

Original Research

Association of Venous Function and Post-Exercise Oxygen Consumption

ERIC TISDELL^{†1}, MATTHEW SCOTT^{†1}, ZEKI BAHADIR^{†1}, TRACIE R. PARISH^{‡2}, ARTURO A. ARCE-ESQUIVEL^{‡3}, DEVON DOBROSIELSKI^{‡4}, and MICHAEL A. WELSCH^{†1}

¹Department of Kinesiology, Louisiana State University, Baton Rouge, LA, USA;

²Department of Kinesiology and Health Studies, Southeastern Louisiana University, Hammond, LA, USA; ³ Department of Health and Kinesiology at The University of Texas at Tyler, Tyler, TX, USA; ⁴ Division of Cardiology at Johns Hopkins Bayview Medical Center, Baltimore, MD, USA

†Denotes graduate student author, ‡Denotes professional author

ABSTRACT

International Journal of Exercise Science 6(1): 63-73, 2013. Excess post-exercise oxygen consumption (EPOC) has been attributed to metabolic, hemodynamic, neuroendocrine, and pulmonary factors. Interestingly, few studies have examined the role of venous system on EPOC. The purpose of this study was to examine the relationship between measures of vascular function and EPOC. Measures of vascular function and VO₂ recovery kinetics were examined in 20 individuals [age=22±2.41 yrs]. Nondominant forearm arterial inflow, venous capacitance and venous outflow were evaluated at rest and after 5 minutes of upper arm occlusion, using strain gauge plethysmography. VO₂ recovery kinetics was assessed using gas exchange analysis following a six-minute constant work rate protocol at 60 percent of VO_{2peak} (VO_{2@60%}), on a cycle ergometer. The average VO_{2peak} was 33.48±8.22 ml·kg⁻¹·min⁻¹ (Range: 18.7 to 46.1 ml·kg⁻¹·min⁻¹). Following the six-minute constant work rate protocol, recovery half-time (T_{1/2}VO₂) and Tau were 17.01±3.51 seconds and 54.45±11.28 seconds, respectively. Arterial resting inflow was 2.77±1.51 ml·100ml⁻¹·min⁻¹, reactive hyperemic blood flow was 17.72±3.65 ml·100ml⁻¹·min⁻¹, venous capacitance was 2.86±0.72 percent, and venous outflow was 34.19±10.03 ml·100ml⁻¹·min⁻¹. Bivariate correlations revealed significant inverse associations between T_{1/2}VO₂ and the reactive hyperemic response (r=-0.48, p=0.03) and T_{1/2}VO₂ and venous outflow post-occlusion (r=-0.50, p=0.02). In conclusion, these findings suggest an important role of both the arterial and venous circulation on EPOC.

KEY WORDS: Acute exercise, oxygen kinetics, hyperemia, venous function

INTRODUCTION

Recovery kinetics following an acute bout of exercise is a prognostic marker in clinical populations (10, 12). Oxygen consumption (VO₂) recovery kinetics following a bout of exercise has been defined as the excess post exercise oxygen consumption (EPOC) (13).

A.V. Hill was the first to report on the post exercise phase (15), which was later termed "O₂ debt" (15). Hill attributed this period to the need to repay the metabolic requirement attained at the onset of exercise (15), termed "O₂ deficit" (17). Based on early work, the O₂ debt was thought to be the result of reconversion of

lactate to glycogen during the recovery period. Subsequent research suggested these explanations were too simplistic to accurately describe this phenomenon (13). In fact, additional factors contribute to EPOC. These factors include resynthesis of phosphocreatine, restoration of O₂ stores, elevated body temperature, post-exercise elevation of heart rate, breathing, and hormonal activity.

VO₂ recovery kinetics is generally slower in individuals who are less fit (20), older (6), or those with chronic heart failure (9). The apparent differences between fitter, younger and/or healthier persons have been attributed to more efficient replenishing systems and metabolic waste removal (2, 4). For instance, Belardinelli et al. (4) suggested a slower rate of recovery could reflect slower phosphocreatine resynthesis. Therefore, early repayment of O₂ debt is more likely to reflect rate of recovery of phosphocreatine and venous blood oxygenation (4). The fact mitochondrial respiration may be affected by a variety of factors, such as catecholamine levels, thyroxin, and the accumulation of other metabolic byproducts (13), suggests prolonged VO₂ recovery kinetics has a large metabolic component.

