Exercise ventilation after balloon dilatation of the mitral valve

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Abstract

Background—Exertional dyspnoea is a limiting symptom in many patients with mitral stenosis but its causes remain incompletely understood. Ventilation during exercise is abnormal in chronic heart failure of all causes and there is increased ventilatory cost of carbon dioxide production.

Patients—23 patients with rheumatic mitral stenosis undergoing percutaneous balloon dilatation of the mitral valve were studied to investigate exercise ventilation.

Methods—Treadmill exercise tests with respiratory gas analysis were performed before and 1 day, 7 days, and 10 weeks after balloon dilatation of the mitral valve. The relation between ventilation ($\dot{V}_E$) and production ($\dot{V}_{CO_2}$) was analysed by linear regression.

Results—The $\dot{V}_E/\dot{V}_{CO_2}$ slope was linear in all patients and before balloon dilatation of the mitral valve it correlated inversely with peak minute oxygen consumption ($\dot{V}_{O_2}$) ($r_s = -0.47$, $P < 0.05$), exercise duration ($r_s = -0.66$, $P < 0.01$), and mitral valve area ($r_s = -0.5$, $P < 0.05$). The $\dot{V}_E/\dot{V}_{CO_2}$ slope declined acutely after balloon dilatation of the mitral valve ($n = 10$) (mean (SD) 41 (4) *v* 36 (2.9), $P < 0.05$) and did not change again thereafter. At 10 weeks ($n = 23$) exercise duration (460 (230) *v* 630 (240) s, $P < 0.01$) and peak $\dot{V}_{O_2}$ (12.7 (4.3) *v* 14.9 (4.8) ml/kg/min, $P < 0.05$) increased significantly.

Conclusions—Patients with rheumatic mitral stenosis have a similar increase in the $\dot{V}_E/\dot{V}_{CO_2}$ slope to that of patients with heart failure from other causes. Successful balloon dilatation of the mitral valve is associated with an acute reduction in the exercise $\dot{V}_E/\dot{V}_{CO_2}$ slope.

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Keywords: balloon dilatation of the mitral valve; exercise ventilation; chronic heart failure; mitral stenosis

The haemodynamic changes that occur during exercise in patients with mitral stenosis are well documented¹ but the causes of exertional hyperpnoea have never been fully elucidated. Possible causes including enhanced central respiratory drive, reduced lung compliance, and pulmonary congestion leading to stimula-

tion of juxtapulmonary capillary receptors have been suggested.²

The ventilatory response to exercise is abnormal in chronic heart failure (CHF) of all causes.³ There is an excess ventilatory cost of carbon dioxide (CO_2) production and this is reflected by an increase in the slope of the linear relation between CO_2 production and ventilation.^{4,5} This increase in slope is related directly to exercise capacity at levels of peak minute oxygen consumption ($\dot{V}_{O_2}$) < 20 ml/kg/min⁴ and reflects increased physiological dead space ventilation.

Patients with mitral stenosis have increased ventilation of physiological dead space at rest.⁶ Unlike conventional surgical treatment for mitral stenosis, percutaneous balloon dilatation of the mitral valve allows assessment of exercise dynamics immediately after palliation of valve disease, without confounding postoperative changes. We have investigated hyperpnoea in mitral stenosis by observing the time course of changes in cardiorespiratory function after balloon dilatation of the mitral valve and their temporal relation to improvement in exercise haemodynamics.

Patients and methods

PATIENTS

The study population consisted of 23 patients (22 women; mean (SD) (range) age (58 (10) (44-74) years) with rheumatic mitral stenosis undergoing routine balloon dilatation of the mitral valve. Two patients were in New York Heart Association class II for heart failure, 19 in class III, and two in class IV. All patients had normal coronary arteries and a left ventricular ejection fraction $> 40\%$ on angiography (normal range 55-70%). Patients with aortic valve disease, evidence of bronchopulmonary disease, or other serious medical conditions were excluded. Medication was continued unchanged throughout the study and is summarised in table 1.

