

Exercise and heart rate recovery

By: Julie S. MacMillan, [Leslie L. Davis](#), Carol F. Durham, and Elizabeth S. Matteson

MacMillan, J.S., Davis, L.L., Durham, C.F., Matteson E.S. (2006). Exercise and heart rate recovery. *Heart and Lung*, 35 (6), 383-90.

***© Elsevier. Reprinted with permission. No further reproduction is authorized without written permission from Elsevier. This version of the document is not the version of record. Figures and/or pictures may be missing from this format of the document.

*** Made available courtesy of Elsevier: <https://doi.org/10.1016/j.hrtlng.2006.07.003>



© 2006. This manuscript version is made available under the CC-BY-NC-ND 4.0 license <http://creativecommons.org/licenses/by-nc-nd/4.0/>

Abstract:

Purpose:

This study examines whether heart rate recovery (HRR) improves as a result of exercise training during cardiac rehabilitation (CR).

Methods:

A retrospective study was performed that included 100 patients who completed phase II CR and had entry and exit exercise stress tests. HRR was compared for the sample. Improvements in HRR were compared between gender and age groups. Correlation between age and HRR was performed.

Results:

The total sample improved HRR ($P = .020$). There was no significant difference in the improvement of HRR based on gender, indicating males and females improve at similar rates ($P = .833$). Similarly, there was no significant difference in the improvement of HRR based on age, indicating older subjects improve similarly to younger subjects ($P = .700$). There was no relationship between age and HRR; therefore, as age increases there is no decrease in HRR.

Conclusion:

HRR improves in patients who complete CR.

Keywords: heart rate recovery | exercise training | cardiac rehabilitation | beta-blockers

Article:

Coronary heart disease is the single largest killer of American adults with more than 13 million having been diagnosed with heart disease.¹ Cardiac rehabilitation (CR) services are designed with the goals of secondary prevention, which include preventing subsequent events and decreasing the progression of heart disease.² Exercise training has been a core component of CR

programs since their inception.³ Regular exercise training in persons with cardiovascular (CV) disease results in changes in the muscular, CV, and neurohormonal systems that lead to an improvement in functional capacity, a decrease in symptoms, and a reduction in CV mortality.^{3,4}

The exact mechanism by which exercise accomplishes these benefits is a subject of much study. Some hypotheses include decreases in blood pressure, reductions in body weight, improvements in lipid levels, favorable changes in the fibrinolytic system, and improvements in vascular endothelium.⁴ Another emerging theory is the effect of physical exercise on autonomic tone, which is defined as the balance between sympathetic and parasympathetic activity.⁴ Heart rate recovery (HRR), the decrease in heart rate (HR) that occurs immediately after exercise, is related to autonomic balance.⁵

In theory, the activation of the sympathetic nervous system (SNS) and withdrawal of the parasympathetic nervous system (PNS) causes the increase in HR during exercise. Conversely, PNS activation and SNS withdrawal causes the HR to decrease immediately after exercise.⁶ Increased PNS activity has been associated with a decrease in the risk of death by protecting the heart against lethal arrhythmias.⁷

Review of the literature

Recent studies have examined the value of HRR immediately after the completion of an exercise stress test as a predictor of outcome.⁸ HRR is defined as the decrease in HR that occurs immediately after exercise.⁵ These studies show that attenuated HRR, reflecting reduced PNS activity, is an independent predictor of mortality in a wide spectrum of patients.⁵ Shetler and colleagues⁹ validated HRR at 1 or 2 minutes as a prognostic measurement that should be recorded as a part of all treadmill tests.

In a cohort of 5234 healthy adults without clinically evident CV disease, HRR after submaximal exercise testing was a powerful predictor of all-cause mortality.¹⁰ The exercise tests were stopped when 85% to 90% of age-predicted peak HR was reached, no cool-down period was performed, and HRR was measured at 2 minutes after exercise. Those with an abnormal HRR had a mortality rate of 10% versus 4% among those with normal HRR after 12 years of follow-up. An association between fitness levels and HRR was also noted in the study, as those with an abnormal HRR were less likely to exercise regularly.