Interestingly, Barclay (2) reported muscle fatigue characteristics were greatly improved by high flow conditions, independent of O₂ and nutrient delivery. He concluded the effects were likely the result of enhanced removal of metabolites (2). Furthermore, Van Beekvelt et al. (24) reported following a bout of heavy exercise, forearm blood flow was inadequate to meet the metabolic demand and waste removal, as indicated by a marked and prolonged

post exercise hyperemia. This points toward an important vascular component in the recovery kinetics following exercise. Given the apparent interest in both metabolite removal and vascular function in VO₂ recovery kinetics, it is interesting there are no apparent studies that have examined the role of the venous system on EPOC. The purpose was to examine possible relationships between measures of vascular function and VO₂ recovery kinetics. It is thought that such information may provide a greater appreciation of the role of the vasculature in EPOC and may eventually lead to a better understanding of replenishment and removal during the recovery phase from an acute bout of exercise. Furthermore, a link between the venous system and exercise recovery may ultimately provide a better understanding into the symptoms of chronic fatigue. Based on previous investigations, we hypothesized individuals with higher VO₂ peaks would have faster recovery kinetics following a single bout of exercise (5, 14). Moreover, based on previous work (1, 25), we hypothesized that those with higher VO₂ peaks also demonstrate better overall vascular function. Finally, we hypothesized measures of venous function would be inversely related to VO₂ recovery kinetics.

METHODS

Participants

Individuals between the ages of 18 and 30 were recruited. Those with any overt manifestations of cardiovascular, metabolic, orthopedic, or neurological disease, as well as individuals on medications, which could affect the results of this study, were excluded. Informed consent, approved by

the host Institution, was obtained before participation.

Protocol

Experimental Design: The study was an observational study involving 3 visits. The first visit involved assessment of vascular function using mercury-in-Silastic strain gauge plethysmography (Hokanson EC-5R plethysmography system, Bellevue, WA). The second visit involved an incremental maximal exercise test using a standard cycle ergometer (Monark). Breath-by-breath respiratory gas analyses (SensorMedics software, Yorba Linda, CA) were used to obtain measures of oxygen consumption, carbon dioxide production and pulmonary ventilation before, during and after exercise. The final visit involved a constant workload exercise bout for 6 minutes, using the same cycle ergometer used for visit 2, performed at an intensity of 60% of the subjects VO_2peak . Time period between visit 1 and 3 was no longer than 2 weeks. Recovery between visit 2 and visit 3 was at least 48 hours.

Vascular Function Assessments: Indices of vascular function were obtained in the non-dominant forearm using mercury-in-Silastic strain gauge plethysmography (1). Prior to the experiment, blood pressure cuffs were positioned around the participants' upper arm and wrist, and a strain gauge was placed around the forearm.

Resting forearm vascular function measures were obtained following 10 minutes of rest in the supine position. Immediately before the measurement, hand circulation was occluded for one minute by inflating the wrist cuff to 240 mmHg. Forearm blood flow was then examined using an upper arm venous occlusion pressure of 7 mmHg

below diastolic blood pressure. Venous occlusion pressure was held for a subsequent five-minute period, after which resting venous capacitance was measured. At the end of the five-minute period, the upper arm cuff was rapidly deflated and resting venous outflow was obtained (1). The second part of the vascular function assessment involved total blood flow occlusion in the non-dominant arm for five minutes in order to determine reactive hyperemic blood flow (RHBF). This involved the inflation of the upper arm cuff to a pressure of 240 mmHg. Following four minutes of occlusion, the wrist cuff was inflated to 240 mmHg. After the fifth-minute of occlusion, the upper cuff was deflated to a pressure equal to 7 mmHg below diastolic blood pressure. Post-occlusion venous capacitance was also measured after holding the venous pressure for an additional five-minute period. Finally, the upper arm cuff was completely deflated allowing for the measurement of post-occlusion venous outflow (1). Blood pressure, heart rate, and electrocardiogram measurements were monitored prior to and during the procedure. Assessments were obtained following 12 hours of restriction from exercise, food, and alcohol consumption.

