Effects of warm-up intensity on kinetics of oxygen consumption and carbon dioxide production during high-intensity exercise in horses

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Objective—To compare effects of low and high intensity warm-up exercise on oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) in horses.

Animals—6 moderately conditioned adult Standardbreds.

Procedures—Horses ran for 2 minutes at 115% of maximum oxygen consumption ($\dot{V}O_2$max), 5 minutes after each of the following periods: no warm-up (NoWU); 10 minutes at 50% of $\dot{V}O_2$max (LoWU); or 7 minutes at 50% $\dot{V}O_2$max followed by 45-second intervals at 80, 90, and 100% $\dot{V}O_2$max (HiWU). Oxygen consumption and $\dot{V}CO_2$ were measured during exercise, and kinetics of $\dot{V}O_2$ and $\dot{V}CO_2$ were calculated. Accumulated O_2 deficit was also calculated.

Results—For both warm-up trials, the time constant for the rapid exponential increase in $\dot{V}O_2$ was 30% lower than for NoWU. Similarly, the rate of increase in $\dot{V}CO_2$ was 23% faster in LoWU and HiWU than in NoWU. Peak values for $\dot{V}O_2$ achieved during the high-speed test were not significantly different among trials (LoWU, 150.2 ± 3.2 ml/kg/min; HiWU, 151.2 ± 4.2 ml/kg/min; NoWU, 145.1 ± 4.1 ml/kg/min). However, accumulated O_2 deficit (ml of O_2 equivalents/kg) was significantly lower during LoWU (65.3 ± 5.1) and HiWU (63.4 ± 3.9) than during NoWU (82.1 ± 7.3).

Conclusions and Clinical Relevance—Both the low- and high-intensity warm-up, completed 5 minutes before the start of high-intensity exercise, accelerated the kinetics of $\dot{V}O_2$ and $\dot{V}CO_2$ and decreased accumulated O_2 deficit during 2 minutes of intense exertion in horses that were moderately conditioned. (*Am J Vet Res* 2000;61:638–645)

Several studies in humans have revealed that warm-up (preliminary exercise) results in acceleration in the kinetics of **oxygen consumption** ($\dot{V}O_2$) during a subsequent bout of intense exertion.[1,2,a] Although the mechanisms for this acceleration have not been clearly elucidated, it has been hypothesized that increases in muscle blood flow and temperature following a warm-up enhance O_2 delivery and utilization in working muscle.[2,3] Because more strenuous exercise would result in a greater increase in muscle temperature, the intensity of the preliminary exercise could contribute to its effect on a subsequent exercise bout. However, a recent study in humans revealed that increased muscle temperature induced by passive heating of the legs before the onset of cycling exercise had no effect on the fast exponential component of $\dot{V}O_2$ kinetics.[4] Alterations in $\dot{V}O_2$ as a result of warm-up will affect the relative contributions of aerobic and anaerobic metabolism to energy expenditure during exercise at similar workloads above the **maximum rate of oxygen consumption ($\dot{V}O_2$max).** Indeed, studies in humans have revealed that prior exercise reduces the anaerobic contribution to energy expenditure during equivalent bouts of intense exercise, as reflected by lower values for accumulated O_2 deficit and concentrations of lactate and hydrogen ions in blood.[2]

Few studies have examined the effects of warm-up on the physiologic responses of horses during high-intensity exertion. Anecdotal evidence indicates that a variety of warm-up strategies are used for competitive horses. For example, Standardbred racehorses usually undertake a 1,600- to 2,000-m warm-up at near racing speed 5 minutes before the start of a race. In addition, these horses often receive an initial warm-up 30 to 45 minutes prior to a race. In contrast, warm-up routines used in Thoroughbreds are more varied but typically undertaken at low work intensities. This substantial difference in prerace strategy raises the question of the most appropriate intensity for warm-up exercise. Tyler et al[5] reported that 5 minutes of exercise at a workload equivalent to 50% of $\dot{V}O_2$max resulted in a substantial acceleration of the kinetics of respiratory gas exchange during 1 minute of intense exertion, undertaken 5 minutes after completion of the warm-up. However, to the authors' knowledge, no study has investigated the effects of warm-up intensity on respiratory gas exchange and energy metabolism in horses during heavy exercise. Studies in humans revealed that the kinetics of respiratory gas exchange are influenced by the intensity of the warm-up exercise. Whereas prior high-intensity exercise (greater than the onset of blood lactate accumulation) consistently results in augmentation of aerobic metabolism during subsequent heavy exercise, low intensity warm-up has little or no effect on the kinetics of gas exchange.[1,2,a]

The objective of the study reported here was to determine the effects of low and high intensity warm-up exercise protocols on respiratory gas exchange in horses during 2 minutes of exercise at a workload equivalent to 115% of the $\dot{V}O_2$max measured during an incremental exercise test. Our hypothesis was that a bout of warm-up exercise would hasten the onset of

Received Nov 23, 1998.
Accepted Aug 16, 1999.
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Supported by a grant from EP Taylor Equine Research Trust, ON, Canada.

