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Kakimoto, Tetsuya

Yamabe, Hiroshi

Yokoyama, Mitsuhiro

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EXERCISE PERFORMANCE AFTER PTMC IN MITRAL STENOSIS: THE RELATION WITH HEMODYNAMICS, VENTILATORY RESPONSE AND SKELETAL MUSCLE FUNCTION

Tetsuya KAKIMOTO, Hiroshi YAMABE, and Mitsuhiro YOKOYAMA

First Division, Department of Internal Medicine,
Kobe University School of Medicine

INDEXING WORDS

PTMC; exercise test; cardiac output; excessive ventilation; muscle strength

SYNOPSIS

We examined the contribution of hemodynamics, ventilatory response and skeletal muscle function to the exercise performance after percutaneous transvenous mitral commissurotomy (PTMC). Nine patients with mitral stenosis (MS) underwent symptom-limited maximal ergometer exercise before, at 1 week and 3 months after PTMC. At 3 months after PTMC four patients (group1) revealed an improved exercise performance (peak oxygen uptake (VO2) 747 ± 145 to 1039 ± 175 ml/min; $p<0.01$), and five patients (group2) showed an unchanged exercise performance. Exercise performance in group1 already increased at 1 week after PTMC. Peak cardiac output (Q) in group1 increased (4.9 ± 0.7 to 6.9 ± 1.0 L/min, $p<0.05$) at 3 months after PTMC. The slope of the minute ventilation (VE)-carbon dioxide production (VCO2) relation during exercise decreased at 1 week after PTMC (40.2 ± 4.2 to 30.5 ± 3.8, $p<0.05$),and decreased slope of VE-VCO2 remained at 3 months after PTMC. However in group2 these parameters did not change. There was a significant correlation between percent change of peak VO2 and percent change of peak Q from before to 3 months after PTMC ($r=0.84, p<0.01$). However there were no differences in

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Authors' names in Japanese : 柿本哲也, 山辺 裕, 横山光宏

reduction of mean pulmonary artery pressure, pulmonary vascular resistance and maximum isometric force (MIF) of left quadriceps between two groups. These findings suggest that the increased Q and improved excessive ventilation were important for increase in exercise performance in patients with MS after PTMC.

INTRODUCTION

Exercise intolerance is a marker of severity and prognosis of chronic heart failure (CHF).[3,6,19] The mechanism of exercise intolerance has been argued. The importance of maximal cardiac output(Q)[15] as similar as in healthy persons[4] controversial because recent study showed that pharmacologic augmentation of Q did not always improve exercise performance in CHF.[13] Other reports proposed that skeletal muscle circulation[20] or skeletal muscle itself,[11,17] and excessive ventilation[18] plays an important role in exercise intolerance in CHF. Percutaneous transvenous mitral commissurotomy(PTMC) is a newly developed less invasive treatment to open mitral valve orifice in patients with MS.[9] Since patients can be ambulatory soon after procedure, PTMC gives a clinical model to assess the change of exercise performance induced by non-pharmacological hemodynamic change. The purpose of this study was to determine which of hemodynamics or ventilation or skeletal muscle is important for change of exercise performance in patients with MS after PTMC.

METHODS

Subjects

The study comprised nine patients (one man and eight women, aged 54 ± 9 years) with MS. Four patients were in New York Heart Association(NYHA) functional class Ⅱ, five patients were in class Ⅲ. Three patients had sinus rhythm and six patients had atrial fibrillation. Mitral regurgitation(MR) judged by angiography was grade 1 in four patients and grade 2 in one patient but none in grade 3 or 4. The patients had taken digoxin, diuretics and anti-coagulants, which were not changed throughout the study. The risks of the study were explained fully to the patients and

informed written consent was obtained.

Study design

The measurements consist of exercise performance, hemodynamics, ventilatory response and skeletal muscle function. These were performed before, at 1 week and 3 months after PTMC. The subjects were divided into 2 groups according to change of exercise performance, that is, group1 consisted of 4 patients with improved exercise performance and group2 consisted of 5 patients with unchanged exercise performance at 3 months after PTMC.

PTMC

All patients underwent PTMC successfully using an Inoue balloon catheter by a transseptal technique described previously in detail.[9] Mitral valve pressure gradient was calculated from left atrial pressure and left ventricular diastolic pressure. Echocardiographic mitral valve area was calculated by pressure half time method with Toshiba SSH 160A system.

