Published in final edited form as:

J Cardiovasc Nurs. 2014 ; 29(1): 38–47. doi:10.1097/JCN.0b013e31827324e2.

Improvements in Heart Rate Recovery Among Women After Cardiac Rehabilitation Completion

Theresa M. Beckie, PhD, FAHA,

Professor, College of Nursing, University of South Florida, Tampa

Jason W. Beckstead, PhD,

Associate Professor, College of Nursing, University of South Florida, Tampa

Kevin E. Kip, PhD, FAHA, and

Professor, College of Nursing, University of South Florida, Tampa

Gerald Fletcher, MD, FAHA

Professor of Medicine, Cardiovascular Diseases, Mayo Clinic College of Medicine, Jacksonville, Florida

Abstract

Background—Heart rate recovery (HRR) after exercise cessation is thought to reflect the rate of reestablishment of parasympathetic tone. Relatively little research has focused on improved HRR in women after completing cardiac rehabilitation (CR) exercise training.

Objective—We examined the influence of exercise training on HRR in women completing a traditional CR program and in women completing a CR program tailored for women.

Methods—A 2-group randomized clinical trial compared HRR between 99 women completing a traditional 12-week CR program and 137 women completing a tailored CR program. Immediately upon completion of a symptom-limited graded exercise test, HRR was measured at 1 through 6 minutes.

Results—Compared with baseline, improvement in 1-minute HRR (HRR_1) was similar ($P = 0.777$) between the tailored (mean [SD], 17.5 [11] to 19.1 [12]) and the traditional CR program (15.7 [9.0] to 16.9 [9.5]). The amount of change in the 2-minute HRR (HRR_2) for the tailored (30 [13] to 32.8 [14.6]) and traditional programs (28.3 [12.8] to 31.2 [13.7]) also was not different ($P = 0.391$). Similar results were observed for HRR at 3 through 6 minutes. Given these comparable improvements of the 2 programs, in the full cohort, the factors independently predictive of post-CR HRR_1 , in rank order, were baseline HRR_1 (part correlation, 0.35; $P < 0.001$); peak exercise capacity, estimated as metabolic equivalents (METs; 0.24, $P < 0.001$); anxiety (-0.17 , $P = 0.001$); and age (-0.13 , $P = 0.016$). The factors independently associated with post-CR HRR_2 were baseline HRR_2 (0.44, $P < 0.001$), peak METs (0.21, $P < 0.001$), and insulin use (-0.10 , $P = 0.041$).

Copyright © 2012 Wolters Kluwer Health | Lippincott Williams & Wilkins

Correspondence, Theresa M. Beckie, PhD, FAHA, College of Nursing, MDC 22, University of South Florida, 12901 Bruce B. Downs Blvd, Tampa, FL 33612 (tbeckie@health.usf.edu).

The authors have no conflicts of interest to disclose.

Conclusions—One to 6 minutes after exercise cessation, HRR was significantly improved among the women completing both CR programs. The modifiable factors positively associated with HRR₁ included peak METs and lower anxiety, whereas HRR₂ was associated with insulin administration and peak METs. Additional research on HRR after exercise training in women is warranted.

Keywords

cardiac rehabilitation; exercise; heart rate recovery; women

Coronary heart disease (CHD) is the single greatest threat to the health of American women.¹ Multi-disciplinary cardiac rehabilitation/secondary prevention (CR/SP) programs are intended to slow CHD progression, improve cardiorespiratory fitness, and reduce subsequent cardiac events.^{2–4} Exercise training is an important core component of CR/SP programs.^{2,5,6} Before CR/SP participation, a symptom-limited graded exercise test (SL-GXT) is completed to assess risk and to provide the initial guidelines for individualized exercise prescriptions.⁷ Although robust evidence suggests that CR/SP programs result in improved exercise capacity of participants,⁴ relatively less research has examined SL-GXT indices during early recovery after exercise. Methodologically rigorous research exploring the efficacy of innovative, motivationally enhanced CR/SP programs for improving exercise capacity and heart rate (HR) recovery (HRR) among women is glaringly absent.

Heart rate recovery, the HR deceleration upon exercise cessation, is thought to reflect parasympathetic nervous system (PNS) reactivation⁷ and sympathetic nervous system (SNS) withdrawal.⁸ Heart rate recovery, reflecting complex interactions between the autonomic nervous system (ANS), metabolic system, and neuroendocrine system, has emerged as an important prognostic factor associated with mortality. Empirical evidence supports the link between blunted HRR after exercise and total and cardiovascular (CVS) mortality and sudden cardiac death in diverse cohorts.^{9–29} Poor ANS control of the CVS system is a plausible mechanism underlying the factors associated with attenuated HRR and increased risk for cardiac events and mortality. The preponderance of HRR research has focused on apparently healthy individuals,^{18,30} men,^{13–16,20,21,29} or patients with heart failure (HF).^{22,31–37}

Although the prognostic importance of HRR has been examined in predominantly healthy or asymptomatic women,^{13,27} few studies, with either small sample sizes and/or few women, have examined the modifiability of HRR with CR/SP exercise training.^{38–41} Retrospective studies primarily comprising men found improved HRR after CR/SP exercise and a strong association between abnormal HRR and all-cause mortality.^{42,43} Efforts to determine the significant predictors of blunted HRR have found that being female,^{9,12,13,27,30,44} having lower exercise capacity,^{13–15,19–21,29,30,45} being older,^{9,12–14} having HF or more comorbidities including diabetes,^{9,12,14,16,45} having multiple traditional risk factors,^{12,29,30,44–46} having more depression and anxiety,^{30,47–49} having a lower level of education,³⁰ and experiencing CVS events^{13,23,50} have been associated with abnormal HRR. In women participating in CR/SP programs, it is less clear which of these predictors are related to attenuated HRR and at what particular minute of HRR.

The extant HRR literature encompasses studies that examine heterogeneous populations and apply heterogeneous operational definitions. Notably, 3 features, (1) active versus passive recovery, (2) varying lengths of time in recovery, and (3) a variety of dichotomized cut points for defining normal/abnormal, have rendered the conduct of systematic reviews of the literature difficult. Evidence of sex differences in HRR is mixed with data suggesting no sex differentials between men and both healthy women and women with CHD^{13,43,46} and with data indicating that HRR in women is lower than in men.^{12,40} Comparisons across studies of both the antecedents and the consequences of HRR are challenging and result in few evidence-based recommendations for clinical practice²⁰ for healthy compared with diseased cohorts or for age-specific and sex-specific populations.

Few prospective, methodologically rigorous studies have examined either the effect of exercise training on HRR in women completing CR/SP or the significant predictors of HRR at 1, 2, and 3 minutes after exercise cessation. No studies were found that examined the efficacy of a motivationally enhanced CR/SP program specifically designed for women on HRR. Therefore, this study sought to examine the influence of exercise training on HRR in women completing a traditional CR/SP program compared with women completing a motivationally enhanced CR/SP program. We also explored the significant predictors of HRR at multiple time points after exercise cessation.

