Oxygen Uptake Kinetics and Cardiopulmonary Performance in Lone Atrial Fibrillation and the Effects of Sotalol*

Ngai-Sang Lok, MB; and Chu-Pak Lau, MD, FCCP

Background: Atrial fibrillation (AF) is associated with impaired exercise capacity. Oxygen uptake (\(\text{VO}_2\)) kinetics determines cardiopulmonary performance during submaximal exercise, which may be impaired in patients with AF.

Aim: To study oxygen kinetics and cardiopulmonary performance in patients with AF without structural heart disease and the effects of oral sotalol on these parameters.

Patients and methods: Twenty consecutive patients (mean age, 56±8 years) with chronic AF were recruited. The protocol design was a randomized, single-blinded, and placebo-controlled trial. Patients received either sotalol or placebo for an 8-week study period, and the alternative treatment in the subsequent period. Cardiopulmonary function tests using constant workload and incremental workload protocols were performed at the end of each phase. Sixteen age-matched normal subjects were included as control subjects.

Results: During constant submaximal exercise, patients with AF had a larger oxygen deficit (425±140 mL vs 289±80 mL in normal subjects; \(p<0.05\)) and the time for achieving 63% of \(\text{VO}_2\) (mean response time) was also delayed (46±15 s vs 33±10 s; \(p<0.05\)). Compared with normal subjects, patients with chronic AF had a higher maximal exercise heart rate (180±34 beats/min vs 153±22 beats/min; \(p<0.05\)), but a lower maximal \(\text{VO}_2\) (20±4 mL/kg/min vs 26±6 mL/kg/min; \(p<0.05\)). Oral sotalol lowered the resting (72±15 beats/min vs 93±22 beats/min; \(p<0.05\)) and exercise heart rate compared with placebo (125±27 beats/min vs 180±34 beats/min; \(p<0.05\), respectively), and normalized oxygen pulse and the heart rate to minute ventilation ratio during maximal exercise. There was no significant difference between those receiving sotalol and those receiving placebo in oxygen deficit (502±150 mL vs 425±140 mL; \(p=0.38\)), maximal \(\text{VO}_2\) (17.2±4.9 mL/kg/min vs 20.4±4.7 mL/kg/min; \(p=0.17\)), and other gas exchange variables. In patients with AF, oxygen deficit has a fair correlation with \(\text{VO}_2\) at the anaerobic threshold \((r^2=0.43; p<0.05)\) and at maximal exercise \((r^2=0.45; p<0.05)\).

Conclusion: In addition to maximal exercise capacity and cardiopulmonary performance, patients with chronic AF without significant structural heart disease had impaired submaximal exercise performance as assessed by \(\text{VO}_2\) kinetics. These parameters were not significantly affected by sotalol used for rate control.

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Key words: atrial fibrillation; cardiopulmonary test; oxygen kinetics; sotalol

Abbreviations: AF=atrial fibrillation; AT=anaerobic threshold; MRT=mean response time; RER=respiratory exchange ratio; \(\dot{V}e\)=minute ventilation; \(\text{VO}_2\)=oxygen uptake

Chronic atrial fibrillation (AF), the most common sustained arrhythmia in daily clinical practice,\textsuperscript{1,5} is an important cause of hospitalization.\textsuperscript{3,4} Apart from the risk of thromboembolism, it is associated with increased mortality due to cardiovascular and noncardiovascular diseases.\textsuperscript{1,5} Previous studies showed conflicting data on the impairment of maximal exercise capacity in patients with AF. It was reported that the peak oxygen uptake (\(\text{VO}_2\)) in patients with lone AF was lower than that of age- and sex-predicted value, and AF itself was considered to be a factor limiting exercise.\textsuperscript{5,7} However, other authors\textsuperscript{8,9} have found no limitation in exercise performance in patients with lone AF when compared with the predicted value for age-matched normal subjects. These differences may be due to concomitant

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antiarrhythmic agents or different calculation methods used for predicted values since a normal control group was rarely used in these studies. While maximal exercise is seldom performed by the usually elderly patients with AF, many activities of daily living involved only constant levels of submaximal exercise.

