similar to those obtained with other agents. The nonblinded design of our study is a limitation, since some patients might have spontaneous conversion. Thus our results encourage us to perform a double-blind randomized study. Finally, the incidence of side effects was low and we did not observe proarrhythmic effects in our study. In conclusion, propafenone is an effective therapy to convert AF to sinus rhythm in about 50% of cases and has a low incidence of side effects. Maintenance of sinus rhythm could also be achieved in almost all patients.

REFERENCES

- 1. Stern S. Conversion of chronic atrial fibrillation to sinus rhythm with combined propranolol and quinidine treatment. AM HEART J 1967;74:170-5.
- Goy JJ, Kaufmann U, Kappenberger L, Sigwart U. Restoration of sinus rhythm with flecainide in patients with atrial fibrillation. Am J Cardiol 1988;62:38D-40D.
- Borgeat A, Goy JJ, Maendly R, Kaufmann U, Grbic M, Sigwart U. Flecainide versus quinidine for conversion of atrial fibrillation to sinus rhythm. Am J Cardiol 1986;58:496-8.
- Fenster PE, Comess KA, Marsh R, Katzenberg C, Hager WD. Conversion of atrial fibrillation to sinus rhythm by acute intravenous procainamide infusion. AM HEART J 1983;106:501-4.
- Hammill SC, Wood DL, Gersh BJ, Osborn MJ, Holmes DR. Propafenone for paroxysmal atrial fibrillation. Am J Cardiol 1988;61:473-4.
- Kerr CR, Klein GJ, Axelson JE, Cooper JC. Propafenone for prevention of recurrent atrial fibrillation. Am J Cardiol 1988;63:914-6.

Evaluation of 85% predicted maximal heart rate as an end point for diagnostic exercise testing

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An exercise test ideally should be limited by symptoms or by signs of significant cardiovascular dysfunction (e.g., exercise-related hypotension, electrocardiographic evidence of marked myocardial ischemia, etc.). In many cases, however, an exercise test is terminated when the patient reaches a previously determined target heart rate. To better understand the implications of target heart rate-limited exercise with respect to potential metabolic stressors, this study was designed to determine the relationship between a ventilatory measurement that approximates lactate December 1991 American Heart Journal

threshold (the respiratory compensation threshold¹⁻³) and a parameter frequently used to denote performance of a "maximal exercise test" (85% age-predicted maximal heart rate). An approximation of lactate threshold was used as a criterion for evaluation because catecholamines, which may be important in the genesis of myocardial ischemia and cardiac arrhythmias,^{4, 5} have a nonlinear increase during incremental exercise that parallels the course of lactate.^{6,7} The rise in lactate (lactate threshold) does not occur at a fixed percentage of maximal heart rate,⁸ resulting in a situation where patients at the same heart rate may experience varying degrees of physiologic stress. As a noninvasive predictor of lactate threshold, determination of respiratory compensation threshold provides a better estimation of physiologic stress than can be appreciated from heart rate alone.

The exercise tests of 131 healthy subjects referred for exercise prescription were retrospectively reviewed. Each subject performed symptom-limited incremental treadmill exercise on a modified Balke protocol with 1-minute stages. Most subjects stopped exercise secondary to fatigue; no subject had signs of exercise-related myocardial ischemia. Subjects were not taking any medication that might influence heart rate. Direct measurements of ventilatory and oxygen consumption were made during exercise. Peak oxygen consumption and the heart rate at respiratory compensation threshold (HR-RCT defined as the heart rate associated with an increase in the ventilatory equivalent for $(CO_2)^{1,2}$ were determined by an observer who was blinded with respect to the purpose of this study. The best method for using exercise ventilatory measurements to assess lactate threshold is controversial; the RCT was used in this study because it has been documented to correspond to a lactate level associated with a marked rise in catecholamines and has been studied in both men and women.1-3 Patients were divided into two groups, based on the relationship between 85% age-predicted maximal heart rate (PMHR = 220-age) and the HR-RCT: group A included subjects with 85% PMHR below HR-RCT; group B included subjects with 85% PMHR above HR-RCT. Nonpaired t tests and chi square tests with Yates' correction were used to determine significant differences between groups. Data are reported as mean (standard error of the mean).

The mean difference between measured peak heart rate and 85% PMHR was 24 beats for the entire study group. Group A included 43 subjects (15 women and 28 men) for whom 85% PMHR occurred below HR-RCT and group B included 88 subjects (12 women and 76 men) for whom 85% PMHR occurred above HR-RCT (Table I). Group A had a significantly higher peak heart rate than group B (180 versus 170 beats/min, p = 0.001), but there was no significant difference in peak oxygen consumption between groups. Similarly, the HR-RCT was significantly higher in group A compared with group B (159 versus 135 beats/min, p = 0.001), and there was no difference in oxygen consumption at RCT between groups. The HR-RCT occurred at a significantly greater percentage of PMHR in group A than in group B (90 versus 77%, p = 0.001). Women were

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	Group A	Group B	р
No.	43	88	
Women/Men	15/28	12/76	
Peak HR (beats/min)	180 (2)	170 (1.7)	0.001
Peak VO ₂ (ml/kg/min)	39.3 (1.7)	38.4(1.2)	NS
HR-RCT	159 (1.7)	135.5(1.4)	0.001
VO ₂ -RCT	29.3 (1.4)	27.6 (0.9)	NS
HR-RCT/PMHR	90% (0.6)	76.9% (0.6)	0.001

