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Original article

Feasibility and Limitations of Mitral Valve Repair, with or without Left Ventricular Reconstruction in Non-Ischemic Dilated Cardiomyopathy

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Abstract

Background: Although non-transplant surgical interventions for non-ischemic dilated cardiomyopathy (NIDCM) are relatively effective, their feasibility and limitations have not been fully elucidated. The aim of this study was to define the feasibility and limitations of mitral valve repair, with or without surgical ventricular reconstruction for patients with NIDCM in terms of postoperative low cardiac output syndrome (LOS).

Methods: Twenty non-transplant candidates (aged 57 ± 13 years) with NIDCM and significant mitral regurgitation had undergone mitral valve repair combined with submitral procedures. Using a 72-mL plastic ellipsoidal sizer, left ventricular reconstruction was performed concomitantly in 14/20 (70%) patients with extremely large ventricles. Total stroke volume, deceleration time of early trans-mitral flow wave, and the slope (Mw) in the preload recruitable stroke-work relationship were assessed using transthoracic echocardiography. LOS was defined as in-hospital death due to heart failure or a cardiac index less than 2.2 L/min/m^2 before discharge.

Results: There were three in-hospital deaths and four patients with postoperative cardiac index less than 2.2 L/min/m^2 [$n = 7$ (35%), LOS group]. Preoperative total stroke volume, deceleration time, and the Mw were significantly lower in the LOS group compared to those in the non-LOS group; the predicted cut-off values for LOS were 84 mL/beat ($p = 0.008$), 133 ms ($p = 0.015$), and $45 \text{ erg} \cdot \text{cm}^{-3} \cdot 10^3$ ($p = 0.036$), respectively. Preoperative left ventricular ejection fraction and ventricular size could not predict postoperative LOS. The one-year survival rate was 0% in the LOS group and 84% in the non-LOS group ($p < 0.001$).

Conclusions: Mitral valve repair, with or without left ventricular reconstruction, could be contraindicated for NIDCM patients with low total stroke volume, deceleration time,

and Mw in terms of high postoperative incidence of LOS. For high-risk patients, other therapeutic strategies might be necessary.

Introduction

Although the definitive therapy for non-ischemic dilated cardiomyopathy (NIDCM) patients with end-stage heart failure is heart transplantation, the number of transplantations are still limited in the Japanese society. Non-transplant surgical interventions including mitral valve repair and left ventricular reconstruction for NIDCM are sometimes effective [1, 2], but their feasibility and limitations have not yet been elucidated.

Mitral valve repair for patients with secondary severe mitral regurgitation (MR) and low left ventricular ejection fraction (LVEF), less than 30%, is still not a gold standard (class IIb) [3]. The elimination of MR increases the left ventricular (LV) afterload and may induce low cardiac output syndrome (LOS) after surgery. However, recent favorable results of MitraClip (Abbott, Menlo Park, California) [4] and other minimally invasive therapies [5, 6] for functional MR underscore the impact of interventions to the mitral valve, even when the LVEF is relatively low. On the other hand, there is a report of ineffectiveness (high early mortality) of MitraClip in patients with end-stage heart failure [7]. Furthermore, there is no contractile functional parameter that can predict postoperative LOS.

Within a decade, we have performed mitral valve repairs, with or without LV reconstruction, for NIDCM patients who were not candidates for transplantation. The aim of this study was to determine the feasibility and limitations of this surgery for NIDCM patients in terms of postoperative LOS predicted by preoperative contractile functional parameters.

Materials and Methods

Subjects

This retrospective longitudinal, observational study was performed at a single center (Hokkaido University Hospital). Initially, 23 patients with NIDCM who had undergone surgery between 2006 and 2013 were potentially eligible for the study. None of the patients had registered for heart transplantation due to old age and/or their personal refusal. Mitral repair was indicated for those with severe functional MR and heart failure symptoms refractory to medications (class IIb in ESC guidelines) [3]. We regarded patients as inoperable when they had severe organ failure due to LOS before surgery (total bilirubin > 3.0 mg/dl and serum creatinine > 3.0 mg/dl). We did not take LVEF into consideration for the decision making. All the patients were consulted for surgical intervention by local cardiologists or those at the University Hospital. The exclusion criteria were emergent surgeries and non-applicability to comprehensive echocardiographic parameters. After implementation of the exclusion criteria, 20 patients were selected. The University Ethics Committee approved of the research protocol and waived the informed consent requirements for the study.

Surgical procedures

Mitral valve annuloplasty, papillary muscle approximation, and suspension (mitral complex reconstruction) were performed for all the patients: 1) The papillary muscles were approximated side-by-side from the bases to the heads using three pledgeted mattress sutures; 2) A CV-4 ePTFE suture was placed between the site of the chordal attachment of the approximated papillary muscles and the anterior mitral

annulus. This suture was then passed through the true-sized semi-rigid total annuloplasty ring [8].

Overlapping left ventriculoplasty was also performed when the LV end-diastolic dimension was greater than 75 mm [9]. A 72-mL plastic ellipsoidal sizer (minimum ventricular size) was inserted into the LV to remodel an ellipsoidal shape. The lateral wall was sutured onto the deep septal wall using 3-0 polypropylene sutures with large specially designed curved needles. The medial wall was then overlapped onto the lateral wall using interrupted 4-0 polypropylene sutures.