During constant workload exercise, a delay in oxygen delivery to the tissues occurs as a result of an inherent inertia of the cardiopulmonary function. In normal individuals, steady-state \( \text{Vo}_2 \) is generally attained within the first 3 min of exercise.\(^\text{10}\) Oxygen deficit reflects the increase in \( \text{Vo}_2 \) before a steady-state level is reached, and this deficit is “repaid” after exercise. In the absence of lung disease, the speed of \( \text{Vo}_2 \) and the size of oxygen deficit are determined by the cardiovascular function and may be used as an indicator of submaximal exercise performance. The use of \( \text{Vo}_2 \) kinetics to evaluate and monitor treatment of several cardiovascular diseases has been studied. For example, in patients with heart failure, there is a delay in \( \text{Vo}_2 \) kinetics at the onset of exercise that reflects impaired cardiovascular performance and predicts decreased submaximal and maximal exercise tolerance.\(^\text{11}\) Similarly in patients with permanent ventricular rate adaptive pacemaker, oxygen kinetics are dependent on the types of sensors used for rate adaptation\(^\text{12}\) and may be a useful objective assessment of pacemaker programming. The status of \( \text{Vo}_2 \) kinetics in patients with AF is unknown. In addition, to our knowledge, the effect of heart rate control on \( \text{Vo}_2 \) kinetics for patients with chronic AF has not been investigated. The aims of this study were (1) to assess \( \text{Vo}_2 \) kinetics and cardiopulmonary performance in patients with chronic AF compared with normal control subjects, and (2) to assess the effect of sotalol as a rate-controlling agent on the above parameters in patients with chronic AF.

**Materials and Methods**

**Patients and Normal Subjects**

Twenty patients (15 men and 5 women) with chronic AF known for 12 to 66 months and 16 volunteers without a history of AF were recruited in this study (Table 1). Chronic AF was defined as ECG evidence of AF found in at least two consecutive follow-up visits separated by an interval of 3 months. Exclusion criteria included the following: (1) significant valvular heart disease; (2) New York Heart Association class III or IV heart failure; (3) those who could not perform incremental treadmill exercise test; and (4) abnormal results of lung function test. For the normal control subjects, those with a history of cardiac arrhythmias, cardiovascular disease, or chronic respiratory diseases were excluded. All patients and normal subjects underwent two-dimensional and M-mode echocardiography to exclude significant valvular heart disease and to assess the left atrial diameter and ejection fraction. Before entering the study, all subjects gave written consent for a protocol that was approved by the ethics committee of the University of Hong Kong.

**Table 1—Demographic Data of Normal Subjects and Patients Who Completed the Study**

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
<th>Normal Subjects</th>
<th>( p ) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>20</td>
<td>16</td>
<td>-</td>
</tr>
<tr>
<td>Age, yr</td>
<td>56±5 (42–71)</td>
<td>58±9 (48–71)</td>
<td>NS</td>
</tr>
<tr>
<td>Sex, M/F</td>
<td>15/5</td>
<td>10/6</td>
<td>NS</td>
</tr>
<tr>
<td>Left atrial diameter, cm</td>
<td>4.2±0.8 (2.9–5.6)</td>
<td>3.5±0.4 (2.8–4.1)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>61.7±9.5 (50–75)</td>
<td>65.3±6.6 (54–77)</td>
<td>NS</td>
</tr>
</tbody>
</table>

**Study Protocol**

The study was a randomized, single-blinded, placebo-controlled, and crossover trial. Treatment with all antiarrhythmic agents was stopped for at least five half-lives before the study. In phase 1, patients were randomly allocated to either placebo or sotalol treatment for an 8-week study period, and then changed to the alternative treatment during phase 2. In the treatment group, sotalol therapy was initiated at a dose of 80 mg twice daily. The dose was titrated to 120 mg bid and 160 mg bid (maximum dose) at 2-weekly intervals, with an aim to reach a resting seated heart rate ≤60 beats/min. The same titration was also performed for patients receiving placebo using matched tablets. \( \text{Vo}_2 \) kinetics and cardiopulmonary test were performed at the end of each phase. Apart from a training session, only one cardiopulmonary test was performed in the normal subjects.

