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BEYOND THE PROSTHETIC VALVE: ONE-YEAR ECHOCARDIOGRAPHIC REVERSE REMODELING AFTER SURGICAL VALVE REPLACEMENT – A RETROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT
Background: Surgical valve replacement corrects valvular obstruction or regurgitation, but postoperative recovery also involves structural and functional adaptation of the cardiac chambers. This original study evaluated echocardiographic reverse remodeling beyond prosthetic valve performance. ***Methods:*** A retrospective observational study included seven patients with paired preoperative and postoperative echocardiographic records. Extracted parameters included aortic root diameter, left atrial diameter, left ventricular internal dimensions, interventricular septal and posterior wall measurements, ejection fraction (EF), fractional shortening (FS), valve Doppler velocities and gradients, and right ventricular systolic pressure (RVSP), where available. Changes were assessed descriptively at the individual-patient level; no inferential statistical testing was performed. ***Results:*** EF increased in all seven patients, with absolute improvements of 10–22 percentage points, while FS also increased in every case. Left atrial diameter decreased in all patients, with reductions ranging from 2.6 to 25.0 mm. Left ventricular internal dimension in diastole showed heterogeneous remodeling, decreasing in three patients and increasing in four. RVSP was available in three patients and decreased in all, from 69 to 35 mmHg, 45 to 35 mmHg, and 60 to 40 mmHg. Patient 1 demonstrated marked improvement in aortic haemodynamics, with Doppler velocity decreasing from 4.19 to 1.64 m/s and the recorded gradient from 70/41 to 11 mmHg. ***Conclusion:*** Surgical valve replacement was associated with consistent improvement in systolic function and left atrial size, with favourable pulmonary pressure changes where measured. Ventricular dimensional remodeling was heterogeneous, highlighting the value of comprehensive echocardiographic follow-up beyond prosthetic valve assessment alone.

KEYWORDS: Surgical valve replacement; echocardiography; reverse remodeling; prosthetic valve; left ventricular function; left atrial remodeling; ejection fraction; RVSP.

INTRODUCTION:

Valvular heart disease (VHD) remains an important cause of cardiovascular morbidity, functional limitation, heart failure, and premature mortality worldwide. Its epidemiological profile varies considerably across regions: degenerative aortic and mitral valve disease predominates in ageing populations, whereas rheumatic valvular disease continues to impose a substantial burden in low- and middle-income countries [1]. Regardless of aetiology, clinically significant valve stenosis or regurgitation alters normal intracardiac loading conditions and initiates a progressive cascade of structural and functional cardiac adaptation. Chronic pressure overload, as occurs in aortic stenosis, promotes concentric left ventricular (LV) hypertrophy and increased myocardial stiffness, whereas chronic volume overload associated with valvular regurgitation commonly produces ventricular dilatation and eccentric remodeling. Mitral valve disease may additionally result in marked left atrial enlargement, pulmonary hypertension, atrial arrhythmias, and eventually right-sided cardiac involvement. Thus, advanced VHD should be regarded not only as a disorder of the valve itself but also as a disease affecting the myocardium and the entire cardiac chamber system [1,2]. Surgical valve replacement remains an established treatment for patients with severe valvular lesions in whom replacement rather than repair is indicated. The immediate objective of surgery is to relieve valvular obstruction or eliminate pathological regurgitant loading, restore effective forward flow, and prevent further haemodynamic deterioration. However, successful implantation and satisfactory haemodynamic performance of a prosthetic valve represent only one component of postoperative recovery. The myocardium, atria, pulmonary circulation, and right ventricle may have undergone substantial structural adaptation before surgery, and these abnormalities do not

necessarily normalize immediately after correction of the valve lesion. Consequently, contemporary evaluation of valve surgery increasingly extends beyond the prosthetic valve to examine the extent to which the heart itself recovers after removal of the abnormal pressure or volume load [2,3].

This postoperative recovery is commonly described as reverse cardiac remodeling. Reverse remodeling represents favourable changes in chamber geometry, myocardial mass, ventricular volumes, systolic and diastolic performance, atrial size, pulmonary pressures, and right ventricular adaptation following correction of the underlying haemodynamic lesion. Transthoracic echocardiography is particularly valuable for documenting this process because it permits serial assessment of prosthetic valve haemodynamics together with ventricular dimensions, LV mass, ejection fraction, atrial size, pulmonary artery pressure, and right ventricular function. Current recommendations identify echocardiography as the principal non-invasive modality for surveillance of prosthetic valves and emphasize assessment not only of prosthetic function but also of ventricular and other cardiac changes during follow-up [2,3].