The mean semi-rigid total ring size was 28 ± 2 mm. LV reconstruction was conducted in 14/20 (70%) (overlapping left ventriculoplasty - 13; Batista type - one). Tricuspid ring annuloplasty was performed for all the patients and MAZE procedure for 8/20 (40%) (chronic atrial fibrillation -seven; paroxysmal atrial fibrillation - one). Cardiopulmonary bypass and aortic cross clamp time were 228 ± 61 and 112 ± 24 minutes, respectively. Intra-aortic balloon pumping was performed preoperatively for 2/20 (10%) and intraoperatively for 5/20 (25%) patients. Histological examination using LV muscle obtained during surgery was performed in 14/20 patients. We introduced a semiquantitative, visual evaluation of the grade of interstitial fibrosis as follows: 0, absent; 1, mild; 2, moderate; 3, severe).

Transthoracic echocardiography

We used a Sonos 5500 ultrasound system (Philips Medical Systems, Andover, MA, USA) with a 3S transducer (3–5 MHz), a Vivid Seven system (GE/Vingmed, Milwaukee, WI, USA) with an M3S (2.5–3.5 MHz) transducer, or an Aplio system (Toshiba Medical Systems, Tokyo, Japan) with a 2.5 MHz transducer. LV end-diastolic

(LVDd) and end-systolic (LVDs) dimensions (mm), interventricular septal thickness (IVST), and LV posterior wall thickness (LVPWT) (mm) were measured from the parasternal long axis view. The MR volume and grade were determined by the volumetric method (1 - mild; 2 - mild-to-moderate; 3 - moderate-to-severe; 4 - severe [10]). The forward stroke volume was calculated using pulse wave Doppler at the LV outflow tract. The total stroke volume was calculated as follows: forward stroke volume + MR volume (mL). The LV end-diastolic volume (EDV), end-systolic volume (ESV), and LV ejection fraction (LVEF) were measured using the modified Simpson method. Deceleration time was determined as the duration from the peak to baseline of the early LV filling velocity (E-wave). Short deceleration time reflects operative ventricular stiffness [11].

The slope (Mw) in preload recruitable stroke work (PRSW) relationship - a relatively load-independent parameter of contractile function was estimated from a formula according to Lee et al: $Mw = (\text{total stroke work}) / [EDV - k \times EDV + (1 - k) \times \text{LV wall}]$ ($\text{erg} \cdot \text{cm}^{-3} \cdot 10^3$) [12, 13]. EDV was derived from the total stroke volume and LVEF. LV wall was estimated by the echocardiography-derived LV mass. Constant k was calculated as follows: $k = 0.0004 \times \text{LV mass} + 0.6408$ [13]. The total stroke work was calculated as follows: $(\text{total stroke volume}) \times (\text{SBP} - \text{LAP})$ ($\text{mL} \cdot \text{mmHg}$), where SBP and LAP indicate systolic blood pressure and left atrial pressure, respectively [14]. LAP was derived from continuous wave Doppler of the MR jet as follows: $(\text{systolic blood pressure}) - (\text{peak pressure gradient of the wave})$ (mmHg) [15]. The single-beat technique for Mw has been reported to closely match that by the invasive catheter method for different LV sizes, LV mass, and the presence of regional wall motion abnormalities [13]. All the specialized examiners were mutually blinded for this study.

Validation of functional parameters in patients registered for heart transplantation

In order to examine whether the contractile functional parameters can predict prognosis also in patients without valve surgery, we analyzed them in NIDCM patients who were newly registered for heart transplantation from 2013 to 2016 in our hospital. Those patients with less than moderate MR and catecholamine infusion at the registration were excluded from the analysis. Nine out of 23 patients were finally analyzed. Using the parameters detected in the surgical group, Cox regression analysis was performed to examine the prediction of LV assist device implantation.

Statistical analysis

All the data were represented as mean $\pm$ standard deviation. Statistical analysis was performed with SPSS version 17.0 software (SPSS Inc. Chicago, Ill). Categorical data were compared using Fisher's exact test. The Mann-Whitney U-test were used to compare the continuous variables between the groups and Wilcoxon test to compare the pre- and post-operative values. Postoperative LOS was defined as either in-hospital death due to heart failure or cardiac index less than 2.2 L/min/m² before discharge (39 $\pm$ 30 days after surgery). The receiver operating characteristic (ROC) curve was used to analyze the sensitivity and specificity of the parameters for detecting postoperative LOS. We selected the cut-off values that maximized sensitivity plus specificity. The area under the curve (AUC) was used to compare predictive parameters. The survival rate was analyzed using the Kaplan-Meier method. A *p* value < 0.05 was considered statistically significant.

Results

Predictors of postoperative LOS

The LOS group included three in-hospital deaths due to heart failure and four patients with postoperative cardiac index < 2.2 L/min/m². A total of seven patients (7/20, 35%) in the LOS group were compared to 13 patients (13/20, 65%) without low cardiac index (non-LOS group). In the three in-hospital deaths, the average cardiac index after surgery by Swan-Ganz catheter was less than 2.0 L/min/m² under catecholamine infusion (Dopamine/Dobutamine: 4-5/4-5 μ g; with or without adrenaline and noradrenaline). One patient needed left ventricular assist device intraoperatively. All the three patients died of multiorgan failure due to LOS on the postoperative day 16, 32, and 37.

