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Dynamic assessment of ventilatory efficiency during recovery from peak exercise to enhance cardiopulmonary exercise testing

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Abstract

Background—While cardiopulmonary exercise testing (CPX) assessment is generally regarded as an optimal means to assess functional capacity in heart failure (HF) patients, strength parameters are omitted. CPX indices collected in recovery may provide additional insight regarding function, including strength.

Design and methods—We performed a cross-sectional controlled study. Systolic HF patients (aged 50 years) and age-matched controls were assessed using CPX and strength evaluations. Standard CPX indices were assessed during exercise (peak oxygen consumption [VO_2], first ventilatory threshold [1stVT], and ventilatory efficiency [VE/VCO_2 slope]) as well as indices at 1-minute recovery (1 min VO_2 , 1 min VE/VCO_2 , and 1 min heart rate recovery [HRR]) and differences between peak and 1-minute recovery (VO_2 and VE/VCO_2). Lower extremity strength was evaluated using the 1-repetition maximum (1RM) and power.

Results—Seventy adults (31 HF; 39 controls), mean age 66.2 ± 9.7 years were evaluated. Peak VO_2 (15.4 ± 4.2 versus $23.4 \pm 6.6 \text{ mL O}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, $p < 0.0001$) and 1stVT (10.9 ± 2.1 versus $14.4 \pm 4.0 \text{ mL O}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, $p < 0.0001$) were diminished in HF versus controls and VE/VCO_2 slope was increased (42.3 ± 12.2 versus 35.4 ± 8.3 , $p < 0.01$). HF patients had reduced 1 min VO_2 (13.1 ± 2.9 versus $16.3 \pm 3.7 \text{ mL O}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, $p < 0.0001$), 1 min HRR (6.7 ± 11.4 versus 12.4 ± 7.6 beats, $p < 0.02$), and VO_2 (2.43 ± 2.3 versus $7.3 \pm 5.0 \text{ mL O}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, $p < 0.0001$) as well as increased 1 min VE/VCO_2 (37 ± 7.5 versus 31.5 ± 4.4 , $p < 0.001$) and VE/VCO_2 (1.17 ± 3.0 versus -0.5 ± 1.3 , $p < 0.0001$). Strength parameters were relatively lower in HF. While CPX

exercise parameters correlated with strength, stronger correlations were observed between CPX recovery parameters and strength.

Conclusions—CPX recovery indices corroborate disease-specific aerobic differences and distinguish differences in strength. Recovery ventilatory efficiency enhances CPX's value as a comprehensive physical function tool.

Keywords

Heart failure; cardiopulmonary functional testing; recovery kinetics; muscular strength

Introduction

Multiple reports support the application of cardiopulmonary exercise testing (CPX) as a gauge of functional capacity and disease severity in systolic heart failure (HF) patients.^{1,2} Pertinent gas exchange indices are collected during exercise, but with relatively less attention focused on gas exchange assessments during the recovery phase (after exercise is completed). However, post-exercise re-equilibration is a dynamic state entailing complex physiology, i.e. cardiovascular (cardiac output and perfusion), pulmonary (congestion and ventilatory dead space), metabolic (inflammation, insulin resistance, oxidative metabolism), neuroautonomic (sympathetic and parasympathetic), neurohormones (angiotensin II and endothelin), and skeletal muscle (chemoreceptors and ergoreflexes) effects,³ which affect CPX recovery dynamics, and which may be applied to complement CPX measures assessed during exercise.^{4,5}

The principle of assessing exercise recovery dynamics as a means to refine clinical evaluation has already been established. Heart rate recovery (HRR) dynamics provide a sensitive marker of autonomic performance; reduced HRR is associated with increased mortality, particularly due to increased propensity for sudden cardiac death.^{5–7}

Utility of CPX recovery dynamics also has precedent. Oxygen uptake (VO_2) usually returns to baseline more slowly in HF patients, i.e. a delay that has been demonstrated to be associated with reduced cardiac output⁸ as well as HF disease severity and poor prognosis.^{4,9–10}

In this study, we extend the concept of CPX recovery assessment by studying post-exercise ventilatory efficiency (VE/VCO_2). VE/VCO_2 involves physiological components that are distinct from VO_2 ¹¹ and which may better characterize features of HF that improve physical function evaluation.¹

While seminal studies by Piepoli et al.^{12,13} and Ponikowski et al.¹⁴ focused on the relationship between skeletal muscle and ventilation during exercise, assessment of muscle–ventilatory physiology during recovery may facilitate greater clinical discrimination since the pertinent physiology may be relatively less confounded by exercise-induced cardiovascular and pulmonary responses.