HR, Heart rate; HR-RCT, heart rate at respiratory compensation threshold; PMHR, predicted maximal heart rate; VO₂-RCT, oxygen consumption at respiratory compensation threshold.

significantly less likely than men to surpass RCT at 85% PMHR (p = 0.01); 55% of the women in the study compared with 27% of the men were in group A (Table II). Men had a significantly higher peak oxygen consumption than women (39.8 versus 34.4 ml/kg/min, p = 0.02), but peak heart rates were the same (173 beats/min) (Table II). Men and women had a similar oxygen consumption at RCT, but men had a significantly lower HR-RCT (141 versus 152, p = 0.01). The HR-RCT occurred at a significantly higher percentage of PMHR in women than in men (85.7% versus 80.1%, p = 0.001).

Exercise tests are performed to evaluate a patient's response to the increased demands of exercise in a controlled laboratory setting. Using respiratory compensation threshold as a reference point, this study was designed to determine the effect of stopping exercise test at 85% PMHR compared with symptom-limited exercise. The results indicate that a large proportion of subjects (43 of 131) who exceeded RCT during symptom-limited exercise would fail to reach this point if exercise is stopped at 85% PMHR.

The data also indicate a significant gender effect on the percentage of PMHR at which RCT occurs, resulting in women being significantly less likely than men to exceed this point if exercise is stopped at 85% PMHR. The gender difference may have resulted from a smaller stroke volume secondary to smaller ventricular size in women^{9, 10} or from lower hematocrit levels in women¹¹; these factors, alone or in combination, would necessitate a higher heart rate at equivalent submaximal loads to meet metabolic needs. Consistent with this, women had a higher heart rate at RCT than men, despite the fact that oxygen consumptions were similar. In addition, women had a similar peak heart rate to men with a concomitant significantly lower peak oxygen consumption. Thus the oxygen pulse (oxygen consumption/beat) was lower for women at both RCT and peak exercise.

The clinical implications of the findings in this study remain to be determined. Stopping exercise at 85% PMHR

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	Men	Women	р
No.	104	27	
Age	43.8 (1.2)	42.6 (2.0)	NS
% in Group A	27%	55%	0.01
Peak HR (beats/min)	173 (2.9)	173 (1.5)	NS
Peak VO ₂ (ml/kg/min)	39.8 (1.1)	34.4(1.7)	0.02
HR-RCT	141 (1.6)	152 (3)	0.01
VO_2 -RCT	27.7 (1.5)	28.3 (0.9)	\mathbf{NS}
HR-RCT/PMHR	80.1% (0.8)	85.7% (1.6)	0.001

Abbreviations as in Table I.

may result in failure to gain all the information available from a symptom-limited exercise test. In particular, the effect of elevated catecholamines and changes in other factors that do not bear a direct linear relationship to heart rate may not be seen. The result is that a test that does not show evidence of cardiovascular dysfunction at 85 % PMHR may not have included the stress of elevated catecholamine levels, a stress that the patient may experience when exercising outside of the laboratory. Additional testing of larger groups of patients, particularly those with cardiac disease, is needed to determine the effect of surpassing RCT on the predictive accuracy of the exercise test for evaluating myocardial ischemia or arrhythmia.

REFERENCES

- 1. Skinner JS, McLeillen TH. The transition from aerobic to anaerobic metabolism. Res Q Exercise Sport 1980;51:238-48.
- Simon J, Young JL, Gutin B, Blood DK, Case RB. Lactate accumulation relative to the anaerobic and respiratory compensation thresholds. J Appl Physiol 1983;54:13-7.
- Iwaoka K, Hatta H, Atómi Y, Miyashita M. Lactate, respiratory compensation thresholds, and distance running performance in runners of both sexes. Int J Sports Med 1988;9:306-9.
- Podrid PJ, Fuschs T, Candines R. Role of the sympathetic nervous system in the genesis of ventricular arrhythmia. Circulation 1990;82(suppl I):I-103-13.
- 5. DeMaria AN, Vera Z, Amsterdam EA, Mason DT, Masumi RA. Disturbances of the cardiac rhythm and conduction induced by exercise. Am J Cardiol 1974;33:732-6.
- Lehman M, Schmid P, Keul J. Plasma catecholamine and blood lactate accumulation during incremental exhaustive exercise. Int J Sports Med 1985;8:78-81.
- Lehman M, Keul J, Huber G, DePrada M. Plasma catecholamines in trained and untrained volunteers during graduated exercise. Int J Sports Med 1981;2:143-7.
- Katch V, Weltman A, Sady S, Freedson P. Validity of the relative percent concept for equating training indices. Eur J Appl Physiol 1978;39:219-27.
- 9. Lewis DA, Kamon E, Hodgson JL. Physiological differences between genders. Sports Med 1986;3:357-69.
- 10. Pate R, Kriska A. Physiological basis of the sex difference in cardiorespiratory endurance. Sports Med 1984;1:87-98.
- 11. Freedson PS. The influence of hemoglobin concentration on exercise cardiac output. Int J Sports Med 1981;2:81-6.