

Effects of mitral valve surgery on static lung function and exercise performance

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ABSTRACT Lung function at rest was assessed in 50 patients before and six months after mitral valve surgery. There were small increases in spirometric volumes (FEV₁ and vital capacity) with decreases in total lung capacity and residual volume, but no change in carbon monoxide transfer factor or transfer coefficient (KCO). Progressive exercise tests performed before and after operation in 19 of the patients confirmed an improved exercise capacity after surgery. The patients with the greatest symptomatic improvement in breathlessness were also those who achieved the greatest increase in maximum work load and the greatest decrease in ventilation for a given oxygen consumption. Depression of the ST segment of the electrocardiogram and frequent ventricular ectopic beats on exercise remained common after surgery and may have been due to digoxin treatment.

Mitral valve disease produces well recognised abnormalities of lung function and a major symptom is effort intolerance. Studies comparing respiratory function before and after surgery have often failed to show any change but the numbers of patients studied have usually been small.¹⁻⁷ There is little information on preoperative and postoperative exercise performance: Gilmour *et al*⁸ in five patients undergoing mitral valve replacement found that subjective improvement was not matched by objective improvement in exercise performance. Reed *et al*⁹ studied 20 patients before and after mitral valvotomy. Twelve patients showed clinical improvement and in these the heart rate and ventilation at a standard oxygen uptake were lower after surgery. The tidal volume during exercise increased, despite a fall in vital capacity; the latter was attributed to the thoracotomy.

The factors determining exercise intolerance in patients with mitral valve disease are likely to be multiple and to include both a low cardiac output and an abnormal drive to breathe resulting from stimulation by pulmonary congestion or by lactic acid production from inadequately perfused muscles; these effects will be compounded by any inefficiency of pulmonary function resulting from reduced compliance, increased airways resistance, or inefficient gas exchange.

Many patients with mitral valve disease have atrial fibrillation so that some postoperative improvement might result from reversion to a normal cardiac rhythm. Failure of the systemic blood pressure to rise on exercise is a well recognised association of ischaemic heart disease¹⁰ and this also might contribute to effort intolerance; there is, however, no evidence on blood pressure changes during exercise in patients with mitral disease or on how the response may be affected by surgical correction.

The present study was designed to re-examine in a large group of patients the effects of mitral valve surgery on standard tests of pulmonary function. In some patients the ventilatory and cardiovascular responses to progressive exercise before and after operation were also studied and the relationships between these changes and the patients' symptoms were examined.

Patients and methods

Fifty seven patients undergoing surgery for mitral valve disease were studied. Of these, seven were subsequently excluded from analysis for the following reasons: three failed to attend for postoperative assessment, one suffered a pulmonary embolus postoperatively, one developed a bronchial carcinoma during the follow up period, and two

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patients were clearly asthmatic. The remaining 50 patients comprised 40 women and 10 men with a mean age of 51 (range 35–65) years. Six patients also had aortic valve disease but in only one was this severe enough to warrant simultaneous aortic valve replacement. In 15 patients mitral valvotomy was performed and the remaining 35 underwent mitral valve replacement. Twenty patients were effectively non-smokers as they had smoked less than five pack years; 21 were smokers, and nine ex-smokers at the time of preoperative assessment.

The patients were studied shortly before operation (median three days preoperatively) and about six months after operation (median 182 days). At the time of postoperative assessment 10 of the 21 cigarette smokers claimed to have reduced their cigarette consumption and two to have stopped smoking. At each visit the patients were questioned about respiratory symptoms and the degree of dyspnoea, classified according to the following grades: 0—not short of breath; 1—short of breath only while hurrying on the level or walking uphill; 2—short of breath while walking with others at an ordinary pace on the level; 3—short of breath while walking at own pace on the level; 4—short of breath while dressing or during minimal exertion.

From a standard posteroanterior chest radiograph at each visit the cardiothoracic ratio was measured. The following pulmonary function tests were performed before and after surgery: FEV₁ and vital capacity (VC) with a bellows spirometer (Vitalograph); single breath carbon monoxide transfer factor (TLCO),¹¹ on the basis of the simultaneously measured single breath alveolar volume (VA), and transfer coefficient (KCO) (in 49 patients); total lung capacity (TLC) and residual volume (RV) in a constant volume body plethysmograph¹² (in 37 patients). The TLCO and KCO were corrected to a haemoglobin concentration of 14.6 g/dl.¹³ All the values recorded were expressed as percentages of predicted values.^{14–18}