Table 1 shows preoperative characteristics of the LOS and non-LOS groups. There was no significant difference in the age, gender, comorbidities, and medications between the two groups. LV reconstruction was performed in 6/7 and 9/13 patients in the LOS and non-LOS groups, respectively ($p = 1.00$).

Table 2 shows preoperative cardiac functional parameters of the LOS and non-LOS groups. Total stroke volume, deceleration time, and the Mw in the LOS group were smaller compared to those in the non-LOS group. LV size, LVEF and cardiac index were comparable between the groups. The grade of interstitial fibrosis which may influence passive stiffness of the LV tended to be greater in the LOS than in the non-LOS group without statistical significance.

Fig. 1 shows the ROC curve analysis of total stroke volume, deceleration time, and the Mw for the prediction of postoperative LOS; the AUC were 0.917 ($p = 0.008$),

0.861 ($p = 0.015$), 0.791 ($p = 0.036$), respectively. The cut-off value of total stroke volume was 84 mL (sensitivity: 100%; specificity: 75%); that of deceleration time was 133 ms (sensitivity: 83%; specificity: 75%); and that of Mw was $45 \text{ erg} \cdot \text{cm}^{-3} \cdot 10^3$ (sensitivity: 100%; specificity: 62%).

Changes in cardiac functional parameters

Fig. 2 shows the changes in cardiac indexes in the low-risk group (A: total stroke volume $> 84 \text{ mL}$, deceleration time $> 133 \text{ ms}$, and $\text{Mw} > 45 \text{ erg} \cdot \text{cm}^{-3} \cdot 10^3$) and the high-risk group (B: either total stroke volume $\leq 84 \text{ mL}$, deceleration time $\leq 133 \text{ ms}$, or $\text{Mw} \leq 45 \text{ erg} \cdot \text{cm}^{-3} \cdot 10^3$) respectively. In the low-risk group, no patient presented with postoperative LOS, whereas most of the patients in the high-risk group had LOS post-surgery.

Fig. 3 shows the changes in cardiac indexes in patients with (A) and without (B) LV reconstruction. Although there was no statistical significance in the increase of cardiac index between the groups ($p = 0.52$), cardiac index decreased after surgery in two patients with LV reconstruction.

Table 3 shows the changes in cardiac functional parameters in the LOS and non-LOS groups. The parameters taken six months post-surgery were only applicable in 11 non-LOS patients. MR grade and total stroke volume were significantly reduced after the surgery in both groups. In the non-LOS group, the significant reductions in LV size, MR grade and total stroke volume were sustained six months post-surgery. LV reconstruction did not influence the increase in deceleration time ($34 \pm 49 \text{ ms}$ and $39 \pm 73 \text{ ms}$ in the LV reconstruction and non-LV- reconstruction group, respectively; $p = 0.90$).

Survival rate

The mean follow-up period was 18 months. There were three in-hospital deaths (15%) due to heart failure and 13 late deaths (12 - heart failure; 1 - pneumonia). The overall survival rates were 64% after six months and 53% one year post-operation.

Fig. 4 shows survival rates of the LOS and non-LOS groups. The one-year survival rates were 0% and 84% in the LOS and non-LOS groups, respectively (log rank $p < 0.001$). The one-year survival rate in the LOS group was significantly lower compared to the predicted value with medical therapy alone according to the Seattle Heart Failure Model (0% vs. 47%). On the other hand, the one-year survival rate in the non-LOS group was higher compared to that of the Seattle Heart Failure Model predicated value (84% vs. 63%).

Frank-Starling's relationship in low- and high-Mw patients

Fig. 5 shows total stroke volume as a function of LV end-diastolic volume of each patient in the present study (corresponding to Frank-Starling's law). The functional curve of low-Mw patients was lying below that of high-Mw patients. This may indicate that the LV pump function is severely compromised in those with lower Mw.

Contractile functional parameters in patients registered for heart transplantation

In nine NIDCM patients registered for heart transplantation, six patients needed LV assist device implantation during the follow up (13 ± 12 months). Although LVEF, total stroke volume, and deceleration time did not predict LV assist device implantation ($p = 0.95, 0.80, \text{ and } 0.45$, respectively), Mw tended to predict the event ($p = 0.067$). The

average Mw in the heart-transplantation candidates was $28 \pm 11 \text{ erg} \cdot \text{cm}^{-3} \cdot 10^3$. All three patients with extremely low Mw ($< 20 \text{ erg} \cdot \text{cm}^{-3} \cdot 10^3$) needed LV assist device implantation within six months from the registration.

Discussion

We demonstrated that: (1) there exists a definite limitation to the surgery for NIDCM in terms of high postoperative LOS rates (35%), and (2) the contraindications could be determined by preoperative assessment of the total stroke volume, deceleration time (ventricular stiffness), and Mw (intrinsic contractile function).