STUDY PROTOCOL

Consecutive patients ($n = 23$) performed maximal exercise testing 24 h before balloon dilatation of the mitral valve and 10 (range 9-15) weeks after the procedure. Table 1 gives patient details. Ten consecutive unselected patients (nine women; mean (SD) (range) age 60 (10) (45-70) years) also performed exercise tests at 24 h and 10 days after the procedure. Exercise testing at 24 h was submaximal and limited to 75% of the

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Table 1 Patient population

Case	Sex	Age (years)	Rhythm	Treatment	MVA		PAP (mm Hg)
					Before (cm ²)	Immediately after (cm ²)	
1	F	63	AF	W,D,F	1.02	1.29	46
2	F	46	SR	F	0.9	1.5	40
3	F	44	SR	W,F,A	1.3	2.09	44
4	F	45	AF	W,D,F	1.08	1.75	45
5	F	58	AF	W,D,F	0.7	1.3	55
6	F	58	SR	F	1.17	1.73	30
7	F	55	AF	W,D,F	1.4	2.2	45
8	F	53	AF	D,F	0.95	1.63	57
9	F	52	AF	D,F	0.8	1.8	50
10	F	74	AF	W,D,F	0.85	1.4	30
11	F	54	AF	W,D	1.17	1.6	38
12*	F	70	AF	W,D,F	0.83	1.07	51
13*	F	67	AF	W,D,F	0.8	1.34	40
14*	M	60	AF	W,D,F	0.6	1.3	70
15	F	71	AF	W,D,F	0.7	0.8	46
16	F	60	SR	W,F	1.08	1.08	70
17*	F	70	AF	W,D,F	0.73	1.05	50
18*	F	45	AF	W,D,F	0.81	1.58	57
19*	F	46	SR	—	0.92	1.13	46
20*	F	55	AF	W,D,F	0.7	1.2	36
21*	F	61	AF	W,D,F	0.65	1	26
22*	F	72	AF	W,D,F	0.64	0.64	50
23*	F	58	SR	F,A	0.9	1.2	44

*Patients exercised at 24 h, 10 days, and 10 weeks after balloon dilatation of the mitral valve. AF, atrial fibrillation; SR, sinus rhythm; W, warfarin; D, digoxin; F, frusemide; A, angiotensin converting enzyme inhibitor; MVA, mitral valve area (Gorlin); PAP, systolic pulmonary artery pressure measured before balloon dilatation of the mitral valve.

maximum workload, before balloon dilatation of the mitral valve, in view of recent femoral arterial cannulation. All participants gave informed consent.

EXERCISE TESTING

After familiarisation patients performed progressive symptom limited exercise testing using the Weber *et al* protocol.⁷ Expired respiratory gases were collected and passed to a mass spectrometer (Airspec 2000; Airspec, Bromley, Kent). Argon dilution was used to give on line measurement of minute ventilation ($\dot{V}_E$), minute CO₂ production ($\dot{V}_{CO_2}$), and $\dot{V}O_2$. Peak $\dot{V}O_2$ was determined from the mean $\dot{V}O_2$ over the final 30 s of exercise. The relation during exercise between $\dot{V}_E$ and $\dot{V}_{CO_2}$ (plotted every 15 s) was analysed by linear regression.

PERCUTANEOUS BALLOON DILATATION OF THE MITRAL VALVE

Cardiac output was measured using the Fick method and mitral valve area was calculated using the Gorlin formula. Balloon dilatation of the mitral valve was performed using the Inoue *et al* technique⁸ without complication. Table 1 presents the changes in mitral valve area. Measurements after balloon dilatation of the mitral valve were repeated with the balloon catheter in place across the atrial septum. This avoided the possibility of a spurious increase in mitral valve area induced by the small left to right shunt.

STATISTICAL ANALYSIS

Data are reported as group mean (SD). The significance of differences was tested using non-parametric statistical analysis (Wilcoxon signed rank for paired data and Mann-Whitney U for unpaired data). Correlation's were examined using the Spearman's rank test (r_s). A p value <0.05 was considered significant.