Another study from the same investigators examined HRR in 2428 adults referred for exercise thallium scintigraphy over a 6-year period.¹¹ An abnormal HRR was defined as a decrease of 12 beats or less at 1 minute after exercise. The group with a value of 12 or less had a mortality of 19%, whereas the group with an HRR greater than 12 had a mortality rate of 5%. The study used a treadmill protocol with a 2-minute cool-down walk. Poor HRR was predictive of death in several important subgroups including the elderly, women, and those taking beta-blockers and correlated with impaired functional capacity.

In addition, HRR was found to be an independent predictor of mortality regardless of severity of coronary artery disease (CAD) in a study of 2935 patients undergoing coronary angiography.¹² Abnormal HRR was defined as 12 beats or less in those patients with a cool-

down period after treadmill exercise and 18 beats or less for patients with no cool-down period after treadmill exercise. HRR was found to be predictive of mortality regardless of severity of coronary disease.

Watanabe and colleagues¹³ studied the effects of HRR in the absence of a cool-down period and taking into account left ventricular systolic function. After a symptom-limited exercise test, 5438 patients were immediately placed in a supine position and recovery HRs were obtained by electrocardiography (ECG). Abnormal HRR was defined as 18 beats or less at 1 minute. Again, an abnormal HRR was a strong and independent predictor of mortality, and those patients are more likely to have left ventricular systolic dysfunction. Other studies have showed that abnormal HRR was found in patients with diabetes or abnormal spirometry.^{14,15}

Three studies have examined improvement in HRR through exercise in the CR population. Entry and exit treadmill stress tests were examined from a group of 55 patients undergoing CR.¹⁶ The patients significantly increased HRR from a mean of 15.4 beats per minute to a mean of 19.4 beats per minute. Subgroups of men, women, older, and younger patients all significantly improved HRR. Patients with recent surgical revascularization were compared with patients diagnosed with stable angina to adjust for possible effects of recovery after a cardiac event. The analysis showed that exercise in the CR setting is associated with improvement in HRR, and this improvement is likely the result of exercise and not solely of recovery from a debilitating event. No control group was available for the study.

A second study of 34 patients undergoing CR demonstrated that exercise training was associated with significant improvement in HRR from baseline to follow-up exercise stress testing relative to a control group of 35 patients.¹⁷ The subjects undergoing CR increased HRR by a mean of 4 beats per minute (standard deviation [SD] 5), whereas the control group decreased HRR by a mean of .6 beats per minute (SD 7). A control group of patients did not participate in exercise training and did not show improvements in HRR.

A third study by Kligfield and colleagues¹⁸ studied whether HRR after submaximal exercise is a modifiable characteristic in patients with heart disease and if so, whether HRR improves during 12 weeks of exercise training during CR in a group of 81 patients. Instead of looking at HRR after a treadmill test, this study evaluated HRR after exercise sessions, three at the beginning of the program and three at the end. Submaximal exercise effort is more likely to be done during routine activities of daily living. This study demonstrated that the effects of exercise training on neurohumoral tone as reflected by HRR are extended to periods of submaximal effort that are associated with routine activities of daily living.

The review of the literature suggests that one of the many potential benefits of exercise training is an improvement in autonomic tone, reflected by an improvement in HRR.⁵ HRR is a simple and readily available parameter for assessing autonomic tone.¹⁷ It may be useful as an outcome measure for risk stratifying patients after treadmill stress tests.¹⁷ It also may be useful for identifying patients at risk for subsequent cardiac events and motivating those patients to adhere to an exercise program.¹⁷

Purpose

Exercise in the CR setting has demonstrated a reduction in mortality from CV causes.² The exact mechanism by which exercise achieves benefits is unknown, but one theory suggests the effect exercise produces on autonomic tone. HRR, a measure of autonomic tone, has been shown to be an independent predictor of mortality, but few studies have shown that HRR can be improved by exercise. Therefore, the purpose of this study was to examine whether HRR improves as a result of the exercise training during CR to add to the current body of knowledge about HRR.

