A T Marcel Gosselink, Harry J G M Crijns, Maarten P Van Den Berg, Stan A J Van Den Broek, Hans Hillege, Martin L J Landsman, Kong I Lie

Abstract

Objective—To evaluate the effect of cardioversion on peak oxygen consumption (peak VO_2) in patients with long-standing atrial fibrillation, to assess the importance of underlying heart disease with respect to the response to exercise, and to relate functional capacity to long-term arrhythmia outcome.

Design—Prospective controlled clinical trial.

Setting—Tertiary referral centre.

Patients—63 consecutive patients with chronic atrial fibrillation accepted for treatment with electrical cardioversion. Before cardioversion all patients were treated with digoxin, verapamil, or a combination of both to attain a resting heart rate ≤ 100 beats per minute.

Interventions—Electrical cardioversion. Main outcome measures—Peak VO_2 measured before and 1 month after electrical cardioversion to compare patients who were in sinus rhythm and those in atrial fibrillation at these times. Maintenance of sinus rhythm for a mean follow up of 19 (7) months.

Results—Mean (1SD) peak Vo_2 in patients in sinus rhythm after 1 month (n = 37) increased from 21.4 (5.8) to 23.7 (6.4) ml/min/kg (+11%, P < 0.05), whereas in patients with a recurrence of atrial fibrillation 1 month after cardioversion (n = 26) peak Vo_2 was unchanged. In patients who were in sinus rhythm both those with and without underlying heart disease improved, and improvement was not related to functional capacity or left ventricular function before cardioversion. Baseline peak Vo_2 was not a predictive factor for longterm arrhythmia outcome.

Conclusion—Restoration of sinus rhythm improved peak Vo_2 in patients with atrial fibrillation, irrespective of the presence of underlying heart disease. Peak Vo_2 was not a predictive factor for long-term arrhythmia outcome after cardioversion of atrial fibrillation. These findings suggest that cardioversion is the best method of improving functional capacity in patients with atrial fibrillation, whether or not they have underlying heart disease and whatever their functional state.

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In atrial fibrillation, loss of atrial contraction and an excessive response of heart rate to exercise may be detrimental to cardiac output because they impair ventricular diastolic filling.¹⁻⁴ As a consequence, functional capacity in patients with atrial fibrillation may be reduced.5 Though cardioversion of atrial fibrillation is often performed its effect on functional status has not been extensively studied. Measurement of peak oxygen consumption (peak Vo₂) by gas exchange analysis is an accurate and objective non-invasive method of determining functional capacity.⁶⁻¹⁰ So far, there have been only three small studies of the effect of cardioversion on peak Vo₂ in atrial fibrillation.11-13

The aim of the present prospective study was to analyse the effect of cardioversion on peak VO_2 a month after cardioversion in a large cohort of patients with long-standing, chronic atrial fibrillation; patients who had a relapse of atrial fibrillation served as a control group. We also studied the importance of underlying heart disease with respect to functional capacity in atrial fibrillation and assessed the predictive value of functional capacity for long arrhythmia outcome by measuring baseline peak VO_2 .

Patients and methods

PATIENTS

All consecutive patients with chronic atrial fibrillation selected between June 1989 and February 1992 to undergo electrical cardioversion were eligible for the study. Exclusion criteria were (a) paroxysmal atrial fibrillation; (b) myocardial infarction within the past 6 months; (c) thyroid dysfunction; (d) use of β adrenergic blocking agents; and (e) angina, chronic obstructive pulmonary disease, claudication, or any other abnormality intefering with the performance of a treadmill exercise test. The study was approved by the Institutional Review Board and informed consent for participation in the study was obtained from all patients.

STUDY DESIGN

Patients underwent exercise testing a day before and 4 weeks after cardioversion. In all patients anticoagulant therapy was started to maintain prothrombin time within a target range of $1.5-2.0 \times \text{control}$ at least 4 weeks before cardioversion. Atrioventricular nodal blocking drugs (digitalis, verapamil, or both) were titrated to attain a resting heart rate

Department of Cardiology, University Hospital Groningen, The Netherlands A T M Gosselink H J G M Crijns M P Van Den Berg S A J Van Den Broek H Hillege M L J Landsman K I Lie

Correspondence to: Dr A T Marcel Gosselink, Department of Cardiology, Thoraxcentre, University Hospital Groningen, PO Box 30001, 9700 RB Groningen, The Netherlands.