Exercise test

All patients underwent symptom-limited maximal ergometer exercise without right heart catheterization before the study to be familiarized themselves with exercise test with respiratory gas analysis. Patients were given their usual cardiac medications 3 hr before testing. After right heart catheterization was prepared, symptom-limited sitting ergometer exercise was performed using electronically braked ergometer (Corival 400, Lode, Netherlands). The exercise was started at unloaded cycling for 2 minutes and followed by ramp protocol of which the increment of workrate was determined individually on the basis of the subject's exercise performance. All continued exercise until they complained of severe fatigue and/or dyspnea corresponding to Borg's scale 19 or 20. Heart rate(HR) was monitored continuously by standard electrocardiographic leads, and systemic blood pressure was measured every 1 minute using a ECG synchronized sphygmomanometer(STBP-680, Nippon Colin, Japan).

Respiratory gas analysis

Breath by breath measurements of respiratory gas exchange variables were determined. continuously throughout exercise with a metabolic measurement cart (RM-300, Minato, Japan). Respiratory gas exchange variables included minute ventilation(VE), oxygen uptake(VO2), carbon dioxide production(VCO2), respiratory rate(RR) and an estimated ventilatory dead space to tidal volume ratio(VD/TV) calculated with the use of end tidal pressure of carbon dioxide. Ventilatory anaerobic threshold(AT) was determined by v-slope method. The slope of the VE-VCO2 relation was calculated by linear regression analysis using the values of VE and VCO2 averaged 30 seconds of exercise.

Invasive hemodynamic measurement

Right heart catheterization was performed with a optically equipped catheter (Model 93A-431 H-7.5F, Edward, USA) from an antecubital vein. The tip of the catheter was placed in the right main pulmonary artery. The fiber optic lumen was attached to the optical module and oximetry processor instrument (SAT-2, Edward, USA). Mixed venous oxygen saturation(SvO2) was continuously measured with the processor instrument. Pulmonary artery pressure(PAP) and mean right atrial pressure were monitored continuously at rest and during exercise. Arterial blood oxygen saturation was obtained with pulse oximeter (N-200, NELLCOR, USA). Cardiac output(Q) was determined by direct Fick method. Pulmonary vascular resistance(PVR) was calculated from standard formulas using the value of Q, diastolic PAP and mean PAP. Invasive hemodynamic measurement at 1 week and 3 months after PTMC was not performed in one patient in group1.

Left ventricular systolic function

Left ventricular systolic function was evaluated by left ventricular end-systolic dimension(LVDs),left ventricular end-diastolic dimension(LVDd), and percent fractional shortening(%FS) using Toshiba SSH 160A system (Japan).

Skeletal muscle function

Skeletal muscle function was estimated by maximum isometric force(MIF) of left quadriceps measured on an isokinetic dynamometer

(CYBEX 2, Lumen, USA). The dynamometer consists of a lever arm, which was fixed at 60° . Three maximum voluntary isometric knee extensions were performed. The highest value was taken as MIF. MIF at 3 months after PTMC was not obtained in 2 patients of group2.

Statistical analysis

All data are expressed as mean ± standard deviation. Intergroup comparisons were analyzed by one way of ANOVA test. Intragroup variables before and after PTMC were compared by paired t test. Difference between variables were considered significant at $p<0.05$.

RESULTS

Patients characteristics (Table I)

Table I summarizes the clinical characteristics of the patients according to subgroups and total patients. Although mitral regurgitation was worsened in five patients after PTMC, none of the patients showed more than grade 3. Mitral valve pressure gradients were significantly decreased immediately after PTMC ($p<0.01$: before vs immediately after PTMC). Mitral valve areas enlarged significantly at 1 week after PTMC ($p<0.05$: before vs 1 week after PTMC) and remained at 3 months after PTMC ($p<0.05$: before vs 3 months after PTMC). NYHA functional class improved in seven patients at 3 months after PTMC, however it did not change in two patients of group2. Small left-to-right shunt, that is pulmonary/systemic blood flow ratio < 1.5, was found in two patients(case 5 and case 8). There were no differences in clinical characteristics between group1 and group2 except for age.