Nineteen of the patients performed a progressive exercise test before and after operation. These patients were chosen at random from the study group before operation and did not differ in any important respect from the group as a whole. Exercise was performed on a cycle ergometer (Lode) with the patient breathing room air. The power output was increased by 10 w each minute until the patient could not exercise further. Expired ventilation (\dot{V}_E l min⁻¹ BTPS) was obtained by integration of air flow at the mouth measured by a Fleisch pneumotachograph and mixed expired gas was sampled at the distal end of a mixing chamber and analysed by a mass spectrometer (Centronic MGA 200). The outputs of mass spectrometer and integrator

were connected to a microprocessor (PK Morgan) that calculated \dot{V}_E and oxygen uptake ($\dot{V}O_2$ l min⁻¹ STPD); the second half minute at each work load was used for analysis of the results. The electrocardiogram (ECG) was continuously monitored and recorded on a Mingograf recorder with a modified CL5 electrode position. From the ECG record heart rate was measured over the second half of each minute of exercise; in 12 patients satisfactory preoperative and postoperative readings of blood pressure were obtained each minute.

Ventilation and heart rate were plotted against oxygen consumption and work load and the slopes of the relationships were determined by linear regression. Ventilation and heart rate were also recorded by interpolation at standard $\dot{V}O_2$ levels of 0.5 and 1.0 l min⁻¹ ($\dot{V}E_{0.5}$, HR_{0.5}, etc). Tidal volume (VT) was related to ventilation and values at a standard \dot{V}_E of 30 l min⁻¹ were interpolated (VT₃₀).

Preoperative and postoperative data were compared by paired *t* tests. Relationships between changes in lung volume (expressed as percentages of predicted values) and changes in cardiothoracic ratio were examined by linear correlation. Differences between groups of patients were sought by analysis of variance.

Results

Mean values for dyspnoea grade, cardiothoracic ratio, and results of static lung function tests before and after operation are shown in table 1. The mean dyspnoea grade was, as expected, lower after surgery. There were significant increases in FEV₁ and VC and reductions in TLC and RV. No changes were found in the FEV₁/VC ratio, TLCO, or KCO. Consideration of smokers and non-smokers separately did not affect these conclusions. Relating the change in FEV₁ to the preoperative value showed a negative correlation ($r = -0.40$, $p < 0.005$). Although there was no overall change in cardiothoracic ratio, inverse correlations were found between the change in cardiothoracic ratio and change in both vital capacity ($r = -0.43$, $p < 0.005$) and total lung capacity ($r = -0.41$, $p < 0.005$).

The exercise data are summarised in table 2. After operation the patients were able to achieve a significantly higher work load and oxygen consumption; the maximum ventilation was correspondingly greater but the maximum heart rate was similar before and after operation. The patients were asked to exercise until they felt unable for any reason to continue; in most cases they stopped because of shortness of breath and in no case was the exercise test terminated by the observer. The reasons for stopping were usually the same in an

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Table 1 Dyspnoea grade, cardiothoracic ratio, and static lung function

	No of patients	Preoperative	Postoperative	p
		Mean (SEM)	Mean (SEM)	
Dyspnoea grade	50	3.0 (0.09)	1.28 (0.19)	<0.001
Cardiothoracic ratio (%)	50	56.7 (0.42)	55.9 (0.39)	NS
FEV ₁ (% predicted)	50	74.3 (2.67)	78.5 (2.52)	<0.02
Vital capacity (VC) (% predicted)	50	78.8 (1.98)	83.4 (2.13)	<0.01
FEV ₁ /VC (% predicted)	50	93.3 (1.55)	93.2 (1.29)	NS
Carbon monoxide transfer factor (% predicted)	49	64.4 (1.95)	62.9 (1.90)	NS
Kco (% predicted)	49	86.2 (2.43)	83.5 (2.38)	NS
Total lung capacity (% predicted)	37	110.0 (3.10)	101.0 (1.85)	<0.01
Residual volume (% predicted)	37	167.0 (8.50)	135.0 (5.10)	<0.001

NS—not significant.

Table 2 Exercise data in 19 patients undergoing mitral valve surgery

	Preoperative Mean (SEM)	Postoperative Mean (SEM)	p
Maximum WL (w)	43.2 (4.9)	64.2 (5.7)	<0.001
Maximum $\dot{V}O_2$ (l (STPD) min ⁻¹)	0.885 (0.059)	1.07 (0.082)	<0.01
Maximum $\dot{V}E$ (l (BTPS) min ⁻¹)	36.0 (2.3)	42.4 (2.7)	<0.01
Maximum HR (min ⁻¹)	150.0 (5.9)	154.0 (5.5)	NS
$\dot{V}E/\dot{V}O_2$ slope	41.3 (3.6)	40.4 (3.3)	NS
$\dot{V}E/WL$ slope	0.54 (0.05)	0.49 (0.03)	NS
HR/ $\dot{V}O_2$ slope	116 (14.9)	82.7 (7.4)	<0.02
HR/WL slope	1.52 (0.21)	0.98 (0.08)	<0.01
$\dot{V}E_{0.5}$ (l BTPS min ⁻¹)	19.7 (1.1)	17.8 (1.3)	NS
HR _{0.5} (min ⁻¹)	112 (5.1)	110 (4.7)	NS
V_{T30} (l)	1.01 (0.035)	1.15 (0.059)	<0.05