**Exercise Protocol and Gas Exchange Analysis**

Two weeks before the study, all subjects underwent a trial cardiopulmonary exercise test to familiarize them with the equipment and the procedure. During the study, all patients and normal subjects underwent cardiopulmonary test in a postabsorptive state. Subjects were asked to exercise on a calibrated motor-driven treadmill (CASE 15; Marquette; Milwaukee) with constant workload and incremental workload exercise protocols. During the test, subjects breathed room air through a low-resistance mask. Expired \( \text{O}_2 \) and \( \text{CO}_2 \) partial pressures were measured with a gas analyzer (Cardiopulmonary Exercise Testing System; MedGraphics; St. Paul, Minn.). The signals underwent analog-to-digital conversion for breath-by-breath gas exchange analysis, and the gas analyzer was calibrated before each test.

**Exercise Protocol:** After collecting 2 min of resting gas exchange, constant workload exercise was performed by walking on the treadmill at a speed of 2.0 mph (0% grade) for 6 min without a warm-up period. Standard 12-lead ECG was recorded every 15 s and BP was measured at 2-min intervals. \( \text{Vo}_2 \) kinetics was measured during this exercise (see below).

After the constant workload exercise and a 30-min rest period, an incremental exercise using the chronotropic assessment exercise protocol\(^\text{13}\) was used to assess cardiopulmonary performance during maximal exercise. Standard 12-lead ECG was recorded at rest, at the end of each exercise stage, and at the third minute of recovery with a paper speed of 25 mm/s. BP was measured by a cuff sphygmomanometer at rest, and at 2-min intervals throughout the study. The indications for exercise termination were...
noted. Anaerobic threshold (AT), gas exchange variables at AT, and peak exercise were automatically determined by the system (see below).

**VO₂ Kinetics Analysis:** VO₂ kinetics was derived from the 6-min constant workload exercise. Breath-by-breath VO₂ was measured, and the rate of rise of VO₂ until plateau was computed by a commercially available software (Oxygen Kinetics Option v1.2, Medgraphics). Two parameters were used to assess oxygen kinetics: oxygen deficit and mean response time (MRT). Oxygen deficit was calculated by measuring the area between the ideal square curve of O₂ consumption at the onset of the constant workload exercise and the actual exponentially shaped curve (Fig 1). The curve, plotting a best-fit line based on the difference between steady-state VO₂ (after subtracting resting VO₂) and VO₂ for each breath during the first 3 min of exercise, was determined by the following equation:

\[ \text{VO}_2(t) = \text{VO}_2_{\text{rest}}(1 - e^{-kt}), \]

where \( \text{VO}_2(t) \) = rate of oxygen consumption at any time (t), \( t \) = time, \( k \) = rate constant of the rise in VO₂ with the dimension of time.

The MRT described the rapidity of VO₂ of a subject to respond to constant workload exercise. In this software, it was defined as the time it took to achieve 63% of the full response, and was calculated by the following equation:

\[ \text{MRT} = \frac{\text{VO}_2 (6 \text{ min}) \times (\text{mL of oxygen})}{\text{oxygen deficit (mL of oxygen)}} - 60 \text{ s} \]

**Gas Exchange Analysis and AT Determination:** Gas exchange variables were measured continuously and averaged at intervals of 30 s throughout the test. Variables measured included VO₂ (mL/min), minute ventilation (VE, L/min), respiratory exchange ratio (RER, VO₂/CO₂), and oxygen pulse (oxygen uptake/heart rate). These parameters were determined at the AT and at peak exercise.

**AT Determination for Graded Exercise:** AT was determined automatically by three criteria: (1) a point on the regression line of CO₂ production vs O₂ uptake at which CO₂ production began increasing disproportionately to the O₂ consumption; (2) an increase in end-tidal O₂ without a corresponding decrease in end-tidal CO₂; and (3) a respiratory exchange ratio >1.0 was reached.14 The threshold was later manually reviewed and readjusted if necessary by one of the investigators (C.P.L.) who was unaware of the treatment sequence.

**Statistical Analysis**

All data were presented as mean ± 1 SD. Unpaired t-test was performed to evaluate oxygen kinetics, gas exchange, and hemodynamic variables during the placebo vs sotalol phase, and to compare the results in both groups with those in normal subjects. Linear regression was used to evaluate the correlation between oxygen deficit, AT, and VO₂ in patients with AF. The difference was considered to be statistically significant if p<0.05.