Research questions

The research questions for this study were as follows: (1) Will the total sample of patients undergoing CR significantly improve HRR after completion of CR? (2) Will changes in HRR differ between men and women? (3) Will changes in HRR differ in those aged less than 65 years (younger) compared with those aged 65 years or more (older)? (4) Is there a relationship between age and HRR?

Methods

This study was a retrospective, quasi-experimental design. The setting for this study was the UNC Hospitals Cardiac Rehabilitation program located in the UNC Wellness Center, Chapel Hill, North Carolina. This setting is part of UNC-Hospitals, a large, southeastern, 665-bed hospital with a CR program that treats 150 patients per year.

The sample for this study included adult patients who completed 12 weeks of phase II CR between August 2003 and August 2004 and completed symptom limited exercise treadmill tests before entry into CR and after completing CR. Patients who had paced heart rhythms, atrial fibrillation, or Wolff-Parkinson-White syndrome were excluded from the analysis. Patients taking beta-blockers, medications that lower HR and blood pressure, were included in the analysis.

Phase II CR was conducted according to established protocols from the American Association of Cardiovascular and Pulmonary Rehabilitation.¹⁹ Patients were referred from their health care provider to the CR program after myocardial infarction, coronary bypass surgery, percutaneous coronary intervention (angioplasty or stent placement), a stable angina diagnosis, or other cardiac-related diagnoses. Before exercising in the rehabilitation program, patients underwent a symptom-limited treadmill stress test, medical evaluation from a cardiac nurse, and exercise evaluation from an exercise physiologist. The results from the exercise test were used to assist with exercise prescription during the CR program.

Patients exercised under medical supervision 3 days per week for 12 weeks. Each exercise session consisted of 10 to 15 minutes of aerobic warm-up and stretching, 30 to 35 minutes of continuous aerobic exercise on standard treadmills, bikes, elliptical trainers, and seated step machines, followed by 15 to 20 minutes of cool-down stretching and progressive relaxation. A second symptom-limited treadmill stress test was obtained at the completion of the program.

The Ramped Bruce Protocol was used for exercise testing for 87% of patients at entry and 100% of patients at exit. Other protocols used at entry were the Standard Bruce and the Modified Bruce. Exercise testing was performed in a similar manner for each patient. Patients were placed on a 12-lead ECG monitor; resting ECG, HR, and blood pressure were obtained. The cardiologist reviewed each patient's medical history and resting values. The exercise protocol was started, and each patient exercised until symptoms prevented further exercise or the test was stopped by the cardiologist because of ECG changes. Immediately after exercise the patients were placed in a supine position for 6 minutes of recovery. There was no cool-down period after exercise. HRs were recorded every minute during the exercise phase and every minute during the recovery phase. ECG tracings were printed for each minute of exercise and recovery.

Retrospective data collection was completed from charts of patients who had finished and had been discharged from the Phase II CR program. Data were obtained from the results of the two exercise treadmill stress tests that were performed before entry into CR and after 3 months of CR.

Measures

Resting HR, peak HR, and HR at 1-minute recovery was measured using standard 12-lead ECG equipment and interpreted by a cardiologist present during the exercise test. Estimated exercise tolerance was determined on the basis of standard tables and reported as the estimated maximal exercise test (MET) level.²⁰

Resting HR was defined as the number of QRS complexes per minute when the patient is seated, before exercise testing. Peak HR was defined as the maximum number of QRS complexes per minute obtained by the patient during exercise. HR at 1-minute recovery was defined as the number of QRS complexes per minute at 1 minute after peak exercise in a supine position. Subtracting HR at 1-minute recovery from the peak HR determined HRR. MET level was defined as an estimate of fitness level achieved during graded exercise tests (where 1 MET equals 3.5 mL of oxygen uptake per kilogram of body weight per minute).²⁰

Statistical methods

HRR and other exercise testing variables were compared before and after CR for all groups with the paired *t* test. To compare differences in improvements between gender and age, an independent *t* test was used. A Pearson correlation was used to test a relationship between age and HRR. For all comparisons, a *P* value less than .05 was required to determine significance.

