

# Massage Impairs Postexercise Muscle Blood Flow and “Lactic Acid” Removal

E. VICTORIA WILTSHIRE, VERONICA POITRAS, MELISSA PAK, TERENCE HONG, JAY RAYNER, and MICHAEL E. TSCHAKOVSKY

*School of Kinesiology and Health Studies, Queen's University, Kingston, Ontario, CANADA*

## ABSTRACT

WILTSHIRE, E. V., V. POITRAS, M. PAK, T. HONG, J. RAYNER, and M. E. TSCHAKOVSKY. Massage Impairs Postexercise Muscle Blood Flow and “Lactic Acid” Removal. *Med. Sci. Sports Exerc.*, Vol. 42, No. 6, pp. 1062–1071, 2010. **Purpose:** This study tested the hypothesis that one of the ways sports massage aids muscle recovery from exercise is by increasing muscle blood flow to improve “lactic acid” removal. **Methods:** Twelve subjects performed 2 min of strenuous isometric handgrip (IHG) exercise at 40% maximum voluntary contraction to elevate forearm muscle lactic acid. Forearm blood flow (FBF; Doppler and Echo ultrasound of the brachial artery) and deep venous forearm blood lactate and  $H^+$  concentration ( $[La^-]$ ,  $[H^+]$ ) were measured every minute for 10 min post-IHG under three conditions: passive (passive rest), active (rhythmic exercise at 10% maximum voluntary contraction), and massage (effleurage and pétrissage). Arterialized  $[La^-]$  and  $[H^+]$  from a superficial heated hand vein was measured at baseline. **Results:** Data are presented as mean  $\pm$  SE. Venoarterial  $[La^-]$  difference ( $[La^-]_{v-a}$ ) at 30 s of post-IHG was the same across conditions (passive =  $6.1 \pm 0.6$  mmol·L $^{-1}$ , active =  $5.7 \pm 0.6$  mmol·L $^{-1}$ , massage =  $5.5 \pm 0.6$  mmol·L $^{-1}$ , NS), whereas FBF was greater in passive ( $766 \pm 101$  mL·min $^{-1}$ ) versus active ( $614 \pm 62$  mL·min $^{-1}$ ,  $P = 0.003$ ) versus massage ( $540 \pm 60$  mL·min $^{-1}$ ,  $P < 0.0001$ ). Total FBF area under the curve (AUC) for 10 min after handgrip was significantly higher in passive versus massage ( $4203 \pm 531$  vs  $3178 \pm 304$  mL,  $P = 0.024$ ) but not versus active ( $3584 \pm 284$  mL,  $P = 0.217$ ).  $La^-$  efflux ( $FBF \times [La^-]_{v-a}$ ) AUC mirrored FBF AUC (passive =  $20.5 \pm 2.8$  mmol vs massage =  $14.7 \pm 1.6$  mmol,  $P = 0.03$ , vs active =  $15.4 \pm 1.9$  mmol,  $P = 0.064$ ).  $H^+$  efflux ( $FBF \times [H^+]_{v-a}$ ) was greater in passive versus massage at 30 s ( $2.2 \pm 0.4e^{-5}$  vs  $1.3 \pm 0.2e^{-5}$  mmol,  $P < 0.001$ ) and 1.5 min ( $1.0 \pm 0.2e^{-5}$  vs  $0.6 \pm 0.09e^{-5}$  mmol,  $P = 0.003$ ) after IHG. **Conclusions:** Massage impairs  $La^-$  and  $H^+$  removal from muscle after strenuous exercise by mechanically impeding blood flow. **Key Words:** LACTATE, ISOMETRIC HANDGRIP, ATHLETIC THERAPY, PÉTRISSAGE, EFFLEURAGE

Sports massage is widely accepted among the athletic community as an aid to promote recovery from training and competition, thereby enhancing optimal performance (8,25). A common belief among therapists or trainers and athletes alike has been that massage enhances muscle blood flow and therefore the removal of metabolites such as “lactic acid” (3). The term “lactic acid” is actually misleading because the lactate dehydrogenase reaction does not generate a  $La^- H^+$  complex where  $H^+$  then dissociates at physiological pH. Instead, it converts pyruvate to  $La^-$ , which is a strong ion, such that  $La^-$  accumulation changes strong ion difference in a manner that elevates  $[H^+]$  (24,32). Therefore, we use the term “lactic acid” to represent  $H^+$  accumulation associated with  $La^-$  accumulation.

The importance of the removal of lactic acid from the exercised muscle for recovery of performance originates

from earlier studies demonstrating that intracellular acidosis contributed to muscle fatigue (7,33). Although recent research in skinned muscle fiber preparations that are closer to *in vivo* body temperature has questioned the role of  $La^-$  and/or  $H^+$  acidosis in muscle fatigue (33), other *in situ* (13) and *in vitro* (17) evidence argues for a role of both in fatigue, such that no conclusive consensus on lactic acid's role in fatigue has been reached (7). Therefore, it still remains that early lactic acid removal from the muscle may be advantageous to improve athletic performance, in particular in athletic events that involve intermittent bursts at a high power output where substantial lactic acid production would occur (30) and rapid recovery is required.

Previous studies have examined the effects of massage and/or active recovery on clearance of  $La^-$  from the systemic circulation (11,12,20,21), and the predominant finding is that massage does not enhance systemic blood lactate disappearance beyond passive recovery whereas active recovery does. However, no study has investigated its efficacy in removing lactic acid from the exercised muscle. This efficacy would require that massage increases muscle blood flow and/or lactic acid movement into the bloodstream. Although massage appears to have no impact on resting muscle blood flow (23) or muscle blood flow between brief pauses in massage postexercise (12), there is a solid physiological basis for predicting that massage could

Address for correspondence: Michael E. Tschakovsky, Ph.D., School of Kinesiology and Health Studies and Department of Physiology, Queen's University, Kingston, Ontario, Canada K7L 3N6; E-mail: mt29@queensu.ca. Submitted for publication July 2009.

Accepted for publication November 2009.

0195-9131/10/4206-1062/0

MEDICINE & SCIENCE IN SPORTS & EXERCISE®

Copyright © 2010 by the American College of Sports Medicine

DOI: 10.1249/MSS.0b013e3181c9214f

elevate muscle blood flow on the basis of recent findings of rapid vasodilatory responses of resistance vessels to repeated compression (5,6,16).

Active recovery (light exercise during recovery) has also been proposed as a means of improving  $\text{La}^-$  removal from muscle postexercise by either maintaining elevated muscle blood flow (20) and/or by maintaining elevated  $\text{La}^-$  uptake in the muscle (1,2). Again, no studies have directly examined this hypothesis. Therefore, the purpose of this experiment was to test the hypothesis that massage and/or active recovery can improve muscle blood flow and lactic acid removal from exercised muscle after intense exercise.

## METHODS

### General Methods

**Subjects.** Twelve healthy male subjects (age =  $24.2 \pm 1.5$  yr (mean  $\pm$  SE)) participated in this study. All subjects reported being nonsmokers with no history of cardiovascular disease. This study was approved by the Health Sciences Human Research Ethics Board at Queen's University according to the terms of the Declaration of Helsinki. After receiving a complete verbal and written description of the experimental protocol and potential risks, each subject provided signed consent.