Mitral valve surgery for non-ischemic dilated cardiomyopathy

Mitral valve repair for patients with secondary severe MR and low LVEF is still controversial [3]. Furthermore, diversity in the etiology of functional MR in previous studies (ischemic, non-ischemic, and valvular) makes the interpretation of results difficult [16, 17]. There have been two reports which included relatively high rates of populations with functional MR due to non-ischemic etiology. In a subgroup analysis (mitral valve replacement stratum) of the Acorn clinical trial (non-ischemic etiology - 78%; mean LVEF - $24 \pm 9\%$), 60% (62/102) of the patients experienced hemodynamic compromise after mitral valve surgery (mitral valve repair for 84%; replacement with chordal preservation for 16% of the patients). Furthermore, it took 18 months post-surgery for significant increase in LVEF and the 5-year total mortality was 30% [1]. In that study, however, risk factors for hemodynamic compromise were not analyzed. Wu et al. reported that restrictive mitral valve annuloplasty for functional MR

had no impact on late event-free survival of 180 patients with non-ischemic etiology. The use of angiotensin converting enzyme inhibitor and digoxin, serum sodium, and QRS interval were the independent predictors for the events (death, LV assist device implantation, or transplantation) [18]. Although some additional submitral procedures after annuloplasty may improve late outcomes, there have been no reports examining or validating the impact of those procedures.

Left ventricular reconstruction for non-ischemic dilated cardiomyopathy

LV volume reduction surgery (partial LV muscle resection) was first introduced by Batista et al. in 1996 [19]. Although the early beneficial effect has been reported [2], late failures precluded its widespread use and the partial left ventriculectomy has now been abandoned in western countries. In a mathematical model analysis, LV volume reduction surgery for diffusely dilated LV did not increase LVEF, and could therefore compromise cardiac output [20]. For a decade, we have performed LV reconstruction without muscle resection i.e., overlapping left ventriculoplasty. LV reconstruction does not guarantee LVEF increase (**Table 3**). After the present study, we have re-strategized mitral valve replacement combined with submitral procedures without LV reconstruction.

Implications of total stroke volume, deceleration time, and Mw in heart failure

In our recent study, total stroke volume (MR plus forward stroke volume) was significantly lower in the LOS group compared to the non-LOS group. Starling et al. reported that external total stroke work efficiency was significantly reduced in patients with chronic MR and New York Heart Association (NYHA) functional class III

compared to those with class I or II symptoms [21]. We therefore hypothesize that those patients with low total stroke volume (< 84 mL/beat) in our cohort already had end-stage LV function and were unresponsive to the surgery.

Deceleration time is the duration from the peak to baseline of the early LV filling velocity (E-wave). Patients with increased LV operative stiffness had more rapid rates of deceleration of early LV filling and shorter deceleration time [11]. Operative diastolic chamber stiffness (K_{lv} , mmHg/ml) is defined by change of LV pressure from time of minimal to late diastolic pressure divided by change of LV volume during this time period. Marino et al. reported that deceleration time in patients with low LVEF was proportional to the inverse square root of K_{lv} , independent of other factors, including LVEF, heart rate, left atrial chamber stiffness, mitral annular area, and Tau [22]. In our recent study, MR volume was rather smaller in the LOS than in the non-LOS group, whereas the difference of LV chamber size was not statistically significant. We therefore speculate that shorter deceleration time in the LOS group may imply increased intrinsic stiffness of the LV wall (passive stiffness) but not volume-overload caused by MR. Ueno et al. reported that the deceleration time significantly decreased and continued to decrease in the mid-term after septal anterior ventricular exclusion with an artificial patch. On the other hand, deceleration time increased after overlapping left ventriculoplasty without a patch. They asserted that the use of a stiff artificial patch could further stiffen the heart after surgery and compromise LV diastolic function [23]. Deceleration time significantly increased after surgery also in the present study. In an individual heart, the increase in deceleration time after surgery indicates reduced operative diastolic chamber stiffness. With decreasing LV volume, the slope of the relaxation (K_{lv}) also decreases. Thus, a reduced K_{lv} may be

because of decrease in ventricular size, decreased intrinsic stiffness of the LV wall, or both [22]. Intrinsic stiffness of the LV wall (passive stiffness) may not decrease early post-surgery. We therefore attribute the postoperative increase in deceleration time (reduced operative stiffness) mainly to unloading of the LV by the elimination of MR.

Preload recruitable stroke work relationship is defined as the relationship between LV stroke work and end-diastolic volume. The relationship is highly linear, reproducible and quantified by the slope (Mw) and x-intercept (Vw) [13]. Mw is recognized as a load-independent contractile functional parameter. Mw cannot be influenced by heart rate and ejection pressure [24]. Although there is no comparative study, Mw is more optimal as a functional parameter than LVEF because the loading conditions in MR can be changed largely according to heart rate, afterload, preload, and the severity of MR. We used total stroke work (TSW) by echocardiography for the calculation of Mw proposed by Lee et al: $Mw = TSW / [EDV - k \times EDV + (1 - k) \times LV \text{ wall}]$ [13]. The TSW was calculated as follows: (total stroke volume) $\times$ (SBP – LAP). This formula is based on the concept that the pressure-volume loop is assumed to be a square. There would be concerns about the difference in LV end-diastolic pressure and LAP, the absence of isovolumic contraction period in patients with severe MR. In our previous experimental validation using MR rats, the value of TSW by echo in which the pressure-volume loop was assumed to be a square could be approximated to that of TSW by catheter [12]. Constant k in the Mw formula was the ratio of the volumes within the epicardial shell ($V_{w,epi}/EDV_{B,epi}$) and validated in patients with wide range of LV volume and LV mass [13]. Taken together, we consider that it is feasible to use this single beat technique for MR patients.