Methods

After we obtained informed consent, the eligible women were randomized to either the gender-specific CR/SP program (henceforth labeled *tailored*) or a traditional CR/SP program. Details of the methodological design, recruitment, retention, and baseline characteristics and depressive symptom, perceived health, and quality-of-life outcomes are provided elsewhere.^{51–56} The institutional review board of the university and the participating hospital approved the study protocol.

Participants

Women referred to a phase II CR/SP program in Florida from 2004 to 2008 were recruited as participants. The sample included women older than 21 years (1) diagnosed with acute myocardial infarction or stable angina or having undergone coronary artery bypass graft surgery or percutaneous coronary intervention within the last year and (2) able to read, write, and speak English. We excluded women with (1) health insurance coverage for fewer than 36 electrocardiogram (ECG)–monitored exercise sessions, (2) cognitive impairment, and (3) an inability to ambulate. We randomly allocated 111 women to the traditional CR/SP and 141 women to the tailored program, of which 99 and 137, respectively, completed the study.

Traditional Cardiac Rehabilitation

The traditional CR/SP program, certified by the American Association of Cardiovascular and Pulmonary Rehabilitation, was delivered by nurses and exercise physiologists using a case management model. The ECG-monitored and supervised exercise protocol included aerobic exercise and resistance training 3 days per week for 12 weeks. The exercise

consisted of 5 minutes of warm-up exercises and 35 to 45 minutes of aerobic exercise (treadmill walking, cycling, or rowing), with exercise HRs maintained at 60% to 80% of the maximal HR calculated from the baseline SL-GXT. Exercise intensity was adjusted to maintain participant Borg scale rating of perceived exertion between “light” and “somewhat hard” (12–14 on a scale of 6–20).⁵⁷ Resistance training included wall pulleys and hand weights, followed by 5 minutes of cooldown exercises. The women in the traditional CR/SP freely selected any scheduled mixed-gender exercise session between 8 AM and 4 PM. The CR/SP staff also conducted education classes focused on CHD risk factor reduction for 8 weeks.

Tailored Cardiac Rehabilitation

The tailored CR/SP exercise protocol was identical to that of the traditional program except for the exclusion of men and the restriction of intervention implementation to 1 period when the CR/SP facility was closed. In addition to exercise training, a clinical nurse specialist and a clinical psychologist facilitated psychoeducational sessions for 10 consecutive weeks. All exercise and education sessions were enhanced with stage-of-change– matched strategies guided by the transtheoretical model of behavior change⁵⁸ and were delivered with a motivational interviewing counseling style⁵⁹ by the nurses and the exercise physiologists.

Physiological and Psychosocial Measures

Body mass index was calculated as weight (kilogram) divided by the square of height (meter). Body fat composition was determined using skinfold measurements of the suprailium, the triceps, and the thigh.⁶⁰ Waist circumference was measured by trained staff and reported in centimeters. Lipid profiles and serum glucose were measured after a 12-hour fast using the Cholestech LDX System (Haywood, California). Urine specimens were screened for exposure to nicotine by immunoassay at a threshold cotinine level of 25 ng/mL; positive samples were confirmed by gas chromatography (MedTox Laboratories, St Paul, Minnesota). The Charlston Comorbidity Index measured the burden of comorbid diseases.⁶¹

The standards for SL-GXT were followed,^{62,63} and 1 cardiologist supervised all tests using 1 treadmill. Cardio-respiratory fitness was evaluated using the modified Bruce protocol and expressed in metabolic equivalents (METs), where a MET typically equals 3.5 mL of oxygen consumption per kilogram per minute; METs were estimated from the final treadmill speed and grade.⁶⁴ Baseline SL-GXTs were obtained within 3 months after the index cardiac event and within 1 week before initiating CR/SP, whereas exit tests were obtained 1 week after CR/SP completion. Medication regimens were maintained on the usual schedule, and all SL-GXTs were conducted in the morning. The participants were encouraged not to grip the handrail and to exert maximal effort to volitional fatigue or until the occurrence of abnormal clinical signs or symptoms. A second cardiologist, masked to the group assignment, interpreted all SL-GXTs. Termination criteria for exercise testing included fatigue, leg discomfort, dyspnea, abnormal blood pressure (BP) responses, or ischemic ECG changes. The patients were immediately seated after the SL-GXT without a cooldown walk.

Blood pressure was measured manually, and HR was measured by continuous ECG monitoring at 1 to 6 minutes after exercise cessation. Heart rate recovery was defined as the maximum HR minus the HR at specified minutes after the exercise and represented the drop in HR during that time interval. Maximum rate pressure product was defined as the product of the highest HR and BP divided by 100 obtained during the last stage of exercise. Exercise attendance was calculated as the number of sessions completed of 36 prescribed sessions. Education attendance was expressed as the percentage of the number of sessions attended because the tailored intervention comprised 10 sessions and the traditional intervention consisted of 8 sessions.

Anxiety was measured with the 20-item State Anxiety Inventory,⁶⁵ with higher scores reflecting more anxiety. The 20-item Center for Epidemiological Studies Depression Scale measured depressive symptoms.⁶⁶ Scores range from 0 to 60, with higher scores reflecting greater depressive symptoms. The SF-36 Health Survey⁶⁷ version 2 comprises 36 questions, measuring health perceptions on 8 subscales. The subscales were totaled and transformed to a 0 to 100 scale, with higher scores representing better-perceived health.

Data Analysis

Data were analyzed using IBM SPSS version 20 for Windows. Univariate and bivariate distributions were examined using frequencies and cross-tabulation procedures to check for outliers and to assess normality. Descriptive statistics including means, standard deviations, Pearson correlations, and percentages describe the participants and the study variables. Categorical variables were compared by random assignment using the χ^2 test and by use of independent-samples *t* tests for continuous variables. Urine cotinine levels were log transformed. Analysis of variance was used to assess the changes by random assignment in the exercise test variables among the 236 women who completed the intervention. In addition, paired *t* tests were used to examine the magnitude of change in the exercise test variables separately for each treatment regimen. To determine the predictors of post-CR/SP HRR for the full cohort, we first examined all bivariate correlations between the HRR variables and more than 100 baseline and post-CR/SP variables including lipid profiles, psychosocial characteristics, comorbidities, medications, anthropometric measures, and sociodemographic characteristics. From these data, 15 variables that were correlated with HRR at 1 through 6 minutes (at $r \geq 0.2$) were selected as potential predictors. Hierarchical linear regression on these 15 predictors (age, systolic BP, Charlston Comorbidity Index, 5 SF-36 Health Survey version 2 subscales [physical functioning, social functioning, bodily pain, role physical, and role emotional], depressive symptoms, anxiety, fasting glucose, insulin use, peak METs, urine cotinine (current smoking status), and left ventricular ejection fraction) was used to predict post-CR/SP HRR, controlling for the baseline levels. All tests were 2-tailed and were evaluated for statistical significance using an α criterion of 0.05.