Evidence from patients undergoing aortic valve replacement demonstrates that removal of chronic LV pressure or volume overload can produce substantial structural recovery. Treibel et al. demonstrated significant regression of LV hypertrophy following valve replacement for severe aortic stenosis, with approximately one-fifth reduction in indexed LV mass at one year together with improvements in myocardial cellular hypertrophy, diffuse fibrosis, diastolic function, functional capacity, and clinical status [4]. Importantly, focal replacement fibrosis did not regress, illustrating that reverse remodeling may be incomplete when irreversible myocardial injury has already developed. These findings reinforce the concept that correction of the valvular lesion does not automatically restore myocardial structure to normal and that the extent of recovery may depend on the severity and duration of preoperative myocardial damage.

The prognostic relevance of postoperative remodeling has also been demonstrated in larger echocardiographic cohorts. Izumi et al., evaluating patients after aortic valve replacement for aortic stenosis or regurgitation, reported significant reductions in LV end-diastolic and end-systolic dimensions and LV mass index, together with improvement in LV ejection fraction at one-year follow-up [5]. Notably, echocardiographic measurements obtained one year after surgery were more strongly associated with subsequent long-term outcomes than preoperative measurements. These findings suggest that one-year echocardiographic assessment is not merely a description of postoperative anatomy; rather, it may provide clinically relevant information regarding residual myocardial burden and longer-term prognosis.

Reverse remodeling is similarly important in chronic aortic regurgitation, although assessment methodology can influence its recognition. Ong et al. demonstrated substantial LV reverse remodeling following aortic valve replacement for severe aortic regurgitation and showed that volume-based measurements may identify postoperative ventricular recovery more accurately than simple linear dimensions [6]. This is particularly relevant because ventricular dilatation represents a central adaptive response to chronic regurgitant volume overload, and incomplete reduction of LV volumes after surgery may signify advanced or persistent myocardial dysfunction.

The remodeling response following mitral valve replacement is more complex because longstanding mitral disease may involve both ventricular and atrial structural changes. In patients with chronic rheumatic mixed mitral valve disease, Topal et al. demonstrated reductions in LV end-diastolic dimension and left atrial size following mitral valve replacement, accompanied by improvement in functional status [7]. Preoperative LV function and dimensions influenced subsequent ventricular performance, highlighting the importance of the preoperative myocardial substrate in determining the degree of postoperative recovery. Left atrial reverse remodeling may be particularly important in mitral valve disease because chronic atrial enlargement is associated with atrial fibrillation, thromboembolic risk, and pulmonary vascular consequences.

Although left-sided remodeling has received the greatest attention, postoperative recovery of the right heart can also carry prognostic significance. Patlolla et al. demonstrated that right ventricular reverse remodeling following isolated tricuspid valve surgery was associated with improved long-term survival, whereas persistent right ventricular dysfunction identified patients with less favourable outcomes [8]. These observations reinforce the broader principle that postoperative assessment should encompass the response of the cardiac chambers rather than focusing exclusively on prosthetic valve gradients or leaflet motion.

Despite this evidence, reverse remodeling following surgical valve replacement is heterogeneous. Patients with technically successful procedures and normally functioning prostheses may demonstrate markedly different trajectories of ventricular, atrial, and pulmonary haemodynamic recovery. Factors such as the original valve lesion, duration of disease, preoperative ventricular enlargement, myocardial fibrosis, ventricular systolic function, pulmonary hypertension, concomitant cardiac pathology, prosthesis–patient interaction, and postoperative loading conditions may influence the magnitude and completeness of reverse remodeling [4,5]. Furthermore, much of the available evidence has examined individual valve lesions or selected patient cohorts, whereas the longitudinal echocardiographic evolution of patients encountered in routine surgical practice may demonstrate a wider spectrum of recovery.

Against this background, systematic examination of echocardiographic changes from the preoperative period to one year after surgical valve replacement can provide clinically meaningful information that extends beyond confirmation of prosthetic valve function. A original-study approach can illustrate individual patterns of structural and functional recovery, identify concordant and discordant remodeling responses, and demonstrate how changes in ventricular

dimensions, systolic function, atrial size, pulmonary pressures, and prosthetic haemodynamics evolve following relief of chronic valvular loading. The present original study therefore aims to characterize one-year echocardiographic reverse remodeling after surgical valve replacement, with particular emphasis on changes beyond the prosthesis itself and on the recovery—or persistence—of pre-existing cardiac structural and functional abnormalities.