**Maximum voluntary contraction (MVC).** Each subject performed three maximal isometric handgrip (IHG) contractions using a calibrated handgrip dynamometer, with 1 min between maximal efforts. The MVC was taken as the peak contraction force observed.

**IHG exercise.** Subjects squeezed a handgrip dynamometer continuously at 40% MVC for 2 min. Subjects received visual feedback of their force output on a computer screen and were verbally encouraged by the researchers to maintain contraction force at 40% MVC.

**Post-IHG passive recovery.** After the 2-min IHG, the subject lay quietly with the forearm at rest for 10.5 min.

**Post-IHG active recovery.** After the 2-min IHG, the subjects lay quietly with the forearm at rest for 30 s, at which time they began 10 min of rhythmic forearm contractions at 10% MVC at a duty cycle of 1:2-s contraction-relaxation in time with a metronome. Visual feedback displaying force output on a computer screen guided the contractions.

**Post-IHG massage.** After 2 min of IHG, the subject lay quietly with the forearm at rest for 30 s, at which time forearm massage began. Massage was administered by a registered massage therapist who had 13 yr of experience in sports massage (J. Rayner, coauthor). The massage strokes applied were chosen to represent common techniques used in postevent massage (15) and consisted of firm effleurage for the first and the last 2.5-min intervals with *pétrissage* during the 5-min period in between. Effleurage is defined as a "gliding or movement over the skin with a smooth continuous motion" directed toward the heart, whereas *pétrissage* is defined as "lifting, wringing, or squeezing of

soft tissues in a kneading motion, or pressing or rolling of the tissues under or between the hands" (31).

**Arterialized blood sampling.** Before the commencement of the experimental protocols, all subjects had their right hand heated with a standard hydrocollator heating pad (Midland Tropic Heater; Therapeutic Equipment Corporation, Clifton, NJ) for 10 min. After the hand was heated, a 1-mL venous blood sample was taken from a superficial dorsal hand vein using a standard venous catheter. Using venous blood samples from a heated vein has been validated to be an accurate representation of arterial levels of lactate and hemoglobin (35). The blood sample was immediately analyzed (StatProfile M Blood Gas Analyzer; Nova Biomedical, Mississauga, Canada). The minimum accepted value of oxygen saturation ( $\text{SO}_2$ ) to indicate appropriate arterializations was 90%; if the sample tested below this cutoff value, heating continued until a minimum of 90%  $\text{SO}_2$  was attained as assessed by further blood sampling. Because the  $\text{La}^-$  produced by a small muscle mass has been shown to have negligible effects on arterial blood [ $\text{La}^-$ ] concentration (4), this baseline arterialized sample was deemed to represent arterial [ $\text{La}^-$ ] and [ $\text{H}^+$ ] throughout the experimental protocol.

**Central hemodynamic monitoring.** HR (standard  $\text{CM}_5$  ECG placement) and mean arterial pressure (MAP; finger photoplethysmography on the middle finger of the resting hand, Finometer Midi; Finapres Medical Systems, Amsterdam, The Netherlands) were measured beat to beat. Arterial oxygen saturation ( $\text{S}_a\text{O}_2$ ) data were estimated via a pulse oximeter (Nellcor N-395, Covidien-Nellcor, Boulder CO) placed over a fingertip of the resting hand.

**Forearm deep venous blood sampling.** A 20-gauge venous catheter was inserted retrograde into an antecubital vein on the experiment arm (14). Confirmation that the vein drained forearm muscle was obtained via Echo ultrasound imaging before catheterization. An initial blood sample of 2 mL was discarded and followed by a 1-mL sample for analysis at all sample times.

**Brachial artery mean blood velocity and diameter.** Brachial artery mean blood velocity (MBV) was measured beat by beat on the experimental arm with a flat, pulsed-Doppler ultrasound probe (Model 500V TCD; Multigon Industries, Mt. Vernon, NY) with an operating frequency of 4 MHz. The probe was fixed to the skin over the brachial artery proximal to the antecubital fossa region of the forearm that would perform IHG. Brachial artery diameter was measured approximately 5 cm proximal to the site of MBV measurement using a 10-MHz linear echo Doppler ultrasound probe operating in B mode (Vingmed System FiVe; GE Medical Systems, London Ontario, Canada) (for more details about velocity and diameter measurement methodology, see Tschakovsky et al. [27,28]).

### Experimental Protocol and Data Acquisition

There were three experimental conditions. Eleven of 12 subjects performed all three on the same day, and the order of conditions was counterbalanced between subjects. All

subjects arrived for testing after having refrained from exercise and caffeine for the previous 12 h and from food and beverages for 4 h. They were positioned supine for the duration of the experiment. The forearm was positioned at heart level for all conditions.

Figure 1 identifies the specific experimental protocol and the timing of data acquisition. The order of experimental conditions was counterbalanced across subjects. Adequate time (typically ~15–20 min) was allowed between trials for all variables to return to original baseline levels before the next trial began.

**Baseline data acquisition.** A 1-mL venous blood sample was taken 1–2 min before the commencement of IHG and immediately analyzed for  $La^-$ ,  $C_vO_2$ , Hb, pH,  $P_vO_2$ , and  $S_vO_2$ . One minute of continuous beat-by-beat data acquisition of central hemodynamic and brachial artery MBV and diameter was performed with the subject in a resting supine position.

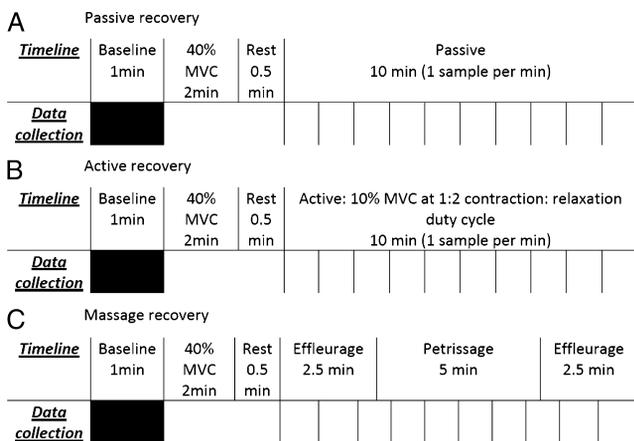
**IHG data acquisition.** Forearm handgrip force was monitored continuously, and central hemodynamic variables were recorded during the final 10 s of IHG.

**Post-IHG data acquisition.** Central hemodynamic measures and brachial artery MBV and diameter were obtained over an approximately 20-s period every minute starting at 30 s post-IHG for a total of 10 min. At these times, blood sampling was also performed as described above.

**Between trials.** The subject was asked to remain relaxed and supine until further venous blood samples ensured that the forearm venous blood  $[La^-]$  was within  $0.4 \text{ mmol}\cdot\text{L}^{-1}$  of baseline values before commencing the next protocol. This required a minimum of 10 min.

**Data Analysis**

**Central hemodynamics.** Beat-by-beat HR and MAP were averaged over 1-min baseline and 20-s periods every minute starting at 30 s after the end of the IHG exercise.



**FIGURE 1**—The experimental protocol timeline and data collection time points. (A) Passive recovery condition, (B) active recovery condition, and (C) massage recovery condition. MVC, maximum voluntary contraction; R, rest. Data collection time points are shown as vertical marks below the horizontal line.