Exercise performance and hemodynamics (Table II, III)

Table II shows average values of exercise performance and hemodynamics in total cases. At resting condition, VO2 and Q did not change after PTMC, while mean PAP and PVR decreased significantly at 3 months after PTMC(both $p<0.01$: before vs 3 months after PTMC). At peak exercise, workrate, VO2 did not change significantly at 1 week and 3 months after PTMC. Q increased significantly at 1 week after PTMC($p<0.05$: before

vs 1 week after PTMC), but at 3 months after PTMC it returned to the level before PTMC. Mean PAP and PVR decreased significantly at 1 week after PTMC($p<0.05$ vs before PTMC) and kept the same level 3 months after PTMC($p<0.05$: before vs 3 months after PTMC). The AT did not change at 1 week after PTMC, and increased at 3 months after PTMC though it was not statistically significant.

Table Ⅲ shows the comparison of exercise performance and hemodynamics between two groups. In group1, peak VO2 increased significantly at 1 week after PTMC($p<0.05$: before vs 1 week after PTMC) and further increased at 3 months after PTMC($p<0.01$: before vs 3 months after PTMC) as shown in Figure 1-A. Peak workrate and AT significantly increased at 3 months after PTMC(both $p<0.05$: before vs 3 months after PTMC) as shown in Figure 1-B. Peak Q and peak HR increased significantly at 3 months after PTMC(both $p<0.05$: before vs 3 months after PTMC). Mean PAP and PVR at peak exercise did not significantly change. However, in group2, exercise performance and all hemodynamic parameters did not significantly change during follow-up period. Significant correlation($r=0.84, p<0.01$) between % change of peak VO2 and % change of peak Q from before PTMC to 3 months after PTMC were found(Figure 2).

Ventilatory response

Figure 3 shows the relationship between RR and TV before PTMC and after PTMC. In group1, RRs at rest and at peak exercise did not change during follow-up period(Figure 3-A). TV at peak exercise significantly increased at 1 week after PTMC(1124 ± 327 to 1370 ± 319 ml, $p<0.05$ vs before PTMC), and remained unchanged at 3 months after PTMC. Thus the relationship between TV and RR was shifted upward at 1 week and 3 months after PTMC compared to before PTMC. In group2, RR and TV did not change after PTMC compared to before PTMC (Figure 3-B). In group1 VD/TV at rest decreased significantly at 1 week after PTMC(0.49 ± 0.03 to 0.45 ± 0.03, $p<0.05$: before vs 1 week after PTMC), and it continued at 3 months after PTMC. VD/TV at peak exercise significantly decreased at 3 months after PTMC compared to before PTMC(0.40 ± 0.04 to 0.37 ± 0.02, $p<0.05$: before vs 3 months after PTMC). In group2, however, VD/TVs at rest and at peak exercise did not change significantly during follow-up period.

Slope of the relation between VE and VCO2(slope of VE-VCO2) are shown in Figure 4. In group1, slope of VE-VCO2 significantly decreased at 1 week after PTMC(40.2±4.2 to 30.5±3.8, $p<0.05$: before vs 1 week after PTMC), and it continued 3 months after PTMC. In group2, slope of VE-VCO2 did not change during follow-up period.

Left ventricular function (Table IV)

Table IV summarizes left ventricular systolic function in echocardiography. Parameters did not change significantly in both groups. Change of % fractional shortening of the left ventricle(△%FS) from before to 3 months after PTMC in group1 was significantly greater than in group2($p<0.05$: before vs 3 months after PTMC).

Skeletal muscle function (Table V)

Table V shows skeletal muscle function. MIF did not change during follow-up period in both groups, and no significant differences between both groups were observed. There was no significant correlation between % change of MIF and % change of peak VO2 from before to 3 months after PTMC(Figure 5).

DISCUSSION

Our study demonstrated that there were different courses of exercise performance after PTMC; four patients whose peak VO2 increased already at one week after PTMC had an improved exercise performance at 3 months after PTMC, while five patients had an unchanged one. Our result suggested that the increased Q and the improved ventilatory response were important. Whereas the significance of skeletal muscle function was not certain.