Results

Demographic data for the sample are found in Table I. There was an improvement in HRR after 3 months of CR for the total sample ($P = .020$), which represents an increase of 13.6%. Improvements for the total group include HRR ($P = .020$), MET level ($P < .000$), resting HR ($P = .020$), and peak HR ($P = .002$) (Table II).

Table I. Demographics

Total sample	N = 100	Age = 64.0 y (SD 12.1)*
Men	n = 69 (69%)	Age = 64.7 y (SD 12.1)*
Women	n = 31 (31%)	Age = 62.5 y (SD 12.4)*
Younger (<65 y)	n = 50 (50%)	Age = 53.7 y (SD 6.8)*
Older (≥65 y)	n = 50 (50%)	Age = 74.3 y (SD 5.8)*
Race		
White	n = 85 (85%)	
Black	n = 12 (12%)	
Latino	n = 3 (3%)	
Referral reason		
Myocardial infarction	n = 38 (38%)	
Revascularization†	n = 48 (48%)	
Medical management	n = 12 (12%)	
CHF/VR	n = 2 (2%)	
Diabetes	n = 23 (23%)	
Beta-blocker use	n = 86 (86%)	

CHF, Chronic heart failure; VR, valve replacement; SD, standard deviation.

* Mean (SD).

† Revascularization includes coronary artery bypass surgery and coronary intervention.

Table II. Exercise testing results for the total sample before and after cardiac rehabilitation

N = 100	Baseline	12 wk	P value	% change
MET level	8.3 (SD 2.9)	10.7 (SD 3.0)	.000*	+28.9%
Resting HR	69.0 (SD 11.0)	64.0 (SD 13.1)	.018*	-7.2%
Peak HR	123.3 (SD 22.0)	128.5 (SD 23.1)	.002*	+4.2%
HRR	21.4 (SD 11.2)	24.3 (SD 11.4)	.020*	+13.6%

HR, Heart rate; HRR, heart rate recovery; MET, maximal exercise test.

* Significant result.

Table III. Exercise testing results by gender before and after cardiac rehabilitation

	Baseline	12 wk	P value	% change
MET level				
M	9.0 (SD 3.1)	11.5 (SD 3.0)	.000*	+27.8%
F	6.8 (SD 1.8)	8.8 (SD 2.1)	.000*	+29.4%
Resting HR				
M	66.0 (SD 11.7)	63.0 (SD 13.6)	.063	-4.5%
F	69.3 (SD 9.1)	66.4 (SD 11.9)	.125	-4.2%
Peak HR				
M	124.6 (SD 23.0)	131.5 (SD 22.4)	.001*	+5.5%
F	120.4 (SD 19.6)	121.7 (SD 23.5)	.627	+1.1%
HRR				
M	22.2 (SD 11.7)	25.2 (SD 11.0)	.047*	+13.5%
F	19.6 (SD 10.1)	22.4 (SD 12.1)	.233	+14.3%

HR, Heart rate; HRR, heart rate recovery; MET, maximal exercise test; SD, standard deviation.

M = male (n = 69).

F = female (n = 31).

* Significant result.

There was an improvement in HRR for the male subgroup ($P = .047$). Improvements for males included HR ($P = .047$), MET level ($P < .0001$), and peak HR ($P = .001$). Overall, HRR increased by 13.5%. Females increased the MET level ($P < .0001$), and overall HRR increased by 14.3%. Although there was an absolute improvement in HRR, this was not statistically significant ($P = .233$). However, there was no significant difference in the improvement of HRR based on gender, indicating males and females improve at similar rates ($P = .833$) (Table III).

Improvements for younger subjects included MET level ($P < .000$) and peak HR ($P = .016$). Similarly, older subjects improved their MET level ($P < .000$). HRR increased by 15% for younger subjects and increased by 12.8% for older subjects. Although there was an absolute improvement in HRR for both younger and older subjects, this was not statistically significant ($P = .064$ and $P = .159$, respectively). There was no significant difference in the improvement of HRR based on age, indicating older subjects improve similarly to younger subjects ($P = .700$) (Table IV).