MATERIALS AND METHODS

Study Design

This study was designed as a retrospective observational study evaluating structural, functional, and haemodynamic echocardiographic changes following surgical valve replacement. Consecutive available patient records containing paired preoperative and postoperative echocardiographic examinations were reviewed. The study was intended to characterize the pattern of cardiac reverse remodeling following surgical correction of valvular heart disease, with particular emphasis on changes occurring beyond prosthetic valve haemodynamics.

The analysis was conducted at the individual-patient level because of the small number of cases and the heterogeneity of the underlying valvular lesions. Accordingly, the study was exploratory and descriptive rather than hypothesis-driven and was not designed to establish treatment efficacy or causal associations.

The current dataset comprised seven patients with paired preoperative and one-year follow-up echocardiographic measurements.

Study Setting and Study Period

The study was conducted in the Department of Cardiothoracic and Vascular Surgery (CTVS), All India Institute of Medical Sciences (AIIMS), Rae Bareli, using archived clinical and echocardiographic records of patients undergoing surgical valve replacement.

Patients undergoing valve replacement during the period from **May 2025 to May 2026** were screened for eligibility.

Study Population

Patients who had undergone surgical valve replacement and had interpretable echocardiographic examinations before surgery and during one-year postoperative follow-up were considered for inclusion. Each patient represented an independent observational unit.

The current original study included seven patients for whom paired echocardiographic information was available. Patient 1 underwent aortic valve replacement, whereas Patients 2–7 underwent mitral valve replacement with or without concomitant tricuspid valve repair. For confidentiality, all patients were assigned anonymized study identifiers (Patient 1 to patient 7), and no directly identifiable patient information was included in the analytical dataset or manuscript.

Eligibility Criteria

Patients were eligible for inclusion when they fulfilled all of the following criteria: they had undergone surgical valve replacement for clinically significant valvular heart disease; a preoperative transthoracic echocardiographic study was available; at least one postoperative echocardiographic study suitable for comparison was available; and the available records contained sufficient structural, functional, or Doppler measurements to permit assessment of postoperative cardiac remodeling.

Patients were excluded when preoperative or postoperative echocardiographic records were unavailable, when the available images or reports were insufficient for meaningful paired comparison, when essential values could not be reliably interpreted from the original record, or when the operative record could not confirm that surgical valve replacement had been performed.

Because this was a retrospective observational study based on the available dataset, no formal sample-size calculation was performed.

Timing of Echocardiographic Assessment

The preoperative echocardiogram was defined as the most recent available transthoracic echocardiographic examination performed before surgical valve replacement.

The postoperative echocardiographic examination represented the one-year follow-up study, performed one year after surgical valve replacement. The complete dataset therefore comprised paired preoperative and one-year follow-up echocardiographic assessments for all seven patients.

Echocardiographic Assessment

Preoperative and postoperative transthoracic echocardiographic data were extracted from the available reports. The analysis was structured to evaluate three complementary domains: cardiac structural remodeling, ventricular functional recovery, and valve/pulmonary haemodynamic response.

The following echocardiographic parameters were recorded where available:

- aortic root diameter;
- aortic valve opening;
- left atrial diameter;
- interventricular septal thickness;
- left ventricular internal dimension;

- left ventricular posterior wall thickness;
- left ventricular ejection fraction;
- fractional shortening;
- mitral valve Doppler velocity and recorded transvalvular gradient;
- aortic valve Doppler velocity and recorded transvalvular gradient;
- tricuspid regurgitation velocity and gradient; and
- estimated right ventricular systolic pressure.

These parameters correspond to those available in the original clinical records.

Measurements were recorded separately for the preoperative and one-year follow-up echocardiographic examinations. Values were transcribed using the units documented in the clinical reports, principally millimetres for chamber and wall dimensions, percentages for ejection fraction and fractional shortening, metres per second for Doppler velocities, and millimetres of mercury for pressure gradients and right ventricular systolic pressure.

Assessment of Cardiac Reverse Remodeling

For the purposes of this study, reverse remodeling was considered a multidimensional postoperative response rather than a single echocardiographic measurement.

Structural remodeling was assessed principally from changes in left atrial diameter, left ventricular internal dimensions, interventricular septal thickness, and left ventricular posterior wall thickness.

Functional remodeling was evaluated using changes in left ventricular ejection fraction and fractional shortening.

Pulmonary haemodynamic recovery was evaluated using changes in estimated right ventricular systolic pressure where this measurement was available.

Valve-related haemodynamic response was assessed using changes in Doppler-derived velocities and reported pressure gradients across the relevant valve.