WL—work load; $\dot{V}O_2$ —oxygen consumption; $\dot{V}E$ —minute ventilation; HR—heart rate; $\dot{V}E_{0.5}$, HR_{0.5}—ventilation, heart rate at $\dot{V}O_2=0.5$ l (STPD) min⁻¹; V_{T30} —tidal volume at $\dot{V}E=30$ l (BTPS) min⁻¹; NS—not significant.

individual before and after surgery. The slopes of the relationships between ventilation and oxygen consumption and between ventilation and work load were similar before and after surgery, but the corresponding slopes of heart rate responses were significantly lower after surgery. Only seven patients achieved a $\dot{V}O_2$ of 1 l min⁻¹ both before and after operation and no significant differences were found in the submaximal indices $\dot{V}E_{0.5}$ and HR_{0.5}. The tidal volume at a standard ventilation of 30 l min⁻¹ was, however, significantly greater after surgery. Thus the more normal cardiac response to exercise after surgery apparently allowed the patients to perform more work and hence they achieved a greater maximum ventilation.

The relationships between change in dyspnoea grade and exercise performance are examined in table 3. Of the 19 patients who exercised, only four had no symptomatic improvement and the remainder improved by either one or two grades. Although the numbers are small, analysis of variance showed that patients whose symptoms improved more also had greater objective increases in exercise capacity and in maximum heart rate during exercise as well as larger decreases in ventilation for a given oxygen consumption.

Table 4 shows the haemodynamic and electrocar-

diographic data. The resting heart rate and systolic blood pressure were similar before and after operation. Apart from atrial fibrillation (which developed postoperatively in one patient) there were no significant arrhythmias at rest and no change after surgery.

In most patients the blood pressure rose on exercise and the highest systolic value was significantly greater after operation. Exercise did not alter the basic cardiac rhythm, which was either sinus rhythm or atrial fibrillation in all cases. Heart rates were faster on exercise in patients with atrial fibrillation, but the maximum values were not significantly different after operation whatever the underlying rhythm. Sixteen patients were taking digoxin before operation and 15 afterwards. ST segment changes on exercise were common but were attributable to digoxin in most cases. Ventricular ectopics and "serious" arrhythmias (defined as more than five ventricular ectopic beats a minute) were common during exercise and their frequency was unaffected by surgery.

Discussion

STATIC TESTS

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Table 3 Relationships between changes in dyspnoea grade and changes in exercise function in 19 patients

Change in dyspnoea grade	0	-1	-2	p*
No of patients	4	8	7	
Mean change in:				
Maximum WL (w)	2.5	16.2	37.1	<0.02
Maximum $\dot{V}O_2$ (l (STPD) min^{-1})	-0.023	0.184	0.311	<0.05
Maximum $\dot{V}E$ (l (BTPS) min^{-1})	7.9	5.8	6.1	NS
Maximum HR (min^{-1})	-27.5	11.3	14.0	-0.05
$\dot{V}E/\dot{V}O_2$ slope	16.2	-0.5	-11.3	<0.005
HR/ $\dot{V}O_2$ slope	-15.7	-13.6	-67.9	NS
$\dot{V}E_{0.5}$ (l BTPS min^{-1})	5.1	-1.6	-6.3	<0.01
HR _{0.5} (min^{-1})	-15.8	10.9	-9.4	NS
VT ₃₀ (l)	-0.041	0.201	0.166	NS

*The p value refers to differences between groups based on analysis of variance. Abbreviations as in table 2.

Table 4 Haemodynamic and electrocardiographic data at rest and during exercise in 19 patients

	Preoperative Mean (SD)	Postoperative Mean (SD)	P
REST			
Heart rate (min^{-1})	83.4 (15.1)	90.6 (20.3)	NS
Systolic blood pressure (mm Hg) (n=12)	124 (21)	136 (27)	NS
Rhythm			
Sinus rhythm	6	5	
Atrial fibrillation	13	14	
ST depression*	3	8	
Arrhythmias†	0	0	
EXERCISE			
Maximum heart rate			
Patients with sinus rhythm	139 (30)	139 (13)	NS
Patients with atrial fibrillation	155 (23)	158 (24)	NS
Maximum systolic blood pressure (mm Hg) (n=12)	153 (32)	191 (33)	<0.01
ST depression* [patients taking digoxin]	13 [12]	12 [10]	
Arrhythmias† [patients taking digoxin]	10 [7]	8 [8]	

*Depression by 1 mV 0.08 second after J point.