Table IV. Exercise testing results by age before and after cardiac rehabilitation

	Baseline	12 wk	P value	% change
MET level				
Y	9.4 (SD 3.1)	11.7 (SD 3.2)	.000*	+24.5%
O	7.3 (SD 2.4)	9.6 (SD 2.5)	.000*	+31.5%
Resting HR				
Y	67.1 (SD 10.5)	63.1 (SD 14.0)	.055	-6.0%
O	66.9 (SD 11.7)	65.0 (SD 12.3)	.168	-2.8%
Peak HR				
Y	128.9 (SD 21.5)	134.6 (SD 22.0)	.016*	+4.4%
O	117.6 (SD 21.2)	122.4 (SD 22.9)	.062	+4.1%
HRR				
Y	23.3 (SD 10.9)	26.7 (SD 11.3)	.064	+14.6%
O	19.5 (SD 11.4)	22.0 (SD 11.1)	.159	+12.8%

HR, Heart rate; HRR, heart rate recovery; MET, maximal exercise test; SD, standard deviation.

Y = <65 y (n = 50).

O = \geq 65 y (n = 50).

* Significant result.

There was no relationship between age and HRR ($r = .009$; $P = .929$). Accordingly, in this sample as age increased, there was not a decrease in HRR.

Discussion

Demographics

This sample was similar to those in other studies of CR in that the majority of subjects are men, with women underrepresented despite similar occurrence rates of CV disease.^{1,21} Women participating in CR tend to be 10 years older than men; however, in this study the average age for women was younger (62.5 years) than the men (64.7 years).²¹

The women, although younger than previous studies, presented with a lower exercise tolerance than the men (6.8 METs vs 9.0 METs) and reached a lower peak HR (120.4 beats/min vs 124.6). This was consistent with other studies of gender differences in CR.²² Despite starting at lower levels of fitness, women increased their exercise tolerance from 6.8 METs to 8.8 METs, a 29.4% increase. Cannistra and colleagues²³ reported similar results in their study in which women increased the MET level by 30%.

Several studies have shown that there was no significant difference in improvement of exercise capacity, represented by MET level, between men and women.²² Men and women in this study improved their HRR at similar rates, which was a consistent finding with other measures of exercise improvement.

Similarly to other studies of CR, peak exercise tolerance before and after training was lower in older patients compared with the younger patients. Older patients improved from 7.3 METs to 9.6 METs after training compared with younger patients who improved from 9.4 METs to 11.7 METs. This represents a 24.5% increase in MET level for younger patients and a 31.5% increase in older patients. Lavie and Milani²⁴ reported similar results with elderly patients (≥ 65 years) increasing exercise tolerance by 43% compared with a 32% increase in the younger patients (< 65 years). In both studies, elderly patients seemed to achieve greater gains in exercise tolerance than younger patients, which may be because younger patients have a higher baseline exercise tolerance.

Although CAD disproportionately affects older patients, CR populations tend to have fewer older patients compared with younger patients.^{1,25} This study showed an equal proportion of older and younger patients (50% each), which was consistent with other studies of CR populations.

Heart rate recovery

HRR after exercise is an easily obtainable clinical variable, reflecting PNS activity, that has shown to be a strong predictor of all-cause mortality in various patient populations including healthy patients, patients with heart failure, and patients with CAD.²⁶ Abnormal HRR has been associated with an increased risk, therefore, modifying HRR may change risk.²⁷ Exercise training has established effects on resting autonomic tone but an emerging benefit may be modulation of the autonomic response to exercise, specifically, endurance training is associated with higher PNS tone and lower SNS tone during exercise.²⁸

Exercise training during CR was associated with an improvement in HRR, which may represent an improvement of autonomic tone. These findings were consistent with previous studies that evaluated the effect of exercise-based CR on HRR that showed improvement in HRR.¹⁶⁻

¹⁸ Results from Hao and colleagues¹⁶ and Tiukinhoy and colleagues¹⁷ permit suitable comparison because these studies used exercise testing to obtain HRR data.