While most clinicians regard CPX as the optimal means to assess functional capacity in HF patients,² it provides no information regarding strength. Whereas strength is increasingly

recognized as a relevant dimension of cardiac health and disease,^{15,16} the omission of strength as an integral part of CPX assessment (the ‘gold standard’) is an ironic shortcoming. In fact, if strength evaluation is to be completed for HF patients, it must be assessed separately from CPX as an additional evaluation. This added burden often contributes to the omission of strength assessment altogether despite its clinical relevance.

Goals of this study were twofold. 1. To study CPX post-exercise recovery indices to clarify their relationship to traditional CPX-exercise indices, i.e. to confirm that greater aerobic capacity would correspond to a more rapid re-equilibration of VO_2 and VE/VCO_2 to resting levels. 2. To provide additional information regarding a relationship between VO_2 and VE/VCO_2 recovery and intrinsic muscle strength.

Methods

Subjects

Male systolic HF patients and age-matched controls were studied. HF patients were eligible if they had a left ventricular ejection fraction (LVEF) of $\geq 40\%$ within 6 months of enrollment due to either ischemic or idiopathic etiology. Patients with moderate or severe valvular disease and/or uncontrolled heart arrhythmia were excluded. Patients with a pacemaker and/or implantable cardioverter defibrillator (ICD) implantation were in sinus rhythm prior to, during, and after the exercise provocation. Patients were required to be receiving optimal medical therapy, including a beta-blocker and either an angiotensin-converting enzyme (ACE) inhibitor or angiotensin receptor blocker (ARB) for at least 3 months at the time of study enrollment. All were assessed by a cardiologist to ensure that they were euvolemic prior to CPX, strength assessments, and other functional testing. Comorbidities of severe pulmonary disease, severe peripheral vascular disease, or neuropathy were excluded.

Control subjects had no history of HF, severe pulmonary disease, or musculoskeletal diseases and were ineligible if they exercised on a regular basis. Control subjects were sedentary for 3 months prior to enrollment.

Comorbidities were evaluated on the basis of diagnoses found in the electronic medical record. Whereas some patients were listed as having peripheral vascular disease and/or chronic obstructive pulmonary disease, only those with mild disease (and which did not confound functional assessments in this study) were enrolled.

The study was approved by the VA Institutional Review Board and informed consent was obtained from each subject.

Aerobic testing

All subjects performed exercise testing on a motorized treadmill using a modified Balke protocol¹⁷ in conjunction with ventilatory expired gas analysis through a snorkel device; patients’ noses were clipped to force all breathing through the snorkel. The ventilatory expired gas analysis system (MedGraphics BreezeSuite, St. Paul, MN) was calibrated

according to manufacturer specifications prior to each test. Continuous gas exchange was assessed for each subject one minute prior to, during, and after the exercise provocation.

The recovery portion of the test included one minute of active cool-down (i.e. continued gas assessments as the subjects continued to walk on the treadmill at 1 mph with no incline). ECG waveforms, blood pressure, O₂ saturation, and subjective symptoms were also assessed before, during, and after exercise, consistent with routine clinical fashion. No subjects demonstrated desaturation, and SpO₂ remained above 95% for all exercise tests.

Exercise and recovery assessments included continuous HR, VO₂, and VE/VCO₂, monitoring. VE/VCO₂ was calculated as a ratio between VE and VCO₂ at peak exercise (peak VE/VCO₂). Peak VO₂ and peak VE/VCO₂ were evaluated as 30-second averages during the final stage of exercise. VE/VCO₂ was also evaluated during exercise; VE and VCO₂ values, acquired from the initiation of exercise to maximal exertion, were input into a spreadsheet software (Microsoft Excel, Microsoft Corp., Bellevue, WA) to calculate the VE/VCO₂ slope via least-squares linear regression ($y = mx + b$, $m = \text{slope}$). First ventilatory threshold (1stVT) was determined by the V-slope method.¹⁸ All of the subjects achieved a minimum peak respiratory exchange ratio (RER) of 1.0, an indicator of good exercise effort.