**Venous blood constituents.**  $La^-$ ,  $C_vO_2$ , Hb, pH,  $P_vO_2$ , and  $S_vO_2$  were analyzed from venous blood samples (both arterialized sample and samples from the deep cubital vein) using the StatProfile M Blood Gas Analyzer (Nova Biomedical, Mississauga, Ontario, Canada). Ongoing blood sample analysis was initiated with the beginning of the experimental conditions so that all samples were analyzed within approximately 30 min of collection. Blood samples were kept on ice between the time they were drawn and the time of the analysis.

**Brachial artery diameter.** The acquired brachial artery images were stored in a Digital Imaging and Communications in Medicine (DICOM) format with custom software on a dedicated computer. The diameter of the brachial artery was analyzed via automated edge-detection software (34), which identifies a region of interest where the vessel walls are the clearest. The arterial walls are then tracked by detection of the greatest change in pixel brightness in the vertical axis. The edge-detection software collects one diameter measurement for every pixel column and uses the median diameter as the diameter for that frame.

**MBV, ECG, IHG, and  $S_aO_2$ .** These were collected via a data acquisition system (Powerlab; ADInstruments, Inc., Bella Vista, Australia) at a sampling frequency of 200 Hz and stored on a personal computer. IHG at 40% MVC was analyzed by averaging the force output over 20-s time bins for the full 2 min of contraction. For MBV, ECG, and  $S_aO_2$ , baseline values were averaged over a 60-s period. Then, starting at 30 s post-IHG, approximately 20-s averages were taken every 60 s for the massage and passive conditions. For the active recovery condition, the IHG exercise at 10% MVC was analyzed over seven cycles of the exercise.

**Calculated variables.** Forearm blood flow (FBF) was calculated from measured brachial artery MBV and diameter using the following equation:

$$FBF = MBV \times 60\pi(d/2)^2 \tag{1}$$

where FBF is in milliliters per minute, MBV is in centimeters per second, 60 represents  $60 \text{ s}\cdot\text{min}^{-1}$ , and  $d$  is the brachial artery diameter in centimeters (22).

Venoarterial  $La^-$  and  $H^+$  difference, representing the amount of  $La^-$  and  $H^+$  in the forearm venous effluent as a result of the exercise stimulus to muscle metabolism, was determined by the following equation:

$$[La^-]_{(v-a)} = [La^-]_{\text{venous}} - [La^-]_{\text{arterial}} \tag{2}$$

The  $La^-$  and  $H^+$  efflux, which represents the removal of either ion from the exercised forearm via the blood in excess of that at baseline rest, was determined by the following equation:

$$\begin{aligned} La^- \text{ efflux} &= [La^-]_{(v-a)}/1000 \times FBF \\ H^+ \text{ efflux} &= [H^+]_{(v-a)}/1000 \times FBF \end{aligned} \tag{3}$$

where  $La^-$  and  $H^+$  efflux is in millimoles per minute and  $[La^-]_{(v-a)}$  and  $[H^+]_{(v-a)}$  are in millimoles per liter of

blood (19). The value 1000 is a correction factor to convert millimoles per liter into millimoles per milliliter so that it can be multiplied by FBF, which is in millimoles per minute.

Forearm oxygen uptake ( $\dot{V}O_2$ ) was calculated using the Fick equation:

$$\dot{V}O_2 = \text{FBF}(C_aO_2 - C_vO_2)/100 \quad [4]$$

where  $\dot{V}O_2$  is in milliliters of  $O_2$  per minute,  $C_vO_2$  is in milliliters of  $O_2$  per deciliter of blood, the value 100 is a factor to convert milliliters of  $O_2$  per deciliter of blood into milliliters of  $O_2$  per milliliters of blood so that it can be multiplied by FBF, which is in milliliters of blood per minute.  $C_aO_2$  is calculated from the following:

$$C_aO_2 = (1.34\text{Hb})(S_aO_2/100) + 3 \quad [5]$$

where  $C_aO_2$  is measured as milliliters of  $O_2$  per deciliter of blood, 1.34 is the physiological oxygen-binding capacity constant for Hb (measured in milliliters of  $O_2$  per gram Hb), Hb is in grams per deciliter of blood,  $S_aO_2$  is measured in percent, and 3 is the dissolved  $O_2$  content in 1 dL of blood at an assumed  $PO_2$  of 100 mm Hg as would be expected at the close to sea-level altitude of our laboratory.

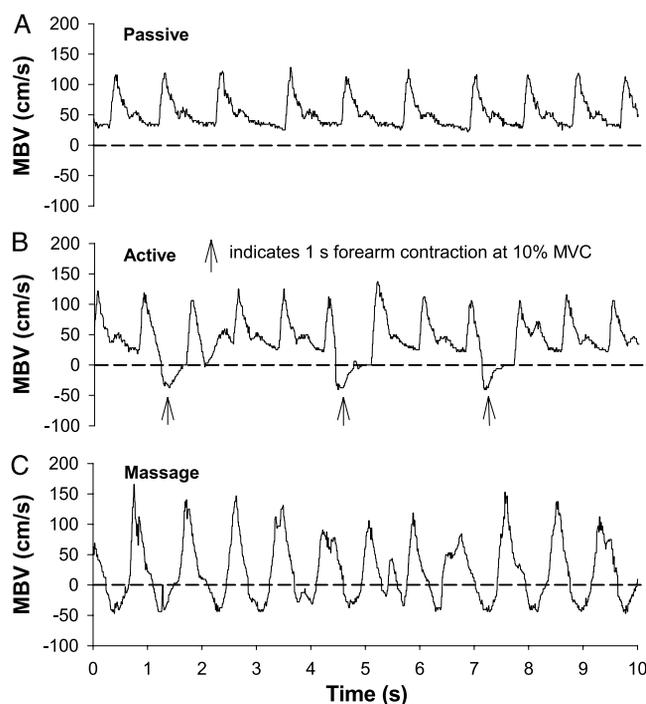
The area under the curve (AUC) was used to quantify the total amount of a variable over the course of the 10.5-min recovery after IHG. The trapezoid rule was used to quantify the AUC between data points and summed to provide total AUC.

## Statistical Analysis

This study was a repeated-measures design where all subjects participated in all experimental conditions and times. The main effects of condition (passive recovery, massage recovery, and active recovery) and time and condition  $\times$  time interactions for IHG contraction intensity and the post-IHG central hemodynamic variables, FBF,  $[La^-]_{(v-a)}$ ,  $[H^+]_{(v-a)}$ ,  $La^-$  and  $H^+$  efflux, and  $\dot{V}O_2$ , were examined with a two-way repeated-measures ANOVA. Subsequent *post hoc* testing was performed using Tukey's test for pairwise comparisons to isolate specific differences if there was an interaction between time and condition. Effects of experimental condition on AUC for FBF,  $[La^-]_{(v-a)}$ ,  $[H^+]_{(v-a)}$ ,  $La^-$  and  $H^+$  efflux, and  $\dot{V}O_2$  were examined with a one-way repeated-measure ANOVA. Statistical significance for all tests was set at  $P < 0.05$ . Data are presented as mean  $\pm$  SE. All data analysis was performed using commercial statistical software (SigmaStat 3.1, SPSS, Chicago, IL).

