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*Original Article*

# End-systolic wall stress is a major determinant of postoperative left ventricular dysfunction in patients with congenital mitral regurgitation

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**Abstract** To clarify the contribution of afterload to left ventricular performance after repair of mitral regurgitation, we evaluated echocardiographically 8 children who had undergone surgical repair for isolated congenital mitral regurgitation. We examined the relationship between left ventricular systolic function and preload, afterload, and contractility. The left ventricular systolic function was strongly correlated with the afterload after the surgical repair. In the postoperative state, reducing afterload by vasodilators could be a useful means of treating cardiac failure, in addition to using catecholamines to increase the contractility.

Keywords: Afterload; afterload mismatch; mitral regurgitation

In patients with chronic volume overload due to severe mitral regurgitation, left ventricular systolic function is usually overestimated when calculated using conventional parameters related to the ejection phase, since the presence of mitral regurgitation reduces the impedance to emptying of the left ventricle.[1] Thus, an unexpected reduction in left ventricular function may sometimes becomes apparent after surgery. Little has been reported, however, on left ventricular function after repair of mitral regurgitation. In particular, at present we do not know whether the decreased left ventricular systolic function is the consequence of impaired contractility. We designed the present study, therefore, to clarify the cause of left ventricular systolic dysfunction seen after the surgical repair of mitral regurgitation.

## Materials and methods

We enrolled 8 patients who had undergone replacement or plasty of the mitral valve because of isolated congenital mitral regurgitation. Prior to surgery, the regurgitation was graded at levels 3 or 4 in the scheme proposed by Sellers and colleagues.[2] The age of the patients at operation ranged from 1 to 19 years (Table 1). After surgery, no patient continued to exhibit significant mitral regurgitation. We evaluated left ventricular function between 11 and 37 days after surgery using cross-sectional echocardiography.

To quantitate left ventricular systolic function, we used the parameters of fractional shortening and the mean velocity of circumferential fiber shortening corrected for heart rate. For establishing left ventricular preload, we used the index of left ventricular

Table 1. Clinical and echocardiographic features of patients.

| Patient | Age at operation | FS | mV cfc (circ/sec) | %LVDd | ESWS | SVI |
|---|---|---|---|---|---|---|
| 1 | 1 | 0.28 | 0.90 | 106 | 97 | +2.5 |
| 2 | 2 | 0.29 | 0.98 | 137 | 74 | +1.9 |
| 3 | 3 | 0.23 | 0.75 | 93 | 94 | +0.27 |
| 4 | 4 | 0.38 | 1.34 | 90 | 35 | +3.7 |
| 5 | 6 | 0.40 | 1.27 | 92 | 37 | +3.0 |
| 6 | 12 | 0.36 | 1.21 | 81 | 72 | +4.7 |
| 7 | 14 | 0.16 | 0.48 | 137 | 161 | +1.6 |
| 8 | 19 | 0.28 | 0.64 | 117 | 113 | +0.12 |

Abbreviations: ESWS: end-systolic wall stress; FS: fractional shortening; %LVDd: left ventricular end-diastolic dimension index; mV cfc: heart rate-corrected mean velocity of circumferential fibre shortening; SVI: stress velocity index

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end-diastolic dimension, and used end-systolic wall stress as the surrogate of left ventricular afterload. The stress velocity index was used as the marker of left ventricular contractility.

An electrocardiogram was recorded simultaneously with the echocardiographic recording, and blood pressure was measured from the right arm using a cuff at the time of each echocardiographic study. Measurements of dimensions and inferior wall thicknesses, at both end-diastole and end-systole, were made according to the recommendations of the American Society of Echocardiography.[3] From these measurements, we obtained the values of left ventricular fractional shortening, the mean velocity of circumferential fiber shortening corrected for heart rate, the index of left ventricular end-diastolic dimension, end-systolic wall stress, and the index of stress velocity as follows:

- The left ventricular end-diastolic dimension was converted to an index of end-diastolic dimension, representing the percentage of "normal" end-diastolic dimension, using the known relationship between body surface area and left ventricular end-diastolic dimension in healthy Japanese children. The estimated left ventricular end-diastolic dimension equals $40.8 + 35.1 \times \log$[body surface area ($m^2$)].[4]
- The end-systolic wall stress was calculated according to the method described by Grossman et al.[5] and Colan et al.[6]
- The index of stress velocity index was calculated using the method of Colan et al.[7] (Table 2).

The values were then used to examine the contributions of preload, afterload and contractility to left ventricular systolic function after surgery.

### *Statistical analysis*

The strength of the relation between fractional shortening or the mean velocity of circumferential fiber shortening corrected for heart rate and the index of end-diastolic dimension, end-systolic wall stress, and the index of stress velocity was assessed using Pearson's correlation coefficient. For multivariate analysis, we employed stepwise regression. A level of probability of less than 0.05 was considered statistically significant.

## Results (Table 1)

Although left ventricular contractility was preserved in all patients, the left ventricular systolic function was impaired in two patients, whose fractional shortening was under 0.28. The end-systolic wall stress in patients with impaired left ventricular systolic function was elevated.

### *Fractional shortening*

Univariate analysis demonstrated the presence of a strong correlation between fractional shortening and end-systolic wall stress ($r = 0.927$). The relationships between fractional shortening and other two factors were weak, the correlation with end-diastolic dimension being $r = 0.665$, and the index of stress velocity being $r = 0.683$. We used simple analysis of linear regression to demonstrate the relationship between left ventricular end-systolic wall stress and left ventricular fractional shortening (Fig. 1).

Multivariate analysis for fractional shortening then confirmed that, after adjustment for end-systolic wall stress, other univariate predictors of fractional shortening had no additional independent influence on the estimation. The F value for end-systolic wall

Table 2. Formulas for calculation.