This approach was selected because the available dataset contained conventional linear echocardiographic measurements but did not consistently include indexed left atrial volume, left ventricular volumes, left ventricular mass index, global longitudinal strain, or other advanced measures of myocardial mechanics. Consequently, the study does not equate changes in individual linear dimensions with complete myocardial reverse remodeling.

Definition of Study Outcomes

The primary outcome was the pattern of echocardiographic reverse remodeling from the preoperative examination to one-year postoperative follow-up.

The principal remodeling domains were:

1. change in left ventricular systolic function, particularly ejection fraction;
2. change in left atrial diameter;
3. change in left ventricular dimensions and wall measurements;
4. change in valve-specific Doppler velocity and transvalvular gradients; and
5. change in estimated right ventricular systolic pressure, where available.

No single parameter was used to classify a patient as having or not having reverse remodeling. Instead, each patient was characterized according to the overall direction and magnitude of changes across the available echocardiographic domains.

Data Extraction and Quality Control

Data were extracted directly from the available echocardiographic records into a structured data sheet. Preoperative and one-year follow-up values were recorded independently for each patient.

Ambiguously labelled measurements were excluded when their intended meaning could not be established confidently.

Particular care was taken not to reinterpret historical Doppler values when the source report used different formats for recording peak/mean gradients or pressures. Such measurements were retained according to the original documentation to minimize retrospective measurement assumptions.

Calculation of Echocardiographic Change

For each paired variable, absolute one-year follow-up change was calculated as:

Absolute change = postoperative value − preoperative value

A negative value therefore represented a one-year follow-up reduction, whereas a positive value represented an increase.

For parameters such as ejection fraction and fractional shortening, differences were expressed in percentage points rather than relative percentages.

Because the central purpose of the study was to demonstrate patient-level remodeling trajectories, changes were interpreted individually rather than combining heterogeneous valve lesions into a single pooled estimate.

Statistical Analysis

Owing to the very small sample size (n = 7), absence of a comparator group, and clinical heterogeneity among patients, statistical hypothesis testing was considered inappropriate. No inferential comparisons, P values, or regression analyses were therefore planned.

Continuous echocardiographic variables were summarized descriptively using individual preoperative and one-year follow-up values and their absolute changes. Where useful for descriptive presentation, median and interquartile range may additionally be reported for variables available in most or all patients; however, patient-level values remain the principal analytical presentation.

RESULTS

Seven patients with paired preoperative and preoperative and one-year follow-up echocardiographic data were included in the final study. The available records demonstrated changes across multiple echocardiographic domains, including left atrial (LA) size, left ventricular dimensions, systolic function, valve-related Doppler haemodynamics, and right ventricular systolic pressure (RVSP).

The most consistent postoperative findings were improvement in left ventricular systolic functional indices and reduction in LA diameter. Ejection fraction (EF) increased in all seven patients, with a median paired improvement of 16 percentage points, while fractional shortening (FS) increased in every patient, with a median improvement of 10 percentage points. Similarly, LA diameter decreased in all seven patients, with a median paired reduction of 19.6 mm. In contrast, changes in left ventricular internal dimension in diastole (LVIDd) were heterogeneous, demonstrating reduction in three patients and increase in four.

Table 1. Descriptive summary of principal echocardiographic parameters

Parameter	Preoperative median (range)	Postoperative median (range)	Median paired change
Aortic root diameter (mm)	25.0 (21.9–27.9)	27.1 (25.9–31.9)	+2.4
LA diameter (mm)	52.4 (30.0–56.0)	29.1 (27.4–46.4)	−19.6
LVIDd (mm)	47.6 (36.0–61.7)	50.4 (32.2–53.7)	+4.9
EF (%)	50 (40–60)	68 (60–71)	+16 percentage points
FS (%)	25 (20–30)	35 (30–36)	+10 percentage points

All seven patient records contained paired structural and functional measurements as available in the source dataset.

Reduction in LA diameter was the most uniform structural remodeling finding. All seven patients demonstrated a smaller postoperative LA diameter.

Among the Patients 1–5, reductions ranged from 2.6 mm in Patient 1 to 25.0 mm in Patient 2. Patient 3 demonstrated a reduction of 19.6 mm, while Patients 4 and 5 showed reductions of 11.0 and 6.0 mm, respectively.

The Patients 6 and 7 demonstrated similarly substantial reductions. LA diameter decreased from 50.8 to 29.1 mm in Patient 6 and from 53.1 to 28.6 mm in Patient 7, corresponding to reductions of 21.7 and 24.5 mm, respectively.

