Further Increase in Oxygen Uptake During Early Active Recovery Following Maximal Exercise in Chronic Heart Failure*

Hiroyuki Daida, MD; Thomas G. Allison, PhD, MPH; Bruce D. Johnson, PhD; Ray W. Squires, PhD; and Gerald T. Gau, MD, FCCP

**Study objective:** Some patients with chronic heart failure manifest a further increase in oxygen uptake (\( \dot{V}O_2 \)) after maximal exercise whereas others do not. The purpose of this study was to determine the characteristics of chronic heart failure patients with further increase in \( \dot{V}O_2 \) in early active recovery following maximal exercise.

**Design:** Retrospective analysis of clinical and exercise testing characteristics in patients with or without a further increase in \( \dot{V}O_2 \) during early active recovery. **Patients:** One hundred forty-two patients with a history of congestive heart failure and left ventricular ejection fraction of 45%, or less who performed a symptom-limited graded treadmill exercise test. **Measurements and results:** Expired gases were monitored breath by breath from rest throughout exercise and during 1 min of active recovery. Patients were defined as having a further increase in \( \dot{V}O_2 \) if the average \( \dot{V}O_2 \) during the initial 30 s of active recovery was greater than or equal to \( \dot{V}O_2 \) during the final 30 s of graded exercise and the instantaneous \( \dot{V}O_2 \) (from the breath-by-breath plot) at 30 s of active recovery was greater than or equal to the instantaneous \( \dot{V}O_2 \) at peak exercise. Thirty patients (21%) showed a further increase in \( \dot{V}O_2 \) following peak exercise (group 1), and 112 had decreased \( \dot{V}O_2 \) at 30 s after peak exercise (group 2). In group 1, treadmill time was significantly shorter, peak \( \dot{V}O_2 \) was significantly lower (16.6±3.6 vs 21.6±6.4 mL/kg/min), and peak ventilatory equivalent for carbon dioxide (Ve/VCO\(_2\)) was significantly higher than those in group 2. There was no difference in etiology of heart failure or functional class and medication status. **Conclusion:** A further increase in \( \dot{V}O_2 \) during early active recovery was associated with poorer exercise tolerance, lower peak \( \dot{V}O_2 \), and higher peak Ve/VCO\(_2\) in chronic heart failure patients. This sign may be a new functional variable for assessment of chronic heart failure. Further investigations are warranted to clarify the mechanisms and clinical implications of this phenomenon. (CHEST 1996; 109:47-51)

**Key words:** chronic heart failure; exercise; oxygen uptake; recovery phase

Chronic heart failure is a syndrome of impaired cardiac performance associated with reduced exercise tolerance and poor prognosis. Exercise testing with respiratory gas analysis has demonstrated that oxygen consumption at maximal exercise (\( \dot{V}O_2 \) max) is an important predictor of survival in chronic heart failure.1-4 Heart failure may also result in delayed recovery oxygen uptake (\( \dot{V}O_2 \) kinetics)4-7 This may play an important role in the prolonged dyspnea or fatigue after exercise in heart failure patients. However, the pathophysiology of the delayed recovery in \( \dot{V}O_2 \) is not yet fully understood.

In our practice, we have sometimes observed not only a delayed recovery in \( \dot{V}O_2 \) kinetics but a further increase in \( \dot{V}O_2 \) during early active recovery in some patients with heart failure rather than the normal decline in \( \dot{V}O_2 \). We speculate that this response may be an indication of a very insufficient cardiopulmonary response to exercise. The purpose of this study was to characterize the clinical and exercise testing characteristics of chronic heart failure patients with and without this further increase in \( \dot{V}O_2 \) in early active recovery following maximal exercise.

**METHODS**

**Subjects**

The study population consisted of 142 patients with a history of...
congestive heart failure and with left ventricular ejection fractions by echocardiography of less than or equal to 45% who underwent symptom-limited, maximal treadmill exercise testing with expired gas analysis. Patients with chronic obstructive or restrictive pulmonary disease or excessive exercise variable artifact (eg, air leak), preventing analysis of the early recovery VO2 kinetics, were excluded. All eligible patients tested between June 1990 and June 1994 were included in this study.