Results

Participants

Of the 252 women randomized, 236 completed the trial, with SL-GXT data on 232 women. Baseline characteristics were similar across the randomized groups. With a mean (SD) age

of 63 (12) years, most women were white and had undergone a percutaneous coronary intervention or a coronary artery bypass graft surgery during their index admission (Table 1). Except for angiotensin-converting enzyme inhibitors, baseline consumption of evidence-based medications did not differ between the women in the 2 treatment groups, and medications remained stable throughout the study. The participants in the tailored group attended nominally more exercise sessions (mean [SD], 33 [8] versus 30 [8]; $F_{1,234} = 6.575$, $P = 0.01$) and had a markedly higher percentage of education sessions than did the traditional group (88.5 [21] versus 60.5 [30]; $F_{1,234} = 71.168$, $P < 0.001$). Compared with baseline, improvements in the SL-GXT parameters were similar between the tailored and traditional CR/SP programs (Table 2).

Heart Rate Recovery

Table 3 shows the amount of change from baseline to post-CR/SP in HRR for 6 minutes after exercise cessation. The women in the tailored group made statistically significant yet relatively modest improvements on most of the HRR measures from baseline to post-CR/SP, whereas those in the traditional CR/SP group seemed to demonstrate less improvement. Nonetheless, improvements in HRR were not statistically different between the 2 groups. Given the comparable improvements of the 2 CR/SP programs, HRR data were combined for subsequent analyses. Figure illustrates that HRR during a 6-minute recovery period for the full cohort was statistically significantly improved from baseline to post-CR/SP ($P < 0.05$ for all). The greatest change was observed between the 1 and 2 minutes into recovery. Compared with baseline, the women achieved improved 1-minute HRR (HRR₁; mean [SD], 16.8 [10.2] to 18.2 [11.1]; $F_{1,230} = 3.91$, $P < 0.05$) and 2-minute HRR (HRR₂; 30 [13] to 32.2 [14.2], $F_{1,230} = 7.52$, $P < 0.05$). Similar improvements were demonstrated for 3-minute (HRR₃) through 6-minute HRRs.

Predictors of HRR for the Total Sample

Separate regression models were constructed to predict HRR₁, HRR₂, and HRR₃ and to facilitate comparisons with previous research. Forward stepwise regression using the 15 predictors resulted in a final model with 4 predictors of HRR₁ shown in Table 4. In rank order, the factors predictive of post-CR HRR₁ were baseline HRR₁ (part [or semipartial] correlation, 0.35; $P < 0.001$), post-CR peak METs (0.24, $P < 0.001$), anxiety (−0.17, $P = 0.001$), and age (−0.13, $P = 0.016$).

The hierarchical regression model shown in Table 5 reveals that the factors independently associated with post-CR HRR₂, in rank order, were baseline HRR₂ (part correlation, 0.44; $P < 0.001$), post-CR peak METs (0.21, $P < 0.001$), and insulin use (−0.10, $P = 0.041$). Post-CR peak METs was the strongest predictor of post-CR HRR₂. The significant predictors of post-CR HRR₃, in rank order, were baseline HRR₃ (part correlation, 0.48; $P < 0.001$), post-CR peak METs (0.23, $P < 0.001$), and post-CR Center for Epidemiological Studies Depression Scale (depression) scores (−0.12, $P = 0.01$; Table 6). Post-CR METs was the strongest predictor of HRR₃. Additional exploratory analyses identified identical predictors of both HRR₃ and 4-minute HRR (data not shown). Further, regression models for predicting 5-minute HRR and 6-minute HRR showed that post-CR peak METs was the strongest predictor, with part correlations of 0.29 ($P < 0.001$) and 0.32 ($P < 0.001$),

respectively. Post-CR peak systolic BP was also significantly associated with 5-minute HRR (0.12, $P = 0.013$) and 6-minute HRR (0.12, $P = 0.012$; data not shown).

Discussion

The purpose of this article was to examine the effects of a traditional compared with a motivationally enhanced CR/SP program on HRR among women. We also sought to explore the sociodemographic and clinical variables that best predicted HRR at 1, 2, and 3 minutes after exercise cessation. To our knowledge, this is the first randomized clinical trial to examine the effects of 2 different methods of CR/SP delivery on HRR in women. The primary findings of the current analyses were that, compared with baseline, HRR improved similarly at each of the 6-minute HRR time points in both CR/SP programs and that HRR was related to different subsets of predictors depending upon time in recovery. Heart rates decreased rapidly within the first 2 minutes after exercise cessation. Our data concur with the view that the intensity of PNS reactivation steadily increases further until 4 minutes into recovery and remains relatively constant thereafter.⁸ In patients with and without CHD, Wang et al⁶⁸ found that PNS effects on HR reached a peak about 2 minutes into recovery from exercise, whereas SNS effects dissipated more slowly, with significant increases in plasma catecholamines and HR even 45 minutes after exercise. Johnson and Goldberger⁹ suggest that late HRR reflects withdrawal of SNS drive of exercise, with lower HRR indicating more persistent SNS excitation. The SNS seems to have a longer latency in returning to baseline after exercise cessation.

A few small studies showed that HRR was modifiable with CR/SP exercise training.^{38–40,69} Two retro-spective studies reported an improved HRR₁ of 4 beats per minute (bpm) after CR/SP completion.^{38,39} Kligfield et al,⁴⁰ measuring HRR after 3 exercise sessions at the beginning and 3 exercise sessions at the end of CR/SP, found that the HRR averaged during the 3 sessions increased a mean of 3.4 bpm, with lower gains achieved by older patients and women ($n = 23$). Three more recent retrospective studies found improved HRR after CR/SP exercise training, all dichotomizing HRR₁ as 12 bpm or lower or higher than 12 bpm to define normalcy but using various cooldown strategies.^{42,43,70}

MacMillan and colleagues⁴³ reported an improved HRR₁ of 2.9 bpm among participants (69% men) completing CR/SP exercise training, with no gender or age differences. Jolly et al⁴² found that, compared with baseline, post-CR/SP HRR₁ improved by 3.4 bpm among 1070 CR/SP participants (77% men). Of the patients with abnormal HRR₁ at entry, 41% normalized after CR/SP completion and 59% did not. Independent predictors of failure to improve HRR₁ included older age, unimproved exercise capacity, previous HF, and peripheral arterial disease. Adams et al⁷⁰ described an improved HRR₁ of 3 bpm after CR/SP ($n = 210$; 59% men; mean age, 58 years). Finally, compared with nonparticipants, Chinese patients (23% women) who had acute myocardial infarction completing a 16-session CR/SP had improved HRR₁ by 1.5 bpm,⁴¹ and those with poorer HRR₁ were older and, more often, diabetic and hypertensive.