The unique design of our study is to measure simultaneously and serially invasive hemodynamics, ventilatory response and muscle function before, at 1 week and 3 months after PTMC, which have been reported to be important to limit exercise performance.[1,14,16,17] . For a reliable measurement of exercise performance several conditions should be considered; patients should be familiarized with the procedure, exercise protocol is proper and patients have a good motivation. As we were very careful about these

conditions as mentioned in the methods, we believe our result reliable enough to interpret.

Change of exercise performance

Unexpectedly, in five of nine patients exercise performance did not improve after PTMC in spite of increased mitral valve area and improved NYHA functional classification in three of five patients in group2, and that in patients with increased exercise performance at 3 months after PTMC it improved already at 1 week after PTMC. The change of exercise performance at 3 months after PTMC depended on Q response. Our finding in group1 agreed with McKay et al who reported that increased Q and reduced mean PAP and PVR at 3 months after PTMC were associated with an increase in exercise performance.[14] But our result of similar reduction in mean PAP and PVR in two groups suggested that hemodynamic change did not directly affect the exercise performance after PTMC. The improved exercise performance at only 1 week after PTMC in group1 differed from the acute effect of pharmacologic therapy reported by Wilson et al[20] We consider that ventilatory response was responsible for this phenomenon as mentioned below.

Unchanged exercise performance after PTMC

The mechanism why exercise performance does not improve after PTMC has rarely reported. Huikuri et al[8] showed that patients' symptom did not improve after surgical treatment of mitral stenosis, but they did not mentioned about the mechanism. In our study, patients of group2 were elder than group1. This may attribute to our results. However Feuvre et al[5] reported that elderly patients revealed increased Q after PTMC as same as younger patients. Thus it was unlikely that unchanged exercise performance was simply due to elder age of group2. Though we could not identify a definite mechanism of the unchanged Q in group2, a possible explanation for this finding is, first, myocardial factor probably due to rheumatic myocarditis.[7] Kitamura et al[10] reported that unchanged exercise performance after mitral valve repair was associated with negative exercise-induced change in %FS in echocardiography. In our study the increase in %FS after PTMC in group2 was significantly smaller than in group1. Myocardial

dysfunction which could not increase left ventricular systolic function after increased preload caused by PTMC may be a factor affecting Q response and exercise performance. The second explanation is right ventricular dysfunction. The increased afterload to right ventricle compromises its function.[2] After PTMC, decrease in right ventricular afterload is expected to increase right ventricular output to meet an increased left ventricular filling. However, advanced right ventricular dysfunction limits output even after PTMC, so was systemic blood flow.

Excessive ventilation

Excessive ventilation is a recent observation in CHF.[16,18] This phenomenon was suggested to relate with exertional dyspnea which is important for exercise intolerance in patients with CHF.[7] Excessive ventilation, decreased lung compliance and augmented air flow resistance increase a respiratory muscle work and induce a symptom of dyspnea under hypoperfusion of respiratory muscle during exercise in CHF.[12] Excessive ventilation is mainly caused by pulmonary ventilation-perfusion mismatching (high VA/Q).[18] Increased Q response in group1 may be a mechanism of improved excessive ventilation which may relieve the perception of dyspnea in combination with increased lung compliance as suggested by upward shift of TV-RR relationship. We consider that this is a mechanism of immediate improvement of exercise performance after PTMC in our group1.

Skeletal muscle function

Histological and biochemical studies demonstrated abnormal skeletal muscle in patients with CHF.[11,17] Lipkin et al[11] reported that type I muscle fiber was atrophic , and Sullivan et al[17] showed a decreased activity of oxidative muscle enzymes. These disorders induce a reduction of muscle strength. Buller et al[1] showed a reduced maximum isometric force of quadriceps and its significant correlation with maximum VO2. However in our study, MIFs before and after PTMC were not different between group1 and group2. And change of MIF did not correlate with change of exercise performance, though MIF tended to increase in group1 from before to after PTMC. Even though atrophic skeletal muscles as seen in patients with

severe mitral stenosis should limit an exercise performance, the change of exercise performance after PTMC was considered to be more strongly affected by a response of Q. Accordingly an influence of Q probably masked a role of muscle function in the change of exercise performance.

CONCLUSION

There were two courses of exercise performance after PTMC. One was improved and the other was unchanged. Increased Q and improved excessive ventilation were important for increased exercise performance.

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Table I. Patient population.