Tiukinhoy and colleagues¹⁷ reported a significant improvement in HRR in the intervention subjects with a mean increase of 4 (SD 5) compared with our result of 2.94 (SD 12.4) (Table V). These authors used a control group for comparison that did not show an improvement in

HRR. For the intervention group, Tiukinhoy and colleagues' intervention group showed an increase in HRR of 22% compared with our result of a 13.6% increase.

Table V. Mean increase in heart rate recovery before and after cardiac rehabilitation

Total sample	2.94 beats/min (SD 12.4)*
Men	3.00 beats/min (SD 12.3)*
Women	2.81 beats/min (SD 12.8)
Younger	3.42 beats/min (SD 12.8)
Older	2.46 beats/min (SD 12.2)

SD, Standard deviation.

* Significant result.

Hao and colleagues¹⁶ compared results from subgroups similar to those in our study. These authors reported a 26% increase in HRR for the total sample (N = 55) compared with a 13.6% improvement in our results. Hao and colleagues¹⁶ also reported improvements for both genders and both age groups, whereas our results found improvement only in the male subgroup.

Results from previous studies and this study are similar and suggest an improvement in HRR. One plausible explanation for the increase in HRR may be the healing and time after an acute event such as a myocardial infarction, bypass surgery, or coronary stent placement rather than a training effect. However, Hao and colleagues¹⁶ reported a significant improvement in HRR in a subgroup of patients with chronic stable angina who were not recovering from an acute event. This suggests that the improvement was not simply the chronologic pattern of recovery from a debilitating event.¹⁶ In addition, patients usually have up to 1 year after an event to begin CR, and patients who have had bypass surgery typically do not start strenuous exercise for 3 months. Data regarding time between the precipitating event and the start of CR were not collected in this study.

Another possible explanation for the increase in HRR after CR is the effect of beta-blocking medications. Beta-blockers decrease resting HR and reduce the ability to achieve maximum HR; consequently, it is possible that there was an impact of beta-blocker use on HRR.²⁶ Previous studies have been inconsistent over the effects of beta-blockers on HRR. Desai and colleagues²⁹ reported that beta-blockers affect HR in recovery most likely through an indirect effect by reduction of chronotropism.

Conversely, Racine and colleagues²⁶ reported that beta-blocker therapy did not seem to significantly improve HRR up to 3 minutes after maximal exercise test. In addition, Shetler and colleagues⁹ found that the administration of beta-blockers had no significant impact on the prognostic value of HRR. The effect of beta-blockers remains controversial and warrants further study.

A third possible explanation of the improvement in HRR in this study and others may be the effect of exercise training on autonomic function. Endurance training significantly affects autonomic control of the heart, increasing PNS activity and decreasing SNS activity at rest.³⁰ Athletes have been shown to have a lower resting HR and a more rapid recovery of HR after exercise as the result of enhanced PNS activity produced by long-term training.³⁰ Aging is

associated with a reduction in PNS control of the heart that is partly the result of a decrease in physical fitness, but the decline in fitness can be modified by regular exercise, which will increase PNS activity.³⁰ Yamamoto and colleagues³¹ studied the effects of endurance training on autonomic control. The authors reported that through endurance training, changes in autonomic control contribute to a decrease in HR at rest and during post-exercise recovery, and that adaptation of autonomic control occurs more rapidly in immediate post-exercise periods than at rest.

Limitations

One limitation of the current study was the lack of a control group who did not participate in CR for comparison of results. The inherent bias of the retrospective design was also a limitation of the study. A prospective design with consistent application of exercise testing protocols and data collection would yield more complete data from which stronger conclusions could be drawn.

The sample size (N = 100) was relatively small, although larger than comparable studies. A larger sample would increase confidence in the results of this study. However, a post hoc power calculation indicated that the study had moderate power to detect differences between the subgroups.

The sample was a convenience sample of patients from one urban facility in North Carolina. The sample was mostly male, and the majority was white.