Our data are in agreement with the collective findings of previous CR/SP studies demonstrating an improvement in HRR₁ of between 1.5 and 4 bpm and an association

between blunted HRR and increased age, poor exercise capacity, and diabetes. Despite methodological design weaknesses and disparate HRR assessments, previous research seems to support the modifiability of HRR after CR/SP exercise training. Although HRR provides complementary evidence of the benefits of CR/SP exercise training beyond improved METs, the clinical significance and antecedents of improved HRR require clarification in large, prospective studies of women.

As mentioned above, peak METs was the strongest independent predictor of post-CR/SP HRR₁, HRR₂, and HRR₃, controlling for baseline values. However, a somewhat different set of variables predicted HRR at 1, 2, and 3 minutes after exercise cessation, with older age, more insulin use, and greater anxiety and depression generally associated with lower HRR. The biological plausibility that different predictors influence different HRR time points is supported by previous studies in both healthy and diseased populations. Arena et al⁴⁴ identified different variables associated with HRR₁ and HRR₂ in healthy individuals. They and others⁷¹ speculate that differences in the relative rates of PNS activation and SNS withdrawal immediately after exercise cessation contribute to the variable strength of correlations with elapsed HRR time.

Inconsistencies across HRR studies are starkly evident in the variability (1) of the approach (active or passive) for obtaining HRR, (2) of the specific minute of recovery evaluated, and (3) in the dichotomous cut point adopted for defining normal/abnormal HRR. First, the methods of HRR₂ assessment have included using a passive recovery period, with the participants placed supine immediately after peak exercise,^{14,21,23,25,26,43} immediately seated,^{18,41,50,70} or standing for 2 minutes and then seated for several minutes.⁴⁴ The methods using an active cooldown recovery have included walking at 1.5 mph and at 2.5% grade for 2 minutes^{15,17,20,42} or at a speed of 1 mph for 1 minute.¹² Second, HRR has been examined during a range of minutes into recovery. Third, the use of numerous dichotomous cut points designating abnormal HRR include 6 beats at 1 minute,^{32,36} 12 beats at 1 minute,^{12,15,17,27,41,42,70} 18 beats at 1 minute,^{25,28,72} 21 beats at 1 minute,⁷³ 25 beats at 1 minute,¹⁹ 22 beats at 2 minutes,^{14,17,18,23,74} 42 beats at 2 minutes,^{18,25} 46 beats at 3 minutes,²⁹ 50 beats at 3 minutes,⁷⁵ and 55 beats at 5 minutes.¹⁶

Thus, these inconsistencies of approaches, time lapse in recovery, and specific dichotomous cut points have resulted in little consensus regarding the most appropriate tactic for obtaining HRR. Although dichotomization is often done for simplicity of statistical analysis and interpretation of results, there are sound arguments that dichotomizing continuous variables leads to a considerable loss of power, should not be applied to independent or dependent variables in regression models, and leads to loss of information.^{76–78} Using dichotomous cut points is also unrealistic with individuals close to but on the opposite sides of the cut point, characterized as having very different rather than very similar outcomes.^{79,80} We have provided detailed summary statistics in our tables including F statistics and effect sizes to assist future researchers in powering their studies and to facilitate future meta-analyses.

Summary and Implications

Heart rate recovery from 1 to 6 minutes after exercise cessation was significantly improved among women completing both the tailored and the traditional CR/SP program. The modifiable factors positively associated with HRR included peak exercise capacity, nonuse of insulin, and fewer anxiety and depressive symptoms. Each minute of recovery was associated with somewhat unique predictors providing support for evaluating HRR for several minutes rather than at a single time. Further investigation into the determinants, measurement, physiological mechanisms, and prognostic significance of ANS function during recovery after exercise cessation is warranted to better delineate its relation to mortality risk, cardiorespiratory fitness, and subsequent cardiac events in women with CHD. Improving modifiable risk factors such as depression and anxiety may improve exercise capacity in women participating in CR/SP programs. The role of exercise training for women participating in CR/SP programs and the patient characteristics associated with changes in HRR over time also require further exploration. Building consensus among researchers and clinicians as to the “best” operational definition of HRR could facilitate systematic reviews and enhance evidence-based treatments.

Acknowledgments

Funding for this study was provided by the National Institute of Nursing Research (5R01 NR07678).

REFERENCES

1. Roger VL, Go AS, Lloyd-Jones DM, et al. Heart disease and stroke statistics—2012 update: a report from the American Heart Association. *Circulation*. 2012; 125(1):e2–e220. [PubMed: 22179539]
2. Balady GJ, Ades PA, Bittner VA, et al. Referral, enrollment, and delivery of cardiac rehabilitation/secondary prevention programs at clinical centers and beyond: a presidential advisory from the American Heart Association. *Circulation*. 2011; 124(25):2951–2960. [PubMed: 22082676]
3. Smith SC Jr, Benjamin EJ, Bonow RO, et al. AHA/ACCF secondary prevention and risk reduction therapy for patients with coronary and other atherosclerotic vascular disease: 2011 update: a guideline from the American Heart Association and American College of Cardiology Foundation. *Circulation*. 2011; 124(22):2458–2473. [PubMed: 22052934]
4. Sandercock G, Hurtado V, Cardoso F. Changes in cardiorespiratory fitness in cardiac rehabilitation patients: a meta-analysis [published online ahead of print]. *Int J Cardiol*. 2011
5. Balady GJ, Williams MA, Ades PA, et al. Core components of cardiac rehabilitation/secondary prevention programs: 2007 update: a scientific statement from the American Heart Association Exercise, Cardiac Rehabilitation, and Prevention Committee, the Council on Clinical Cardiology; the Councils on Cardiovascular Nursing, Epidemiology and Prevention, and Nutrition, Physical Activity, and Metabolism; and the American Association of Cardiovascular and Pulmonary Rehabilitation. *J Cardiopulm Rehabil Prev*. 2007; 27(3):121–129. [PubMed: 17558191]
6. Corra U, Piepoli MF, Carre F, et al. Secondary prevention through cardiac rehabilitation: physical activity counselling and exercise training: key components of the position paper from the Cardiac Rehabilitation Section of the European Association of Cardiovascular Prevention and Rehabilitation. *Eur Heart J*. 2010; 31(16):1967–1974. [PubMed: 20643803]
7. Balady GJ, Arena R, Sietsema K, et al. Clinician’s guide to cardiopulmonary exercise testing in adults: a scientific statement from the American Heart Association. *Circulation*. 2010; 122(2):191–225. [PubMed: 20585013]
8. Lahiri MK, Kannankeril PJ, Goldberger JJ. Assessment of autonomic function in cardiovascular disease: physiological basis and prognostic implications. *J Am Coll Cardiol*. 2008; 51(18):1725–1733. [PubMed: 18452777]