Limitations

We encountered several limitations during this study. Firstly, this was a retrospective study with a relatively small number of patients; therefore, no direct comparison could be made to other surgical procedures or medical therapies alone. Secondly, we still cannot ascertain whether low-risk patients should undergo surgery or medical therapy alone. Further studies are necessary to compare the clinical outcomes of non-transplant surgery and medical therapies followed by implantable LV assist device or transplantation in our society. Also, some major complications after implantation of LV assist devices (infection - 50%; neurological events - 40%; malfunction - 40%; bleeding - 20% within one year) (Japanese registry for Mechanically Assisted Circulatory Support 2014) should be reconsidered. Thirdly, pulmonary artery wedge pressure data would not be sufficient for making discussion regarding the issue of diastolic function of the heart with massive MR. We could not compare LV end-diastolic pressure between the groups because it was applicable in only a few patients.

Conclusion

Mitral valve repair with or without left ventricular reconstruction could be contraindicated for those with low total stroke volume, deceleration time and Mw in terms of high postoperative incidence of LOS. For high-risk patients, other therapeutic strategies including submitral procedures without left ventricular reconstruction or left ventricular assist device might be necessary.

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Disclosures

The Authors declare that there is no conflict of interest.

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Figure legends

Figure 1. Receiver operating characteristic (ROC) curve for detecting postoperative LOS. MR, mitral regurgitation grade; Mw, slope in preload recruitable stroke work.

Figure 2. Changes of cardiac index in the low-risk group (A: total stroke volume > 84 mL, deceleration time > 133 ms, and $Mw > 45 \text{ erg} \cdot \text{cm}^{-3} \cdot 10^3$) and the high-risk group (B: either total stroke volume ≤ 84 mL, deceleration time ≤ 133 ms, or $Mw \leq 45 \text{ erg} \cdot \text{cm}^{-3} \cdot 10^3$) respectively.

Figure 3. Changes of cardiac index in those with (A) and without (B) left ventricular reconstruction surgery.

Figure 4. Survival curve in the LOS and non-LOS groups. LOS, low cardiac output syndrome.

Figure 5. Frank-Starling relationship in the low Mw and high Mw groups. Mw, slope in preload recruitable stroke work.