The effect of beta-blockers on HRR remains an area of considerable debate.²⁷ Doses of beta-blockers were not routinely held the day of testing. Beta-blocker use could not be controlled during the period between the two exercise stress tests. Once started on beta-blockers, most patients did not discontinue them; however, doses may have been changed in the period between tests. Doses of beta-blockers were not reported because documentation was not optimal.

Finally, this study included only patients who had the physical ability to complete two exercise stress tests, which requires the capability to walk on a treadmill. Some patients were excluded because of the inability to complete a stress test. These patients were prescribed exercise by other means and were able to perform aerobic exercise during CR, but were not included in this study. Patients with more physical limitations probably gain equivalent benefit from CR, but they were excluded from this study.

Implications for future research

HRR has been shown to be a predictor of mortality, and this study demonstrated that HRR improved in a sample of patients who completed CR. It is not known whether improving HRR will have a meaningful effect on survival.²⁷ Future research is needed to more adequately address whether improving HRR improves mortality.

The effect of beta-blockers on HRR also needs to be investigated further. Studies have differed in their conclusions as to whether beta-blockers affect HRR. Until this issue is resolved or beta-blocker use is better controlled, this could be a limitation to any study of HRR.

The samples that have been used to show that exercise improves HRR are all from CR programs. To generalize results, future studies need to include larger, non-CR subjects, more minorities, and more women. Clearly, more research is needed to determine how to incorporate HRR into the management of patients with and without heart disease.⁵

Conclusion

HRR improves in patients who complete CR. Improving HRR strengthens the rationale for promoting exercise. Nurses should encourage participation in CR programs for all patients with CAD but especially for groups known to be underrepresented, such as women, minorities, and older patients.

References

1. American Heart Association. Heart disease and stroke statistics—2004 update. Dallas, TX: American Heart Association; 2003.
2. Ades PA. Cardiac rehabilitation and secondary prevention of coronary heart disease. *N Engl J Med* 2001;345(12):892-902.
3. Ades PA, Coello CE. Effects of exercise and cardiac rehabilitation on cardiovascular outcomes. *Med Clin North Am* 2000;84(1):251-65.
4. Shepard RJ, Balady GJ. Exercise as cardiovascular therapy. *Circulation* 1999;99:963-72.
5. Lauer MS. Is heart rate recovery a modifiable risk factor? *J Cardiopulm Rehabil* 2003;23:88-9.
6. Imai K, Sato H, Hori M, Kusuoka H, Ozaki H, Yokoyama H, et al. Vagally mediated heart rate recovery after exercise is accelerated in athletes but blunted in patients with chronic heart failure. *J Am Coll Cardiol* 1994;24(6):1529-35.
7. Schwartz PJ, LaRovere MT, Vanoli E. Autonomic nervous system and sudden cardiac death: experimental basis and clinical observations for post myocardial infarction risk stratification. *Circulation* 1992;85(Suppl I):I77-I91.
8. Gibbons RJ. Abnormal heart rate recovery after exercise. *Lancet* 2002;359:1536-7.
9. Shetler K, Marcus R, Froelicher VF, Vora S, Kalisetti D, Prakash M, et al. Heart rate recovery: validation and methodologic issues. *J Am Coll Cardiol* 2001;38:1980-7.
10. Cole CR, Foody JM, Blackstone EH, Lauer MS. Heart rate recovery after sub-maximal exercise testing as a predictor of mortality in a cardiovascularly healthy cohort. *Ann Intern Med* 2000;132:552-5.