Table 2. Patient-level changes in left atrial diameter

Patient	Preoperative LA diameter (mm)	Postoperative LA diameter (mm)	Absolute change (mm)
1	30.0	27.4	−2.6
2	52.4	27.4	−25.0
3	56.0	36.4	−19.6
4	54.4	43.4	−11.0
5	52.4	46.4	−6.0
6	50.8	29.1	−21.7
7	53.1	28.6	−24.5

Thus, LA remodeling showed a uniform direction of change, although the magnitude of reduction varied considerably among individual patients.

Improvement in systolic function was observed consistently across all seven patients. EF increased by between 10 and 22 percentage points.

Patients 1 and 3 demonstrated 10-percentage-point increases, while Patient 4 also improved by 10 percentage points. Patients 2 and 5 demonstrated increases of 20 percentage points.

Among Patients 6 and 7, Patient 6 improved from an EF of 52% preoperatively to 68% postoperatively, representing a 16-percentage-point increase. Patient 7 showed the greatest absolute improvement in the entire series, from 49% to 71%, corresponding to an increase of 22 percentage points.

FS followed the same overall direction. It increased in every patient, with improvements ranging from 5 to 12 percentage points.

Table 3. Changes in left ventricular systolic function

Patient	EF pre-op (%)	EF post-op (%)	EF change*	FS pre-op (%)	FS post-op (%)	FS change*
1	60	70	+10	30	35	+5
2	50	70	+20	25	35	+10

3	50	60	+10	24	30	+6
4	55	65	+10	25	35	+10
5	40	60	+20	20	30	+10
6	52	68	+16	26	34	+8
7	49	71	+22	24	36	+12

The consistency of improvement in both EF and FS represented one of the strongest functional patterns observed in the study.

Unlike LA size and systolic function, LVIDd did not show a uniform postoperative response.

Among the original five patients, LVIDd decreased only in Patient 4, from 61.7 to 53.7 mm. LVIDd increased in Patients 1, 2, 3, and 5. The largest increase was observed in Patient 1, from 36.5 to 53.3 mm.

In contrast, Patients 6 and 7 demonstrated marked decreases in LVIDd. Patient 6 decreased from 47.6 to 33.5 mm, whereas Patient 7 decreased from 49.0 to 32.2 mm. These represented reductions of 14.1 and 16.8 mm, respectively.

Table 4. Patient-level changes in left ventricular internal dimension in diastole

Patient	Preoperative LVIDd (mm)	Postoperative LVIDd (mm)	Absolute change (mm)	Direction
1	36.5	53.3	+16.8	Increased
2	48.4	53.3	+4.9	Increased
3	36.0	41.8	+5.8	Increased
4	61.7	53.7	−8.0	Decreased
5	43.8	50.4	+6.6	Increased
6	47.6	33.5	−14.1	Decreased
7	49.0	32.2	−16.8	Decreased

Consequently, three of seven patients demonstrated a reduction in LVIDd, whereas four demonstrated an increase. These findings indicate that ventricular dimensional remodeling was heterogeneous despite consistent improvement in systolic functional indices.

For the patients in whom systolic LV dimensions were clearly available, Patient 4 showed the largest reduction in LVIDs, from 43.9 to 34.4 mm (−9.5 mm), followed by Patient 5 from 34.8 to 29.9 mm (−4.9 mm). The supplied dataset did not provide comparable LVIDs measurements consistently for all seven patients, and missing values were therefore not imputed.

Valve-specific Doppler measurements demonstrated important postoperative haemodynamic changes, although the completeness and recording format varied among patients.

The most striking documented change occurred in Patient 1. Aortic valve Doppler velocity decreased from 4.19 m/s preoperatively to 1.64 m/s postoperatively, accompanied by a reduction in the recorded aortic gradient from 70/41 mmHg to 11 mmHg.

Mitral Doppler velocity also decreased in several cases. Patient 2 demonstrated a reduction from 2.08 to 0.94 m/s, while Patient 4 decreased from 2.65 to 1.59 m/s and Patient 5 from 2.70 to 2.01 m/s.

Table 5. Selected valve-specific Doppler changes as recorded in the source data

Patient	Valve	Pre-op velocity (m/s)	Post-op velocity (m/s)	Pre-op gradient/pressure (mmHg)	Post-op gradient/pressure (mmHg)
1	Aortic	4.19	1.64	70/41	11
1	Mitral	1.60	0.94	2–6	1.5
2	Mitral	2.08	0.94	22/11	11/5
3	Aortic	1.20	0.79	NR	2.5–4
4	Mitral	2.65	1.59	28/12	10/4
5	Mitral	2.70	2.01	29/15	16/15

The Patient 6 and Patient 7 records contain several Doppler velocity entries; however, some valve labels are repeated or ambiguous in the supplied table. They should therefore not be reclassified or pooled without confirmation from the original echocardiographic reports. This follows the predefined methodology of not inferring unclear parameters.