Recovery VO₂ and ventilatory efficiency (1 min VO₂ and 1 min VE/VCO₂, respectively) were measured at 1 minute of recovery after exercise. Change scores for VO₂ (ΔVO_2) was calculated by subtracting values obtained at peak exercise from values obtained at 1 minute of recovery. $\Delta \text{VE/VCO}_2$ was calculated as the difference between peak VE/VCO₂ and 1 min VE/VCO₂. HRR was also determined as the difference between peak HR and HR at 1 minute of recovery (1 min HRR). All recovery kinetics data were measured as 10 second averages.

Muscle strength and power testing

Muscle strength and power were measured using a pneumatic leg press (Keiser A420, Fresno, CA). Participants performed a leg press [measured by the full range of motion (i.e. knee extension and flexion)] using a baseline weight of 45 kg to assess maximal strength capacity. The Borg scale¹⁹ was used following each leg press to assess the rating of perceived exertion (RPE). Relative to the RPE, the weight was incrementally increased until subjects were no longer able to complete a full repetition or maintain proper form. The final successfully completed repetition was recorded as the one repetition maximum (1RM). The weight was then set to 60% of the 1RM and subjects completed up to 30 repetitions continuously. A power curve was generated for each repetition on a desktop computer that was connected to the Keiser leg press. Upon completion of the evaluation, the peak of each curve was determined and then an average was taken to assess sub-maximal power (power).

Statistical analysis

SAS statistical software version 9.0 (SAS, Cary, NC) was used to analyze the data and values are reported as mean $\pm$ SD (unless otherwise indicated). Comparison of HF and controls was determined using non-paired *t*-tests and Pearson correlations were used to

evaluate the correlation coefficients. A p -value of <0.05 was used to define statistical significance.

Results

A total of 70 subjects (mean age 66.2 ± 9.6 years) were enrolled, including 31 HF patients (mean LVEF 29%) and 39 controls. Demographics are listed in Table 1. HF patients were significantly more likely than controls to be using cardiac medications (beta-blockers, ACE-inhibitors, statins, and diuretics). HF patients also had more cardiac events and procedures (7 patients had coronary artery bypass graft [CABG] and 10 had a pacemaker/ICD implantation, meanwhile these were absent in control subjects). HF patients had increased incidence of chronic kidney disease (6 versus 0 patients, $p < 0.01$), hyperlipidemia (21 versus 17 patients, $p < 0.05$), myocardial infarction (6 versus 1 patients, $p < 0.05$), and peripheral vascular disease (3 versus 0 patients, $p < 0.05$) as compared with control subjects. HF patients and controls had similar incidence of diabetes mellitus type 2 (15 versus 11 patients, $p = 0.08$), chronic obstructive pulmonary disease (7 versus 3 patients, $p = 0.08$), and hypertension (19 versus 16 patients, $p = 0.09$).

Table 2 shows resting baseline and exercise data comparing HF and control groups. HF patients demonstrated significantly diminished VO_2 compared with controls and higher VE/VCO_2 with similar RER (indicating both groups achieved high exertion). Table 3 shows recovery dynamics. HF patients had prolonged HRR and reduced VO_2 recovery capacity as compared with control subjects. Likewise, both 1 min VO_2 and VO_2 were reduced in HF patients versus controls, consistent with a relatively delayed exercise recovery. VE/VCO_2 was also higher in HF patients indicating that HF-related ventilatory inefficiency was relatively prolonged. Table 4 shows that strength and power were also lower in HF patients.

Table 5 lists correlations between peak exercise and recovery assessments. 1 min VO_2 , 1 min VE/VCO_2 , and 1 min HRR correlated with all of the peak CPX indices in HF patients, corroborating recovery parameters. 1 min VE/VCO_2 showed stronger correlations with peak VO_2 ($r = -0.60$, $p < 0.001$) and VE/VCO_2 slope ($r = 0.74$, $p < 0.001$) as compared with 1 min HRR (with peak VO_2 : $r = 0.52$, $p < 0.01$ and VE/VCO_2 slope: $r = -0.52$, $p < 0.01$) in HF patients. Peak exercise correlated better with 1 min VO_2 and 1 min VE/VCO_2 as compared with VO_2 and VE/VCO_2 suggesting that evaluating indices at 1 minute of recovery are better at substantiating peak exercise and provide a more accurate reflection of pathophysiology, as compared with change scores.