Exercise Test

All subjects performed a symptom-limited graded treadmill exercise test. The protocol of the treadmill exercise test was as follows: from an initial workload of 2 mph at 0% grade, the workload increased by about 7 mL/kg/min (2 METS) every 2 min. There was a 3-min period of active recovery at a speed of 1.7 mph with 0% grade, then a minimum of 3 additional minutes of seated recovery. Patients were encouraged to exercise to maximal or near-maximal levels. Expired gases were monitored breath by breath from rest throughout exercise and 1 min of active recovery using a metabolic cart (Medical Graphics CPX; Medical Graphics Corporation; St. Paul, Minn). ECG was monitored continuously during exercise and recovery in six leads; standard 12-lead ECGs were run at each minute of exercise and recovery. BP was measured by sphygmomanometer in the left arm at 1/2 min of every stage, at peak exercise, at 30 s and 2½ min of active recovery, and at 2½ min of seated recovery.

Criteria for Further Increase in VO2 During Early Active Recovery

Criteria for a further increase in VO2 were that average VO2 during the initial 30 s of active recovery was greater than or equal to VO2 during the final 30 s of graded exercise and that the instantaneous VO2 (from the breath-by-breath plot) at 30 s of active recovery was greater than or equal to instantaneous VO2 at peak exercise. A typical example of further increase in VO2 during recovery is shown in Figure 1.

Statistics

Patients were divided into two groups based on the early active recovery VO2 kinetics. For the two-group comparisons, Pearson's x² contingency test was used in categorical variables and Student's t test was used for continuous variables. To assess the correlation between the change in VO2 during recovery and other variables, Pearson's correlation analysis was used. A p value less than 0.05 was considered statistically significant.

RESULTS

Of 142 total patients, 30 patients (21%) showed a further increase in VO2 following peak exercise (group 1). The other 112 patients had decreased VO2 at 30 s after peak exercise (group 2). The baseline characteristics of each group are shown in Table 1. There were no differences between groups for any clinical variable except presence of third heart sound (S₃). S₃ was heard more frequently in group 1 than group 2. The cause of left ventricular dysfunction was idiopathic dilated car-

![Figure 1: Typical example of VO2 kinetics with (left) or without (right) further increase of VO2 during recovery in patients with chronic heart failure. In the time plot of VO2 on the left, VO2 was greater at 30 s of active recovery than at peak exercise; the usual response, an immediate decrease in VO2 as the exercise intensity is reduced, is shown on the right.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21726/ on 04/29/2017)
diomyopathy in 60% in group 1 and 75% in group 2 (p=NS). Left ventricular ejection fraction averaged 20±7% in group 1 and 23±9% in group 2, not significantly different between groups.

Results of exercise testing are shown in Figure 2. In group 1, treadmill time was 1.9 min shorter and peak VO₂ was 427 mL/min lower than those in group 2. Peak VO₂ adjusted by body weight was 16.6±3.6 mL/kg/min in group 1, which was significantly lower than 21.6±6.4 mL/kg/min in group 2 (p=0.0001). Peak VE/VO₂ was significantly higher and oxygen pulse was lower in group 1 than that in group 2. Respiratory exchange ratio, systolic BP, and peak heart rate were similar in both groups.

In group 1, heart rate at peak exercise averaged 143±24 beats/min and remained at 142±22 beats/min.

**Figure 2.** Peak exercise variables in patients with (group 1) or without (group 2) further increase in VO₂ during recovery. VO₂=oxygen consumption; VE/VO₂=ventilatory equivalent for CO₂; NS=not significant.
at 30 s of active recovery; in group 2, heart rate averaged 150±30 beats/min at peak exercise and was also relatively unchanged at 30 s of active recovery at 148±30 beats/min. V̇O₂ increased from 1,360±418 mL/min at peak exercise to 1,395±431 mL/min in early recovery in group 1, whereas V̇O₂ decreased from 1,757±633 to 1,866±608 mL/min at the two time points in group 2. The oxygen pulse averaged 9.7±2.9 mL/beat at peak exercise and 9.9±2.8 mL/beat in early recovery in group 1; however, oxygen pulse decreased from 12.1±4.2 at peak exercise to 11.6±4.2 mL/beat in early recovery in group 2.

The absolute changes in V̇O₂ from peak exercise to 30 s postexercise (V̇O₂ mL/kg/min at peak −V̇O₂ mL/kg/min at 30 s of recovery) were inversely correlated with exercise time (r = -0.44, p = 0.0001), peak V̇O₂ mL/kg/min (r = -0.50, p = 0.0001), and positively correlated with peak VE/V̇CO₂ (r = 0.26, p = 0.005), but not with left ventricular ejection fraction.

