

# Mitral Regurgitation After Pericardiectomy for Constrictive Pericarditis

**We report a case of constrictive pericarditis in which trace mitral valve regurgitation was detected preoperatively and temporarily worsened after a pericardiectomy was performed. The early postoperative data suggested that the increased mobility of the lateral wall, in conjunction with an increase in the left ventricular volume, might be one of the causes of the perioperative mitral valve dysfunction. The mitral valve function returned to the preoperative baseline thirteen months after the pericardiectomy. (JJTCVS 1999; 47: 27–30)**

**Index words:** constrictive pericarditis, pericardiectomy, mitral valve insufficiency

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**T**he initial clinical and hemodynamic responses to a pericardiectomy are not always remarkable in some patients with constrictive pericarditis.<sup>1</sup> Less-than-optimal results are often caused by incomplete procedures,<sup>2</sup> resultant cardiomyopathy<sup>3</sup> and transient perioperative mitral valvular insufficiency.<sup>4</sup>

For this paper we evaluated perioperative mitral regurgitation, which transiently increased after a pericardiectomy for constrictive pericarditis was performed.

## Case

A 41-year-old man diagnosed with constrictive pericarditis was referred to the surgical department of the University of Tsukuba Hospital. Noted symptoms were progressive shortness of breath, fatigue, and edema. The liver was palpated 8 cm below the costal margin and an ascites was detect-

ed. An echocardiography showed trace mitral valve regurgitation, and cardiac catheterization revealed a dip-and-plateau pattern in the right ventricular waveform. The end-diastolic levels of pressure were found to be elevated and equal in the four cardiac chambers.

A phrenic-to-phrenic pericardiectomy with an inferoposterior pericardial release was performed through a median sternotomy incision. A cardiopulmonary bypass was not undertaken. (Estimated blood loss was 380 ml; fluid replacement, 3400 ml).

Hemodynamic features in the course of treatment were evaluated by cardiac catheterization and left ventriculography (Table I).

Two weeks after the pericardiectomy, the levels of pressure were similar to those recorded prior to the pericardial release, and the mitral regurgitation increased to grade 3/4. The ventriculographic regional shortening data of the anterior, lateral, posterior and diaphragmatic wall registered an increase as compared with the preoperative data ( $p < 0.01$ ). The patient required medication with digitalis and diuretics for two months.

Thirteen months after the pericardiectomy, the mitral regurgitation had returned to the preoperative grade of 1/4. The pressure levels were within normal ranges, and the regional wall motion had returned to the preoperative baseline.

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**Table I.** Summary of hemodynamic variables, grade of mitral regurgitation, and left ventricular function

		Preop	Operation		Postop	
			Before Sternotomy	After	2 weeks	13 months
Intracardiac pressure levels						
PCWP	(mmHg)	25	21	12	27	10
mean PAP	(mmHg)	27	24	18	30	18
RVEDP	(mmHg)	20	18	4	25	4
mean RAP	(mmHg)	20	21	10	23	3
LVEDP	(mmHg)	22			25	8
Ao or radial artery	(mmHg)	100/72	111/58	94/49	130/90	112/75
Grade of mitral regurgitation <sup>§</sup>		1/4			3/4	1/4
Left ventricular function						
CI	(L/min/m <sup>2</sup> )	2.6	2.7	3.0	2.5	3.1
LVEF	(%)	52.0			70.0	43.5
LVEDVI	(ml/m <sup>2</sup> )	112.8			130.5	94.5
LVESVI	(ml/m <sup>2</sup> )	54.4			40.0	53.4
regional shortening <sup>§§</sup>						
anterobasal	(%)	30.0± 3.6			45.4± 7.0**	20.0± 5.1**
anterolateral		15.8± 7.0			46.1± 4.7**	9.9± 2.1**
apical		16.5±15.3			27.2± 9.6**	23.8± 5.0
diaphragmatic		31.3± 7.6			51.7± 4.6**	34.5± 0.4
posterobasal		15.2± 7.1			31.7±22.5**	20.8±14.5
septal wall		32.7± 4.9			36.8± 9.7**	31.5± 3.3
posterolateral		29.5± 5.8			41.4± 5.4**	30.1± 3.9

PCWP, pulmonary capillary wedge pressure; PAP, pulmonary artery pressure; RVEDP, right ventricular end-diastolic pressure; LVEDP, left ventricular end-diastolic pressure; Ao, aorta; CI, cardiac index; LVEF, left ventricular ejection fraction; LVEDVI, left ventricular end-diastolic volume index; LVESVI, left ventricular end-systolic volume index.