Cardiorespiratory Assessments

(VO_2peak): A cycle ergometer test, modeled after Cohen-Solal et al. (9) was used to determine the individuals' cardiorespiratory exercise capacity. Following a resting period of five minutes, the participant began pedaling at a speed of 60 revolutions per minute (rpm) with a resistance of 0 watts. Every two minutes the resistance was increased by 30 watts until the subject requested to stop, could not maintain 60 rpm, or exhibited signs or

symptoms requiring termination of the test. During the test, blood pressure, heart rate, and ratings of perceived exertion (RPE) were obtained at the end of each minute using a standard sphygmomanometer, Polar heart rate monitor (Woodbury, NY), and the Borg Scale, respectively. Following the test, heart rate, and blood pressure were monitored for 10 minutes.

Constant Work Rate Exercise Test (Submaximal): The workload used for the test was 60% of the subjects VO_{2peak} ($VO_{2@60\%}$), determined from the maximal test. The VO_{2peak} was determined from a 30-second average at the end of the test. Prior to the constant work rate test, baseline measures were obtained for five minutes. After this period, the subject began pedaling at the pre-calculated submaximal intensity for six-minutes modeled after Belardinelli et al. (2). After six minutes the participant was monitored for a 10-minute recovery period. Throughout the test, breath-by-breath respiratory gases and volumes were obtained. In addition, blood pressure, heart rate, and RPE were obtained as described in visit 2. During the recovery period heart rate and blood pressure were also monitored for 10 minutes.

Data Analysis: Indices of vascular function were reported in terms of resting forearm blood flow, RHBF, venous capacitance, venous capacitance post-occlusion, venous outflow, and venous outflow post-occlusion. Resting forearm blood flow was recorded at a speed of $5 \text{ mm}\cdot\text{sec}^{-1}$ and values were derived from the slope drawn at a best-fit tangent using the first three pulses. Calculations were made as a function of 60 seconds divided by the horizontal distance (mm) needed for the slope to rise vertically from baseline to the

top of the recording paper and multiplied by the full chart range. RHBF was recorded at a paper speed of $25 \text{ cm}\cdot\text{sec}^{-1}$. Analyses were performed using a slope drawn at a best-fit tangent to the curves of the first two pulses of the flow curve post cuff release. The blood flows were then calculated from 60 seconds multiplied by the paper speed ($25 \text{ cm}\cdot\text{sec}^{-1}$) divided by the horizontal distance (mm) needed for the volume slope to increase by 20 mm vertically.

Venous capacitance was measured as the vertical distance measured (mm) representing the increase in forearm-volume graph after the designated period for venous filling. Analysis of venous outflow was derived from a tangent line that represents the vertical drop in volume-graph from the excursion line and drawn at 0.5 seconds and 2 seconds after the release of pressure from the upper arm cuff (1).

The time course for the decay in VO_2 following cessation of the six-minute constant work rate protocol exercise was characterized by determining the time required to return 50% ($T_{1/2}VO_2$) of the way from the VO_2 value obtained at the end of the sixth-minute of exercise to the pre-exercise baseline value and the time constant Tau (τ) (14).

Statistical Analysis

Values are reported as mean \pm SD. Pearson product moment correlations were used to assess the relationships of $T_{1/2}VO_2$ and τ with VO_{2peak} , reactive hyperemic blood flow and venous outflow post-occlusion using the SPSS system. In a subsequent analysis, participants were separated in tertiles on the basis of their VO_{2peak} , which enabled statistical examination of the

dependent variables using independent t-tests. Alpha was set a priori at 0.05.

RESULTS

Participant Characteristics: Twenty adults (8 Men and 12 Women) participated in the study. Baseline characteristics are shown in Table 1.

Table 1. Participant Characteristics.