Patient		age	gender	NYHA		E C G	mitral valve area(cm2)			pressure gradient(mmHg)		mitral regurgitation	
				begore	3M	rhythm	before	1W	3M	before	after	before	after
group1	1	53	M	3	2	Af	1	1.8	1.7	8	4	1	2
	2	36	F	2	1	Af	0.7	1.2	1.2	19	8	2	2
	3	46	F	2	1	sinus	1.3	1.6	2	21	7	0	1
	4	52	F	3	2	Af	0.9	2	1.9	12	6	1	2
group mean ± sd		47 ± 8		3 ± 1	2 ± 1		1.0 ± 0.2	1.6 ± 0.3'	1.7 ± 0.4*	15 ± 6	6 ± 2*	1 ± 1	2 ± 1
group2	5	53	F	2	2	Af	0.8	1.5	1.8	8	4	0	2
	6	65	F	2	1	Af	1.6	2.2	2.2	7	5	0	0
	7	55	F	3	2	Af	0.8	1.6	1.6	16	6	0	1
	8	61	F	3	3	sinus	0.9	1.2	1.5	19	11	1	1
	9	65	F	3	2	sinus	1	1.5	1.6	10	6	1	1
group mean ± sd		60 ± 6#		3 ± 1	2 ± 1		1.0 ± 0.3	1.6 ± 0.4'	1.8 ± 0.3*	12 ± 6	6 ± 3*	1 ± 1	1 ± 1
total mean ± sd		54 ± 9		3 ± 1	2 ± 1		1.0 ± 0.3	1.6 ± 0.3'	1.7 ± 0.3*	13 ± 6	6 ± 2**	1 ± 1	1 ± 1

*p<0.05 before vs after PTMC;#p<0.05 group1 vs group2;NYHA,New York Heart Association;ECG,electrocardiogram;Af,atrial fibrillation;Sinus,sinus rhythm;
1W,1week after PTMC;3M,3 months after PTMC;Grading of mitral regurgitation(by angiography): 0,none;1,mild;2,moderate;3,moderate to severe;4,severe.

Table II. Exercise capacity and hemodynamic data during exercise before and after PTMC in all patients.

	before	1W	3M
peak workrate(watt)	58 ± 15	62 ± 13	69 ± 16
peak VO2(ml/min)	783 ± 106	824 ± 166	872 ± 205
VO2 at AT(ml/min)	508 ± 74	502 ± 74	565 ± 91
Q (L/min)			
rest	2.7 ± 0.6	3.1 ± 0.7	2.7 ± 0.5
peak	5.7 ± 1.0	6.9 ± 1.3*	6.1 ± 0.9
mPAP (mmHg)			
rest	14.6 ± 4.2	17 ± 11.1	10.2 ± 2.6**
peak	40.5 ± 13.3	31.9 ± 12.9*	30.7 ± 8.0*
PVR(dyne.s.cm-5)			
rest	156 ± 23	140 ± 47	129 ± 49
peak	205 ± 98	147 ± 79*	136 ± 43*

PTMC,percutaneous transvenous mitral commissurotomy;VO2,oxygen uptake;AT,ventilatory anaerobic threshold;
HR,heart rate;Q,cardiac output;mPAP,mean pulmonary artery pressure;PVR,pulmonary vascular resistance;
before,before PTMC;1 W;1 week after PTMC;3 M,3 months after PTMC;*p<0.05 before vs after PTMC,
** P < 0.01 before vs after PTMC.

Table III. Exercise capacity and hemodynamic data during exercise before and after PTMC in two groups.

	group1(n=4)			group2(n=5)		
	before	1W	3M	before	1W	3M
peak workrate(watt)	55 ± 22	71 ± 13	81 ± 16**	60 ± 8	55 ± 8	59 ± 6
peak VO2(ml/min)	747 ± 145	921 ± 156*	1039 ± 175**	811 ± 65	745 ± 140	740 ± 109
VO2 at AT (ml/min)	465 ± 53	486 ± 60	609 ± 65*	543 ± 72	515 ± 88	529 ± 100
peak Q (L/min)	4.9 ± 0.7	6.5 ± 1.6	6.9 ± 1.0*	6.1 ± 0.9	7.2 ± 1.3	5.7 ± 0.6
peak HR (beats/min)	130 ± 32	162 ± 6	154 ± 32*	163 ± 31	155 ± 28	146 ± 38
peak mPAP (mmHg)	42.2 ± 23.3	41.8 ± 16.8	35.2 ± 12.1	39.5 ± 5.7	25.9 ± 5.7	27.9 ± 3.6
peak PVR(dyne.s.cm-5)	248 ± 148	183 ± 129	142 ± 69	179 ± 61	124 ± 32	131 ± 28