9. Johnson NP, Goldberger JJ. Prognostic value of late heart rate recovery after treadmill exercise. *Am J Cardiol.* 2012; 110(1):45–49. [PubMed: 22463837]
10. Guazzi M, Myers J, Peberdy MA, Bensimhon D, Chase P, Arena R. Heart rate recovery predicts sudden cardiac death in heart failure. *Int J Cardiol.* 2010; 144(1):121–123. [PubMed: 19193457]
11. Freeman JV, Dewey FE, Hadley DM, Myers J, Froelicher VF. Autonomic nervous system interaction with the cardiovascular system during exercise. *Prog Cardiovasc Dis.* 2006; 48(5):342–362. [PubMed: 16627049]
12. Maddox TM, Ross C, Ho PM, et al. The prognostic importance of abnormal heart rate recovery and chronotropic response among exercise treadmill test patients. *Am Heart J.* 2008; 156(4):736–744. [PubMed: 18926155]
13. Mora S, Redberg RF, Sharrett AR, Blumenthal RS. Enhanced risk assessment in asymptomatic individuals with exercise testing and Framingham risk scores. *Circulation.* 2005; 112(11):1566–1572. [PubMed: 16144993]
14. Myers J, Tan SY, Abella J, Aleti V, Froelicher VF. Comparison of the chronotropic response to exercise and heart rate recovery in predicting cardiovascular mortality. *Eur J Cardiovasc Prev Rehabil.* 2007; 14(2):215–221.
15. Nishime EO, Cole CR, Blackstone EH, Pashkow FJ, Lauer MS. Heart rate recovery and treadmill exercise score as predictors of mortality in patients referred for exercise ECG. *JAMA.* 2000; 284(11):1392–1398. [PubMed: 10989401]
16. Cheng YJ, Lauer MS, Earnest CP, 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(7):2052–2057. [PubMed: 12832312]
17. Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MS. Heart-rate recovery immediately after exercise as a predictor of mortality. *N Engl J Med.* 1999; 341(18):1351–1357. [PubMed: 10536127]
18. Cole CR, Foody JM, Blackstone EH, Lauer MS. Heart rate recovery after submaximal exercise testing as a predictor of mortality in a cardiovascularly healthy cohort. *Ann Intern Med.* 2000; 132(7):552–555. [PubMed: 10744592]
19. Jouven X, Empana JP, Schwartz PJ, Desnos M, Courbon D, Ducimetiere P. Heart-rate profile during exercise as a predictor of sudden death. *N Engl J Med.* 2005; 352(19):1951–1958. [PubMed: 15888695]
20. Kokkinos P, Myers J, Doumas M, et al. Heart rate recovery, exercise capacity, and mortality risk in male veterans. *Eur J Prev Cardiol.* 2012; 19(2):177–184. [PubMed: 21450594]
21. Lipinski MJ, Vetrovec GW, Froelicher VF. Importance of the first two minutes of heart rate recovery after exercise treadmill testing in predicting mortality and the presence of coronary artery disease in men. *Am J Cardiol.* 2004; 93(4):445–449. [PubMed: 14969619]
22. Lipinski MJ, Vetrovec GW, Gorelik D, Froelicher VF. The importance of heart rate recovery in patients with heart failure or left ventricular systolic dysfunction. *J Card Fail.* 2005; 11(8):624–630. [PubMed: 16230267]
23. Morshedi-Meibodi A, Larson MG, Levy D, O'Donnell CJ, Vasan RS. Heart rate recovery after treadmill exercise testing and risk of cardiovascular disease events (The Framingham Heart Study). *Am J Cardiol.* 2002; 90(8):848–852. [PubMed: 12372572]
24. 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(5):831–838. [PubMed: 12957428]
25. Shetler K, Marcus R, Froelicher VF, et al. Heart rate recovery: validation and methodologic issues. *J Am Coll Cardiol.* 2001; 38(7):1980–1987. [PubMed: 11738304]
26. Leeper NJ, Dewey FE, Ashley EA, et al. Prognostic value of heart rate increase at onset of exercise testing. *Circulation.* 2007; 115(4):468–474. [PubMed: 17242274]
27. Daugherty SL, Magid DJ, Kikla JR, et al. Gender differences in the prognostic value of exercise treadmill test characteristics. *Am Heart J.* 2011; 161(5):908–914. [PubMed: 21570521]
28. Yanagisawa S, Miki K, Yasuda N, Hirai T, Suzuki N, Tanaka T. The prognostic value of treadmill exercise testing in very elderly patients: heart rate recovery as a predictor of mortality in octogenarians. *Europace.* 2011; 13(1):114–120. [PubMed: 21084358]