Participants (n=20)	Minimum	Maximum	Mean	SD
Age (yrs.) (F/M)	18/20	24/27	21/24	1.42/2.33
Height (cm) (F/M)	155/170	170/193	164/179	3.99/7.96
Weight (kg) (F/M)	52.00/61.4	97.5/118.00	62.84/79.40	10.06/14.11
SBP _{rest} (mmHg)	99.00	139.00	116.55	12.36
DBP _{rest} (mmHg)	57.00	89.00	69.60	8.80
RHR (bpm)	61.00	103.00	82.85	12.93
VO _{2peak} (ml·kg ⁻¹ ·min ⁻¹)	18.70	46.10	33.48	8.22

Vascular Function Assessments: Indices of vascular function are shown in Table 2. Average resting blood flow for the group was 2.77±1.51, average venous capacitance, 2.86±0.72 and venous outflow 34.19 ± 10.03 ml·100ml⁻¹·min⁻¹, respectively. The average for RHBF was 17.72±3.65 ml·100ml⁻¹·min⁻¹.

Oxygen Consumption Recovery Kinetics: Values for VO_{2@60%}, and the T_{1/2}VO₂, and Tau are expressed in Table 3. The VO_{2@60%} of the participants in this study ranged from 12.50.7 to 30.77 ml·kg⁻¹·min⁻¹. The

average T_{1/2}VO₂ for the participants was 17.01 sec and Tau 54.45 sec.

Table 2. Indices of Vascular Function.

Participants (n=20)	Minimum	Maximum	Mean	SD
Resting forearm blood flow (ml·100ml ⁻¹ ·min ⁻¹)	1.36	7.50	2.77	1.51
Reactive hyperemic blood flow (ml·100ml ⁻¹ ·min ⁻¹)	12.63	23.63	17.72	3.65
Venous capacitance (%)	1.10	4.05	2.86	0.72
Venous outflow (ml·100ml ⁻¹ ·min ⁻¹)	16.75	50.00	34.19	10.03
Venous capacitance post-occlusion (%)	0.70	3.35	1.75	0.87
Venous outflow post-occlusion (ml·100ml ⁻¹ ·min ⁻¹)	16.21	56.60	36.25	11.32

Table 3. Indices for VO₂ Recovery Kinetics.

Participants (n=20)	Minimum	Maximum	Mean	SD
VO _{260%} (ml·kg ⁻¹ ·min ⁻¹)	12.50	30.77	20.29	5.36
T _{1/2} VO ₂ (sec)	7.36	23.32	17.01	3.51
Tau (sec)	24.00	76.00	54.45	11.28

Table 4. Pearson Product Moment Correlations.

	RBF	RHBF	VO	VOPO	VO _{2peak}	T1/2VO2	TAO	VC	VCPO
RBF (ml·100ml ⁻¹ ·min ⁻¹)	1.000								
RHBF (ml·100ml ⁻¹ ·min ⁻¹)	0.49*	1.000							
VO (ml·100ml ⁻¹ ·min ⁻¹)	0.41	0.68**	1.000						
VOPO (ml·100ml ⁻¹ ·min ⁻¹)	0.23	0.65**	0.87**	1.000					
VO _{2peak} (ml·kg ⁻¹ ·min ⁻¹)	0.52*	0.21	0.44*	0.46*	1.000				
T1/2VO2 (sec)	-0.32	-0.48*	-0.48*	-0.50*	-0.45*	1.000			
Tau (sec)	-0.26	-0.42	-0.46*	-0.44*	-0.45*	0.95**	1.000		
VC (%)	0.30	0.68**	0.78**	0.65**	0.18	-0.32	-0.28	1.000	
VCPO (%)	0.05	0.57**	0.50*	0.57**	0.02	-0.35	-0.30	0.71**	1.000

Resting forearm blood flow= RBF; Reactive hyperemic blood flow= RHBF; Venous Outflow=VO; Venous Outflow post-occlusion=VOPO; Recovery Half time= T1/2VO2; Venous Capacitance=VC; Venous Capacitance post-occlusion=VCPO. * Correlation is significant at the 0.05 level (2-tailed). ** Correlation is significant at the 0.01 level (2-tailed).

Pearson Product Moment Correlations:

Analyses of associations between vascular function and VO₂ recovery kinetics are shown in Table 4. Data show a significant relationship between RHBF and T_{1/2}VO₂ (r=-0.48, p=0.03, see Figure 1).