PTMC,percutaneous transvenous mitral commissurotomy;VO2,oxygen uptake;AT,ventilatory anaerobic threshold;Q,cardiac output;HR,heart rate; SV,stroke volume;mPAP,mean pulmonary artery pressure;PVR,pulmonary vascular resistance; before,before PTMC;1 W;1 week after PTMC; 3 M,3 months after PTMC;*p<0.05 before vs after PTMC;** p<0.01 before vs after PTMC.

Table IV. Left ventricular systolic function before and after PTMC.

	group 1			group 2		
	Before	1W	3M	Before	1W	3M
LVDd (mm)	47.0 ± 6.7	47.5 ± 5.4	47.5 ± 6.1	46.6 ± 2.4	46.0 ± 6.8	47.3 ± 6.0
LVDs (mm)	35.0 ± 8.2	34.0 ± 4.9	32.3 ± 7.4	31.0 ± 2.5	32.4 ± 6.7	32.4 ± 4.7
%FS (%)	26.1 ± 8.9	28.5 ± 6.0	32.7 ± 8.1	33.5 ± 4.2	29.9 ± 4.7	31.5 ± 4.8
Δ%FS (%)		2.3 ± 4.8	6.6 ± 5.2#		-3.6 ± 5.0	-2.0 ± 4.2

PTMC:percutaneous transvenous mitral commissurotomy, before:before PTMC,1 W:1 week after PTMC, 3 M:3 months after PTMC, LVDd:left ventricular end diastolic dimension, LVDs:left ventricular end systolic dimension, %FS:percent fractional shortening of the left ventricle, Δ%FS:change of percent fractional shortening of the left ventricle before to after PTMC, *p<0.05 :before PTMC vs after PTMC, ** p<0.01:before PTMC vs after PTMC, #p<0.05 group1 vs group2.

Table V. Skeletal muscle function before and after PTMC in seven patients.

		group1	group2
MIF(FT.LBS)	before	53 ± 33	43 ± 7
	3M	68 ± 49	48 ± 7
	% change	25.5 ± 14.1	11.7 ± 9.1

MIF,quadriceps maximum isometric force;FT.LBS,foot.pounds;before,before PTMC; 3 M, 3 months after PTMC;% change, percent change from before to 3 months after PTMC.

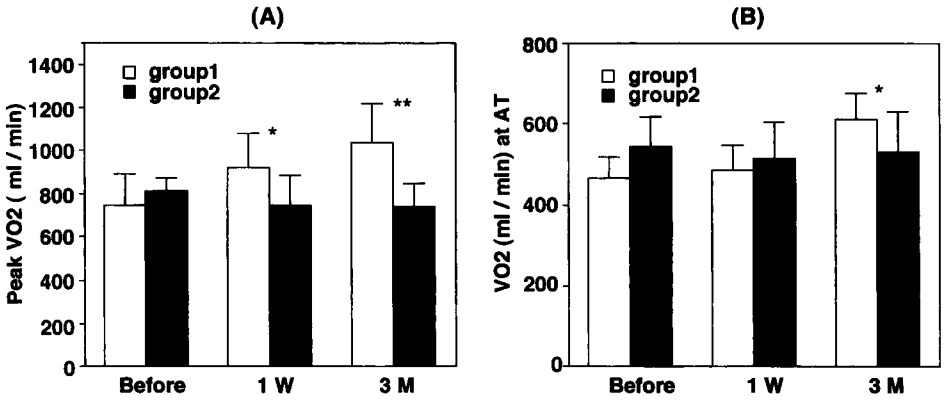

Figure 1. Plots of serial changes of peak oxygen uptake (A) and ventilatory anaerobic threshold(B) in group 1(□) and group 2(■) before, at 1week (1W) and at 3 months(3 M) after percutaneous transluminal mitral commissurotomy. * $p < 0.05$ before vs after PTMC, ** $P<0.01$ before vs after PTMC.