$\dot{V}O_2$max and reduce accumulated oxygen deficit during intense exercise of defined duration. We further hypothesized that, compared with low intensity warm-up, the larger increase in body temperature following a high intensity warm-up would result in greater augmentation of aerobic metabolism.

Materials and Methods

All experiments were conducted after approval by the Institutional Laboratory Animal Care and Use Committee of The Ohio State University and were performed in compliance with their recommendations.

Experimental design—The effects of prior exercise on pulmonary gas exchange during intense exercise were examined in a 3-way balanced randomized crossover study. Six horses participated in each of 3 trials: a control or **no warm-up (NoWU)** trial in which there was no exercise prior to the sprint, a **low-intensity warm-up (LoWU)** trial in which horses completed 10 minutes of trotting at a speed equivalent to 50% $\dot{V}O_2$max prior to the sprint, and a **high-intensity warm-up (HiWU)** trial in which horses trotted for 7 minutes at a speed equivalent to 50% $\dot{V}O_2$max, followed by 45-second intervals during which horses galloped at speeds calculated to elicit 80, 90, and 100% $\dot{V}O_2$max. For an individual horse, the distance covered during the low- and high-intensity warm-up was the same (approx 2,700 m). Each horse completed each protocol twice for a total of 6 experiments for each subject, with ≥ 3 days between trials for individual horses.

Horses—Six Standardbreds (1 gelding and 5 mares) that were 3 to 7 years old and weighed 398 to 442 kg (mean ± SEM, 421.8 ± 7.3 kg) were used. All horses were housed indoors during the experimental period, fed a diet of timothy grass-alfalfa hay and mixed grain, and had access to a mineral block. All horses were conditioned and had regular treadmill exercise for at least 3 months prior to the study.

Measurement of $\dot{V}O_2$max—For each horse, $\dot{V}O_2$max and the relationship between $\dot{V}O_2$ and speed was determined during an incremental exercise test 3 days prior to the first experiment. The incremental exercise test consisted of the horse trotting on a high speed treadmill[b] inclined at 4° for 90 seconds at 4 m/s, after which the treadmill speed was increased by 1 m/s every 90 seconds until the horse was no longer able to maintain its position on the treadmill. The $\dot{V}O_2$ was measured every 10 seconds during the exercise test. The $\dot{V}O_2$max was defined as the value at which $\dot{V}O_2$ reached a plateau, despite further increases in speed and when **respiratory exchange ratio (RER)** was > 1.0. A plateau was defined as a change in $\dot{V}O_2$ of < 4 ml/kg/min with an increase in speed. From linear regression analysis (including measurements from speeds between 4 m/s and the speed immediately prior to the plateau in $\dot{V}O_2$ values), the running speed that elicited **115% of the $\dot{V}O_2$max (115% $\dot{V}O_2$max [st])**, as defined by this step test, was calculated for each horse.[6] A representative graph of the linear regression analysis of the $\dot{V}O_2$ and speed relationship was developed (**Fig 1**).

Experimental protocol—Food was withheld for 12 hours before each experiment. After aseptic preparation and induction of local anesthesia of the overlying skin, a copper constantan thermocouple[c] was inserted through a catheter[d] placed in the right jugular vein and attached to a thermometer[e]; the thermocouple protruded approximately 3 mm beyond the distal end of the catheter. The catheter-thermocouple was of sufficient length to reach the level of the right side of the heart, although the actual location of the thermocouple was not verified by examination of pressure waveforms. Following measurement of body weight[f] (± 0.5 kg), horses were positioned on a high-speed treadmill for collection of baseline respiratory gas exchange measurements and **blood temperature (T_{bl})** at the level of the right side of the heart. During the exercise test, a fan mounted 0.5 m in front of the treadmill was used to maintain an air velocity of 3.5 to 4 m/s over the cranial and dorsal aspects of the horse. A thermohygrometer[g] was used to monitor ambient conditions during all trials. Ambient conditions were similar for all trials; mean (± SE) values for room temperature and relative humidity during the experiments were 20.8 ± 0.2 C and 32 ± 1%, respectively.