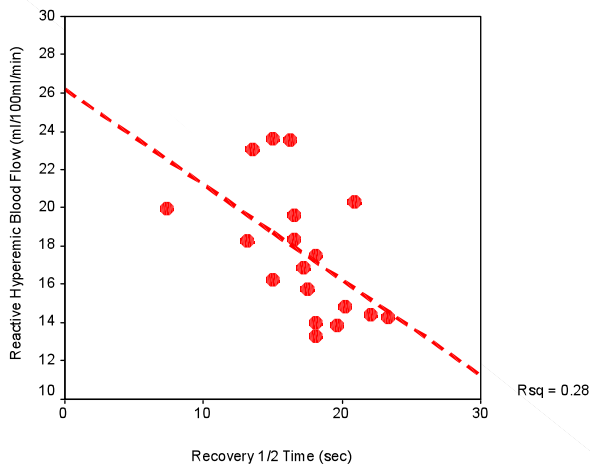


Figure 1. Relationship between reactive hyperemic blood flow and half-time of recovery.

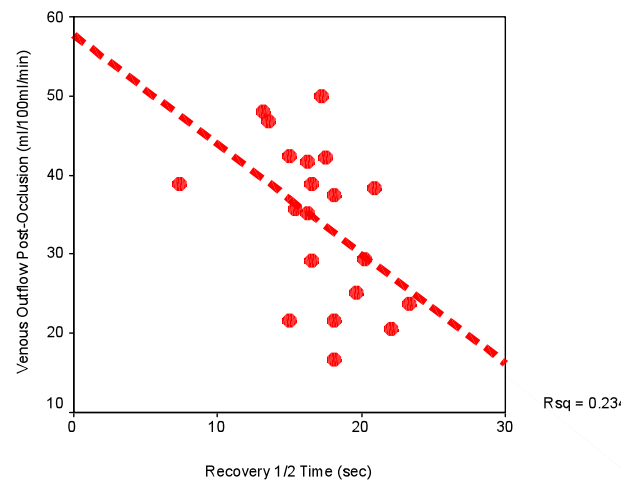


Figure 2. Relationship between venous outflow post-occlusion and the half-time of recovery.

Furthermore, there is a significant relationship between measures of venous outflow and exercise capacity (r=0.46, p=0.04) and T_{1/2}VO₂ (Venous outflow: r=-0.50, p=0.02; and Venous outflow post-

occlusion: $r=-0.48$, $p=0.03$, see Figure 2). These associations were still evident after controlling for body size ($BMI=wt(kg)/ht(m^2)$).

Differences Between Groups of High and Low VO_{2peak} : When participants were separated in tertiles on the basis of their VO_{2peak} (Higher Fitness: $VO_{2peak} >40 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$; Lower Fitness: $VO_{2peak} <35 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) additional differences were noted (see Table 5).

Table 5. Differences between High and Low VO_{2peak} .

Variable	Fitness Category	N	Mean	SD
VO_{2peak} ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	High	8	40.98*	3.08
	Low	7	23.96	4.15
$T_{1/2}VO_2$ (Sec)	High	8	15.34†	4.05
	Low	7	19.11	3.04
Tau (Sec)	High	8	49.50†	13.15
	Low	7	60.00	10.71
VC (%)	High	8	2.98	0.94
	Low	7	2.65	0.39
VO ($\text{ml}\cdot 100\text{ml}^{-1}\cdot\text{min}^{-1}$)	High	8	39.46*	10.55
	Low	7	28.96	6.55
RHBF ($\text{ml}\cdot 100\text{ml}^{-1}\cdot\text{min}^{-1}$)	High	8	19.43*	3.38
	Low	7	15.40	2.44

Recovery Half time= $T_{1/2}VO_2$; Venous Capacitance=VC; Venous Outflow=VO; Reactive hyperemic blood flow= RHBF; * $p < 0.05$; † $P < 0.01$.

Data show a significant difference between the $T_{1/2}VO_2$ (Higher VO_{2peak} : 15.34 sec, Lower VO_{2peak} : 19.11 sec; $p < 0.01$), and for venous outflow and RHBF between groups (Higher VO_{2peak} : 39.46 and 19.43 $\text{ml}\cdot 100\text{ml}^{-1}\cdot\text{min}^{-1}$, Lower VO_{2peak} : 28.96 and 15.4 $\text{ml}\cdot 100\text{ml}^{-1}\cdot\text{min}^{-1}$; $p < 0.05$), respectively.