To control the exercise time, we selected 58 patients whose exercise time was less than 6 min. In this subset of patients, exercise time averaged 4.5±0.8 min vs 4.7±0.7 min in groups 1 and 2, respectively (p = 0.05). However, peak V̇O₂ mL/kg/min remained significantly lower in group 1 vs group 2 (14.8±2.3 vs 17.4±3.9 mL/kg/min; p = 0.005), while peak VE/V̇CO₂ remained higher (45.6±9.9 vs 38.6±6.5; p = 0.01). Left ventricular ejection fraction was not different between the two groups in this low exercise time subset (21±7% vs 23±10%).

**DISCUSSION**

In 1927, Meakins and Long⁷ reported that in circulatory disease, V̇O₂ kinetics in response to exercise were slow during increasing intensity of exercise and recovery. Siessema et al.⁶ recently confirmed this delayed recovery of V̇O₂ in chronic heart failure patients. In chronic heart failure patients, recovery of V̇O₂ was prolonged for the same absolute and relative work rate compared with normal subjects. The delay in recovery V̇O₂ was associated with severity of heart failure and its symptoms, especially with fatigue after exercise.⁷,⁸ Those investigations were made mainly using a cycle ergometer with a steady-state exercise test. V̇O₂ kinetics in active recovery for graded treadmill exercise tests in chronic heart failure have not been well described previously.

In the present study, a further increase in V̇O₂ during early active recovery was observed in 21% of our study population of chronic heart failure patients. This phenomenon was associated with more frequent presence of S₃, poorer exercise tolerance, lower peak V̇O₂, and higher peak VE/V̇CO₂. Lower peak V̇O₂ implies lower peak cardiac output, whereas higher peak VE/ V̇CO₂ suggests increased dead-space ventilation due to ventilation-perfusion mismatch in the lung.⁹ The sustained elevation in V̇O₂ during early active recovery was not characterized by a differential heart rate response in group 1 vs group 2. There was a relative increase in V̇O₂ in early active recovery of 2.6% with a simultaneous decrease in heart rate of 0.7% in group 1. In group 2, V̇O₂ decreased by 5.7% while heart rate decreased by 1.3% in early active recovery. This suggests that stroke volume and/or arteriovenous oxygen difference must be increasing during early active recovery in group 1 while decreasing in group 2.

In severe heart failure, the increase in stroke volume with exercise is limited by poor myocardial function (systolic and diastolic) and restrained ventricular filling by the pericardium. It has been suggested that during exercise in chronic heart failure, stroke volume may progressively decrease above the anaerobic threshold due to excessive preload, pushing the heart onto the “descending limb” of the Frank-Starling curve, and high afterload due to inadequate systemic vasodilation.¹⁰ Stroke volume may thus increase temporarily in early active recovery in severe heart failure patients due to rapid relief from unmatched preload and afterload.

Another possible mechanism for the sustained elevation of V̇O₂ during early active recovery is an increase in arteriovenous oxygen (A-V O₂) difference for a short time after peak exercise. In the rapid phase of excess postexercise oxygen consumption, restoration of creatine phosphate takes a few minutes after exercise stops.¹¹ Using ³¹P magnetic resonance spectroscopy, Mancini et al.¹² demonstrated that the recovery of phosphagens was delayed in chronic heart failure.

A longer circulatory delay from the lower extremities (where oxygen is being consumed) to the lungs (where oxygen is being taken up) due to lower cardiac output could also explain the transient increase in A-V O₂ difference in early active recovery. In chronic heart failure, lower cardiac output would prolong the time required for blood with the widened A-V O₂ difference to reach the lungs.

By any of these mechanisms, the increase in V̇O₂ in early recovery in group 1 patients implies more severe heart failure with more limited cardiac reserve during exercise. The lack of significant differences in resting left ventricular ejection fraction between group 1 and group 2 is not surprising in light of previous studies showing a poor correlation between resting ejection fraction and exercise tolerance or V̇O₂max.¹³,¹⁴

**Limitations**

We investigated the V̇O₂ kinetics only for 1 min of recovery, so we could not address how this further increase in V̇O₂ during early active recovery affects oxygen consumption in the later phase of recovery. Moreover, because of the retrospective nature of the present study, we have no invasive measurements to
validate any of the proposed mechanisms. Finally, we do not yet have follow-up data to determine if this further increase in VO2 in early active recovery is associated with a poorer prognosis, especially after adjustment for VO2max. The mechanism and clinical significance of further increase in VO2 in early active recovery must be addressed in future investigations.

CONCLUSIONS

In the present study, a further increase in VO2 during early active recovery was associated with poorer exercise tolerance, lower peak VO2, and higher peak VE/VCO2 in chronic heart failure patients but not any of their clinical characteristics except for presence of S3. Further investigations are warranted to clarify the mechanism and clinical implication of this phenomenon.

REFERENCES

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