29. Gayda M, Bourassa MG, Tardif JC, Fortier A, Juneau M, Nigam A. Heart rate recovery after exercise and long-term prognosis in patients with coronary artery disease. *Can J Cardiol.* 2012; 28(2):201–207. [PubMed: 22336522]
30. Carnethon MR, Sternfeld B, Liu K, et al. Correlates of heart rate recovery over 20 years in a healthy population sample. *Med Sci Sports Exerc.* 2012; 44(2):273–279. [PubMed: 21796053]
31. Zavin A, Arena R, Joseph J, et al. Dynamic assessment of ventilatory efficiency during recovery from peak exercise to enhance cardiopulmonary exercise testing [published online ahead of print]. *Eur J Prev Cardiol.* 2012
32. Arena R, Guazzi M, Myers J, Peberdy MA. Prognostic value of heart rate recovery in patients with heart failure. *Am Heart J.* 2006; 151(4):851. e7-851.e131. [PubMed: 16569547]
33. Arena R, Myers J, Abella J, et al. The prognostic value of the heart rate response during exercise and recovery in patients with heart failure: influence of beta-blockade. *Int J Cardiol.* 2010; 138(2): 166–173. [PubMed: 18804882]
34. Arena R, Myers J, Abella J, et al. Prognostic characteristics of heart rate recovery according to sex in patients with heart failure. *Int J Cardiol.* 2010; 145(2):293–294. [PubMed: 19910060]
35. Guazzi M, Myers J, Ann Peberdy M, et al. Heart rate recovery and tissue Doppler echocardiography in heart failure. *Clin Cardiol.* 2010; 33(2):E61–E64. [PubMed: 20043341]
36. Myers J, Arena R, Dewey F, et al. A cardiopulmonary exercise testing score for predicting outcomes in patients with heart failure. *Am Heart J.* 2008; 156(6):1177–1183. [PubMed: 19033016]
37. Driss AB, Tabet JY, Meurin P, et al. Heart rate recovery identifies high risk heart failure patients with intermediate peak oxygen consumption values. *Int J Cardiol.* 2011; 149(2):284–285. [PubMed: 21450358]
38. 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(7):763–765. [PubMed: 12356394]
39. Tiukinhoy S, Beohar N, Hsie M. Improvement in heart rate recovery after cardiac rehabilitation. *J Cardiopulm Rehabil.* 2003; 23(2):84–87. [PubMed: 12668927]
40. 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(5):600–603. [PubMed: 12943886]
41. Hai JJ, Siu CW, Ho HH, Li SW, Lee S, Tse HF. Relationship between changes in heart rate recovery after cardiac rehabilitation on cardiovascular mortality in patients with myocardial infarction. *Heart Rhythm.* 2010; 7(7):929–936. [PubMed: 20346416]
42. Jolly MA, Brennan DM, Cho L. Impact of exercise on heart rate recovery. *Circulation.* 2011; 124(14):1520–1526. [PubMed: 21947293]
43. MacMillan JS, Davis LL, Durham CF, Matteson ES. Exercise and heart rate recovery. *Heart Lung.* 2006; 35(6):383–390. [PubMed: 17137939]
44. Arena R, Arrowood JA, Fei D, Shelar S, Helm S, Kraft KA. The influence of sex on the relationship between heart rate recovery and other cardiovascular risk factors in apparently healthy subjects. *Scand J Med Sci Sports.* 2010; 20(2):291–297. [PubMed: 19522754]
45. Neves VR, Kiviniemi AM, Hautala AJ, et al. Heart rate dynamics after exercise in cardiac patients with and without type 2 diabetes. *Front Physiol.* 2011; 2:57. [PubMed: 21922009]
46. Nilsson G, Hedberg P, Jonason T, Lonnberg I, Ohrvik J. Heart rate recovery is more strongly associated with the metabolic syndrome, waist circumference, and insulin sensitivity in women than in men among the elderly in the general population. *Am Heart J.* 2007; 154(3):460. e1-460.e7. [PubMed: 17719290]
47. Hughes JW, Casey E, Luyster F, et al. Depression symptoms predict heart rate recovery after treadmill stress testing. *Am Heart J.* 2006; 151(5):1122. e1-122.e6. [PubMed: 16644348]
48. Hughes JW, York KM, Li Q, Freedland KE, Carney RM, Sheps DS. Depressive symptoms predict heart rate recovery after exercise treadmill testing in patients with coronary artery disease: results from the Psychophysiological Investigation of Myocardial Ischemia study. *Psychosom Med.* 2008; 70(4):456–460. [PubMed: 18434491]

49. Gordon JL, Ditto B, Lavoie KL, et al. The effect of major depression on postexercise cardiovascular recovery. *Psychophysiology*. 2011; 48(11):1605–1610. [PubMed: 21806634]
50. Mora S, Redberg RF, Cui Y, et al. Ability of exercise testing to predict cardiovascular and all-cause death in asymptomatic women: a 20-year follow-up of the lipid research clinics prevalence study. *JAMA*. 2003; 290(12):1600–1607. [PubMed: 14506119]
51. Beckie TM. A behavior change intervention for women in cardiac rehabilitation. *J Cardiovasc Nurs*. 2006; 21(2):146–153. [PubMed: 16601534]
52. Beckie TM, Beckstead JW. Predicting cardiac rehabilitation attendance in a gender-tailored randomized clinical trial. *J Cardiopulm Rehabil Prev*. 2010; 30(3):147–156. [PubMed: 20216324]
53. Beckie TM, Beckstead JW. The effects of a cardiac rehabilitation program tailored for women on global quality of life: a randomized clinical trial. *J Womens Health (Larchmt)*. 2010; 19(11):1977–1985. [PubMed: 20846018]
54. Beckie TM, Beckstead JW. The effects of a cardiac rehabilitation program tailored for women on their perceptions of health: a randomized clinical trial. *J Cardiopulm Rehabil Prev*. 2011; 31(1):25–34. [PubMed: 21037482]
55. Beckie TM, Fletcher GF, Beckstead JW, Schocken DD, Evans ME. Adverse baseline physiological and psychosocial profiles of women enrolled in a cardiac rehabilitation clinical trial. *J Cardiopulm Rehabil Prev*. 2008; 28(1):52–60. [PubMed: 18277832]
56. Beckie TM, Beckstead JW, Schocken DD, Evans ME, Fletcher GF. The effects of a tailored cardiac rehabilitation program on depressive symptoms in women: a randomized clinical trial. *Int J Nurs Stud*. 2011; 48(1):3–12. [PubMed: 20615504]
57. Borg GA. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc*. 1982; 14(5):377–381. [PubMed: 7154893]
58. Prochaska, JO.; Norcross, JC.; DiClemente, CC. *Changing for Good*. New York, NY: HarperCollins; 1994.
59. Rollnick, S.; Miller, W.; Butler, C. *Motivational Interviewing in Health Care: Helping Patients Change Behavior*. New York, NY: Guilford Press; 2008.
60. Jackson AS, Pollock ML, Ward A. Generalized equations for predicting body density of women. *Med Sci Sports Exerc*. 1980; 12(3):175–181. [PubMed: 7402053]
61. Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chronic Dis*. 1987; 40(5):373–383. [PubMed: 3558716]
62. Gibbons RJ, Balady GJ, Bricker JT, et al. ACC/AHA 2002 guideline update for exercise testing: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1997 Exercise Testing Guidelines). *Circulation*. 2002; 106(14):1883–1892. [PubMed: 12356646]
63. Myers J, Arena R, Franklin B, et al. Recommendations for clinical exercise laboratories: a scientific statement from the American Heart Association. *Circulation*. 2009; 119(24):3144–3161. [PubMed: 19487589]
64. American College of Sports Medicine. *Guidelines for Exercise Testing and Prescription*. Philadelphia, PA: Lippincott, Williams & Wilkins; 2009.
65. Spielberger, CD.; Gorsuch, RL.; Lushene, RE. *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists; 1983.
66. Radloff L. The CES-D scale: a self-report depression scale for researching the general population. *Appl Psychol Meas*. 1977; 1(3):385–401.
67. Ware, JE.; Kosinski, M.; Dewey, JE. *How to Score Version 2 of the SF-36 Health Survey*. Lincoln, RI: QualityMetric Incorporated; 2000.
68. Wang NC, Chicos A, Banthia S, et al. Persistent sympathetic excitation long after submaximal exercise in subjects with and without coronary artery disease. *Am J Physiol Heart Circ Physiol*. 2011; 301(3):H912–H920. [PubMed: 21666114]
69. Tsai SW, Lin YW, Wu SK. The effect of cardiac rehabilitation on recovery of heart rate over one minute after exercise in patients with coronary artery bypass graft surgery. *Clin Rehabil*. 2005; 19(8):843–849. [PubMed: 16323383]