## RESULTS

**Brachial artery blood velocity profile.** Figure 2 illustrates the typical effect that passive recovery, active re-

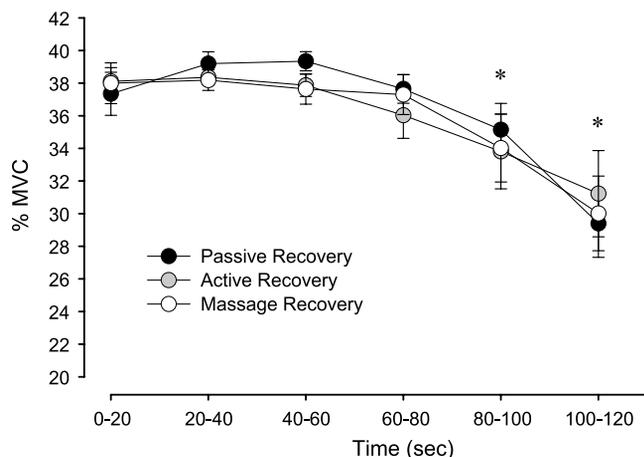


**FIGURE 2—**Examples of beat-by-beat mean blood velocity (MBV) for one subject during the three recovery conditions after IHG. (A) Passive recovery condition illustrating the continuous unimpeded pulsatile postexercise hyperemia. (B) Active recovery condition (10% MVC at a duty cycle of 1:2-s contraction–relaxation) illustrating the rhythmic retrograde brachial artery FBF occurring during forearm contractions. (C) Massage recovery condition (pétrissage in this example) illustrating the rhythmic retrograde brachial artery FBF occurring with each massage stroke.

covery, and massage recovery (in this case pétrissage) have on the velocity of blood flow through the brachial artery in a sample subject. Under passive rest conditions, there is an uninterrupted pulsatile flow. With mild-intensity (rhythmic 10% MVC contractions) active recovery exercise, there is rhythmic retrograde brachial artery blood velocity (negative velocity), consistent with the muscular compression of the vasculature during contraction. With pétrissage, consistent retrograde flow with each massage stroke followed by increases in blood flow velocity during brief pauses is clearly evident. The same was observed during effleurage.

**Isometric hand grip (40% MVC).** Figure 3 illustrates the mean percentage of MVC among subjects in 20-s intervals over the 2-min period. The strength of contraction declined over time, as is evident with a decreasing percentage of MVC in the second minute, which was significantly reduced by 80 s of IHG despite visual feedback and handgrip force and verbal encouragement from the investigators to maintain 40% MVC.

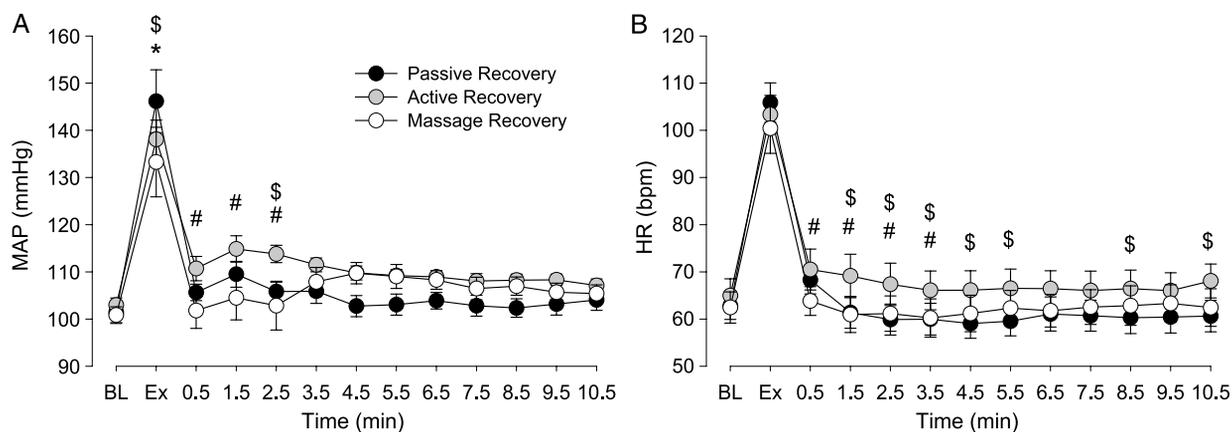
**Central hemodynamics (MAP and HR).** A summary of the MAP and HR responses is illustrated in Figure 4. There was no difference between conditions at baseline for either MAP or HR, confirming a return to baseline between trials.



**FIGURE 3**—Percentage of maximum voluntary contraction (% MVC) during the isometric hand grip (IHG) exercise portion of the experiment before the commencement of the recovery conditions. Values are averages of 20-s time blocks. \*Significantly different from 20- to 40-s time period for all recovery conditions ( $P < 0.05$ ). Data are presented as mean  $\pm$  SE.

The MAP by the end of IHG had increased more in the passive recovery versus both massage and active recovery ( $P < 0.001$  and  $P = 0.038$ , respectively). However, by 30 s post-IHG, MAP had returned to baseline levels in the passive and massage recovery conditions. In contrast, the maintenance of light forearm exercise resulted in a MAP that remained higher than massage recovery during the first 2.5 min postexercise and achieved statistical significance at 2.5 min versus passive recovery.

By the end of the IHG, HR had increased substantially and to the same degree in all three recovery conditions. However, by 30 s post-IHG, it had returned to baseline in both the passive and the massage recovery trials. In contrast, it remained elevated in the active recovery condition relative to the other recovery conditions for part of the recovery period.

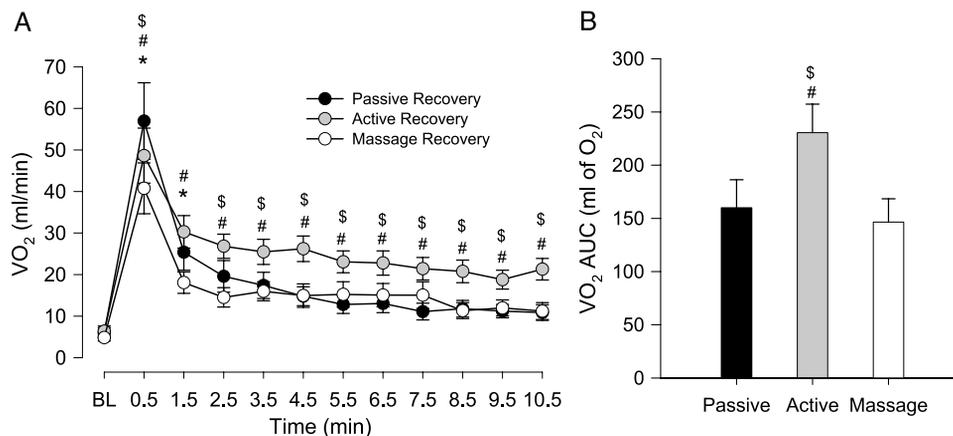


**FIGURE 4**—Central hemodynamic response to IHG and during recovery conditions. (A) MAP; (B) HR. BL, average of 1-min baseline before onset of IHG; Ex, average of the last 10 s of IHG. \*Significant difference between passive recovery and massage recovery. #Significant difference between active recovery and massage recovery. \$Significant difference between passive recovery and active recovery (all  $P < 0.05$ ). Data are presented as mean  $\pm$  SE.