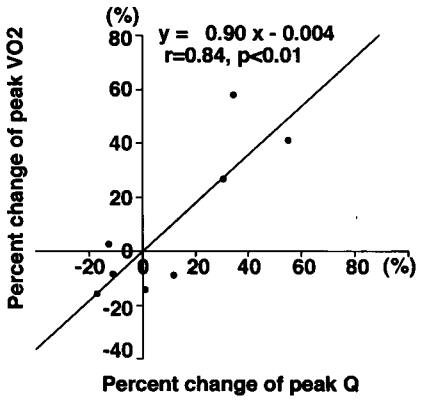

Figure 2. Graphs showing correlation between percent change of peak cardiac output(Q) and peak oxygen uptake(VO2) from before to 3 months after PTMC in eight patients.

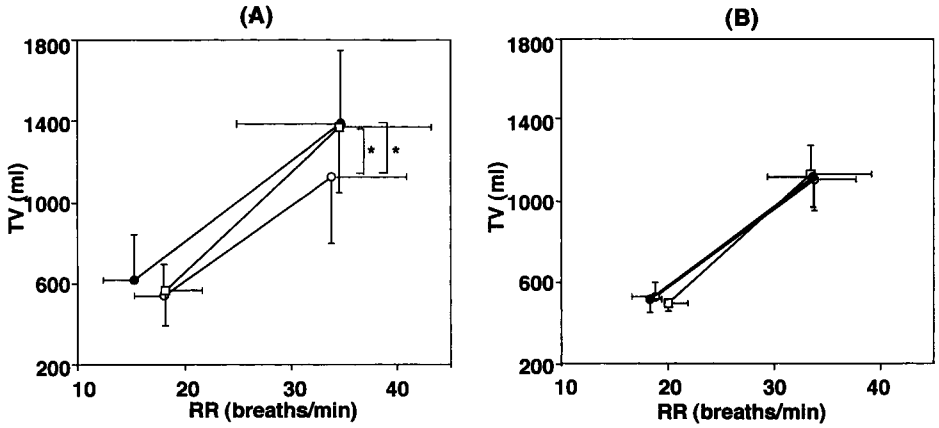

Figure 3. Relation between respiratory rate(RR) and tidal volume(TV) before(○),1 week(□) and 3 months(●) after percutaneous transvenous mitral commissurotomy(PTMC) in group1(A) and group2(B) . *P<0.05 before vs after PTMC.

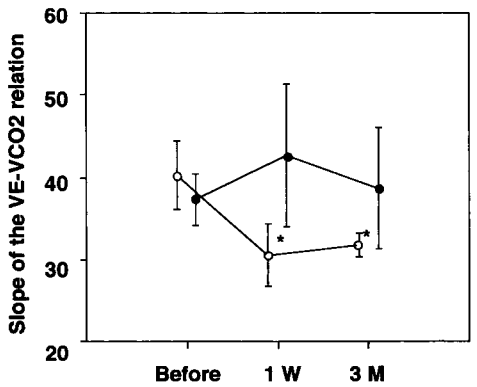

Figure 4. Plots of serial changes of slope of the relation between minute ventilation(VE) and carbon dioxide production(VCO2) in group1 (○) and group2 (●) before, at 1week after(1W) and at 3 months(3 M) after percutaneous transvenous mitral commissurotomy(PTMC) . * p<0.05 before vs after PTMC.

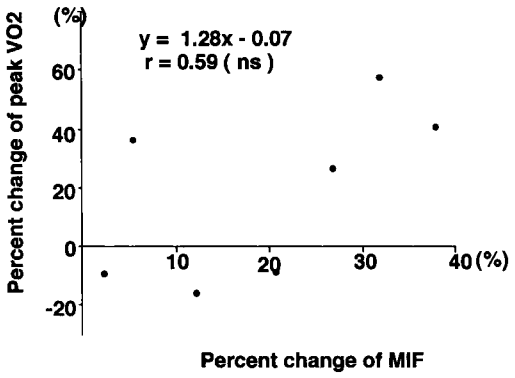

Figure 5. Correlation between percent change of peak oxygen uptake(VO2) and percent change of quadriceps maximum isometric force(MIF) from before to 3 months after percutaneous transvenous mitral commissurotomy(PTMC) in seven patients.