Results

EXERCISE TESTING BEFORE BALLOON DILATATION OF THE MITRAL VALVE

The limiting symptom in the 23 patients was breathlessness. Figure 1 shows a representative example of the exercise relation between $\dot{V}_E$ and $\dot{V}_{CO_2}$. The $\dot{V}_E/\dot{V}_{CO_2}$ slope was linear in all patients ($r > 0.92$ in every test) and, as in chronic heart failure from other causes, it correlated inversely with peak $\dot{V}O_2$ ml/kg/min (fig 2, $r_s = -0.47$, $P < 0.05$). The $\dot{V}_E/\dot{V}_{CO_2}$ slope also correlated inversely with exercise duration (fig 3, $r_s = -0.66$, $P < 0.01$) and with mitral valve area measured before balloon dilatation of the mitral valve (fig 4, $r_s = -0.5$, $P < 0.05$). There was no relation between $\dot{V}_E/\dot{V}_{CO_2}$ slope and pulmonary artery systolic pressure.

EXERCISE TESTING AFTER BALLOON DILATATION OF THE MITRAL VALVE

Submaximal exercise testing at 24 h ($n = 10$) was performed without complication. The

Figure 1 Representative example of a patient's $\dot{V}_E/\dot{V}_{CO_2}$ slope relation before and 24 h after balloon dilatation of the mitral valve (PMV).

Figure 2 $\dot{V}_E/\dot{V}_{CO_2}$ slope plotted as a function of peak $\dot{V}O_2$ in 23 patients with mitral stenosis before balloon dilatation of the mitral valve.

Figure 3 $\dot{V}_E/\dot{V}_{CO_2}$ slope plotted as a function of exercise duration in 23 patients with mitral stenosis before balloon dilatation of the mitral valve.

Figure 4 $\dot{V}_E/\dot{V}_{CO_2}$ slope plotted as a function of mitral valve area in 23 patients with mitral stenosis before balloon dilatation of the mitral valve.

$\dot{V}_E/\dot{V}_{CO_2}$ slope, peak $\dot{V}O_2$, and exercise duration were significantly lower after balloon dilatation of the mitral valve (table 2). Maximal exercise testing at 10 days documented an increase in exercise duration and peak $\dot{V}O_2$ but no further change in the $\dot{V}_E/\dot{V}_{CO_2}$ slope. The respiratory exchange ratio at maximum exercise was > 1.0 in all patients. Figure 5 shows the change in $\dot{V}_E/\dot{V}_{CO_2}$ slope after balloon dilatation of the mitral valve.

Twenty of 23 patients reported an improvement in their symptoms of exertional dyspnoea 10 weeks after balloon dilatation of the mitral valve. Repeat exercise testing in 23 patients demonstrated significant increases in exercise duration (from 460 (230) to 630 (240) s, $P < 0.01$) and peak $\dot{V}O_2$ (12.7 (4.3) v 14.9 (4.8) ml/kg/min, $P < 0.05$) compared with the baseline before balloon dilatation of the mitral valve. The $\dot{V}_E/\dot{V}_{CO_2}$ slope was significantly reduced (from 38 (7) to 34 (9), $P < 0.01$), but remained inversely correlated with peak $\dot{V}O_2$ ($r_s = -0.62$, $P < 0.01$). A weak non-significant correlation existed between absolute change in valve area and reduction in $\dot{V}_E/\dot{V}_{CO_2}$ slope.

There were no significant differences in the change in heart rate with exercise (53 (5) v 55 (5) beats/min), change in systolic blood pressure with exercise (20 (4) v 17 (3) mmHg), or respiratory exchange ratio at maximum exercise (1.02 (0.1) v 1.05 (0.01)).

Balloon dilatation of the mitral valve increased the mitral valve area < 0.25 cm² in four patients (nos 12, 15, 16, and 22). Repeat exercise testing at 10 weeks in these patients demonstrated no change in $\dot{V}_E/\dot{V}_{CO_2}$ slope (before: 47 (8); after: 47 (12)) and a small non-significant increase in exercise duration (before: 250 (90); after: 300 (70) s).