Accordingly, the valve-specific findings are best interpreted descriptively rather than as a uniform quantitative measure of prosthetic function.

RVSP was explicitly available for Patients 3, 4, and 5. All three demonstrated one-year follow-up reductions.

Patient 3 had the greatest reduction, from 69 to 35 mmHg, corresponding to an absolute decrease of 34 mmHg. Patient 5 decreased from 60 to 40 mmHg, while Patient 4 decreased from 45 to 35 mmHg.

Table 6. Changes in right ventricular systolic pressure

Patient	Preoperative RVSP (mmHg)	Postoperative RVSP (mmHg)	Absolute change (mmHg)
3	69	35	−34

4	45	35	−10
5	60	40	−20

RVSP was not clearly available for Patients 1, 2, 6, and 7 and was therefore not estimated. Among the three patients with paired measurements, the direction of change was consistently favourable.

Taken together, the seven patients demonstrated a multidimensional but non-uniform postoperative remodeling response. Improvement in EF and FS was observed in every patient, and LA diameter decreased consistently across all seven patients. These represent the most reproducible functional and structural findings within the dataset.

In contrast, LV dimensional remodeling varied substantially. Patients 4, 6, and 7 demonstrated reductions in LVIDd, whereas Patients 1, 2, 3, and 5 showed increases. This divergence between improved systolic function and heterogeneous chamber-size response indicates that postoperative recovery cannot be characterized adequately using a single ventricular dimension.

The three patients with available RVSP measurements also showed reductions in estimated pulmonary pressure, while the clearest valve-specific haemodynamic response was observed in Patient 1, with marked reduction in aortic Doppler velocity and gradient.

DISCUSSION

The present original study demonstrates that the echocardiographic response following surgical valve replacement extends considerably beyond normalization of prosthetic valve haemodynamics. Across the seven patients, the most reproducible one-year follow-up changes were improvement in left ventricular systolic function and reduction in left atrial (LA) diameter. Ejection fraction (EF) increased in every patient, with absolute improvements ranging from 10 to 22 percentage points, while fractional shortening (FS) also increased in all seven patients. LA diameter decreased consistently, whereas left ventricular internal dimension in diastole (LVIDd) showed a heterogeneous response, decreasing in three patients and increasing in four. Among the three patients for whom paired right ventricular systolic pressure (RVSP) measurements were available, RVSP decreased in every case. These findings support the concept that reverse remodeling after valve replacement is multidimensional and that improvement in one domain, such as systolic function, does not necessarily imply parallel normalization of all structural indices. The paired measurements underlying these observations are documented in the supplied dataset.

One of the most striking findings was the uniform increase in EF and FS. EF increased from 60% to 70% in Patient 1, 50% to 70% in Patient 2, 50% to 60% in Patient 3, 55% to 65% in Patient 4, and 40% to 60% in Patient 5. The two Patients 6 and 7 showed a similar pattern: Patient 6 improved from 52% to 68%, while Patient 7 improved from 49% to 71%.

This functional recovery is physiologically plausible following correction of chronic abnormal loading conditions. In pressure-overload lesions, relief of outflow obstruction reduces afterload, whereas correction of severe regurgitant or stenotic lesions may improve loading conditions, ventricular filling, and effective forward stroke volume. However, the postoperative trajectory of LVEF is not uniform across valve lesions. Maharaj et al. evaluated patients undergoing mitral valve replacement for severe rheumatic mitral regurgitation and demonstrated that LVEF may fall early after surgery and subsequently recover during follow-up, illustrating that the timing of postoperative assessment substantially influences interpretation of ventricular function [11].

The current findings therefore likely represent a later phase of functional adaptation rather than the immediate postoperative response. This distinction is important because removal of regurgitant unloading, perioperative myocardial stunning, cardiopulmonary bypass effects, changes in preload, and altered ventricular geometry may transiently influence early postoperative EF. Longer-term studies also indicate that the trajectory of LV systolic function depends on baseline ventricular status. In patients with rheumatic heart disease undergoing combined mitral and aortic valve replacement, An et al. reported different longitudinal LVEF trajectories according to preoperative ventricular function, with patients who had preserved baseline LVEF reaching a plateau and those with reduced baseline LVEF showing a more prolonged pattern of recovery followed by later change [12].