DISCUSSION

The purpose was to examine potential relationships between measures of vascular function and post exercise recovery. The

study confirms previous findings that RHBF is associated with VO_2 recovery kinetics. Uniquely, measures of venous function are also associated with VO_2 recovery kinetics. Finally, the study indicates individuals with higher VO_{2peak} appear to have greater RHBF and venous function and faster VO_2 recovery kinetics.

Previous research acknowledges the contribution of several factors to the magnitude of EPOC (13). However, the role of the venous system has not been examined, despite speculation of its importance in metabolite removal (2). Indeed, Barclay (2), observed lower rates of fatigue in muscle receiving high flow versus low flow, independent of O_2 content, in part, due to removal of metabolites (1). The present data support an important role of the venous system in EPOC. If so, alterations in the venous system could affect these kinetics.

In terms of the vascular responses our findings appear to follow previous results (1, 25, 26). The RHBF appear similar to those seen in lower fitness groups (25), perhaps reflecting the below average VO_{2peak} for this age group. Measures for venous function appear similar to previous studies. For example, Alomari et al. (1) reported venous capacitances of 3.8 and 4.0 for lower and higher fitness groups of similar age. Moreover, venous outflow measures for the lower fit participants (Venous outflow: 40.7 ± 7.9) in Alomari's study are similar to those observed in this study (Venous outflow: 35.2 ± 10.03) (1). Mean total venous outflow for the current study are similar to those reported by Wecht et al. (25). Thus vascular responses reflect fitness status of these individuals, as is generally accepted. Finally, venous responses in individuals in the highest

tertile of VO₂peak were significantly higher vs. those in the lowest tertile.

The values for the VO₂ recovery kinetics in this study are difficult to compare to existing literature secondary to a lack of consensus on protocols and analyses performed. However, the direction of these data is similar to other investigators. Several studies have examined VO₂ recovery kinetics in populations similar to participants in this study. For example, Hagberg et al. (14) and Billat et al. (5) examined the effects of endurance training on recovery kinetics. The average maximal oxygen consumption (VO₂peak) of the two study groups was 41.46 and 56.0 ml·kg⁻¹·min⁻¹, respectively, which were higher than the mean VO₂peak in this study (5,14). However, it is important to emphasize that both Hagberg et al. (14) and Billat et al. (5) examined recovery kinetics following high intensity submaximal exercise (much higher than used in this study). Consequently, half-times (26.5±2.1 and 29.0±8.0, respectively) were longer than those in the present study. Importantly, Hagberg et al. (14) reported exercise training resulted in significantly faster recovery kinetics.

Additional studies have addressed post exercise VO₂ recovery kinetics in clinical populations. For example, Belardinelli et al. (4) studied recovery kinetics in 10 patients with chronic heart failure (CHF) and 8 healthy controls, following a similar protocol used in this study. The findings by Belardinelli et al. (4) indicate half-time of recovery was inversely related to the VO₂peak of the participants (VO₂peak: Controls: 22.58, CHF: 13.95 ml·kg⁻¹·min⁻¹, p<0.001; Time Constants: Control: 48.0, CHF: 66.8 sec, p<0.05)(4). These findings

are similar to this study, demonstrating a relationship between exercise tolerance and recovery half-time. In addition, Cohen-Solal et al. (9) confirmed an inverse relationship between VO₂ recovery kinetics in patients with CHF, and added that recovery times were strongly associated with severity of the disease, with slower recoveries in patients with greater dysfunction.

The influence of the vasculature on VO₂ recovery has been well documented in terms of the O₂ delivery (2, 3, 24). Consistent with those findings, the present study reports a direct association between RHBF and the half-time of recovery. Interestingly, this study suggests the arterial system may account for as much as 29% of the recovery phase. In addition, those with the greatest RHBF appear to have the fastest VO₂ recovery kinetics. This implies that the recovery of VO₂ is in part determined by the delivery of O₂ and substrate. In fact, PCr resynthesis is highly dependent on an adequate blood supply.