Discussion

We have demonstrated that patients with rheumatic mitral stenosis have an increased

Figure 5 Time course of the mean (SD) change in $\dot{V}_E/\dot{V}_{CO_2}$ slope after balloon dilatation of the mitral valve ($n = 10$). * $P < 0.01$.

ventilatory response to exercise. This is indicated by an increase in the slope of the linear relation between ventilation ($\dot{V}_E$) and $\dot{V}O_2$ during exercise. As with CHF from other causes, the slope of this relation in mitral stenosis is related directly to exercise capacity expressed as peak $\dot{V}O_2$. Improved exercise duration after balloon dilatation of the mitral valve was associated with an increase in peak $\dot{V}O_2$ and a decrease in the exercise $\dot{V}_E/\dot{V}_{CO_2}$ slope. The change in slope occurred within 24 h of balloon dilatation and did not change again thereafter.

The exercise $\dot{V}_E/\dot{V}_{CO_2}$ slope is increased in CHF of all causes.³ It reflects increased dead space ventilation which comprises anatomical dead space, physiological dead space, and apparatus dead space. In normal individuals the ratio of total ventilation to dead space ventilation decreases during exercise as tidal volume increases. When lung compliance is reduced by pulmonary congestion, tidal volumes are smaller and the ratio of total ventilation to dead space ventilation is increased.¹⁰ This increased anatomical dead space ventilation in patients with CHF accounts for only one third of the increased $\dot{V}_E/\dot{V}_{CO_2}$ slope.⁵ The remainder reflects increased physiological dead space ventilation secondary to mismatching between ventilation and perfusion during exercise.⁵ As the $\dot{V}_E/\dot{V}_{CO_2}$ slope and peak $\dot{V}O_2$, are similarly related in patients with CHF and in those with mitral stenosis, the increased exercise ventilation in mitral stenosis probably reflects mismatching between ventilation and perfusion.

The distribution of resting lung ventilation and perfusion in patients with mitral stenosis has been studied extensively. There is an increase in relative perfusion to the apex of the lung^{11 12} and a reduction in ventilation of the lung bases.^{11 13} These abnormalities are exaggerated after exercise.¹⁴ The resulting resting ventilation/perfusion ratios which are normally high in the lung apices and low in the bases are reversed, with a relatively uniform distribution of ventilation/perfusion ratio throughout the lung. This pattern of ventilation/perfusion abnormality is similar to patients with CHF from other causes.¹⁵

Static lung function may be abnormal in patients with mitral stenosis. The abnormalities are usually non-specific but include reductions in vital capacity and dynamic lung compliance, peripheral airway

Table 2 Cardiorespiratory data obtained before and after balloon dilatation of the mitral valve ($n = 10$)

	Before balloon dilatation of the mitral valve	After balloon dilatation of the mitral valve		
		24 h	10 days	10 weeks
Exercise duration (s)	380 (200)	355 (210)	543 (208)*	685 (256)*
Peak $\dot{V}O_2$ (ml/kg/min)	12 (3.5)	12 (3.7)	15 (5.5)	15 (4.1)†
$\dot{V}_E/\dot{V}_{CO_2}$	41 (4)	36 (2.9)*	36 (3)*	34 (2.9)*
RER _{max}	1.08 (0.1)	0.96 (0.1)	1 (0.1)	1.08 (0.1)

Values are mean (SD). * $P < 0.01$ change from baseline; † $P < 0.05$ change from baseline.; Peak $\dot{V}O_2$, oxygen consumption at peak exercise; RER_{max}, respiratory exchange in ratio at peak exercise; $\dot{V}_E/\dot{V}_{CO_2}$, $\dot{V}_E/\dot{V}_{CO_2}$ slope (laboratory normal range 21–31°).

obstruction, and reduced pulmonary diffusion capacity.¹⁶

Improvement in the $\dot{V}_E/\dot{V}_{CO_2}$ slope could reflect a primary improvement in lung function. However, abnormalities in the alveolar/capillary membrane are not readily reversed and increases in vital capacity after balloon dilatation of the mitral valve do not correspond to the decrease in mean pulmonary artery pressure.¹⁶ These data and the rapid change we have observed in the $\dot{V}_E/\dot{V}_{CO_2}$ slope suggest that abnormality of the slope in mitral stenosis reflects impairment of the haemodynamic response to exercise and is not a feature of long standing pulmonary vascular change or a reflection of primary improvement in lung function.