**Forearm oxygen uptake ( $\dot{V}O_2$ ).** The data for  $\dot{V}O_2$  at baseline before IHG and after IHG are illustrated in Figure 5. At baseline,  $\dot{V}O_2$  was virtually identical across conditions, indicating complete metabolic recovery between trials. Immediately post-IHG,  $\dot{V}O_2$  in passive recovery was greater than active recovery and massage recovery ( $P = 0.002$  and  $P < 0.001$ , respectively), and active recovery was greater than massage recovery ( $P = 0.004$ ). The active recovery condition resulted in the maintenance of a higher  $\dot{V}O_2$  during the entire post-IHG recovery period versus passive and massage recovery ( $P < 0.001$ ), with the exception of 1.5 min post-IHG versus passive recovery (where  $P = 0.108$ ). As is expected, the total oxygen consumption during the post-IHG period (AUC) was substantially greater in active recovery versus both passive recovery and massage recovery ( $P < 0.001$ ).

**Forearm blood flow (FBF).** Figures 6A and B illustrate the FBF at baseline before IHG and after IHG. Baseline was not different between conditions, indicating complete recovery between trials. FBF was reduced in massage recovery versus passive recovery for the first 3.5 min post-IHG ( $P < 0.012$ ) and was reduced in active recovery versus passive recovery for the first 1.5 min post-IHG ( $P < 0.013$ ). FBF was elevated in active recovery versus massage recovery at 1.5–2.5 min post-IHG ( $P < 0.023$ ). There were no statistically significant differences between recovery conditions after 4.5 min post-IHG. The AUC for FBF (total FBF during the post-IHG recovery period) was significantly reduced in massage recovery versus passive recovery ( $P = 0.024$ ) but not versus active recovery ( $P = 0.504$ ). Active recovery AUC was not different from passive recovery AUC ( $P = 0.217$ ).

**Forearm lactic acid removal.** Figures 6C and D illustrate the post-IHG venoarterial blood lactate concentration difference  $[La^-]_{(v-a)}$ . There was no difference between recovery conditions immediately after IHG, indicating that the same metabolic stress was achieved in all trials.



**FIGURE 5—(A)** Forearm oxygen uptake ( $\dot{V}O_2$ ) change over time. **(B)** Total area under the curve (AUC) during recovery. BL, average of 1-min baseline before onset of IHG. \*Significant difference between passive recovery and massage recovery. #Significant difference between active recovery and massage recovery. \$Significant difference between passive recovery and active recovery (all  $P < 0.05$ ). Data are presented as mean  $\pm$  SE.

By 4.5 min post-IHG, there was a significantly lower  $[La^-]_{(v-a)}$  level in active recovery versus passive recovery with the exception of time 6.5 min, where  $P = 0.072$ . In contrast, there was no difference between the passive recovery versus the massage recovery condition at any time point after IHG ( $P = 0.79$ ). The  $[La^-]_{(v-a)}$  (AUC) was approximately 17% lower in active recovery versus passive recovery, just missing statistical significance ( $P = 0.057$ ). This was likely a type I error (false negative) because of inadequate statistical power.

$La^-$  efflux was higher for the first 1.5 min post-IHG in passive recovery versus active recovery ( $P < 0.003$ ) and for the first 2.5 min post-IHG versus massage recovery ( $P < 0.001$ ) (Fig. 5E). Figure 5F illustrates the total  $La^-$  efflux (AUC) during the post-IHG period. This was significantly lower in massage recovery versus passive recovery ( $P = 0.033$ ). The  $La^-$  efflux AUC was approximately 25% lower in active recovery versus passive recovery but failed to reach statistical significance ( $P = 0.064$ ). Again, this likely represents a type I error.

Figure 7A illustrates forearm venous pH. There was significant acidosis apparent in the venous blood after IHG. This acidosis gradually diminished over the 10-min recovery period to the same degree in all recovery conditions such that it was no longer different from baseline by 9.5 min for passive recovery ( $P = 0.107$ ) and active recovery ( $P = 0.146$ ) and by 10.5 min for massage ( $P = 0.197$ ). Baseline pH was the same across conditions ( $P = 0.967$ ), indicating complete recovery of venous acid/base before the start of each trial.

Figures 7B and C illustrate the  $[H^+]_{(v-a)}$  over time and the AUC, respectively. Despite isolated differences between recovery conditions, the AUC for  $[H^+]_{(v-a)}$  was virtually identical across conditions ( $P = 0.744$ ). Figures 7D and E illustrate forearm  $H^+$  efflux. This was reduced in massage recovery versus passive recovery for the first 1.5 min post-IHG ( $P < 0.003$ ) and in active recovery versus passive recovery for the first 30 s post-IHG ( $P < 0.001$ ). However,

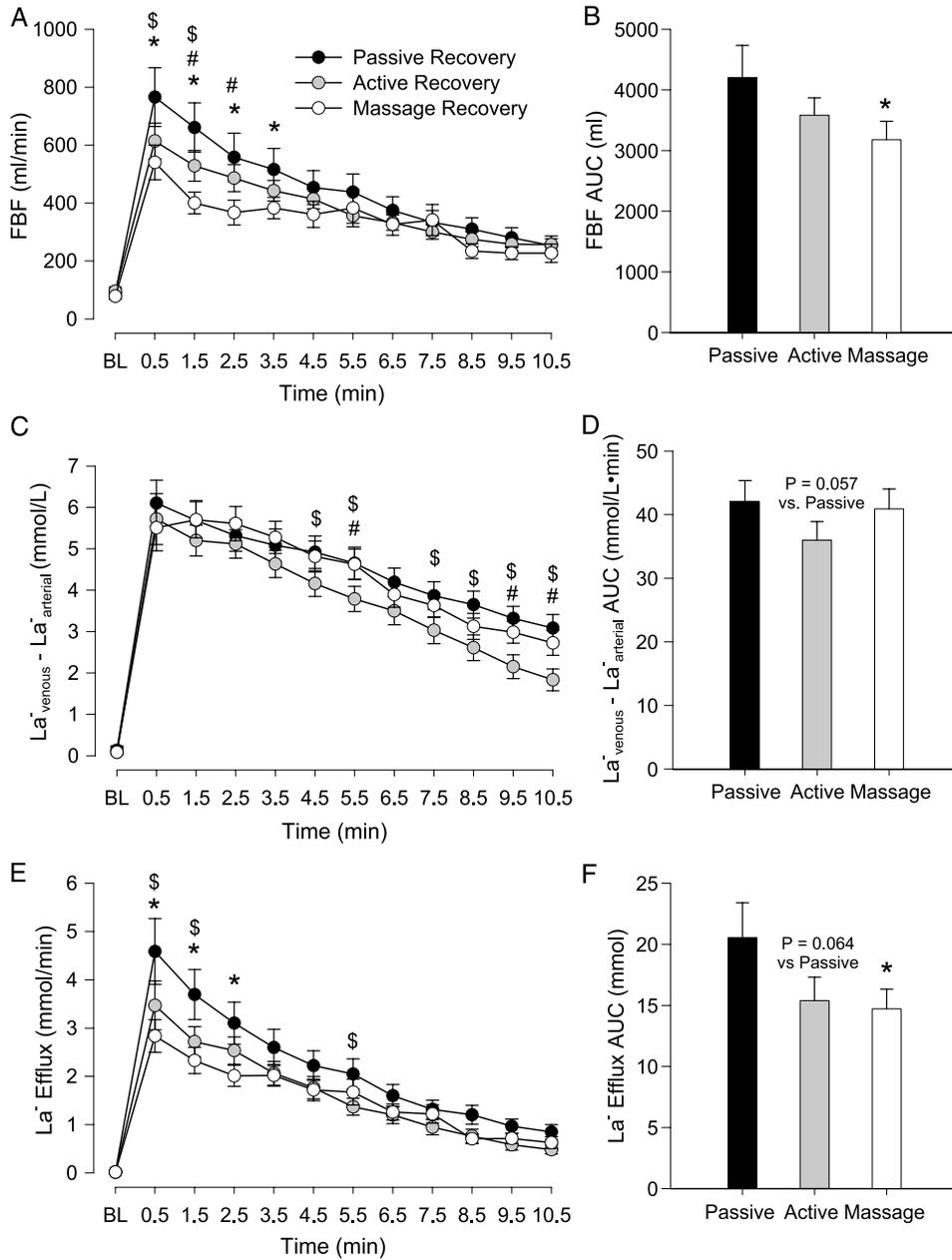
the  $H^+$  efflux AUC was not different between conditions ( $P = 0.191$ ).