**Fractional shortening**

$$\frac{[\text{End-diastolic dimension}] - [\text{End-systolic dimension}]}{[\text{End-diastolic dimension}]}$$

**End-systolic wall stress**

$$\frac{1.35 \times [\text{End-systolic dimension}] \times [\text{End-systolic blood pressure}]}{4 \times [\text{End-systolic posterior wall thickness}] \times \left\{1 + \frac{[\text{End-systolic posterior wall thickness}]}{[\text{End-systolic dimension}]}\right\}} \times \frac{1332}{1000}$$

**Mean velocity of circumferential fiber shortening corrected for heart rate**

$$\frac{[\text{End-diastolic dimension}] - [\text{End-systolic dimension}]}{[\text{End-diastolic dimension}] \times [\text{Ejection time}]} \times [\text{Heart rate}]^{0.5}$$

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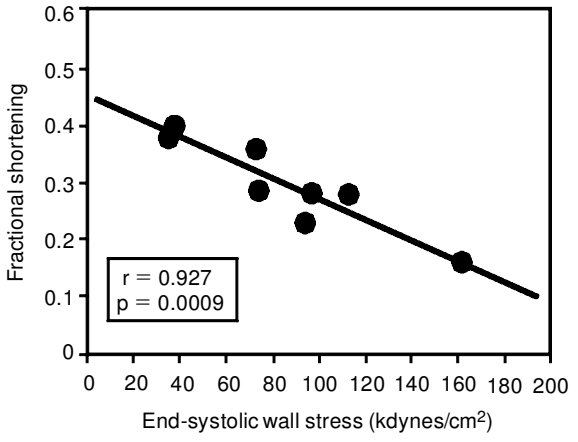


**Figure 1.**
*The correlation between left ventricular end-systolic wall stress and fractional shortening after surgery as shown using single linear regression analysis.*

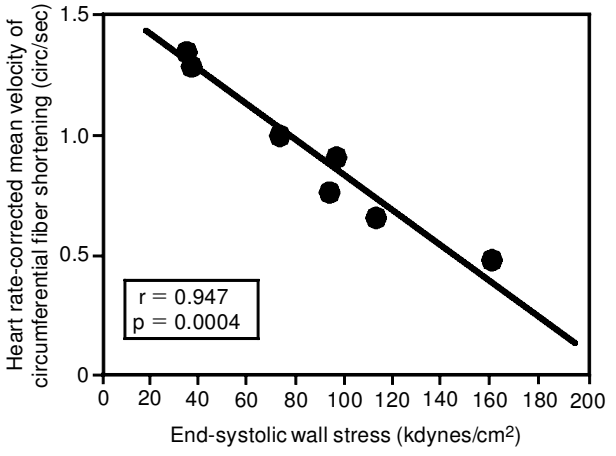


**Figure 2.**
*The correlation between left ventricular end-systolic wall stress and the mean velocity of circumferential fiber shortening corrected for heart rate after surgery as shown using single regression analysis.*

stress was 36.42, as opposed to 4.76 and 5.25 for the indexes of end-diastolic dimension and stress velocity, respectively.

### *Mean velocity of circumferential fiber shortening corrected for heart rate*

Univariate analysis demonstrated a strong correlation ($r = 0.947$) between the mean velocity of circumferential fiber shortening corrected for heart rate and end-systolic wall stress. The index of stress-velocity also had a significant correlation to the corrected mean velocity ($r = 0.803$). We again used simple linear regression to show the relationship between left ventricular end-systolic wall stress and mean velocity of circumferential fiber shortening corrected for heart rate (Fig. 2).

Multivariate analysis of the mean velocity of circumferential fiber shortening corrected for heart rate confirmed that end-systolic wall stress and the index of stress-velocity retained significance. The F value for end-systolic wall stress was 53.39, being 10.85 for the index of stress velocity, and 4.77 for the index of end-diastolic dimension.

## Discussion

Left ventricular systolic function is impaired in some patients after surgical repair of mitral regurgitation.[1] The cause of the dysfunction, however, has still to be clarified. Kennedy et al.[8] suggested that the impaired postoperative ventricular function could be related to permanent myocardial damage produced preoperatively by volume overload, the abnormality not becoming manifest until confronted with the increased afterload occurring after valvar replacement. Gaasch and Zile[9] demonstrated a postoperative increase of end-systolic wall stress in patients with "decompensated" mitral regurgitation. Our present results endorse these reports, confirming an increase of afterload. Moreover, we found that contractility remains unimpaired in the setting of impaired systolic function. Many investigators[10–13] have reported that left ventricular systolic function is strongly influenced not only by left ventricular contractility but also by afterload. Our data revealed that patients undergoing repair of mitral regurgitation are confronted by this situation. In the postoperative state of low output, therefore, reduction of afterload by vasodilators could improve the failing heart, just as catecholamines can increase the contractility.

Although impaired contractility has been reported previously in patients after surgical repair of mitral regurgitation,[8] we found that contractility was preserved in our patients. The differences perhaps reflect the period after surgery when the measurements were taken. In our study, echocardiographic evaluation was performed much earlier than in the previous reports. Because the high afterload causes "remodeling" of the ventricle,[14] the increased afterload may well impair contractility some time after surgical repair of mitral regurgitation. We also performed echocardiographic examinations in 6 patients enrolled in the present study at much later times after surgery – between 3 and 12 years. In all patients, the indexes of stress-velocity were lower than those found in our present study. Although it is impossible to make strong conclusions on the basis of such small numbers, it seems that the increased afterload might well impair the left ventricular contractility after repair of mitral regurgitation. A much larger scale study is now

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needed to clarify the mechanism of the perioperative change in cardiac function occurring in patients undergoing surgical repair of mitral regurgitation.

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