Figure 1

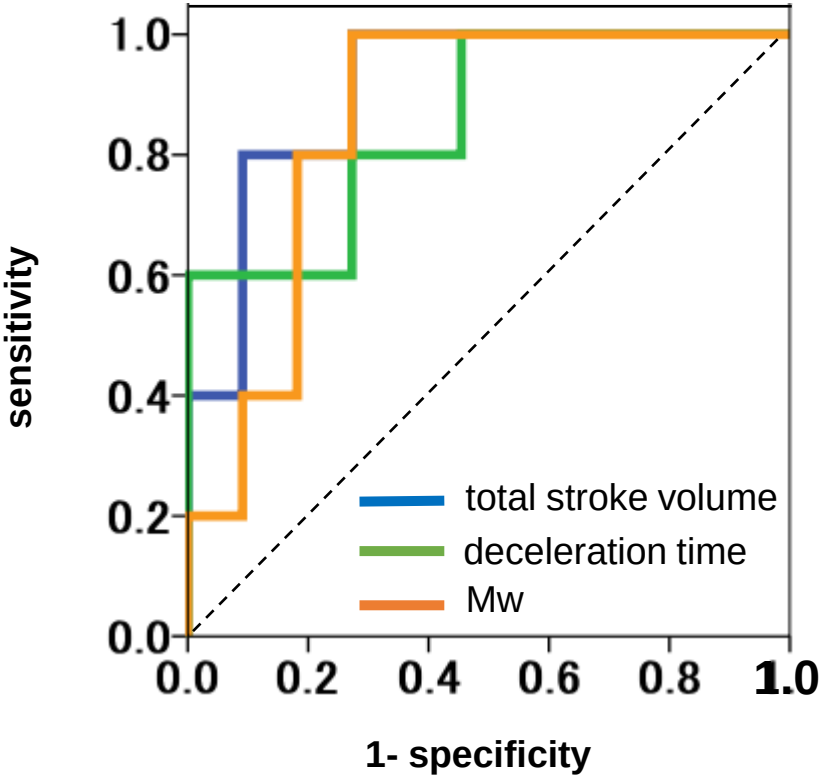


Figure 2

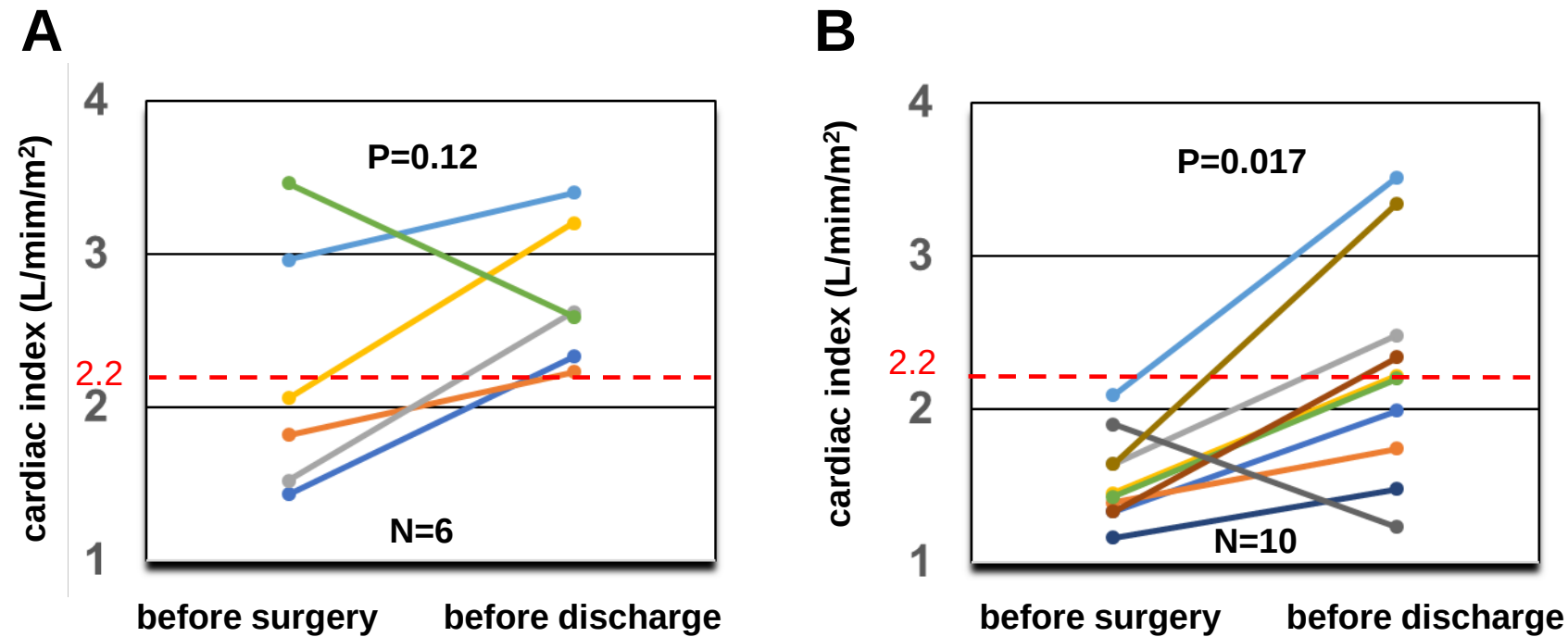
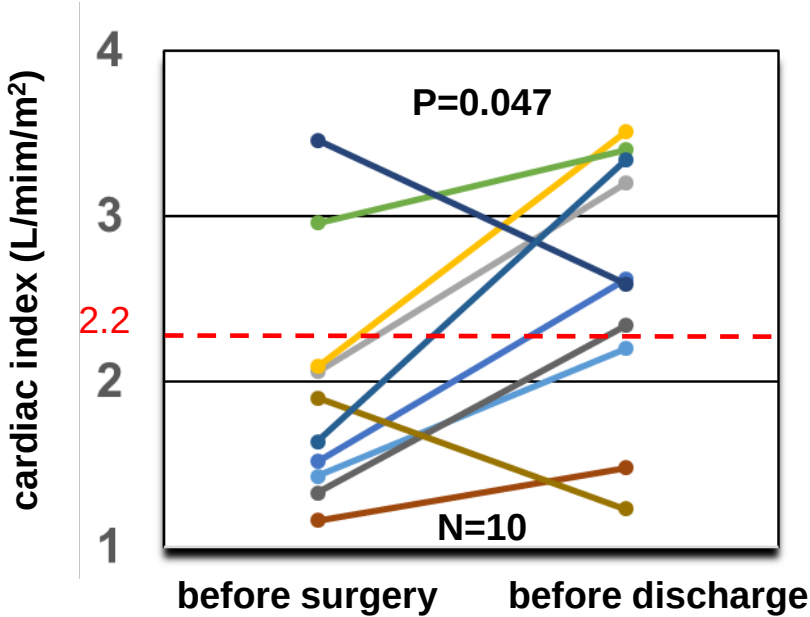