The mechanisms involved in greater arterial function in individuals with higher exercise capacity are not completely understood, but thought to include modifications in neural, local, structural and metabolic components (1, 7, 26). The role of exercise training on venous function has received considerable less attention. This is surprising considering the venous system indirectly controls arterial inflow. For example, when the veins empty, a transient arterial vasodilation occurs, which results in an immediate increase in arterial inflow (22, 23). Most studies have attributed training induced improvements in venous function to greater blood volume, associated with an increased VO₂peak (11, 16). Other studies report increased cross-

sectional area (18), and density (21) of venular structures in response to training. Wecht et al. (25) suggested improved venous vascular function in endurance-trained individuals was due to improved venous capacitance, venous outflow, vasomotor tone, and venous compliance.

Previous findings from our laboratory report an association between forearm arterial and venous function indices ($r=0.43$, $p=0.014$) (1). Our present findings suggest that venous function may account for as much as 23% of the recovery period, supporting the role of the venous system as more than a "passive volume reservoir." In fact, Tschakovsky and Hughson (22) reported a greater arterial inflow with increased venous emptying following arm elevation, and highlighted the importance of a local venoarteriolar sympathetic reflex (22). These findings emphasize the important relationship between arterial and venous function (25), and confirms the role of venous function in cardiovascular control, muscle perfusion, and exercise tolerance (1, 22, 26).

The clinical relevance of this study is still speculative. However, recognizing the cardinal symptoms of most patients is chronic fatigue and their inability to recover from one task to the next, the present findings suggest a target for examination and eventually treatment may well be the venous system.

Previous studies have related diminished venous function to respiratory-cardiovascular hemodynamics, plasma norepinephrine, and severity of disease (8, 9, 26). In the study by Wecht et al. (25), a lower venous capacitance was observed in a sedentary group when compared to an

active group. Welsch et al. (26) suggested that factors that contribute to exercise impairment in the CHF population might extend to the venous system. Thus, future studies should examine whether changes in fatigue and recovery, in patient populations are, in part, a consequence of changes in venous function. If so the typical treatment strategies used to increase O₂ and nutrient delivery may need to include strategies to enhance venous function to ensure improved metabolite removal.

In conclusion, the contributions of this experiment have allowed us to look beyond the accepted explanations for recovery VO₂. Our findings suggest an important role for both arterial and venous circulation on EPOC. These findings provide the basis for subsequent studies to examine whether improvements in arterial and venous function contribute to accelerated recovery kinetics. We speculate exercise training may have the potential to not only improve delivery, but may also play a significant role in metabolite removal, culminating in enhanced recovery kinetics. This could have implications for athletes who have to recover from repeated exercise bouts, or those who suffer from chronic fatigue related symptoms.

REFERENCES

1. Alomari MA, Welsch MA, Prisby RD, Lee CM, Wood RH. Modification of forearm vascular function following short-term handgrip exercise training. *Int J Sports Med* 22(5):361-5, 2001.
2. Barclay JK. A delivery-independent blood flow effect on skeletal muscle fatigue. *J Appl Physiol* 61(3):1084-90, 1986.
3. Barstow TJ, Lamarra N, Whipp BJ. Modulation of muscle and pulmonary O₂ uptakes by circulatory dynamics during exercise. *J Appl Physiol* 68(3):979-89, 1990.