Correlations between strength/power capacity and CPX indices are presented in Tables 6 (exercise) and 7 (recovery), respectively. Strength indices correlated with CPX indices during exercise, but stronger correlations between 1RM and both 1 min VO_2 and 1 min VE/VCO_2 were observed. 1 min VE/VCO_2 correlated significantly with the 1RM ($r = -0.62$, $p < 0.001$) in HF patients and approached significance with power ($r = -0.36$, $p = 0.058$). Moreover, correlations between 1 min VE/VCO_2 and strength parameters were also significant in controls, but remained relatively greater in HF. 1 min VO_2 correlated to 1RM ($r = 0.48$, $p < 0.01$) and power ($r = 0.39$, $p < 0.01$) in HF patients, suggesting that if assessed in aggregate, 1 min VO_2 and 1 min VE/VCO_2 may provide even greater diagnostic

discrimination. 1 min HRR was not associated with strength in HF or controls, suggesting that while HRR recovery has well-recognized utility as a prognostic indicator of cardiac arrhythmia, it relates to a physiology that differs from that which determines aerobic function or strength. Change scores showed no significant correlations with strength capacity in HF patients.

Discussion

Whereas VO_2 and HR recovery dynamics after exercise have been analyzed in prior studies,^{4,7,20} this is the first study to demonstrate utility of VE/VCO_2 after exercise. Moreover, this is the first study in which VO_2 and VE/VCO_2 recovery indices have been demonstrated to correlate with muscular strength. Such correlation is physiologically sound as ventilation entails physiological relationships integrating pulmonary, cardiovascular, autonomic, and skeletal muscle components.^{11,21}

Among healthy adults, recovery dynamics are an intuitive aspect of training when increased conditioning improves metabolism and thereby enables adults to achieve higher physical performance as well as a more rapid restoration of resting equilibrium after exercise.²² This has obvious medical implications. Delayed restoration of VO_2 after exercise has been described and correlates to low cardiac output.^{9,10,23} Prolonged elevation of VE/VCO_2 after exercise is consistent with this logic. HF patients tend to be more fatigued and/or dyspneic than healthy adults after exercise, requiring longer resting periods between activities.

Whereas VE/VCO_2 during exercise has been established as an important gauge of disease severity and pathophysiology,^{11,24,25} it has not been applied in recovery. However, it seems likely that VE/VCO_2 could serve as a sensitive marker of disease during recovery particularly because ventilatory efficiency provides such an important marker of performance. It certainly seems likely to corroborate physiological perturbations that also impact performance during exercise. Low cardiac output would, for example, likely increase end-diastolic pressures, pulmonary vascular congestion and ventilation.²⁶ Likewise, disease-related differences in pulmonary, metabolic, neuroautonomic, and neurohormonal physiologies would all likely affect multiple CPX parameters in exercise and recovery.^{26,27} However, elevated recovery VE/VCO_2 may be particularly useful in distinguishing peripheral abnormalities associated with HF. Direct skeletal muscle myopathy from HF and/or its interrelationship to underperfusion²⁸ contribute to elevated ergoreflexes and abnormal chemoreflexes²⁹ that may particularly affect recovery VE/VCO_2 and delay re-equilibration to resting values.

This capacity of VE/VCO_2 seems to have particular utility in distinguishing properties of muscle strength. Whereas strength assessment is often omitted from HF management due to the inherent challenge of assessment, our data suggest that recovery assessments provide perspective on muscular strength as a convenient and easily implemented modification of the CPX assessment.