70. Adams BJ, Carr JG, Ozonoff A, Lauer MS, Balady GJ. Effect of exercise training in supervised cardiac rehabilitation programs on prognostic variables from the exercise tolerance test. *Am J Cardiol.* 2008; 101(10):1403–1407. [PubMed: 18471449]
71. Rosenwinkel ET, Bloomfield DM, Arwady MA, Goldsmith RL. Exercise and autonomic function in health and cardiovascular disease. *Cardiol Clin.* 2001; 19(3):369–387. [PubMed: 11570111]
72. Zaim S, Schesser J, Hirsch LS, Rockland R. Influence of the maximum heart rate attained during exercise testing on subsequent heart rate recovery. *Ann Noninvasive Electrocardiol.* 2010; 15(1): 43–48. [PubMed: 20146781]
73. Georgoulas P, Orfanakis A, Demakopoulos N, et al. Abnormal heart rate recovery immediately after treadmill testing: correlation with clinical, exercise testing, and myocardial perfusion parameters. *J Nucl Cardiol.* 2003; 10(5):498–505. [PubMed: 14569243]
74. Curtis JM, Horton ES, Bahnson J, et al. Prevalence and predictors of abnormal cardiovascular responses to exercise testing among individuals with type 2 diabetes: the Look AHEAD (Action for Health in Diabetes) study. *Diabetes Care.* 2010; 33(4):901–907. [PubMed: 20056948]
75. Adabag AS, Grandits GA, Prineas RJ, Crow RS, Bloomfield HE, Neaton JD. Relation of heart rate parameters during exercise test to sudden death and all-cause mortality in asymptomatic men. *Am J Cardiol.* 2008; 101(10):1437–1443. [PubMed: 18471455]
76. MacCallum RC, Zhang S, Preacher KJ, Rucker DD. On the practice of dichotomization of quantitative variables. *Psychol Methods.* 2002; 7(1):19–40. [PubMed: 11928888]
77. Beckstead JW, Beckie TM. How much information can metabolic syndrome provide? An application of information theory. *Med Decis Making.* 2011; 31(1):79–92. [PubMed: 20729508]
78. Beckstead JW. To be, or to be two, the question of dichotomizing variables. *Int J Nurs Stud.* 2012; 49(6):635–636. [PubMed: 22591545]
79. Altman DG, Royston P. The cost of dichotomising continuous variables. *BMJ.* 2006; 332(7549): 1080. [PubMed: 16675816]
80. Royston P, Altman DG, Sauerbrei W. Dichotomizing continuous predictors in multiple regression: a bad idea. *Stat Med.* 2006; 25(1):127–141. [PubMed: 16217841]

What's New and Important

- Heart rate recovery is an easily attainable exercise tolerance test parameter for evaluating the improvements in health functioning gained after cardiac rehabilitation.
- Each minute of recovery was associated with somewhat unique predictors providing support for evaluating heart rate recovery for several minutes rather than at a single time.
- Improving modifiable risk factors such as depression and anxiety may improve exercise capacity in women participating in cardiac rehabilitation programs.
- Building consensus among researchers and clinicians as to the “best” operational definition of heart rate recovery could facilitate systematic reviews and enhance evidence-based treatments.

FIGURE.

Heart rate recovery before and after cardiac rehabilitation. $P < 0.05$ for all heart rate recovery time points from baseline to post-cardiac rehabilitation.

TABLE 1

Baseline Characteristics by Treatment Group

Characteristic	Traditional CR (n = 99)	Tailored CR (n = 137)
Age, mean (SD), y	64 (11)	63 (11)
Race or ethnicity, n (%)		
Non-Hispanic white	61 (62)	95 (69)
African American	17 (17)	22 (16)
Hispanic	20 (20)	19 (14)
Asian	1 (1)	1 (1)
Education, n (%)		
High school or lower	42 (42)	58 (42)
Some college	41 (41)	46 (34)
Baccalaureate degree	9 (9)	24 (17)
Graduate degree	7 (7)	9 (6.5)
Diagnostic eligibility for cardiac rehabilitation, n (%)		
PCI	42 (42.4)	76 (55.5)
CABG	39 (39.4)	40 (29.2)
Stable angina	14 (14.1)	14 (10.2)
MI	4 (4.1)	7 (5.1)
Cardiovascular history		
Previous MI	16 (16.2)	26 (19)
Previous CABG	9 (9.1)	9 (6.6)
Previous PCI	14 (14.1)	18 (13.1)
Previous stroke	8 (8.1)	8 (5.8)
Medications, n (%)		
β -Blockers	83 (84)	110 (80)
Statins	89 (90)	121 (88)
Aspirin	87 (88)	115 (84)
Clopidogrel	56 (57)	93 (68)
Angiotensin-converting enzyme inhibitor	30 (30)	60 (44)
Angiotensin receptor blocker	28 (28)	30 (22)
Oral hypoglycemic agents	35 (35)	34 (25)
Nonsteroidal anti-inflammatory agents	23 (23)	35 (25)
Insulin	14 (14)	22 (16)
Ejection fraction, mean (SD)	54.1 (11.3)	54.1 (10.4)
Charlson Comorbidity Index, mean (SD)	2 (1.7)	1.8 (1.4)
Urine cotinine, mean (SD), ng/mL	166 (389)	106 (261)

χ^2 and t test were used for discrete and continuous variables, respectively; $P > 0.05$ for all except for angiotensin-converting enzyme inhibitor ($P = 0.042$).

Abbreviations: CABG, coronary artery bypass graft; CR, cardiac rehabilitation; MI, myocardial infarction; PCI, percutaneous coronary intervention.

TABLE 2
Improved Exercise Capacity by Treatment Group After Cardiac Rehabilitation (CR) (n = 232)

Variable	Traditional (n = 99)				Tailored (n = 137)			
	F ^a	η ²	Baseline	Post-CR	F ^b	η ²	Baseline	Post-CR
Resting HR, bpm	0.66	0.003	72.6 (13)	69.8 (12)	6.56 ^d	0.028	73.6 (6)	69.6 (10)
Resting SBP, mm Hg	0.28	0.001	124.3 (17.6)	119.6 (16)	6.93 ^d	0.029	121.2 (16.4)	115.2 (15)
Resting DBP, mm Hg	0.13	0.001	73.8 (10.7)	71.9 (9)	2.43	0.010	72.6 (9.6)	70.2 (9.5)
Peak HR, bpm	2.08	0.009	115.9 (23.6)	117.7 (24)	0.93	0.004	121.7 (22)	120.0 (21)
Peak SBP, mm Hg	0.23	0.001	166.3 (22.7)	161.4 (23)	3.87	0.017	162.3 (21.7)	159.0 (24)
Peak DBP, mm Hg	0.60	0.003	83.7 (12.2)	80.7 (12)	6.00 ^d	0.025	80.8 (12.4)	79.0 (12)
Peak RPP ((HR × SBP)/100)	0.60	0.003	195.5 (57)	192.5 (52)	0.35	0.002	199.0 (52)	191.0 (52)
Peak METs	<1	0.000	5.6 (2.3)	7.1 (2.8)	68.82 ^d	0.230	6.0 (2.7)	7.6 (2.8)
Peak treadmill time, mins	2.00	0.009	8.0 (3.3)	10 (3.7)	60.51 ^d	0.208	8.3 (3.7)	10.8 (3.3)
								133.45 ^d

Values are presented as mean (standard deviation). η² is a standardized effect size measure.