11. Cole CR, Blackstone EH, Pashkow FJ, Sander CE, Lauer MS. Heart-rate recovery immediately after exercise as a predictor of mortality. *N Engl J Med* 1999;341(18):1351-7.
12. Vivekananthan DP, Blackstone EH, Pothier CE, Lauer MS. Heart rate recovery after exercise is a predictor of mortality, independent of the angiographic severity of coronary disease. *J Am Coll Cardiol* 2003;42:831-8.
13. Watanabe J, Marcus R, Froelicher VF, Vora S, Kalisetti D, Prakash M, et al. Heart rate recovery immediately after treadmill exercise and left ventricular systolic dysfunction as predictors of mortality. *Circulation* 2001;104:1911-6.
14. Cheng YJ, Lauer MS, Earnest CP, Church TS, Kampert JB, Gibbons LW, et al. Heart rate recovery following maximal exercise testing as a predictor of cardiovascular disease and all-cause mortality in men with diabetes. *Diabetes Care* 2003; 26:2052-57.
15. Seshadri N, Gildea TR, McCarthy K, Pothier C, Kavuru MS, Lauer MS. Association of an abnormal heart rate recovery with pulmonary function abnormalities. *Chest* 2004;125:1286-91.
16. Hao SC, Chai A, Kligfield P. Heart rate recovery response to symptom-limited treadmill exercise after cardiac rehabilitation in patients with coronary artery disease with and without recent events. *Am J Cardiol* 2002;90:763-5.
17. Tiukinhoy S, Beohar N, Hsie M. Improvement in heart rate recovery after cardiac rehabilitation. *J Cardiopulm Rehabil* 2003;23:84-7.
18. Kligfield P, McCormick A, Chai A, Jacobson A, Feuerstadt P, Hao SC. Effect of age and gender on heart rate recovery after submaximal exercise during cardiac rehabilitation in patients with angina pectoris, recent acute myocardial infarction, or coronary bypass surgery. *Am J Cardiol* 2003;92:601-3.
19. Balady GJ, Ades PA, Comoss P, Limacher M, Pina IL, Southard D, et al. Core components of cardiac rehabilitation/secondary prevention programs: a statement for health care professionals from the American Heart Association and the American Association of Cardiovascular and Pulmonary Rehabilitation. *Circulation* 2000;102:1069-73.
20. American College of Sports Medicine. ACSM's guidelines for exercise testing and prescription. 6th ed. Baltimore: Lippincott Williams & Wilkins; 2000.
21. Todaro JF, Shen BJ, Niaura R, Tilkemeier PL, Roberts BH. Do men and women achieve similar benefits from cardiac rehabilitation? *J Cardiopulm Rehabil* 2004;24:45-51.
22. Carhart RL, Ades PA. Gender differences in cardiac rehabilitation. *Cardiol Clin* 1998;16(1):37-42.
23. Cannistra LB, Balady GJ, O'Malley CJ, Weiner DA, Ryan TJ. Comparison of the clinical profile and outcome of women and men in cardiac rehabilitation. *Am J Cardiol* 1992;69:1274-9.

24. Lavie CJ, Milani RV. Effects of cardiac rehabilitation programs on exercise capacity, coronary risk factors, behavioral characteristics, and quality of life in a large elderly cohort. *Am J Cardiol* 1995;76:177-9.
25. Ades PA, Maloney A, Savage P, Carhart RL. Determinants of physical functioning in coronary patients. *Arch Intern Med* 1999;159:2357-60.
26. Racine N, Blachet M, Ducharme A, Marquis J, Boucher J, Juneau M, et al. Decreased heart rate recovery after exercise in patients with congestive heart failure: effect of B-blocker therapy. *J Card Fail* 2003;9(4):296-302.
27. Morise AP. Heart rate recovery: predictor of risk today and target of therapy tomorrow? *Circulation* 2004;110:2778-80.
28. Goldsmith RL, Bloomfield DM, Rosenwinkel ET. Exercise and autonomic function. *Coron Artery Dis* 2000;11:129-35.
29. Desai MY, Pena-Almaguer ED, Mannting F. Abnormal heart rate recovery after exercise as a reflection of abnormal chronotropic response. *Am J Cardiol* 2001;87:1164-9.
30. Carter JB, Banister EW, Blaber AP. Effect of endurance exercise on autonomic control of heart rate. *Sports Med* 2003; 33(1):33-46.
31. Yamamoto K, Miyachi M, Saitoh T, Yoshioka A, Onodera S. Effects of endurance training on resting and post-exercise cardiac autonomic control. *Med Sci Sports Exerc* 2001;33(9):1496-1502.