Accordingly, the uniform increase in EF observed in this seven-patient study is encouraging but should not be interpreted as evidence that all patients achieved complete myocardial recovery. EF is load dependent and may remain insensitive to persistent abnormalities of myocardial mechanics. Recent work using speckle-tracking echocardiography has demonstrated that global longitudinal strain and mechanical dispersion can identify patients at risk of adverse LV remodeling after valve replacement even when conventional parameters provide less complete information [13]. This supports incorporating myocardial strain into future prospective studies of postoperative reverse remodeling.

The most consistent structural finding in the present study was the reduction in LA diameter in all seven patients. The largest reductions occurred in Patient 2 (25.0 mm), Patient 7 (24.5 mm), Patient 6 (21.7 mm), and Patient 3 (19.6 mm). The Patients 1–5 already demonstrated a consistent reduction in recorded LA diameter. Across all seven patients, this pattern was consistently observed, with LA diameter decreasing from 50.8 to 29.1 mm and from 53.1 to 28.6 mm, respectively.

LA enlargement in significant mitral valve disease reflects chronic pressure and/or volume loading and is frequently accompanied by changes in atrial compliance, fibrosis, electrical remodeling, and susceptibility to atrial arrhythmias. Relief of the abnormal valvular burden therefore creates the haemodynamic conditions necessary for atrial reverse remodeling. Chipeta et al. studied patients with chronic severe mitral regurgitation after mitral valve surgery and demonstrated a substantial reduction in LA volume early after intervention, with additional remodeling during subsequent follow-up [9]. Importantly, their serial assessment showed that LA remodeling is dynamic rather than a single postoperative event.

Candan et al. similarly found significant LA reverse remodeling after mitral valve surgery and showed that preoperative atrial mechanical characteristics assessed by speckle-tracking imaging were related to the magnitude of subsequent remodeling [10]. Together, these studies provide biological support for the consistent reduction in LA size observed in the current study.

Nevertheless, the present study used LA linear diameter rather than indexed LA volume. This distinction is methodologically important. LA remodeling is three-dimensional, and a single anteroposterior diameter may not fully represent changes in atrial geometry. Therefore, the current data support a reduction in recorded LA dimension but should not be interpreted as proof of complete normalization of atrial size or function. Future studies would benefit from LA volume index and LA reservoir strain.

In contrast to the uniform improvement in EF and LA diameter, LVIDd changed in different directions. Patients 4, 6, and 7 demonstrated reductions in LVIDd, whereas Patients 1, 2, 3, and 5 showed increases. Patient 7 demonstrated the largest reduction, from 49.0 to 32.2 mm, followed by Patient 6, from 47.6 to 33.5 mm. Conversely, Patient 1 increased from 36.5 to 53.3 mm. The source records therefore do not support a statement that LV chamber size uniformly regressed after valve replacement.

This heterogeneity is clinically plausible. Remodeling is influenced by the underlying valve lesion, duration of pressure or volume overload, baseline chamber geometry, myocardial fibrosis, loading conditions, rhythm, concomitant ventricular disease, and prosthesis haemodynamics. Furthermore, changes in a single linear LV dimension do not necessarily parallel changes in LV volume, mass, strain, or EF.

The distinction is particularly relevant in light of contemporary studies of adverse remodeling. Zhang et al. defined postoperative adverse LV remodeling using changes in LV end-diastolic volume and EF and found that impaired preoperative global longitudinal strain and increased mechanical dispersion predicted an unfavourable postoperative response in rheumatic mitral stenosis [13]. In the present study, EF improved in every patient; therefore, an increase in LVIDd alone should not be labelled “adverse remodeling” without volumetric or strain data.

The discordance between EF improvement and LVIDd behaviour also illustrates why reverse remodeling should be viewed as a multidimensional phenotype rather than a binary outcome. In future investigations, biplane LV volumes, indexed LV mass, relative wall thickness, global longitudinal strain, and diastolic parameters would provide a substantially more robust characterization.

The clearest demonstration of haemodynamic relief was observed in Patient 1, in whom the recorded aortic velocity decreased from 4.19 to 1.64 m/s and the recorded gradient fell from 70/41 mmHg to 11 mmHg. Reductions in recorded mitral velocities and gradients were also evident in several cases, including Patients 2, 4, and 5.

These observations illustrate the first stage of reverse remodeling: removal or substantial reduction of the abnormal haemodynamic load. However, successful valve implantation does not guarantee identical myocardial recovery among patients. Residual gradients, prosthesis size, effective orifice area, prosthesis–patient mismatch, systemic hypertension, and pre-existing myocardial disease can modify the remodeling response.