## DISCUSSION

This study was the first to test the hypothesis that sport massage and/or active recovery can improve muscle blood flow and lactic acid removal from exercised muscle after intense exercise. The major findings of this study are 1) both massage and active recovery resulted in rhythmic retrograde brachial artery flow post-IHG exercise, 2) FBF post-IHG exercise was substantially reduced during massage recovery and somewhat reduced during active recovery compared with passive recovery, 3)  $La^-$  efflux from the forearm post-IHG exercise was substantially reduced and  $H^+$  efflux was transiently reduced by massage recovery compared with passive recovery because of compromised FBF, and 4) active recovery appears to result in forearm muscle consumption of  $La^-$  as evidenced by significantly lower  $[La^-]_{(v-a)}$ .

These results suggest that sport massage actually impairs removal of lactic acid from exercised muscle and that this is due to a mechanical impairment to postexercise muscle blood flow from rhythmic compression of muscle tissue. Active recovery appears to increase muscle utilization of  $La^-$  that had been generated in the exercised muscle.

**IHG effect on cardiovascular and muscle metabolic and contractile function.** On the basis of pilot work, we chose 40% MVC IHG contraction for 2 min to incur a substantial metabolic acidosis and inability to maintain the desired muscle contraction force (fatigue), as might occur with short-duration intense sports events (e.g., ice hockey shift). The progression of fatigue in the second minute of the IHG was the same across all three post-IHG conditions. Furthermore, there was a substantial increase in  $[La^-]_{(v-a)}$  to approximately  $6 \text{ mmol}\cdot\text{L}^{-1}$ . This is actually an underestimate of venous lactate concentration because it

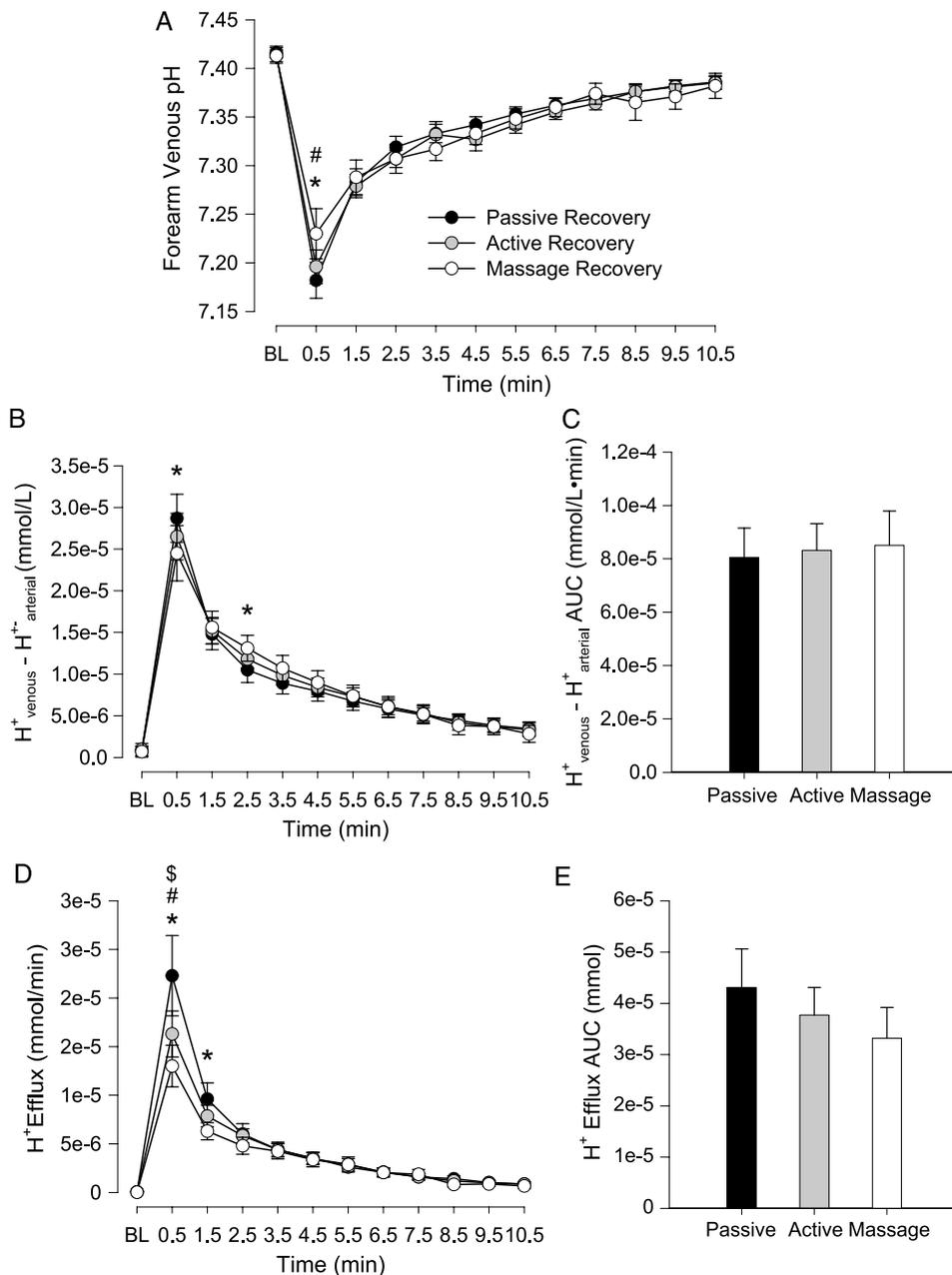


**FIGURE 6—(A) Forearm blood flow (FBF). (B) FBF AUC. (C) Difference between venous and arterial lactate concentrations ( $[La^-]_{venous} - [La^-]_{arterial}$ ). (D)  $[La^-]_{venous} - [La^-]_{arterial}$  AUC. (E) Lactate efflux ( $La^-$  efflux). (F)  $La^-$  efflux AUC. BL, average of 1-min baseline before onset of IHG. \*Significant difference between passive recovery and massage recovery. #Significant difference between active recovery and massage recovery. \$Significant difference between passive recovery and active recovery (all  $P < 0.05$ ). Data are presented as mean  $\pm$  SE.**

represents the difference between venous and arterialized samples, the latter being approximately  $1 \text{ mmol}\cdot\text{L}^{-1}$  (data not shown). A further demonstration of the magnitude of metabolic demand with this level of IHG was the forearm  $\dot{V}O_2$  immediately post-IHG, which had increased approximately 8- to 10-fold. Finally, we observed substantial elevations in MAP and HR despite the limited muscle mass involved in exercise. Importantly, all metabolic and cardiovascular variables returned to baseline between trials. Thus, we conclude that IHG in our study provided a consistent and substantial effect on cardiovascular indicators of

exercise intensity and muscle metabolic and contractile function between recovery conditions. Therefore, differences in the removal of lactic acid between conditions can be ascribed specifically to the modality of recovery.