4. Belardinelli R, Barstow TJ, Nguyen P, Wasserman K. Skeletal muscle oxygenation and oxygen uptake kinetics following constant work rate exercise in congestive heart failure. *Am J Cardiol* 80:1319-1324, 1997.
5. Billat BL, Mille-Hamard L, Demarle A, Koralsztein JP. Effect of training in humans on off- and on-transient oxygen uptake kinetics after severe exhausting intensity runs. *Eur J Physiol* 87:496-505 2002.
6. Chilibeck PD, Paterson DH, Cunningham DA, Taylor AW, Noble EG. Muscle capillarization O₂ diffusion distance, and VO₂ kinetics in old and young individuals. *J Appl Physiol* 82(1):63-9, 1997.
7. Clarkson P, Montgomery HE, Mullen MJ, Donald AE, Powe AJ, Bull T, Jubbs M, World M, Deanfield JE. Exercise training enhances endothelial function in young men. *J Am Coll Cardiol* 33:1379-85, 1999.
8. Cohen-Solal A, Chabernaude JM, Gourgon R. Comparison of oxygen uptake during bicycle exercise in patients with chronic heart failure and in normal subjects. *J Am Coll Cardiol* 16:80-85, 1990.
9. Cohen-Solal A, Laperche T, Morvan D, Geneves M, Caviezel B, Gourgon R. Prolonged kinetics of recovery of oxygen consumption after maximal graded exercise in patients with chronic heart failure. *Circulation* 91:2924-2932, 1995.
10. Cole CR, Blackstone CH, Pashkow FJ, Snader CE, Lauer MS. Heart-rate recovery immediately after exercise as a predictor of mortality. *N Engl J Med* 341:1351-7, 1999.
11. Convertino VA, Mack GW, Nadel ER. Elevated central venous pressure: a consequence of exercise-training induced hypervolemia? *Am J Physiol* 260:R273-277, 1991.
12. De Groote P, Millaire A, Decoux E, Nugue O, Guimer P. Kinetics of oxygen consumption during and after exercise in patients with dilated cardiomyopathy. New markers of exercise intolerance with clinical implications. *J Am Coll Cardiol* 28(1):168-75, 1996.
13. Gaesser GA, Brooks GA. Metabolic bases of excess post-exercise oxygen consumption: a review. *Med Sci Sports Exerc* 16(1):29-43, 1984.
14. Hagberg JM, Hickson RC, Ehsani AA, Holloszy JO. Faster adjustment to and recovery from submaximal exercise in the trained state. *J Appl Physiol* 48(2):218-24, 1980.
15. Hill AV, Long CNH, Lupton H. Muscular exercise, lactic acid and the supply and utilisation of oxygen. Pt. IV-VI *Proc B Soc B* 97:84-138, 1924.
16. Louisy F, Jouanin JC, Guezennec CY. Filling and emptying characteristics of lower limb venous network in athletes. Study by postural plethysmography. *Int J Sports Med* 18:26-9, 1997.
17. Margaria R, Edwards HT, Dill DB. The possible mechanisms of contracting and the role of lactic acid in muscular contraction. *Am J Physiol* 106:689-715.49, 202-210, 1933.
18. Miyachi M, Tanaka H, Yamamoto K, Yoshioka A, Takahashi K, Onodera S. Effects of one-legged endurance training on femoral arterial and venous size in healthy humans. *J Appl Physiol* 90:2439-44, 2001.
19. Pavia L, Myers J, Cesare R. Recovery kinetics of oxygen uptake and heart rate in patients with coronary artery disease and heart failure. *Chest* 116(3):808-13, 1999.
20. Short KR, Sedlock DA. Excess postexercise oxygen consumption and recovery rate in trained and untrained subjects. *J Appl Physiol* 83(1):153-9, 1997.
21. Suzuki J, Kobayashi T, Uruma T, Koyama T. Time-course changes in arteriolar and venular portions of capillary in young treadmill-trained rats. *Acta Physiol Scand* 171:77-86, 2001.
22. Tschakovsky ME, Hughson RL. Venous emptying mediates a transient vasodilation in the human forearm. *Am J Physiol Heart Circ Physiol* 279:H1007-H1014, 2000.
23. Tschakovsky ME, Shoemaker JK, Hughson RL. Vasodilation and muscle pump contribution to immediate exercise hyperemia. *Am J Physiol Heart Circ Physiol* 271:H1697-H1701, 1996.
24. Van Beekvelt MCP, Shoemaker JK, Tschakovsky ME, Hopman MTE, Hughson RL. Blood flow and

ASSOCIATION OF VENOUS FUNCTION

muscle oxygen uptake at the onset of moderate and heavy dynamic forearm exercise. *Am J Physiol Regulatory Integrative Comp Physiol* 280:R1741-7, 2001.

25. Wecht JM, De Messerman RE, Weir JP, Bauman WA, Grimm DR. Effects of autonomic disruption and inactivity on venous vascular function. *Am J Physiol Heart Circ Physiol* 278:H515-H520, 2000.

26. Welsch MA, Alomari M, Parish TR, Wood RH, Kalb D. Influence of venous function on exercise tolerance in chronic heart failure. *J Cardiopulm Rehabil* 22(5):321-326, 2002.