Figure 3

A



B

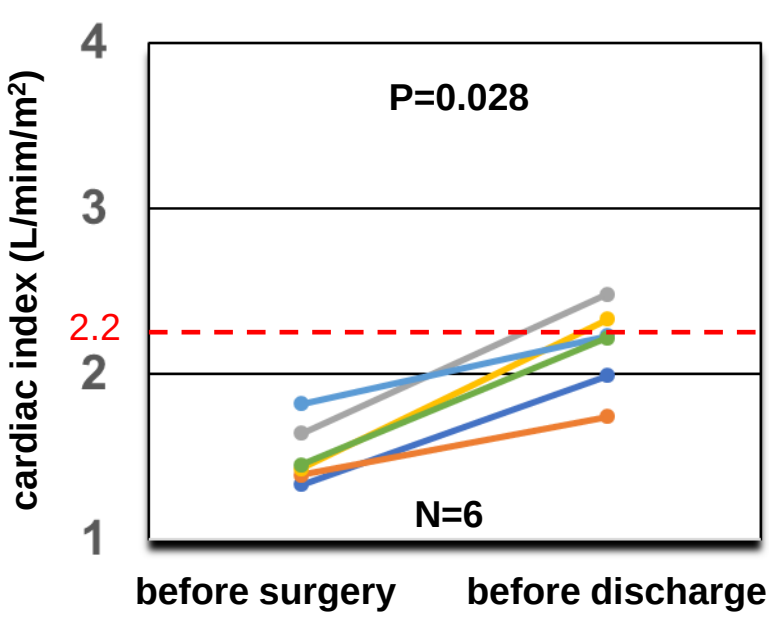


Figure 4

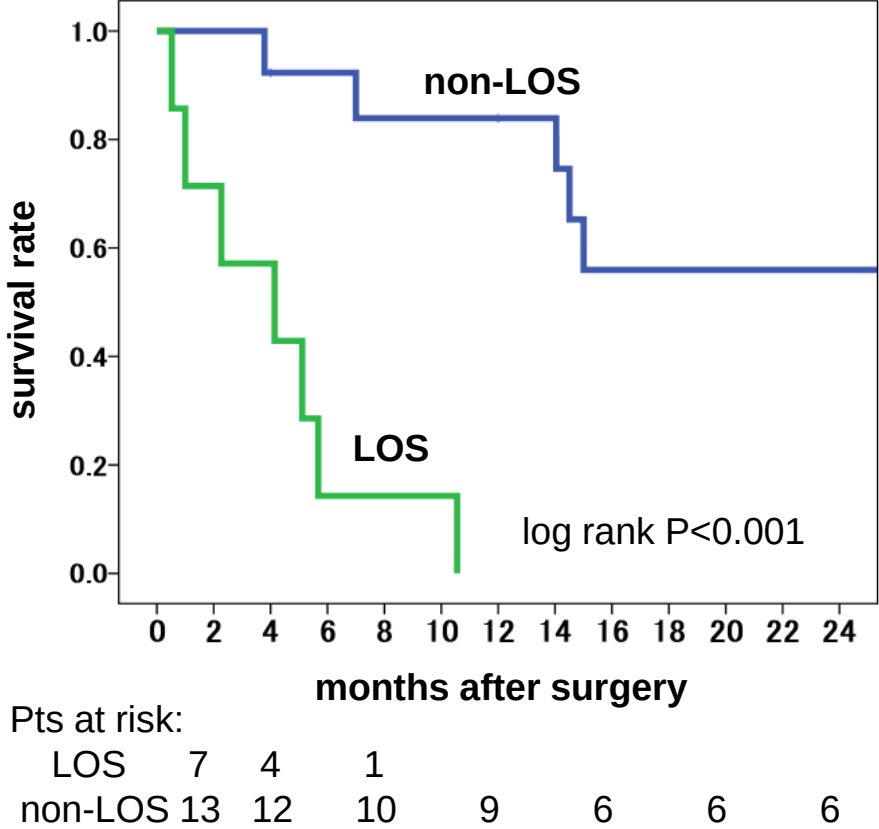


Figure 5

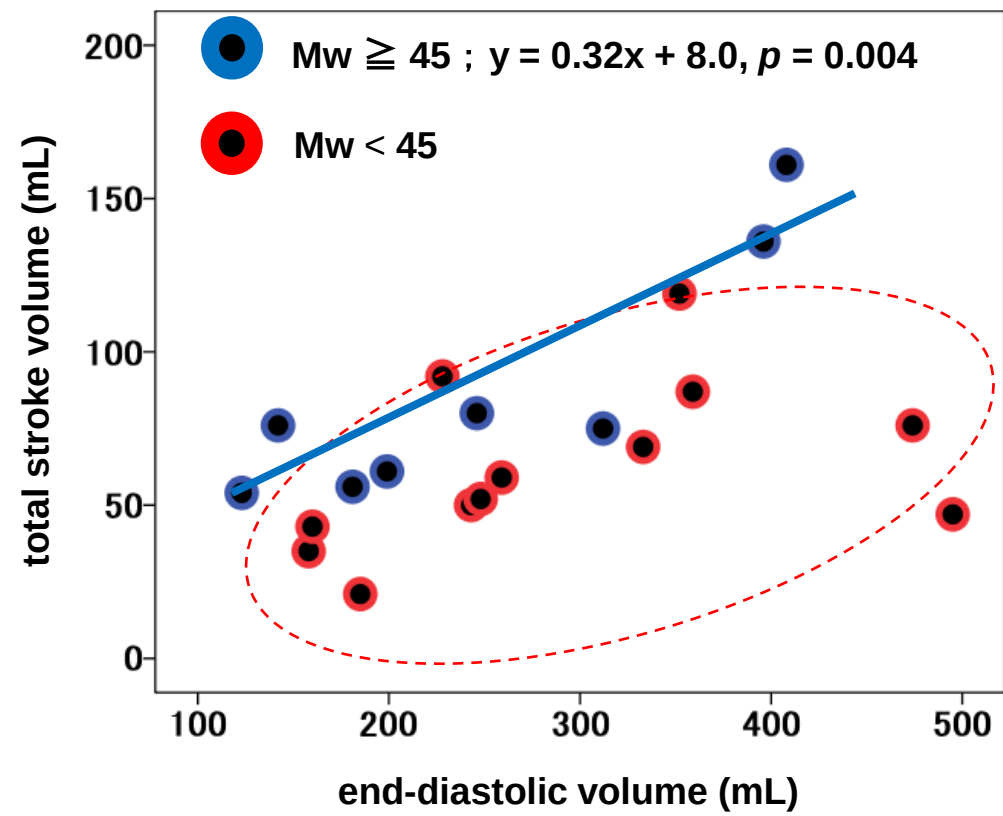


Table 1. Preoperative characteristics in postoperative LOS and non-LOS groups

	Total (n=20)	LOS (n=7)	non-LOS (n=13)	P value
Age	57±13	59±11	55±15	0.58
Male	16 (80%)	7	9	0.25
Body surface area, m ²	1.64±0.14	1.71±0.15	1.60±0.13	0.11
NYHA class	3.5±0.5	3.6±0.5	3.5±0.5	0.65
BNP, pg/mL	1499±1407	1372±909	1567±1645	0.72
Heart rate	72±14	76±14	69±14	0.20
Systolic blood pressure, mmHg	91±13	88±6	92±15	0.84
Diastolic blood pressure, mmHg	55±12	56±6	55±14	0.45
Dyslipidemia	4 (20%)	8	5	1.00
Diabetes mellitus	6 (30%)	2	4	1.00
Chronic renal failure	7 (35%)	3	4	0.65
Atrial fibrillation	6 (30%)	3	3	0.34
CRT	8 (40%)	4	4	0.32
ICD	7 (35%)	3	4	0.62
Medications				
Beta blockers	18 (90%)	7	11	0.52
ARB	9 (45%)	2	7	0.37
ACE-I	6 (30%)	2	4	1.00
Seattle Heart Failure Mode				
estimated one-year survival	58±25	47±29	63±22	0.19
estimated two-year survival	39±26	29±25	44±26	0.19

Values±SD. ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BNP, brain natriuretic peptide; CRT, cardiac resynchronization therapy; ICD, implantable cardioverter defibrillator; LOS, low cardiac output syndrome; NYHA, New York Heart Association.

Table 2. Preoperative cardiac function in postoperative LOS and non-LOS groups

	Total (n=20)	LOS (n=7)	non-LOS (n=13)	P value
Right heart catheter				
sPA, mmHg	47±17	55±15	43±16	0.12
PCWP, mmHg	24±10	26±8	24±11	0.75
Cardiac index, L/min/m ²	2.0±0.4	1.8±0.3	2.1±0.4	0.15
Echocardiography				
LVDd, mm	75±12	80±9	72±12	0.15
LVDs, mm	67±13	74±10	63±13	0.08
IVST, mm	9.7±1.4	9.0±1.3	10±1.3	0.13
PWT, mm	8.7±1.4	8.4±1.4	8.8±1.5	0.62
E wave, cm/s	103±30	95±15	108±36	0.19
DcT, ms	158±67	112±25	181±71	0.009
EDV, ml	335±134	320±138	251±90	0.19
ESV, ml	256±123	255±128	174±67	0.08
MR grade	3.9±0.3	3.7±0.5	4.0±0.0	0.17
Total stroke volume, ml/beat	43±19	67±15	105±30	0.019
Forward stroke volume, ml/beat	42±19	35±13	47±22	0.28