†More than five ventricular ectopics per minute.

function are well known and include a reduction in spirometric volumes and CO transfer factor with an increase in residual volume.¹⁹ The present study has shown that some of these abnormalities improve after surgery. In particular there were increases in FEV₁ and VC and a reduction in RV. A small mean reduction was also seen in total lung capacity six months after operation, but there were no changes in TLCO or KCO. The postoperative values in general continued to show the same qualitative abnormalities and some of the changes are likely to be not completely reversible.

The negative correlation between preoperative FEV₁ and the increase in FEV₁ found postoperatively shows that a low FEV₁ value per se need not imply a poor functional outcome. On the basis of the criteria previously suggested¹⁹ it is, however, probable that few of our patients had significant primary airway disease since FEV₁/VC was below 80% of the predicted value in only five of 50 patients and TLC exceeded 120% predicted in only one of 37; the patients described here were not preselected for

the purpose of the study and are probably representative of those undergoing mitral valve surgery in this unit. Other patients with clinical or functional evidence of more severe airway disease may, however, have been excluded from consideration of surgery during the period of this study.

Most previous studies, usually with smaller numbers of patients, have failed to show conclusive changes in static lung function after mitral valve surgery.¹⁻⁷ There may over the years have been a tendency for surgeons to operate at an earlier stage of the disease, but only seven of our 50 patients had preoperative dyspnoea graded less than 3—that is, the great majority were breathless even when walking slowly on the level. In earlier studies the operation performed was usually mitral valvotomy via left thoracotomy, rather than valve replacement via median sternotomy, but the changes seen in the 15 patients in the present series who underwent valvotomy were not different from those in the total group. The mean change in VC in all 50 patients was 189 ml (SD 42 ml) and the negative correlation

between change in vital capacity and in cardiothoracic ratio suggests that some of the post-operative improvement in VC (and therefore also in FEV₁) may be related directly to the reduction in cardiac size; relief of pulmonary congestion was presumably also relevant in some individuals. The latter mechanism may also explain the fall in residual volume, which measured by plethysmography is virtually always increased in patients with significant mitral valve disease¹⁹ and probably results from pulmonary vascular engorgement.²⁰ The fall in total lung capacity was less expected but might represent a residual effect of thoracotomy; if this were still relevant six months post operation it might also tend to counter any improvement in vital capacity, and a larger increase in VC might have been seen if the patients had been studied after a longer interval after surgery.

The failure of TLCO and KCO to increase is disappointing and suggests that the effects of mitral valve disease on small pulmonary vessels and on the alveolar capillary membrane may have been no longer reversible in the patients we studied. We did not, however, measure the membrane (Dm) and pulmonary capillary volume (Vc) components of the transfer factor, and it is theoretically possible that relief of pulmonary vascular engorgement with a corresponding fall in Vc could have masked a postoperative rise in Dm.

EXERCISE RESULTS

The exercise study confirmed that most patients were capable of more exercise after surgery. Those patients who reported the greatest improvement in dyspnoea tended to show the greatest objective changes during exercise. Surgical treatment seems likely to improve stroke volume, thus allowing a lower heart rate for a given cardiac output and hence an increase in the capacity to perform exercise. Reed *et al*⁹ showed a reduction in ventilation during exercise at a standard oxygen consumption. Exercise ventilation in our patients was also on average lower after operation and, although the difference was not significant for the group as a whole, it was most obvious in those with the greatest improvement in symptoms (table 3). We confirmed the finding⁹ of an increased tidal volume at a standard ventilation during exercise.

The greater maximum systolic blood pressure after operation also suggests an improvement in stroke volume and therefore in cardiac output as the main mechanism for the improvement in exercise capacity. All the patients who had atrial fibrillation before operation remained in atrial fibrillation and one other patient developed atrial fibrillation post-operatively. The maximum heart rate was higher in

the patients with atrial fibrillation than in those with sinus rhythm; but this theoretical disadvantage did not appear to affect postoperative exercise capacity, which was similar in patients in sinus rhythm and in atrial fibrillation. There was therefore no evidence that alteration in cardiac rhythm postoperatively contributed to improved exercise performance. ST segment depression during exercise was common. Coronary angiograms had been performed in seven of the 19 patients who had exercise tests; all showed normal coronary arteries but five of the seven showed ST depression during exercise. Coronary artery disease is therefore unlikely to be responsible and digoxin was probably the cause in most of the patients. The number of patients with "serious" arrhythmias during exercise fell only slightly after operation, from 10 to eight; postoperatively such arrhythmias were seen only in patients taking digoxin and therefore they also may be related to therapy.

This study has demonstrated significant improvement in both static lung function and exercise performance as a result of mitral valve surgery. Many of the abnormalities of lung function related to mitral valve disease are likely to be at best only partly reversible. Electrocardiographic abnormalities during exercise remained frequent after apparently successful surgery and may be the result of digoxin treatment.

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