**RESULTS**

During the study, two patients developed intolerable fatigue and shortness of breath after taking sotalol (120 mg bid). Sinus rhythm was restored in two other patients when the maximal dose (160 mg bid) was used. Only the exercise data in the placebo phase were available for comparison in two of these four patients. The remaining 16 patients completed the study uneventfully. During incremental exercise test, exercise was terminated because of either fatigue or dyspnea in all patients and normal subjects.

**Oxygen \( \text{VO}_2 \) Kinetics**

The heart rate changes during constant workload exercise are shown in Figure 2. Steady-state heart rate was attained at 3, 4, and 3 min for normal subjects, patients with AF receiving placebo, and patients with AF receiving sotalol, respectively. At steady state, VO₂ between the normal subjects and patients with AF receiving placebo and sotalol was 11.9±4 mL/kg/min, 9.4±2 mL/kg/min, and 8.9±2 mL/kg/min, respectively (p=NS). Patients with AF had a larger oxygen deficit when compared with that in the normal control subjects (425±140 mL vs 289±80 mL; p<0.05), and showed a prolonged mean response time (46±15 s vs 33±10 s; p<0.05) (Fig 3). Oxygen deficit and MRT were further increased by oral sotalol, although the
Heart rate (beat/min)

<table>
<thead>
<tr>
<th></th>
<th>AF + Placebo</th>
<th>Normal</th>
<th>AF + Sotalol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P&lt;0.05 when compared with normal</td>
<td>*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Figure 2.** Heart rate changes during constant workload exercise in normal subjects and patients with AF receiving either placebo or sotalol.

The difference between sotalol and placebo groups did not reach statistical significance (p=0.38 and p=0.12, respectively).

**Cardiopulmonary Performance on Incremental Exercise**

**Patients With AF vs Normal Subjects:** Patients with AF had a higher heart rate at the AT and maximal exercise than normal subjects (Fig 4). The BP change was similar in patients with AF and normal subjects (Table 2). Compared with normal subjects, patients with AF had a lower \( \dot{V}O_2 \) at AT and maximal exercise (Fig 5). Oxygen pulse was lower in patients with AF at the AT and at maximal exercise when compared with normal subjects. While \( V_E \) and RER in patients with AF were similar to those of normal subjects, the heart rate to \( V_E \) ratio was higher in patients. There was a significant reduction in work performance at the AT and at maximal exercise in patients with AF compared with normal subjects.

**Sotalol vs Placebo:** The heart rate of patients with AF at different exercise levels was significantly lowered by sotalol but the change in BP was insignificant between sotalol and placebo groups. There was an insignificant trend of further reduction of \( \dot{V}O_2 \) at AT and maximal exercise while patients were receiving sotalol. Because of rate control, sotalol normalized the oxygen pulse and the heart rate to \( V_E \) ratio at AT and at maximal exercise. Workloads at AT and maximal exercise were also similar in both sotalol and placebo groups.

In patients with AF, oxygen deficit significantly correlated with \( \dot{V}O_2 \) at AT and maximal exercise \((r^2=0.43, p<0.05, \text{ and } r^2=0.45, p<0.05)\). None of the other clinical factors and gas exchange variables were found to be related to oxygen deficit.

**Discussion**

The main findings in this study are that patients with AF without significant structural heart disease have a higher resting and exercise heart rate, and impaired \( \dot{V}O_2 \) kinetics and cardiopulmonary perfor-
mance. Oral sotalol lowered the resting and exercise heart rate in patients with AF, but tended to impair exercise performance in these patients.

Table 2—Hemodynamic and Gas Exchange Variables at AT and Maximal Exercise in Patients With Chronic AF (Treated With Either Placebo or Sotalol) and in Normal Subjects