**Impairment of muscle blood flow postexercise by massage and/or active recovery.** The view among the athletic community (therapists and athletes alike) that massage acts to improve muscle blood flow is commonly attributed to the effects of direct compression on vasculature, localized release of vasodilators, and decrease in sympathetic tone as a response to mechanical pressure on tissue (10,20).



**FIGURE 7—(A) Forearm venous pH. (B) Difference between venous and arterial hydrogen ion concentrations ( $[H^+]_{\text{venous}} - [H^+]_{\text{arterial}}$ ). (C)  $[H^+]_{\text{venous}} - [H^+]_{\text{arterial}}$  AUC. (D) Hydrogen ion efflux ( $H^+$  efflux). (E)  $H^+$  efflux AUC. BL, average of 1-min baseline before onset of IHG. \*Significant difference between passive recovery and massage recovery. #Significant difference between active recovery and massage recovery. \$\$Significant difference between passive recovery and active recovery (all  $P < 0.05$ ). Data are presented as mean  $\pm$  SE.**

Indeed, there are both mechanical and vasodilatory mechanisms that respond to the compressive effects of muscle contraction and are thought to be important contributors to increased muscle blood flow during exercise (for a review, see Tschakovsky and Sheriff [29]).

Although some earlier studies seemed to support a net enhancement effect of massage (as reviewed by Goats [10] and Weerapong et al. [31]), more recent investigations using ultrasound measurements of arterial inflow to a massaged limb at rest found massage had no effect on blood flow (23,26). Furthermore, Hinds et al. (12) also found no effect of mas-

sage on blood flow immediately after leg exercise, albeit measurements appear to have been taken during brief breaks in massage. However, none of these studies have provided insight into the effect of massage, or for that matter active recovery, on muscle blood flow to the exercised muscle after exercise.

A critical consideration regarding net enhancement effects of muscle compression on muscle blood flow comes from an elegant study by Lutjmeier et al. (18). These investigators measured leg blood flow during increasing contraction intensities and partitioned flow into that during contraction versus

relaxation versus the average across contraction or relaxation duty cycles. They demonstrated that there are impairment effects to blood flow during contraction (i.e., mechanical compression of muscle resistance vessels) and enhancement effects above that of underlying vasodilation during relaxation (possible muscle pump and/or rapid vasodilation effects [29]).

In the present study, we measured FBF during massage and active recovery for the period after intense forearm exercise. Blood flow to the forearm was clearly impaired during the first 4.5 min of recovery after exercise by massage. This would have to be a function of impairment effects outweighing enhancement effects at higher muscle blood flows early in the postexercise period, whereas later on, the impairment and enhancement effects cancel each other out. The latter observation is consistent with other investigations of muscle blood flow in massage (12,23,26).

With regard to active recovery, the maintenance of low-intensity contractions also had a net impairment effect early in the postexercise recovery period. However, this was eventually cancelled out at lower FBF by the enhancement effect during relaxation. Nevertheless, FBF was never greater during recovery from IHG with active recovery compared with passive recovery.

Given that exercise (albeit mild intensity) was occurring during active recovery, it may at first seem puzzling that this did not result in enhancement of recovery FBF. However, there is a simple explanation for this. Two minutes of IHG at 40% results in approximately maximal exercise-evoked forearm vasodilation (see Fig. 6, where FBF is  $\sim 800 \text{ mL}\cdot\text{min}^{-1}$  30 s into recovery, consistent with the highest FBF during peak forearm exercise commonly observed in our laboratory). Further, the magnitude of this dilation remains very high for several minutes (Fig. 6). The oxygen delivery requirement of 10% MVC rhythmic exercise is much less than this so that the lower FBF during rhythmic exercise in recovery versus passive recovery earlier in the recovery period still represents a relative hyperperfusion in excess of the demand for  $\text{O}_2$ . Therefore, it is likely that no additional vasodilatory signal indicating hypoperfusion relative to metabolic demand is present. However, later in the recovery period, the flow is identical in passive and active recovery, although there is impedance due to contraction. This indicates that a greater vasodilation in active recovery over this period must be occurring to offset the contraction-induced impedance. This would be consistent with matching  $\text{O}_2$  delivery to the maintained elevation in metabolic demand.

**Effects of massage and active recovery on lactic acid removal from exercised muscle.** Previous studies examining massage and lactate removal did not look at removal of lactate from the exercised muscle tissue but instead determined the removal of lactate from the systemic circulation (11,12,20,21). To the best of our knowledge, this is the first study that examined venous  $\text{La}^-$  and  $\text{H}^+$  concentration from the deep veins that drained the exercising (and massaged muscles), thus allowing us to investigate

the impact of massage and active recovery on lactic acid removal from exercised muscle.

We found that lactic acid efflux was reduced by massage as a direct consequence of impairment to FBF. The same was true early on during active recovery. The net effect of massage was a 25% reduction in  $\text{La}^-$  efflux over the 10.5-min recovery period after IHG and a transient early  $\text{H}^+$  efflux reduction. It must be recognized that efflux through the blood is not the only process by which muscle lactic acid levels can be reduced. However, this study specifically tested the hypothesis that massage and/or active recovery improve removal of lactic acid via effects on blood flow. In this context, massage clearly impaired lactic acid removal rather than enhancing it.

Active recovery resulted in a gradual reduction in venous lactate concentration, such that as early as 4.5 min into recovery,  $[\text{La}^-]_{(v-a)}$  was reduced relative to the other recovery conditions.  $\dot{V}\text{O}_2$  remained significantly elevated in active recovery, suggesting that the muscle was likely using lactate it had previously produced during IHG as a fuel for oxidative metabolism, consistent with the well-known lactate shuttle hypothesis (1,9). Thus, although active recovery did not enhance lactic acid efflux from muscle via the blood, its impact on local metabolism may provide an additional benefit in this regard.

**Limitations.** The massage strokes used in this study (effleurage and pétrissage) do not represent the only massage techniques used in postevent recovery, and thus the results of this study cannot be generalized to other massage techniques. However, these do represent common postevent massage techniques (12,15), and therefore the results of our study are relevant to current sports massage treatment approaches.

This study was restricted to the use of a forearm model because to measure lactate efflux from exercised muscle, we required venous blood samples from that muscle. This was not possible to obtain from the femoral vein in our laboratory because of the serious invasive nature of such a catheterization site. As such, the percentage of the total forearm muscle mass affected by each massage stroke would likely have been larger than for the leg. However, the effect of compression on leg muscle tissue under the massage stroke would be the same, and therefore restriction to those areas would likely be no different.