Abbreviations: DBP, diastolic blood pressure; HR, heart rate; METs, metabolic equivalents; RPP, rate pressure product; SBP, systolic blood pressure.

^aF test of group by time interaction (df = 1,230).

^bF test of simple main effect of time for traditional group (df = 1,230).

^cF test of simple main effect of time for tailored group (df = 1,230).

^dP < 0.05.

TABLE 3

Recovery Heart Rate Before and After Cardiac Rehabilitation (CR) ($n = 232$)

Variable	Between Group		Traditional (<i>n</i> = 99)		Tailored (<i>n</i> = 137)		<i>F</i> ^c	η^2
	<i>F</i> ^a	η^2	Baseline	Post-CR	Baseline	Post-CR		
Recovery HR, bpm								
1 min HR	2.10	0.009	100 (22)	101 (22)	104 (20)	101 (18)	3.82	0.016
2 min HR	0.65	0.003	88 (17)	86.5 (18)	90 (17)	87 (16)	4.86 ^d	0.021
3 min HR	2.19	0.010	82 (15)	81 (16)	85 (15)	82 (14)	7.48 ^d	0.032
4 min HR	1.43	0.006	80 (15)	79 (15)	82.5 (14)	79 (13)	8.14 ^d	0.035
5 min HR	1.64	0.007	78 (14)	77 (15)	81 (13.5)	78 (13)	6.51 ^d	0.029
6 min HR	2.68	0.012	77 (13)	77 (14)	81 (14)	78 (13)	6.02 ^d	0.027
HRR								
1min HRR	0.08	0.000	15.7 (9)	16.9 (9.5)	17.5 (11)	19.1 (12)	3.11	0.013
2 min HRR	0.74	0.003	28.2 (13)	31.2 (13.7)	5.51 ^d	31.3 (13)	2.15	0.009
3 min HRR	0.06	0.000	34.4 (14.8)	36.7 (14.7)	3.34	36.4 (14.5)	3.35	0.014
4 min HRR	0.56	0.002	36.2 (15.7)	38.9 (15)	4.01 ^d	39.3 (15)	1.53	0.007
5 min HRR	1.18	0.005	37.6 (15.8)	40.3 (15.9)	3.69	40.8 (15)	0.36	0.002
6 min HRR	0.03	0.000	38.3 (16.6)	40.5 (16.6)	2.20	40.5 (15)	2.18	0.010

Values are presented as mean (standard deviation). Two minutes and 4 minutes, $n = 224$; 5 minutes and 6 minutes, $n = 217$.

Abbreviations: bpm, beats per minute; HR, heart rate; HHR, heart rate recovery.

^a F test of group by time interaction ($df = 1,230$).

^b F test of simple main effect of time for traditional group ($df = 1,230$).

^c F test of simple main effect of time for tailored group ($df = 1,230$).

^d $p < 0.05$.

TABLE 4

Predictors of 1-Minute Heart Rate Recovery (HRR) After Cardiac Rehabilitation (CR)

Predictor	Unstandardized Coefficient (β)	Standard Error	Standardized Coefficient (β)
Step 1			
Intercept	8.897 ^a	1.218	
Baseline HRR ₁	0.556 ^a	0.062	0.509
Step 2			
Intercept	17.779 ^a	5.229	
Baseline HRR ₁	0.413 ^a	0.061	0.379
Age	-0.139 ^a	0.057	-0.138
State anxiety	-0.176 ^a	0.055	-0.171
Post-CR peak	1.077 ^a	0.234	0.273
METs			

Step 1, $R^2 = 0.259$, $F_{1,229} = 80.044$, $P < 0.001$; $R^2 = 0.132$, $F_{3,226} = 16.264$, $P < 0.001$. Final model (step 2), $R^2 = 0.391$, $F_{4,226} = 36.210$, $P < 0.001$.

Abbreviation: METs, metabolic equivalents.

^a $P < 0.05$.

TABLE 5

Predictors of 2-Minute Heart Rate Recovery (HRR) After Cardiac Rehabilitation (CR)

Predictor	Unstandardized Coefficient (β)	Standard Error	Standardized Coefficient (β)
Step 1			
Intercept	12.601 ^a	1.913	
Baseline HRR ₂	0.652 ^a	0.058	0.593
Step 2			
Intercept	7.961 ^a	2.378	
Baseline HRR ₂	0.530 ^a	0.061	0.483
Post-CR peak METs	1.205 ^a	0.291	0.238
Insulin use	-4.216 ^a	2.048	-0.108

Step 1, $R^2 = 0.351$, $F_{1,229} = 124.117$, $P < 0.001$; $R^2 = 0.069$, $F_{2,227} = 13.616$, $P < 0.001$. Final model (step 2), $R^2 = 0.421$, $F_{3,227} = 55.009$, $P < 0.001$.

Abbreviation: METs, metabolic equivalents.

^a $P < 0.05$.

TABLE 6

Predictors of 3-Minute Heart Rate Recovery (HRR) After Cardiac Rehabilitation (CR)

Predictor	Unstandardized Coefficient (β)	Standard Error	Standardized Coefficient (β)
Step 1			
Intercept	14.317 ^a	1.975	
Baseline HRR ₃	0.657 ^a	0.051	0.646
Step 2			
Intercept	10.850 ^a	2.502	
Baseline HRR ₃	0.534 ^a	0.053	0.525
Post-CR CES-D	-0.176 ^a	0.068	-0.124
Post-CR Peak METs	1.358 ^a	0.277	0.258

Step 1, $R^2 = 0.418$, $F_{1,228} = 163.494$, $P < 0.001$; $R^2 = 0.076$, $F_{2,226} = 16.945$, $P < 0.001$. Final model (step 2), $R^2 = 0.494$, $F_{3,226} = 73.417$, $P < 0.001$.

Abbreviations: CES-D, Center for Epidemiological Studies Depression Scale; METs, metabolic equivalents.

^a $P < 0.05$.