<table>
<thead>
<tr>
<th></th>
<th>AF+Placebo</th>
<th>AF+Sotalol</th>
<th>Normal Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Resting</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR, p/beat/min</td>
<td>93±20</td>
<td>72±15</td>
<td>78±13</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>132±24</td>
<td>116±19</td>
<td>125±15</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>79±26</td>
<td>70±12</td>
<td>82±7</td>
</tr>
<tr>
<td><strong>AT</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time of exercise, min</td>
<td>8.3±1.8</td>
<td>7.8±2.4</td>
<td>9.9±2.9</td>
</tr>
<tr>
<td>VO₂, mL/kg/min</td>
<td>13.7±2.4†</td>
<td>11.7±3.3†</td>
<td>17.8±7.1</td>
</tr>
<tr>
<td>O₂ pulse, mL/beat</td>
<td>6.8±1.4†</td>
<td>7.9±2.1</td>
<td>9.6±3.1</td>
</tr>
<tr>
<td>Ve, L/min</td>
<td>34.1±9.5</td>
<td>31.1±6.2</td>
<td>34.5±9.2</td>
</tr>
<tr>
<td>RER</td>
<td>0.92±0.08</td>
<td>0.93±0.11</td>
<td>0.93±0.14</td>
</tr>
<tr>
<td>HR/ Ve, beat/L</td>
<td>2.5±0.89†</td>
<td>1.95±0.75</td>
<td>1.84±0.71</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>145±19</td>
<td>128±18</td>
<td>146±21</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>84±19</td>
<td>81±8</td>
<td>89±8</td>
</tr>
<tr>
<td><strong>Maximal exercise</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time of exercise, min</td>
<td>12.5±1.2</td>
<td>11.2±1.5†</td>
<td>13.5±2.1</td>
</tr>
<tr>
<td>VO₂, mL/kg/min</td>
<td>20.4±4.7†</td>
<td>17.2±4.9†</td>
<td>11.4±3.7</td>
</tr>
<tr>
<td>O₂ pulse, mL/beat</td>
<td>1.8±2.1†</td>
<td>10±2.5</td>
<td>11.6±3.7</td>
</tr>
<tr>
<td>Ve, L/min</td>
<td>58±18</td>
<td>51.2±17.2</td>
<td>61.3±19.9</td>
</tr>
<tr>
<td>RER</td>
<td>1.10±0.15</td>
<td>1.08±0.19</td>
<td>1.10±0.50</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>165±12</td>
<td>150±32</td>
<td>173±24</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>86±16</td>
<td>82±19</td>
<td>87±9</td>
</tr>
</tbody>
</table>

*HR = heart rate.
†p<0.05 when compared with placebo group.
‡p<0.05 when compared with normal subjects.

**Figure 4.** Heart rate changes in normal subjects and in patients with chronic AF receiving either placebo or sotalol.

**Vo₂ Kinetics and Cardiopulmonary Performance in Patients with Chronic AF**

In patients with AF, alterations in cardiac output may be less obvious because of a higher heart rate at rest and during exercise despite the loss of an effective atrial contraction. It was suggested that the degree of exaggerated chronotropic response is dependent on the underlying cardiac function, and patients with impaired functional capacity had a higher heart rate to minor exercise compared with patients who had preserved function capacity. However, in the presence of AF, heart rate alone is a poor indicator of cardiac output or oxygen delivery to tissues. Oxygen deficit reflects the ability for rapid acceleration of circulatory oxygen transport during the initiation of exercise. Slower Vo₂ kinetics and a greater oxygen deficit have been found in patients with heart failure. To our knowledge, oxygen deficit in patients with AF has not been evaluated previously. Prolonged Vo₂ kinetics was considered to indicate increased tissue anaerobiosis and lactic acidosis, and is a sensitive indication of cardiovascular performance during submaximal exercise. In the present study, despite a rapid heart rate during the initial part of exercise, patients with AF had an increased oxygen deficit and MRT compared with normal control subjects. This finding suggests patients with AF have impaired oxygen deficit similar to patients with mild heart failure. Like patients with heart failure, oxygen kinetics is significantly correlated to AT and maximal Vo₂, although the correlation coefficient...
we observed was only fair. It is of interest to note that in artificial pacemakers with heart rate driven by implantable sensor, oxygen kinetics is reduced with a faster heart rate response kinetics. A similar situation did not occur in our patients with AF despite a higher initial heart rate response to exercise. As the left ventricular function of the patients was normal, this suggests that the loss of effective atrial contraction and/or the irregularity of AF significantly impaired patients’ performance during submaximal exercise.