<sup>§</sup>According to Seller's classification<sup>5</sup> on a scale of 4.

<sup>§§</sup>Regional wall motion of left ventricle. The outline of the left ventricle in 30-degree right anterior oblique projection was divided into five regions and the regional shortening was calculated by a radial method using a left ventricular wall motion analyzer. (Anterobasal=from segment 11 to 30; anterolateral=from segment 31 to 50; apical=from segment 51 to 70; diaphragmatic=from segment 71 to 90; posterobasal=from segment 91 to 110.) The outline of the left ventricle in 60-degree left anterior oblique projection was divided into two regions. (Septal wall=from segment 61 to 90; posterolateral=from segment 21 to 60.)

\*p<0.05, \*\*p<0.01 compared to the preoperative data.

## Discussion

This case showed the discrepancy between the patient's clinical course and postoperative hemodynamic features. Soon after the operation, the hemodynamic picture was essentially the same as before the pericardiectomy. The elevated levels of right- and left-side filling pressure could be ex-

plained by postoperative fluid retention leading to ventricular dilatation. The improved regional shortening could be due to the pericardial release and also be influenced by the unloading effect of the mitral insufficiency. The late postoperative data would in turn reflect the return of the patient's fluid balance to normal.

The increased mobility of the lateral wall of the

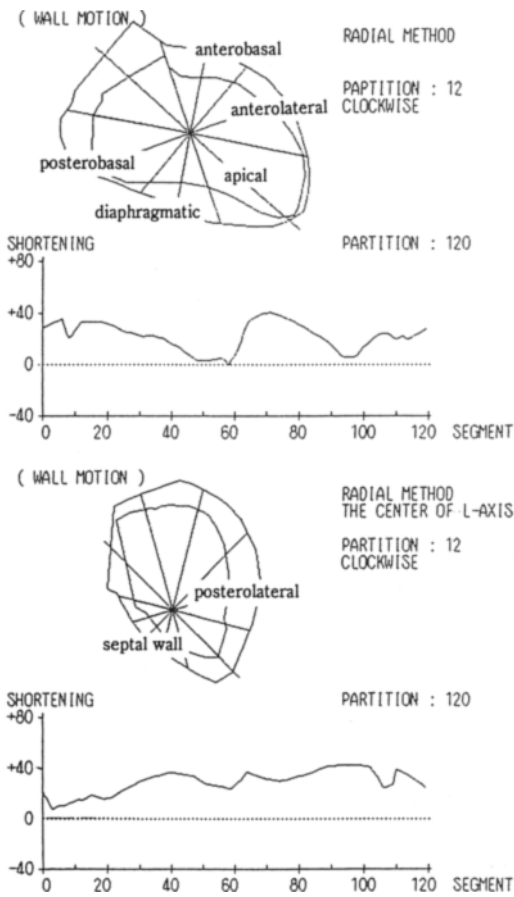


Fig. 1A. Preoperative left ventricular wall motion.