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 ≤ 100 beats per minute at least 2 months before cardioversion. Twenty patients were antiarrhythmic drugs taking (Vaughan-Williams class I, 15 patients; class III, 5 patients). Drug treatment was unchanged throughout the study. Before cardioversion a standard cross sectional echocardiogram was performed in all patients to assess left atrial size and left ventricular function as measured by left ventricular fractional shortening. Cardioversion was carried out according to a previously described protocol.14 Patients visited the outpatient department 1, 3, and 6 months after cardioversion and every 6 months thereafter. At each visit a 12 lead electrocardiogram and 24 hour Holter monitoring (Marquette Laser Holter System, Milwaukee, Wisconsin, USA) were performed to determine the rhythm. Exercise data for patients who were in sinus rhythm 1 month after cardioversion were compared with those of patients who could not be cardioverted or had a relapse of atrial fibrillation 1 month after cardioversion.

EXERCISE TESTING WITH RESPIRATORY GAS ANALYSIS

Exercise testing with measurement of respiratory gas exchange was performed while patients exercised on a treadmill according to a modified Naughton protocol.^{15 16} Oxygen consumption, carbon dioxide production, and respiratory exchange ratios were measured continuously during exercise by an automated system for measuring gas exchange (Sensormedics system 2900, SensorMedics Corp, Anaheim, California, USA). Values were recorded every 20 s through an on-line

Table 1 Baseline characteristics of the 63 study patients

Variable	Value
Age (y) (mean (1SD))	57 (13)
Men (n(%))	44 (70)
Underlying heart disease (n(%))*:	
Ischaemic heart disease	14 (22)
Rheumatic valve disease	3 (5)
Mitral valve disease	15 (24)
Aortic valve disease	6 (10)
Systemic hypertension	7 (11)
Congenital heart disease	5 (8)
Chronic pericardial disease	1 (2)
Lone arrĥythmia	22 (35)
Medication (n(%)):	· · /
Digoxin only	13 (21)
Verapamil only	14 (22)
Both digoxin and verapamil	36 (57)
Antiarrhythmic drugs	20 (32)
Anticoagulants	63 (100)
Total arrhythmia duration (median (range)) (mnth)	35 (1 to 288)
NYHA class, I,II,III (n(%))	29 (46), 23 (36), 11 (18)
Echocardiographic measurements (mean (1SD)):	
Left atrial size, long axis view (mm)	44 (7)
Left ventricular end diastolic diameter (mm)	52 (7)
Left ventricular end systolic diameter (mm)	37 (8)
Left ventricular fractional shortening (%)	30 (7)
Exercise testing before cardioversion (mean (1SD)):	50 (1)
Rest	
HR (beats per minute)	93 (19)
SBP (mm Hg)	133 (20)
DBP (mm Hg)	84 (10)
Maximal exercise (mean (1SD))	01(10)
HR (beats per minute)	174 (30)
SBP (mm Hg)	171 (24)
DBP (mm Hg)	85 (12)
Exercise duration (min)	19.0 (4.3)
Peak VO ₂ (ml/min/kg)	21.3 (6.1)
% predicted Vo ₂	77 (19)

* More than one per patient; CABG, coronary artery bypass grafting; DBP, diastolic blood pressure; HR, heart rate; NYHA, New York Heart Association; SBP, systolic blood pressure; VO₂, oxygen consumption; % predicted VO₂ percentage of age and sex predicted peak oxygen consumption.