## CONCLUSIONS

In summary, sports massage results in severe impairment to blood flow during the massage stroke, and this impairment has a net effect of decreasing muscle blood flow early in the recovery period after strenuous exercise. Similar effects occur with active recovery. This effect is responsible for impairing lactic acid removal from exercised muscle. Although active recovery also does not improve muscle blood flow postexercise, it appears to increase lactate uptake by

muscle and in that way improves lactate removal from muscle tissue. On the basis of these results, sports massage would not be indicated for optimal lactic acid removal from exercised muscle in situations where acute bouts of repeated exercise are occurring.

## REFERENCES

- Brooks GA. Intra- and extra-cellular lactate shuttles. *Med Sci Sports Exerc.* 2000;30(4):790–9.
- Brooks GA. Lactate shuttles in nature. *Biochem Soc Trans.* 2002; 30(2):258–64.
- Callaghan MJ. The role of massage in the management of the athlete: a review. *Br J Sports Med.* 1993;27(1):28–33.
- Chudalla R, Baerwalde S, Schneider G, Maassen N. Local and systemic effects on blood lactate concentration during exercise with small and large muscle groups. *Pflugers Arch.* 2006;452(6):690–7.
- Clifford PS, Kluess HA, Hamann JJ, Buckwalter JB, Jasperse JL. Mechanical compression elicits vasodilatation in rat skeletal muscle feed arteries. *J Physiol.* 2006;572(Pt 2):561–7.
- Clifford PS, Tschakovsky ME. Rapid vascular responses to muscle contraction. *Exerc Sport Sci Rev.* 2008;36(1):25–9.
- Fitts RH. The cross-bridge cycle and skeletal muscle fatigue. *J Appl Physiol.* 2008;104(2):551–8.
- Galloway SD, Watt JM. Massage provision by physiotherapists at major athletics events between 1987 and 1998. *Br J Sports Med.* 2004;38(2):235–6.
- Gladden LB. Muscle as a consumer of lactate. *Med Sci Sports Exerc.* 2000;32(4):764–71.
- Goats GC. Massage—the scientific basis of an ancient art: part 2. Physiological and therapeutic effects. *Br J Sports Med.* 1994;28(3): 153–6.
- Hemmings B, Smith M, Graydon J, Dyson R. Effects of massage on physiological restoration, perceived recovery, and repeated sports performance. *Br J Sports Med.* 2000;34(2):109–14.
- Hinds T, McEwan I, Perkes J, Dawson E, Ball D, George K. Effects of massage on limb and skin blood flow after quadriceps exercise. *Med Sci Sports Exerc.* 2004;36(8):1308–13.
- Hogan MC, Gladden LB, Kurdak SS, Poole DC. Increased [lactate] in working dog muscle reduces tension development independent of pH. *Med Sci Sports Exerc.* 1995;27(3):371–7.
- Hughson RL, Shoemaker JK, Tschakovsky ME, Kowalchuk JM. Dependence of muscle  $\dot{V}O_2$  on blood flow dynamics at onset of forearm exercise. *J Appl Physiol.* 1996;81(4):1619–26.
- Jonhagen S, Ackermann P, Eriksson T, Saartok T, Renstrom PA. Sports massage after eccentric exercise. *Am J Sports Med.* 2004; 32(6):1499–503.
- Kirby BS, Carlson RE, Markwald RR, Voyles WF, Dinunno FA. Mechanical influences on skeletal muscle vascular tone in humans: insight into contraction-induced rapid vasodilatation. *J Physiol.* 2007;583(Pt 3):861–74.
- Kristensen M, Albertsen J, Rentsch M, Juel C. Lactate and force production in skeletal muscle. *J Physiol.* 2005;562(Pt 2):521–6.
- Lutjemeier BJ, Miura A, Scheuermann BW, Koga S, Townsend DK, Barstow TJ. Muscle contraction-blood flow interactions during upright knee extension exercise in humans. *J Appl Physiol.* 2005;98(4):1575–83.
- MacInnes A, Timmons JA. Metabolic adaptations to repeated periods of contraction with reduced blood flow in canine skeletal muscle. *BMC Physiol.* 2005;5:11.
- Martin NA, Zoeller RF, Robertson RJ, Lephart SM. The comparative effects of sports massage, active recovery, and rest in promoting blood lactate clearance after supramaximal leg exercise. *J Athl Train.* 1998;33(1):30–5.
- Robertson A, Watt JM, Galloway SD. Effects of leg massage on recovery from high intensity cycling exercise. *Br J Sports Med.* 2004;38(2):173–6.
- Saunders NR, Pyke KE, Tschakovsky ME. Dynamic response characteristics of local muscle blood flow regulatory mechanisms in human forearm exercise. *J Appl Physiol.* 2005;98(4):1286–96.
- Shoemaker JK, Tiidus PM, Mader R. Failure of manual massage to alter limb blood flow: measures by Doppler ultrasound. *Med Sci Sports Exerc.* 1997;29(5):610–4.
- Stewart PA. Modern quantitative acid-base chemistry. *Can J Physiol Pharmacol.* 1983;61(12):1444–61.
- Tiidus PM. Manual massage and recovery of muscle function following exercise: a literature review. *J Orthop Sports Phys Ther.* 1997;25(2):107–12.
- Tiidus PM, Shoemaker JK. Effleurage massage, muscle blood flow and long-term post-exercise strength recovery. *Int J Sports Med.* 1995;16(7):478–83.
- Tschakovsky ME, Rogers AM, Pyke KE, Saunders NR, Glenn N, Lee SJ, et al. Immediate exercise hyperemia in humans is contraction intensity dependent: evidence for rapid vasodilation. *J Appl Physiol.* 2004;97(2):739–47.
- Tschakovsky ME, Saunders NR, Webb KA, O'Donnell DE. Muscle blood-flow dynamics at exercise onset: do the limbs differ? *Med Sci Sports Exerc.* 2006;38(10):1811–8.
- Tschakovsky ME, Sheriff DD. Immediate exercise hyperemia: contributions of the muscle pump vs. rapid vasodilation. *J Appl Physiol.* 2004;97(2):739–47.
- Van Beekvelt MC, Shoemaker JK, Tschakovsky ME, Hopman MT, Hughson RL. Blood flow and muscle oxygen uptake at the onset and end of moderate and heavy dynamic forearm exercise. *Am J Physiol Regul Integr Comp Physiol.* 2001;280(6):R1741–R47.
- Weerapong P, Hume PA, Kolt GS. The mechanisms of massage and effects on performance, muscle recovery and injury prevention. *Sports Med.* 2005;35(3):235–56.
- Weinstein Y, Magazanik A, Grodjinovsky A, Inbar O, Dlin RA, Stewart PA. Reexamination of Stewart's quantitative analysis of acid-base status. *Med Sci Sports Exerc.* 1991;23(11): 1270–5.
- Westerblad H, Allen DG, Lannergren J. Muscle fatigue: lactic acid or inorganic phosphate the major cause? *News Physiol Sci.* 2002;17:17–21.
- Woodman RJ, Playford DA, Watts GF, Cheatham C, Reed C, Taylor RR, et al. Improved analysis of brachial artery ultrasound using a novel edge-detection software system. *J Appl Physiol.* 2001;91(2):929–37.
- Zavorsky GS, Lands LC, Schneider W, Carli F. Comparison of fingertip to arterial blood samples at rest and during exercise. *Clin J Sport Med.* 2005;15(4):263–70.