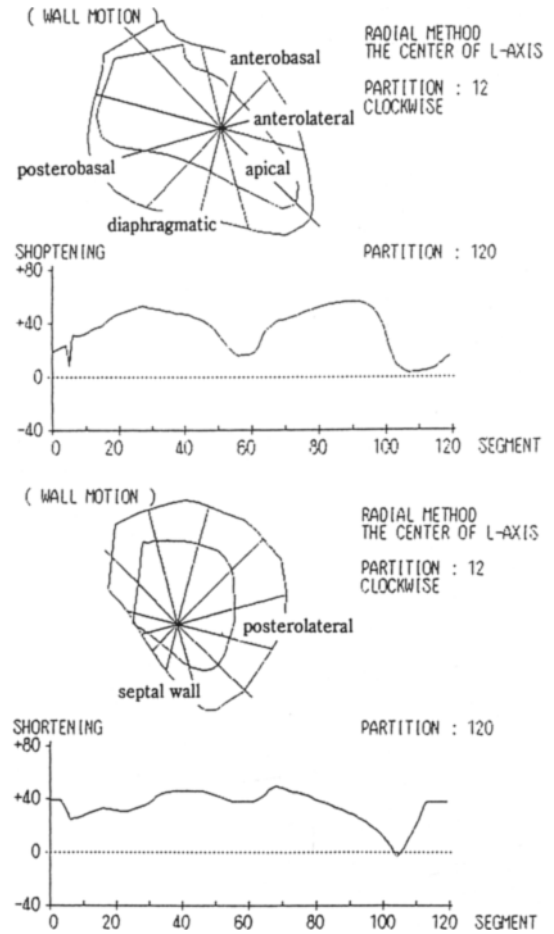


Fig. 1B. Left ventricular wall motion 2 weeks after the pericardiectomy showing the increased shortening of anterobasal, anterolateral, diaphragmatic, posterobasal and posterolateral wall.

left ventricle would be at least one of the causes of perioperative mitral valve regurgitation. An inward movement of the lateral wall increases, and the anterolateral papillary muscle becomes relatively too long functionally to allow for the coaptation of the valve leaflets (hypothesis one in the report by Buckingham, et al.<sup>4</sup>). The early postoperative data could not strongly suggest that the interventricular septum moved outward to a more normal position and that the posteromedial papillary muscle became relatively too short to produce normal coaptation (hypothesis two by Buckingham, et al.<sup>4</sup>). High right ventricular pressure continued to persist shortly after the operation.

We have no data concerning the size of the mitral annulus; however, the dilatation of the mitral annulus due to the increase in the left ventricular

volume might be another factor that influenced the perioperative mitral regurgitation.

After the pericardiectomy, compensation for the altered ventricular wall motion did occur over many months. The data suggests that the pathophysiology of mitral regurgitation following a pericardiectomy may be associated with multiple changes in the hemodynamics as well as the increased shortening of the lateral aspect of the left ventricle.

### Conclusion

The increased shortening of the lateral wall of

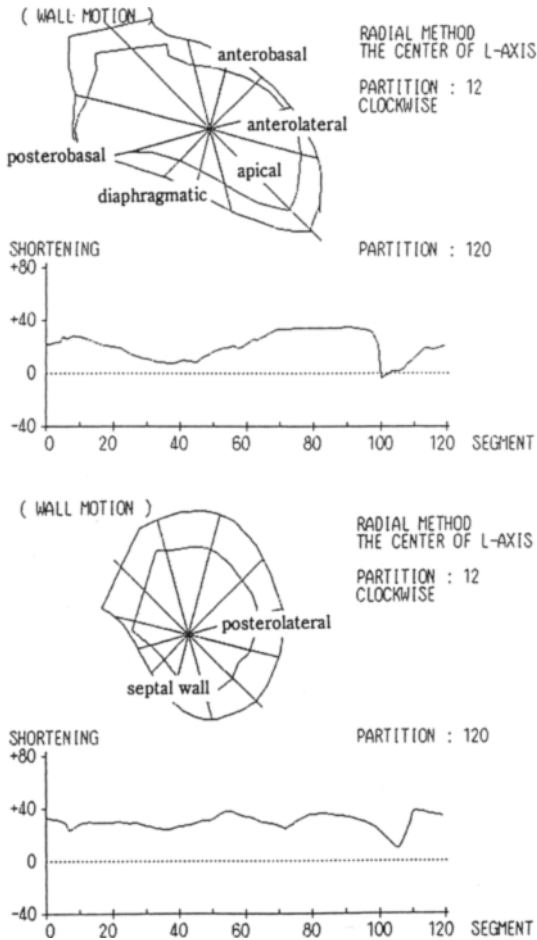


Fig. 1C. Left ventricular wall motion 13 months after the operation showing the regional wall motion had returned to the preoperative baseline.

the left ventricle, in conjunction with the left ventricular dilatation, might be one of the causes of a mitral valve dysfunction soon after performing a pericardiectomy.

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