computer assembly (IBM computer systems, IBM Corp, Austin, Texas, USA). Before each exercise test, the flow meter was calibrated with 31 syringe and the gas analysers were calibrated with nitrogen and a standard oxygen/carbon dioxide mixture. Blood pressure was measured with a mercury sphygmomanometer. The electrocardiogram was monitored continuously with a computer-assisted system (Marquette Electronics, Milwaukee, Wisconsin, USA). Twelve lead electrocardiogram recordings were obtained at rest, at the end of each 2 minute stage, and at peak exercise. Before entering the study, patients underwent at least one test to familiarise themselves with this gas exchange analysis. Patients were encouraged to exercise until symptoms forced them to stop. All patients stopped the test because of dyspnoea or fatigue; and in all patients the gas exchange anaerobic threshold (the point at which carbon dioxide production increased disproportionately in relation to oxygen consumption) and a respiratory exchange ratio > 1.0 were reached. Peak VO2 was defined as oxygen consumption (ml/min/kg) at peak exercise calculated as the mean of the values measured during the last minute of exercise. Heart rate during atrial fibrillation was determined by measuring the average heart rate for a 15 s period. The predicted peak Vo2 was calculated by the following formulas^{17 18}:

Men: peak Vo₂ (ml/min/kg) = $60 - (age \times 0.55)$ Women: peak Vo₂ (ml/min/kg) = $48 - (age \times 0.37)$.

Maximal heart rate was compared with the predicted maximal heart rate.¹⁹

PEAK OXYGEN CONSUMPTION AND LONG-TERM ARRHYTHMIA OUTCOME

To determine the effect of precardioversion functional capacity on long-term maintenance of sinus rhythm we categorised patients in two groups according to the peak VO₂ they attained before cardioversion. We used the age and sex predicted peak VO₂ of 70% as the cutoff point.²⁰ We also studied other factors known to influence long-term arrhythmia outcome: arrhythmia duration,^{21–23} left atrial size,^{21 23 24} left ventricular function,²⁵ presence and type of underlying heart disease,^{23 26 27} and New York Heart Association class.²⁸

LONE ATRIAL FIBRILLATION VERSUS UNDERLYING HEART DISEASE

To study the influence of underlying heart disease on response to exercise we categorised patients as those with lone atrial fibrillation and those with underlying heart disease. Exercise data before and after cardioversion were compared in both groups.

DEFINITION OF TERMS

Chronic atrial fibrillation was defined as atrial fibrillation lasting > 6 months, without intercurrent sinus rhythm. Underlying heart disease was determined from the patient's history and physical examination, as well as from the chest x ray, echocardiography, and coronary angiography when available. Total

Table 2Clinical characteristics of patients in sinus rhythm and those in atrial fibrillation1month after cardioversion

Variable	Sinus rhythm (n = 37)	Atrial fibrillation (n = 26)
Age (y) (mean (1SD))	56 (12)	57 (13)
Men	25 (68)	19 (73)
Underlying heart disease (%)	28 (76)	13 (50)
Lone arrhythmia (%)	9 (24)	13 (50)
Medication (%):	. ,	. ,
Digoxin only	8 (22)	5 (19)
Verapamil only	8 (22)	6 (23)
Both digoxin/verapamil	21 (56)	15 (58)
Antiarrhythmic drugs	12 (32)	8 (31)
Arrhythmia history, total duration (median (range)) (mnth) NYHA class (precardioversion) (%):	34 (1·5–279)	36 (1–88)
I	16 (43)	13 (50)
П	14 (38)	9 (35)
III	7 (19)	4 (15)
Echocardiographic measurements (mean (1SD)):	. ,	()
LA, long axis view (mm)	45 (8)	44 (7)
LVEDD (mm)	51 (8)	52 (6)
LVESD (mm)	36 (9)	36 (7)
Fractional shortening (%)	31 (10)	30 (9)

LA, left atrial size; LVEDD, left ventricular end diastolic diameter; LVESD, left ventricular end systolic diameter; NYHA, New York Heart Association. None of the variables was significantly different.

arrhythmia duration was defined as the duration of the arrhythmia history from the first episode of chronic atrial fibrillation. It was determined by questioning the patient, by examining the patient's medical record, and by reviewing all available previous electrocardiograms.