MR volume, ml/beat	51±21	30±10	58±19	0.006
Cardiac index, L/min/m ²	1.8±0.6	1.5±0.3	1.9±0.7	0.14
LVEF, %	27±9	22±7	27±9	0.07
Mw, erg·cm ⁻³ ·10 ³	46±23	32±10	53±25	0.050
Myocardial fibrosis grade	1.4±1.2 (n=14)	2.3±1.2	1.2±1.1	0.14

Values±SD. DcT, deceleration time; EDV, end-diastolic volume; ESV, end-systolic volume; IVST, interventricular septal thickness; LOS, low cardiac output syndrome; LVDd, left ventricular end-diastolic dimension; LVDs, left ventricular end-systolic dimension; LVEF, left ventricular ejection fraction; MR, mitral regurgitation; PCWP, pulmonary artery wedge pressure; PWT, LV posterior wall thickness; sPA, systolic pulmonary artery pressure.

Table 3. Changes in cardiac functional parameters

	LOS (n=4)			non-LOS (n=11)		
	before surgery	before discharge	6 months after surgery	before surgery	before discharge	6 months after surgery
Heart rate	80±14	93±14	NA	67±12	79±11**	76±10*
SBP, mmHg	90±6	85±17	NA	92±15	89±12	91±20
DBP, mmHg	57±7	54±13	NA	54±14	58±8	56±13
BNP, pg/mL	1278±882	732±322	NA	1410±1730	645±516	759±653
Echo						
LVDd, mm	81±12	71±12	NA	72±12	65±10**	67±12*
LVDs, mm	66±14	59±10	NA	62±13	57±10*	59±13
IVST, mm	8.3±1.0	8.5±1.3	NA	10±1.4	10±1.4	10±1.4
PWT, mm	8.5±1.0	8.3±1.0	NA	9.0±1.5	9.0±1.3	9.1±1.2
DcT, ms	116±30	156±75	NA	191±73	220±60	224±77
MR grade	3.7±0.5	0.5±0.5**	NA	4.0±0.0	0.4±0.5**	0.7±0.8**
TSV, mL/beat	65±16	29±7**	NA	110±29	55±14**	54±19**
CI, L/min/m ²	1.4±0.3	1.6±0.3	NA	2.0±0.7	2.7±0.6**	2.6±0.9*
LVEF, %	18±6	17±4	NA	31±8	28±7	28±7

Values±SD. BNP, brain natriuretic peptide; CI, cardiac index; DBP, diastolic blood pressure; DcT, deceleration time; IVST, interventricular septal thickness; LOS, low cardiac output syndrome; LVDd, left ventricular end-diastolic dimension; LVDs, left ventricular end-systolic dimension; LVEF, left ventricular ejection fraction; MR, mitral regurgitation; NA, not applicable; PWT, LV posterior wall thickness; SBP, systolic blood pressure; TSV, total stroke volume. *P<0.05 and **P<0.01 vs. before surgery.