While symptom-limited exercise capacity was once assumed to be determined primarily by central hemodynamic dysfunction, an expanding body of literature reveals the impact of

skeletal muscle (and associated effects on ventilation) on functional capacity. Skeletal muscle pathophysiology (particularly overactive ergoreflexes) corresponds to ventilatory inefficiency during exercise in HF^{13,14,30} and increased likelihood of exercise intolerance and associated symptoms of dyspnea and fatigue. We extended these findings to show that reduced muscular strength also correlates with ventilatory inefficiency in recovery. The recovery period not only constitutes a physiological transition period in which important physiological relationships are active, but it provides a convenient window for skeletal muscle assessment that is less likely to be confounded by the effects of exercise-induced ischemia, hemodynamic perturbations, and/or intrinsic lung abnormalities.

It is notable that 1 min HRR correlated with CPX exercise indices but not to strength. This discrepancy suggests the relatively unique utility of ventilatory parameters to distinguish skeletal muscle responses. Despite its important neuroautonomic value, the 1 min HRR seems to provide much less insight regarding muscle performance.

Despite the increasingly growing body of literature indicating the prognostic value of assessing recovery after CPX, the optimal strategy to characterize recovery remains unclear. Kriatselis et al.³¹ assessed recovery VO_2 and HR time constants with a monoexponential curve and showed that HF patients had increased VO_2 and HR time constants as compared with healthy controls, indicative of a slower recovery from peak exercise among HF patients. They also showed that patients with reduced functional capacity had prolonged recovery time constants as compared with HF patients who exhibited a higher functional capacity. de Groote et al.²³ showed that the ratio between VO_2 during exercise and in recovery was predictive of survival in HF patients. Nanas et al.⁴ evaluated recovery kinetics through linear regression models and half time recovery (i.e. the time it takes patients to reach 50% of their peak VO_2). Still, others^{10,32} assessed time constants during recovery and mean response times. We assessed both VO_2 and VE/VCO_2 at 1 minute of recovery as well as change scores (i.e. the difference between peak exercise and recovery). While the change scores did not correlate with either strength or power, our data suggest that VO_2 and VE/VCO_2 , at 1 minute of recovery are most reflective of muscular physiology.

Limitations

The most significant limitation of this study is its small sample size, yet the data still show significantly prolonged VO_2 recovery and HRR in HF, consistent with prior literature and suggesting high specificity of these data, such that our conclusions regarding VE/VCO_2 in recovery seem well substantiated. We were also limited in our analysis to a male cohort due to the VA HF population, limiting generalization to females diagnosed with HF. Hormonal effects may also be pertinent to potential sex-based differences in our findings as they are known to affect pulmonary pressures.³³ Thus, assessments on CPX recovery ventilation are needed to analyze women separately. Finally, this study did not include a longitudinal component with which to assess prognosis. It is not certain if ventilatory recovery assessment adds to established CPX prognostic discrimination. Further analysis of CPX-based ventilatory efficiency recovery data are needed in large populations over time to better clarify the diagnostic and prognostic implications of the recovery data.

Conclusion

Ventilatory efficiency recovery dynamics can be applied as a means to augment CPX evaluation, providing perspective regarding strength that complement standard aerobic indices.

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Table 1

Baseline characteristics

	HF (n = 31)	Controls (n = 39)	Significance
Age (years)	67.4±8.9	65.3±10.1	0.37
Weight (kg)	86.0±27.4	86.6±14.9	0.91
Height (meters)	1.7±0.07	1.7±0.09	0.98
Body mass index (kg/m ²)	28.9±7.4	30.2±7.8	0.46
Medications [<i>n</i> (percentage)]			
Beta-blocker	25 (36%)	2 (3%)	<0.0001
ACE-inhibitor	26 (37%)	6 (9%)	<0.0001
ARB 2	2(3%)	2 (3%)	0.80
Statin	25 (36%)	17 (24%)	<0.001
Diuretics	20 (29%)	5 (7%)	<0.0001

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; HF, heart failure.

Table 2

Baseline characteristics during rest and maximal exercise

	HF	Controls	Significance
Resting baseline			
Heart rate (bpm)	68.3±11.1	73.5±12.3	0.07
VO ₂ (mlO ₂ ·kg ⁻¹ ·min ⁻¹)	4.1±1.3	4.1±1.2	0.86
VE/VCO ₂	46.1±6.4	41.8±6.9	<0.05
Maximal exercise			
Peak VO ₂ (mlO ₂ ·kg ⁻¹ ·min ⁻¹)	15.4±4.2	23.4±6.6	<0.0001
1stVT (mlO ₂ ·kg ⁻¹ ·min ⁻¹)	10.9±2.1	14.4±4.0	<0.0001
Peak VE/VCO ₂	38.1±8.8	31.3±4.5	<0.001
VE/VCO ₂ slope	35.7±10.6	29.1±4.6	<0.01
Peak Heart Rate (bpm)	105.7±20.4	133.9±19.0	<0.0001
Peak RER	1.09±0.1	1.1±0.1	0.25

HF, heart failure; 1stVT, first ventilatory threshold; VE/VCO₂, ventilatory efficiency; RER, respiratory exchange ratio.