STASTICAL ANALYSIS

Data are expressed as mean (1 SD). Median values were used for variables with a nonuniform distribution. To compare the two groups, continuous normally distributed variables were tested by the one way analysis of variance. Variables with an asymmetrical distribution were tested by the Mann-Whitney U-test. Frequencies were compared by the χ^2 test for equality of proportions, with Yates's correction for small numbers (< 10). Stepwise logistic regression analysis was used to deter-

Table 3 Exercise data and New York Heart Association classification before and 1 month after cardioversion in patients still in sinus rhythm (sinus rhythm maintained) and patients in atrial fibrillation (atrial fibrillation recurred) 1 month after cardioversion (mean (1SD))

	Before cardioversion	1 month	P value
Sin	us rhythm maintained ($n =$	37)	
Rest:			
HR (beats/min)	96 (21)	75 (18)	NS
SBP (mm Hg)	127 (19)	125 (21)	NS
DBP (mm Hg)	82 (9)	81 (11)	NS
Peak exercise:			
HR (beats/min)	175 (33)	140 (25)	< 0.001
SBP (mm Hg)	164 (21)	176 (24)	< 0.05
DBP (mm Hg)	82 (13)	82 (12)	NS
Exercise duration (min)	19·3 (4·9)	23.7 (6.0)	< 0.05
Peak Vo ₂ (ml/min/kg)	21.4 (5.8)	23.7 (6.4)	< 0.05
% predicted VO ₂	77 (19)	85 (1 9) ´	< 0.05
NYHA class	1.8 (0.7)	1.4 (0.5)	< 0.05
Atr	ial fibrillation recurred ($n =$	26)	
Rest:	, ,		
HR (beats/min)	89 (15)	88 (15)	NS
SBP (mm Hg)	125 (18)	126 (19)	NS
DBP (mm Hg)	78 (8)	80 (9)	NS
Peak exercise:			
HR (beats/min)	172 (26)	165 (27)	NS
SBP (mm Hg)	182 (25)	183 (29)	NS
DBP (mm Hg)	89 (11)	88 (8)	NS
Exercise duration (min)	19·Ò (Ś·O)	20.3 (5.2)	NS
Peak VO ₂ (ml/min/kg)	21.2 (5.2)	21.3 (5.6)	NS
% predicted Vo ₂	78 (18)	78 (18)	NS
NYHA class	1·7 (Ó·7)	1.6 (0.7)	NS

AF, atrial fibrillation; DBP, diastolic blood pressure; HR, heart rate; NYHA, New York Heart Association; SBP, systolic blood pressure; Vo₂, oxygen consumption; % predicted Vo₂, percentage of age and sex predicted peak oxygen consumption. mine predictors of peak Vo_2 before cardioversion. Life-table analysis was carried out by the Kaplan-Meier method. Survival curves were compared by the log rank test. A P value < 0.05 was regarded as significant.

Results

PATIENTS

Sixty three patients were eligible for participation in the study. Table 1 shows their baseline characteristics. Most patients had underlying heart disease. The mean total arrhythmia duration was long.

CARDIOVERSION: IMMEDIATE AND 1 MONTH RESULTS

Cardioversion was uncomplicated in all patients. Fifty five (87%) of 63 patients were successfully cardioverted to sinus rhythm. The clinical characteristics of those patients who could be cardioverted and those who could not were similar. During the first month after cardioversion atrial fibrillation recurred in 18 of those in whom cardioversion had been successful. Thus after a month 37 patients (59%) had maintained sinus rhythm and 26 (41%) were in atrial fibrillation. The clinical characteristics of patients in sinus rhythm and those in atrial fibrillation after 1 month were similar (table 2).

EXERCISE DATA BEFORE AND AFTER

CARDIOVERSION

Table 3 shows exercise data before and after cardioversion in patients who were still in sinus rhythm and those who were in atrial fibrillation 1 month after cardioversion. Exercise testing before cardioversion did not show statistically significant differences between both groups. In patients who had maintained sinus rhythm 1 month after cardioversion, mean peak Vo2 increased by 11%, from 21.4 (5.8) to 23.7 (6.4) ml/min/kg (P < 0.05). Figure 1 shows peak Vo₂ before and after cardioversion in each patient. Improvement of peak VO₂ after cardioversion was not correlated with precardioversion peak Vo₂, left ventricular fractional shortening, or changes in (maximal) heart rate after cardioversion. In contrast, peak Vo₂ and other exercise variables did not change in patients who were in atrial fibrillation 1 month after cardioversion.