In general, exercise capacity in patients with AF associated with significant underlying cardiac diseases such as valvular disease, ischemic heart disease, or a history of congestive heart failure was impaired compared with age-predicted control subjects. Different results have been reported about the cardiopulmonary performance of patients with lone AF. However, patient populations were heterogeneous and many patients were receiving antiarrhythmic treatment in these studies. In addition, none of these studies incorporated the results of normal subjects for comparison. In the present study, \( VO_2 \) kinetics, exercise capacity, and oxygen pulse were impaired in patients with lone AF when compared with normal control subjects. Since none of these patients had significant underlying cardiac disease and abnormal ejection fraction, the result suggests that AF alone limits oxygen consumption at AT and peak exercise consumption.

**Effect of Oral Sotalol on Hemodynamic and Gas Exchange in Patients with Chronic AF**

Synthesized for more than 3 decades, sotalol was first characterized as a \( \beta \)-blocking agent. It was later documented that sotalol has class 3 antiarrhythmic properties and could prolong refractory periods in the atria, ventricles, Purkinje system, and atrioventricular bypass tracts of the heart. Because of these properties, sotalol has been used in patients with AF, both for rate control and for maintenance of sinus rhythm.

To our knowledge, the hemodynamic and cardiopulmonary effect of sotalol on chronic AF has not been studied. In subjects with sinus rhythm, the hemodynamic effects of sotalol have been considered to be similar to other \( \beta \)-blockers. After IV or oral sotalol, heart rate and systolic BP at rest and exercise were reduced without a change in diastolic and mean BP. In another study involving 17 patients with paroxysmal AF, sotalol had no significant effect on systemic arterial pressures (systolic, mean, and diastolic) during either sinus rhythm or induced AF. To our knowledge, our study is the first to examine the effect of oral sotalol on hemodynamic and gas exchange in patients with chronic AF. The results showed that sotalol lowered the resting and exercise heart rate without significant effect on BP.

The effect of class 3 antiarrhythmic agents on gas exchange in patients with AF has not been evaluated in previous studies. Since oxygen deficit appeared to be related to anaerobic metabolism, exercise intensity, and endurance, enlarged oxygen deficit could be due to either increased anaerobic metabolism or decreased exercise capacity. In a study assessing exercise performance in patients with chronic AF, there was a 19% reduction in treadmill time and a 16% reduction in maximal \( VO_2 \) in patients receiving \( \beta \)-adrenergic blockade when compared with placebo. Impaired exercise
capacity was considered to be related to both blunted heart rate response and negative inotropic effect caused by β-adrenergic blockade. In this study, there was an insignificant trend for attenuation in oxygen kinetics and cardiopulmonary performance. As oxygen pulse (which reflects stroke volume) is normalized after sotalol; this suggests that our patients without significant structural heart disease are able to utilize the stroke volume reserve to compensate the negative inotropic and chronotropic effect of sotalol.

Limitations

The present study involved patients with lone AF and normal ejection fraction. Whether the results are applicable to the general patients with AF who may have impaired cardiovascular function requires further investigation. However, we believe that the present study is the first to show that even in patients with AF and normal ejection fraction, oxygen kinetics and cardiopulmonary performance are impaired compared with those in normal control subjects. In the present study, two patients were withdrawn owing to shortness of breath after taking sotalol, and sinus rhythm was restored in two other patients. The extent of how these factors influence our results is unclear. In addition, although ventricular proarrhythmia was not observed in our study, the use of oral sotalol may need close supervision because of the definite incidence of sotalol-induced torsades de pointes.22

CONCLUSION

Despite an exaggerated exercise heart rate, VO2 kinetics, cardiopulmonary performance, and maximal exercise capacity are impaired in patients with AF even without significant underlying heart disease when compared with those of age-matched normal subjects. Oxygen deficit may be used as an indicator of cardiopulmonary performance during submaximal exercise in patients with chronic AF and may be used to objectively assess functional capacity of patients with AF and the effects of drug treatment. In this group of patients without structural heart disease, sotalol treatment reduced the resting and exercise heart rate, without major effect on oxygen kinetics, exercise capacity, and gas exchange responses to exercise.

ACKNOWLEDGMENT: Rebecca Chiu and staff in the Diagnostic Laboratory of Tung Wah Hospital are gratefully acknowledged for their assistance in this study.

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