Table 3

Differences between HF patients and controls during recovery

	HF	Controls	Significance
1 min VO_2 ($\text{mlO}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$)	13.1 $\pm$ 2.9	16.3 $\pm$ 3.7	<0.0001
1 min VE/VCO_2	37.0 $\pm$ 7.5	31.5 $\pm$ 4.4	<0.001
1 min HRR	6.7 $\pm$ 11.4	12.4 $\pm$ 7.6	<0.02
VO_2 ($\text{mlO}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$)	2.43 $\pm$ 2.3	7.3 $\pm$ 5.0	<0.0001
VE/VCO_2	1.17 $\pm$ 3.0	-0.5 $\pm$ 1.3	<0.0001

HF, heart failure; VE/VCO_2 , ventilatory efficiency; HRR, heart rate recovery.

Table 4

Differences in strength/power between HF patients and controls

	HF	Controls	Significance
1RM (kg)	154.8±52.0	195.3±56.8	<0.01
Power (Watts)	226.4±99.2	313.3±130.6	<0.01

HF, heart failure; 1RM, 1-repetition maximum.

Table 5

Correlations between 1-minute recovery ventilatory variables and peak exercise

	1 min VO ₂		1 min VE/VCO ₂		1 min HRR		VO ₂		VE/VCO ₂	
	HF	Controls	HF	Controls	HF	Controls	HF	Controls	HF	Controls
Peak VO ₂	0.86 ^{***}	0.65 ^{***}	-0.60 ^{***}	-0.34 [*]	0.52 ^{**}	0.20	0.76 ^{***}	0.82 ^{***}	-0.37 ^{***}	-0.05
1stVT	0.67 ^{***}	0.65 ^{***}	-0.48 ^{**}	-0.35 [*]	0.51 ^{**}	-0.08	0.58 ^{**}	0.35 [*]	-0.16	0.01
VE/VCO ₂ slope	-0.59 ^{***}	-0.12	0.74 ^{***}	0.85 ^{***}	-0.52 ^{**}	-0.05	-0.14	-0.20	0.73 ^{***}	0.16

HF, heart failure; VE/VCO₂, ventilatory efficiency; HRR, heart rate recovery; 1stVT, first ventilatory threshold.

* $p<0.05$

** $p<0.01$

*** $p<0.001$.

Table 6

1 minute recovery in relation to strength capacity

	1 min VO ₂		1 min VE/VCO ₂		1 min HRR		VO ₂		VE/VCO ₂	
	HF	Controls	HF	Controls	HF	Controls	HF	Controls	HF	Controls
1RM	0.48 ^{**}	0.14	-0.62 ^{***}	-0.34 [*]	0.09	-0.07	0.19	0.16	-0.15	0.33 [*]
60%RM power	0.39 [*]	0.12	-0.36	-0.34 [*]	0.01	0.08	0.08	0.12	0.05	0.10

HF, heart failure; VE/VCO₂, ventilatory efficiency; HRR, heart rate recovery; 1RM, one repetition maximum.^{*}
p<0.05^{**}
p<0.01^{***}
p<0.001.

Table 7

Correlations between peak exercise indices and strength capacity

	Peak VO ₂		1stVT		VE/VCO ₂ slope	
	HF	Controls	HF	Controls	HF	Controls
1RM	0.44 [*]	0.26	0.34	0.25	-0.37 [*]	-0.25
60%RM power	0.32	0.22	0.26	0.20	-0.13	-0.26

HF, heart failure; 1stVT, first ventilatory threshold; VE/VCO₂, ventilatory efficiency; HRR, heart rate recovery; 1RM, one repetition maximum.

^{*} $p<0.05$