PRECARDIOVERSION PEAK OXYGEN

CONSUMPTION AND LONG-TERM ARRHYTHMIA OUTCOME

Mean follow up after cardioversion was 19 (7) months. During this follow up atrial fibrillation recurred in 47 (75%) of all patients who underwent cardioversion (including eight patients who could not be cardioverted). Precardioversion peak Vo₂ was $\leq 70\%$ of the age and sex predicted value in 28 patients (44%), whereas it was > 70% in the remaining 35 patients. Kaplan-Meier life table analysis showed that there was no significant correlation between functional capacity (that is, age and sex predicted peak Vo₂ > 70% $v \leq 70\%$) before cardioversion and long term arrhyth-

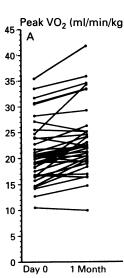


Table 4 Clinical and exercise data (mean (1SD)) of patients with underling heart disease compared with those with lone atrial fibrillation before cardioversion

Variable	Heart disease	Lone AF (n = 22)	P value
Variable	(n=41)		
Age (yrs)	57 (13)	57 (14)	NS
LA, long axis view (mm)	46 (7)	42 (7)	NS
LVEDD (mm)	53 (8)	50 (6)	NS
LVESD (mm)	37 (9)	34 (6)	NS
Fractional shortening (%)	29 (10)	32 (10)	NS
	Exercise testing before cardiovers	ion	
Rest:	0.1		
HR (beats per minute)	94 (20)	92 (17)	NS
SBP (mm Hg)	135 (21)	130 (19)	NS
DBP (mm Hg)	83 (11)	85 (7)	NS
Maximal exercise:			
HR (beats per minute)	166 (26)	187 (31)	< 0.01
SBP (mm Hg)	170 (24)	173 (23)	NS
DBP (mm Hg)	85 (13)	85 (11)	NS
Exercise duration (min)	18·6 (5·1)	20·4 (4·6)	< 0.05
Peak VO ₂ (ml/min/kg)	19·5 (4·4)	24.7 (6.0)	< 0.01
% predicted Vo ₂	72 (18)	87 (16)	< 0.01

AF, atrial fibrillation; DBP, diastolic blood pressure; HR, heart rate; LA, left atrial size; LVEDD, left ventricular enddiastolic diameter; LVESD, left ventricular endystolic diameter; SBP, systolic blood pressure; VO₂ oxygen consumption; % predicted VO₂, percentage of age and sex predicted peak oxygen consumption.

mia outcome. Thus peak Vo2 was not a predictive factor for the maintenance of sinus rhythm. Also none of the established clinical variables including echocardiographic left atrial size and New York Heart Association class were of predictive value in terms of longterm arrhythmia outcome in the present study.

LONE ATRIAL FIBRILLATION VERSUS UNDERLYING HEART DISEASE

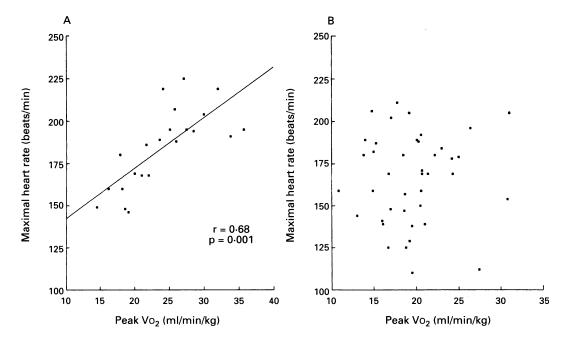
Before cardioversion

Before cardioversion mean peak Vo₂ in patients with lone atrial fibrillation was 24.7 (6.0) ml/min/kg and 87 (16%) when expressed as a percentage of the age and sex predicted maximum (table 4). Mean peak VO₂ in patients with underlying heart disease was significantly lower than in patients with lone atrial fibrillation: 19.5 (4.4) ml/min/kg (P < 0.01) and 72 (18%) (P < 0.01) of that predicted. Maximal heart rate was significantly higher in patients with lone atrial fibrillation than in patients with underlying heart disease. In patients with lone atrial fibrillation

there was a significant correlation between maximal heart rate and peak Vo_2 (r = 0.68, P = 0.001, figure 2A). There was no correlation in patients with underlying heart disease (figure 2B).

