 2D view on the left) and basal and mid lateral wall segments (red curve; corresponding to red boxed segments) in apical 4 chamber view. P = Peak positive strain and onset of lateral wall contraction. S = Peak septal contraction. G = Peak lateral wall contraction occurring after aortic valve closure (AVC). Vertical white arrow denotes opposing wall motion at the time of onset of contraction in the lateral wall. White horizontal arrows illustrate time to P, S and G.

Table 1

Baseline characteristics and TAVI procedure in complete cohort (n=140)

Age	82.8±7.1
Blood pressure, diastolic, mmHg	72.8±10.7
Blood pressure, systolic, mmHg	139.5±23.9
Body surface area, m ²	1.8±0.2
Bodymass index, kg/m ²	24.6±4.3
CAD history, n (%)	73(52)
Creatinine, µmol/L	99±3
Logistic Euroscore	16.2±10.2
Male/female, n (% female)	72/68(49)
NYHA class	2.5±0.6
PCI before TAVI, n(%)	25(18)
Previous CABG, n(%)	22(16)
Valvular regurgitation > moderate	
Aortic, n(%)	1(0.7)
Mitral, n(%)	2(1.4)
Tricuspid, n(%)	5(3.6)
TAVI procedure	
CoreValve, n(%)	8(6)
Edwards Sapien, n(%)	122(87)
Symetis, n(%)	10(7)
Valve sizes	
23mm, n(%)	43(31)
25mm, n(%)	3(2)
26mm, n(%)	56(40)
27mm, n(%)	5(4)
29mm, n(%)	33(24)
Post-TAVI paravalvular regurgitation	
Absent/trace, n(%)	59(42)
Mild, n(%)	54(39)
Mild to moderate, n(%)	20(14)
Moderate, n(%)	7(5)
Severe, n(%)	0(0)

Values are mean $\pm$ standard deviation unless otherwise indicated. BMI, body mass index; BSA, body surface area; CAD, coronary artery disease; CABG, Coronary artery bypass graft; LBBB, left bundle branch block; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; TAVI, transcatheter aortic valve implantation

Table 2

Echocardiographic results in all patients and subgroups stratified according to conduction abnormalities

	Baseline	After TAVI	p value*
Aortic valve area, cm ²	0.7±0.2	1.8±0.5	< 0.01
Normal QRS	0.7±0.2	1.8±0.5	< 0.01
New LBBB	0.6±0.2	2.0±0.6	< 0.01
Pre-existing conduction abnormalities	0.7±0.2	1.8±0.4	< 0.01
Aortic valve mean pressure gradient, mmHg	54±18	12±5	< 0.01
Normal QRS	55±18	12±5	< 0.01
New LBBB	59±15	11±3	< 0.01
Pre-existing conduction abnormalities	46±19	13±4	< 0.01
Aortic valve peak velocity, m/s	4.5±0.7	2.3±0.5	< 0.01
Normal QRS	4.5±0.7	2.3±0.5	< 0.01
New LBBB	4.7±0.5	2.1±0.4	< 0.01
Pre-existing conduction abnormalities	4.3±0.8	2.4±0.5	< 0.01
E/e'	21±11	23±11	0.15
Normal QRS	19±11	20±10	0.35
New LBBB	26±13	27±14	0.91
Pre-existing conduction abnormalities	21±9	26±13	0.07
GLS, %	-15.1±4.3	-16.1±3.9	<0.01
Normal QRS	-16.1±4.2	-17.0±3.8	<0.01
New LBBB	-14.5±3.9	-15.6±3.0	0.03
Pre-existing conduction abnormalities	-13.0±4.4	-14.9±3.7	< 0.01
LVEF, %	52±11	53±9	0.03
Normal QRS	55±9	55±9	0.95
New LBBB	50±10	52±8	0.12
Pre-existing conduction abnormalities	45±13	48±9	0.05

Stroke volume index, ml/m ²	40±10	42±9	<0.01
Normal QRS	39±9	41±9	0.34
New LBBB	39±8	43±9	0.11
Pre-existing conduction abnormalities	39±11	43±8	0.04
TAPSE, mm	21±6	23±6	<0.01
Normal QRS	22±6	23±6	0.08
New LBBB	20±6	23±6	0.03
Pre-existing conduction abnormalities	19±6	24±8	<0.01
TR peak velocity, m/s	3.0±0.5	3.1±0.5	0.04
Normal QRS	2.8±0.5	3.0±0.4	0.02
New LBBB	3.1±0.4	3.2±0.5	0.34
Pre-existing conduction abnormalities	3.1±0.5	3.1±0.6	0.95
Valvulo-arterial impedance, mmHg m ² mL ⁻¹	5.1±1.1	3.6±0.9	<0.01
Normal QRS	5.2±1.1	3.7±0.9	<0.01
New LBBB	5.2±1.0	3.4±0.8	<0.01
Pre-existing conduction abnormalities	4.9±1.2	3.3±0.9	< 0.01

Values are mean±standard deviation unless otherwise indicated. Comparisons performed by paired student's t-test. E, early diastolic mitral inflow velocity; e', early diastolic mitral annular tissue velocity; GLS, global longitudinal strain; LBBB, left bundle branch block; TAPSE, tricuspid annular plane systolic excursion; TAVI, transcatheter aortic valve implantation; TR, tricuspid regurgitation;

Table 3

Regional strain analysis after TAVI

	Maintained normal QRS after TAVI (n=81)	New strict LBBB without classic strain pattern (n=26)	p value
Time to onset of septal contraction, ms (IQR)	29(4-24)	11 (0-25)	0.01
Time to onset of lateral wall contraction, ms (IQR)	67 (35-107)	113 (62-145)	0.01
Difference in time to onset of contraction, ms (IQR)	38 (18-75)	91 (45-129)	0.01
Time to peak septal contraction, ms	383±63	381±48	0.87
Time to peak lateral wall contraction, ms	426±71	452±63	0.10
Difference in time to peak contraction, ms	68(30-111)	78(43-125)	0.82
Opposing wall motion, %	2.6±2.4	4.7±3.4	<0.01
Mean peak positive septal strain, %	0.7±1.1	0.4±0.8	0.14
Mean peak positive lateral wall strain, %	2.3±2.0	2.6±2.2	0.55
Mean peak negative septal strain, %	-12.9±3.7	-12.9±3.4	0.97
Mean peak negative lateral wall strain, %	-13.7±5.1	-13.7±4.5	0.96
Mean septal end systolic strain, %	-12.3±5.2	-11.5±4.7	0.49
Mean lateral wall end systolic strain, %	-12.7±5.2	-11.5±4.7	0.30

Values are mean±standard deviation unless otherwise indicated. Comparisons performed by one-way ANOVA or Mann-Whitney U test. IQR, interquartile range; LBBB, left bundle branch block; TAVI, transcatheter aortic valve implantation