Tasca et al. demonstrated that prosthesis–patient mismatch after aortic valve replacement was associated with higher postoperative gradients and less regression of LV mass, highlighting the relationship between residual haemodynamic burden and incomplete reverse remodeling [16]. Similarly, Haverich et al., in a multicentre study of surgical aortic valve replacement, documented sustained haemodynamic performance accompanied by significant LV mass regression during follow-up, demonstrating that favourable prosthetic haemodynamics and myocardial structural recovery can evolve together [17].

The current study does not contain indexed effective orifice area, prosthesis size, or consistently standardized peak and mean gradients. Therefore, prosthesis–patient mismatch cannot be assessed reliably, and the recorded Doppler values should be interpreted as descriptive evidence of haemodynamic change rather than as a complete evaluation of prosthetic performance.

Paired RVSP values were available in only three patients, but all showed a postoperative decrease. RVSP fell from 69 to 35 mmHg in Patient 3, 45 to 35 mmHg in Patient 4, and 60 to 40 mmHg in Patient 5. The largest reduction of 34 mmHg in Patient 3 suggests substantial relief of the upstream haemodynamic burden associated with the valve lesion.

Reduction in pulmonary pressure after correction of left-sided valvular disease is expected when pulmonary hypertension is predominantly driven by elevated left atrial and pulmonary venous pressure. Nevertheless, pulmonary hypertension may persist after apparently successful valve surgery because of fixed pulmonary vascular remodeling, residual valve gradients, LV dysfunction, concomitant tricuspid disease, or other cardiopulmonary conditions.

Walls et al. examined matched preoperative and at least one-year postoperative RVSP estimates following mitral valve surgery and reported that pulmonary hypertension remained common after intervention despite an overall tendency toward reduced pulmonary pressures [15]. More recently, Collins et al., using the National Echocardiography Database of Australia, demonstrated that pulmonary hypertension following mitral valve replacement remained frequent and was associated with adverse long-term outcomes, emphasizing the importance of continued postoperative assessment rather than assuming complete pulmonary vascular normalization after valve correction [14].

The RVSP reductions observed in the current study are therefore clinically relevant, but the limited availability of this parameter prevents conclusions regarding the prevalence or completeness of pulmonary vascular reverse remodeling across the entire cohort.

The principal implication of this study is that the success of surgical valve replacement should not be evaluated solely by the presence of a normally functioning prosthesis. A comprehensive postoperative echocardiographic examination should integrate valve haemodynamics with assessment of LV systolic function, ventricular geometry, LA size, pulmonary pressures, and, where feasible, advanced measures of myocardial and atrial mechanics.

The current cases illustrate different remodeling phenotypes. Some demonstrated concordant improvement across several domains, whereas others showed improved EF and reduced LA size despite an increase in recorded LV diastolic dimension. Such patterns may be overlooked when follow-up focuses only on transprosthetic velocity and gradient.

These observations also support serial rather than single-time-point follow-up. LA remodeling, LV geometry, myocardial recovery, and pulmonary pressure may evolve at different rates [9,11,12]. Accordingly, the concept of being “beyond the prosthetic valve” is clinically relevant: the prosthesis corrects the primary mechanical lesion, but postoperative cardiac recovery represents an ongoing biological process.

Study limitations

Several limitations should be considered when interpreting these findings. First, the sample comprised only seven patients; therefore, the results are descriptive and cannot be generalized or subjected to meaningful inferential statistical analysis. Second, the retrospective design made the study dependent on measurements documented in existing echocardiographic reports.

Third, the cases were heterogeneous with respect to valve lesion and operative characteristics, and precise prosthesis types, sizes, indexed effective orifice areas, and surgical techniques were not consistently available. Fourth, advanced measures of remodeling—including LV mass index, LV volumes, LA volume index, global longitudinal strain, and atrial strain—were unavailable. The increase in LVIDd observed in four patients therefore cannot be equated directly with adverse volumetric remodeling.

Fifth, RVSP was available in only three patients. Sixth, some Doppler measurements were recorded using different or unclear formats; consequently, the manuscript appropriately retains the reported values rather than retrospectively reassigning them as peak or mean gradients.

CONCLUSION

This seven-patient study demonstrates that the echocardiographic response after surgical valve replacement extends beyond prosthetic valve performance and includes important structural, functional, and haemodynamic changes. The most consistent findings were improvement in left ventricular systolic function and reduction in left atrial diameter, with ejection fraction and fractional shortening increasing in all patients and left atrial size decreasing across the entire cohort. Reductions in right ventricular systolic pressure were also observed in all patients for whom paired measurements were available, while selected cases demonstrated marked improvement in valve-specific Doppler velocities and gradients.

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