One month after cardioversion

In patients in sinus rhythm after 1 month, peak Vo₂ improved both in patients with (n = 28) and without (n = 9) underlying heart disease, from 19.8 (4.8) to 22.2 (5.2) ml/min/kg (P < 0.05) and from 26.1 (6.6) to 28.3 (7.5) ml/min/kg (P < 0.05) respectively (fig 3). The difference in improvement between both groups of patients (12% and 8% respectively) was not statistically significant. In patients without underlying heart disease peak Vo₂ after cardioversion was similar to that predicted for controls, whereas in patients with underlying heart disease it was 78% of that in controls. Resting heart rate and heart rate at peak exercise decreased by 20% in both groups. Whereas maximal heart rate after cardioversion in patients without underlying heart disease was similar to



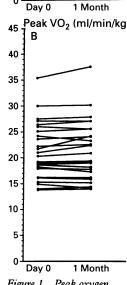
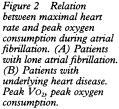


Figure 1 Peak oxygen consumption before and after cardioversion. (A) Patients still in sinus rhythm 1 month after cardioversion. (B) Patients having atrial fibrillation 1 month after cardioversion. Peak VO2, peak oxygen consumption.



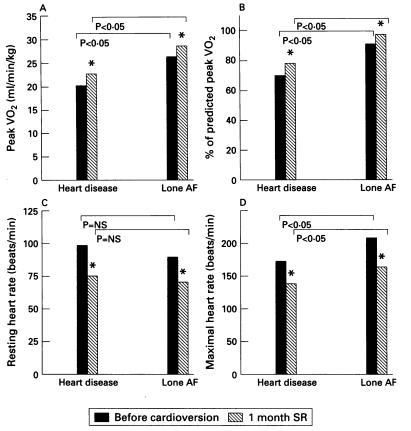


Figure 3 (A) Peak oxygen consumption, (B) peak oxygen consumption expressed as a percentage of the age and sex predicted maximum, (C) resting heart rate, and (D) maximal heart rate in atrial fibrillation (AF) patients (n = 28) and without underlying heart disease (n = 9) before cardioversion and after 1 month of sinus (SR) rhythm. P < 0.05 versus values before cardioversion. Peak VO₂, peak oxygen consumption.

that of controls, in patients with underlying heart disease it was only 80% of that in controls.

PREDICTORS OF PEAK OXYGEN CONSUMPTION DURING ATRIAL FIBRILLATION

Stepwise regression analysis, with pre-cardioversion peak VO_2 as the dependent variable, indicated that sex, age, and the presence of underlying heart disease were significant multivariate predictors of peak VO_2 during atrial fibrillation.

Discussion

Three previous studies used gas exchange analysis to study the effect of cardioversion on exercise capacity in patients with atrial fibrillation.¹¹⁻¹³ As in our study, they showed an increase in peak VO₂ after restoration of sinus rhythm. When Lundström and Karlsson studied 16 patients with atrial fibrillation they found an improvement in maximal oxygen uptake 1 month after successful cardioversion.¹³ This was accompanied by increased efficiency of ventilation. These four studies all showed a remarkably consistent improvement (approximately 10%) in peak VO₂ after restoration of sinus rhythm.

Unlike the studies of Atwood *et al* and Lipkin *et al* we found that the improvement in peak VO_2 in patients who maintained sinus rhythm was not related to peak VO_2^{12} or left ventricular function.^{12 29} Because we used similar methods and selection of patients, these

differences probably reflect the small numbers of patients included in these studies. We believe that peak VO_2 should not be used to select patients who are expected to benefit most from cardioversion in terms of improvement in functional capacity.