In healthy individuals and patients with mild CHF and a peak $\dot{V}O_2 > 20$ ml/kg/min there is no relation between the $\dot{V}_E/\dot{V}_{CO_2}$ slope and peak $\dot{V}O_2$.⁴ This study demonstrates a persistence of the inverse relation between peak $\dot{V}O_2$ and the $\dot{V}_E/\dot{V}_{CO_2}$ slope after balloon dilatation of the mitral valve. Reductions in pulmonary capillary wedge pressure, pulmonary artery pressure, and mitral valve gradient after balloon dilatation of the mitral valve are well recognised.¹⁷ These changes are associated with increases in exercise cardiac output, increased exercise duration, and improved symptomatic status at follow up.¹⁷ Mitral flow dynamics¹⁸ and right ventricular function¹⁷ also improve rapidly after successful balloon dilatation of the mitral valve but not to normal. We postulate that continuing abnormalities in right ventricular function during exercise and residual valvar obstruction despite balloon dilatation of the mitral valve account for the persistence of the relation between peak $\dot{V}O_2$ and $\dot{V}_E/\dot{V}_{CO_2}$ slope.

We conclude that patients with rheumatic mitral stenosis have a similar increase in the $\dot{V}_E/\dot{V}_{CO_2}$ slope as patients with heart failure from other causes and that this slope is directly related to exercise capacity. Successful balloon valvotomy is associated with an acute reduction in exercise $\dot{V}_E/\dot{V}_{CO_2}$ slope and this may reflect increased ventilation/perfusion matching secondary to improved exercise haemodynamics.

STUDY LIMITATIONS

Measurement of arterial partial pressure of CO_2 and exercise cardiac output are necessary

to formally elucidate the mechanisms responsible for our observations. These measurements were not performed in this study as ethical approval was not available for invasive arterial monitoring.

APB is a British Heart Foundation junior research fellow.

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before any clinical component of their course; recent recommendations that all applicants to medical school should be screened, and immunised if appropriate, have far-reaching implications and have provoked controversy.^{17 18}

In conclusion, among invasive cardiologists in the United Kingdom there is clear scope for improvement in vaccine uptake, particularly in completion of the immunisation regimen. Screening of patients, as recommended, should also be performed. Careful practice to avoid needle stick injury should continue and immunisation should also be offered to nursing and other ancillary staff caring for patients undergoing invasive cardiac procedures.

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CORRECTIONS

Failure of "effective" treatment for heart failure to improve normal customary activity. *J T Walsh, R Andrews, A Evans, A J Cowley (Br Heart J 1995;74: 373-6).*

Exercise ventilation after balloon dilatation of the mitral valve. *A P Banning, N P Lewis, J S Elborn, R J C Hall (Br Heart J 1995;74:386-9).*

We regret that the incorrect volume number was given in the strap lines and at the foot of the abstracts of these papers. The correct volume number is 74.

Permanent pacemaker practice at a Scottish district general hospital between 1987 and 1994. *J G Doherty, F Dawson, F Kerr*

We regret that an error appeared in the second sentence of the discussion on page 477 of this article (*Br Heart J* 1995;73:475-8) which should have read, "Our average implantation rate over the 79 months (130/million population/year) compares with the United Kingdom national 1989 value of 148/million population/year."⁵

NOTICES

The 1996 Annual General Meeting of the **British Cardiac Society** will take place at the Scottish Exhibition & Conference Centre, Glasgow from 7 to 9 May.

The **Fourth Annual Molecular Symposium at UCL Medical School on Ischaemic Preconditioning and Adaptation to Ischaemia** will take place on 12 December 1995 in London. For further information please contact Jan Wenley, Symposium Administrator, Department of Molecular Pathology, 46 Cleveland Street, London W1P 6DB (tel: +44 171 380 9343; fax: +44 171 387 3310).