LONE ATRIAL FIBRILLATION VERSUS UNDERLYING HEART DISEASE

Atwood et al showed that before cardioversion patients with lone atrial fibrillation had significantly higher peak Vo₂ values and maximal heart rates than those with underlying heart disease.⁵ This accords with our findings. However, unlike the study of Atwood et al baseline functional capacity in our patients with lone atrial fibrillation was lower than predicted for age and sex matched controls. Therefore, our findings suggest that the arrhythmia itself may be a factor limiting exercise. This is supported by the observation that peak Vo2 increased after successful cardioversion in these patients. Apparently, the excessive heart rate response during atrial fibrillation may not always adequately compensate for the loss of atrial contribution to ventricular filling in patients with lone atrial fibrillation. Increase in functional capacity after restoration of sinus rhythm may also have been promoted by an improvement in left ventricular function, caused by a more favourable heart rate response in these patients with relatively high heart rates during atrial fibrillation.30

EXERCISE CAPACITY AND ARRHYTHMIA OUTCOME

The present study did not show that peak VO₂ was a predictive factor for long-term arrhythmia outcome after cardioversion. In contrast, one previous study reported that a better functional state, expressed as New York Heart Association class, was associated with a longer arrhythmia-free episode after cardioversion.28 One possible explanation for the present finding is that the high arrhythmia recurrence rate, presumably caused by the long total duration of arrhythmia, precluded detection of peak Vo₂ as a predictive factor for arrhythmia outcome. This is supported by the observation that none of the established risk factors for predicting arrhythmia relapses was statistically significant in our study. An alternative explanation may be that patients with a relatively high peak Vo₂—that is, patients with lone atrial fibrillation-have an unfavourable long-term arrhythmia outcome³¹ (probably because of primary electrophysiological abnormalities of the atria), whereas patients with a relatively low peak VO₂-that is, patient with coexistent heart disease-have a high recurrence rate caused by the underlying heart disease. This would also explain the inability to discriminate between a favourable and unfavourable long-term arrhythmia outcome by means of peak Vo₂. Nevertheless, from a clinical point of view, our findings suggest that the opportunity to restore sinus rhythm should not be withheld from patients with atrial fibrillation and a low functional capacity merely because of an impaired functional status.

PREDICTORS OF PEAK OXYGEN CONSUMPTION DURING ATRIAL FIBRILLATION

In the present study the only clinical factors predicting peak Vo₂ during atrial fibrillation were age, sex, and presence of underlying heart disease. These findings contrast with the results of an earlier study, which stressed the importance of maximal heart rate in predicting maximal oxygen consumption.5 In our study a significant relation between maximal heart rate and peak Vo2 was found only in patients with lone atrial fabrillation (figure 2A). The absence of a similar relation in patients with underlying heart disease was mainly the result of an excessive heart rate combined with a low peak Vo₂ in many of these patients (upper left quadrant of fig 2B). Obviously, in patients with underlying heart disease and a low peak VO₂ the "normal" relation between functional capacity and heart rate response is lost. Presumably, an enhanced adrenergic drive32 and adaptive mechanisms in the peripheral circulation^{33 34} may be responsible for this low peak VO₂ despite a high heart rate during exercise in these patients.

STUDY LIMITATIONS

Although we compared results of patients maintaining sinus rhythm after cardioversion with a control group of patients remaining in atrial fibrillation, patients were not randomised to cardioversion or pharmacological rate control only. This may have introduced a bias leading to selection of patients in the control group who did not improve their exercise capacity for reasons other than arrhythmia outcome. However, at baseline, patient groups were similar, and established clinical indices for predicting relapse were similar in the two groups.

CONCLUSIONS AND CLINICAL IMPLICATIONS Restoration of sinus rhythm improved peak

Vo₂ in patients with chronic atrial fibrillation. This holds both for patients with underlying heart disease and without. Also, functional state was not a predictive factor for long-term maintenance of sinus rhythm after cardioversion. Our findings suggest that cardioversion is the best method of improving functional capacity in patients with atrial fibrillation, irrespective of the presence of underlying heart disease and the functional state.

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