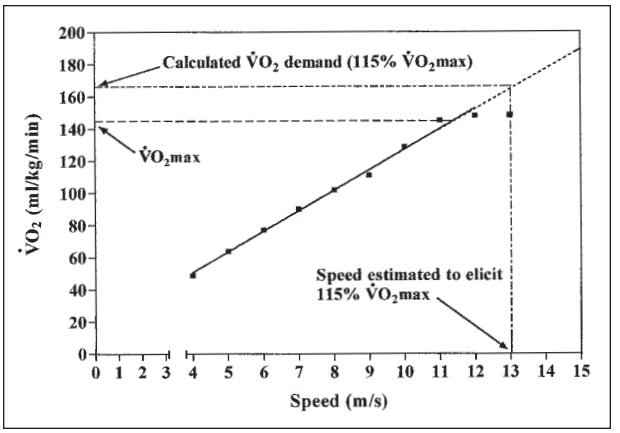

Figure 1—Relationship between oxygen consumption ($\dot{V}O_2$) and speed during an incremental exercise test in a horse. A linear regression equation of $\dot{V}O_2$ versus speed ($\dot{V}O_2$ = 0.222 + [12.67•speed]) was calculated by use of $\dot{V}O_2$ values between 4 and 11 m/s, at which there was a linear relationship between speed and $\dot{V}O_2$. The linear regression was extrapolated to obtain estimates of $\dot{V}O_2$ demand at a workload equivalent to 115% of maximum $\dot{V}O_2$ ($\dot{V}O_2$max) and treadmill belt speed that would elicit 115% of $\dot{V}O_2$max.

Exercise test—With the treadmill set at a 4° incline, horses completed 1 of the 3 warm-up protocols, then stood at rest for 5 minutes. Horses then walked (1.8 m/s) for 2 minutes, followed by acceleration of the treadmill to a speed calculated to elicit a running speed equivalent to 115% $\dot{V}O_2$max (st) (mean [± SEM] running speed, 11.7 ± 0.3 m/s). For each horse, the speed for each exercise test was the same, and the transition from the walk to the high speed required for the exercise test was accomplished in 5 to 6 seconds. Horses ran at this speed for 2 minutes. Respiratory gas exchange variables ($\dot{V}O_2$, **CO_2 output** [$\dot{V}CO_2$], and RER) were measured continuously during the warm-up and the sprint exercise test.

Respiratory gas measurements—The $\dot{V}O_2$ and $\dot{V}CO_2$ were measured by use of an open-circuit calorimeter,[h] as described.[7] Flow through the system was approximately 1,500 L/min at **standard temperature and pressure (STP)** with the horse stationary and 9,000 L/min during running. Expired O_2[i] and CO_2[j] concentrations were measured with gas analyzers at a sample rate of 40 Hz. Data were recorded by a computer-based data acquisition system[h] and reported at 10-second intervals; each measurement represents mean gas concentration determined during the 10-second interval. The gas analysis system was calibrated before the start of each exercise test by use of gas mixtures with O_2 and CO_2 concentrations that spanned the measurement range. Transit delay of the system was determined by bleeding known amounts of nitrogen gas into the system (through the face mask) and measuring the response time; > 90% of the response was achieved within the 10-second measurement interval. Overall accuracy of the system was verified repeatedly by the nitrogen dilution method.[8] Discrepancy between simulated $\dot{V}O_2$ produced by nitrogen

dilution, and the value measured by the system was ± 3% at nitrogen flow rates equivalent to a $\dot{V}O_2$ of 54 L/min (approx 140 ml/kg/min for a 385-kg horse). Standard equations were used to calculate $\dot{V}O_2$ and $\dot{V}CO_2$, and RER values were calculated by dividing $\dot{V}CO_2$ by $\dot{V}O_2$.[9]

Blood temperature—For each horse, T_{bl} was measured at rest prior to the warm-up, 5 minutes after completion of the warm-up, and at 15-second intervals during the sprint exercise test. The thermocouple that was placed within the catheter, with the thermocouple tip protruding 3 mm beyond the end of the catheter, was calibrated in a heated water bath with a precision thermometer[k]; under these conditions, the thermocouple had a response time of approximately 1 C/s.

Calculation of gas exchange kinetics and O_2 deficit—For each horse, the identical tests within a warm-up treatment were superimposed and mean calculated to give a single data set per warm-up protocol. Results of previous studies in horses indicated that the rapid rise in $\dot{V}O_2$ during the early part of intense exercise is well described by a monoexponential function.[10] Therefore, the overall time courses for $\dot{V}O_2$ and $\dot{V}CO_2$ between 10 and 120 seconds of exercise were estimated by fitting a single exponential model to the mean individual response.[l] The model was defined by least-squares criteria, as determined by a computerized nonlinear regression algorithm that used the technique of Marquardt.[11] The single exponential model was defined as:

$$y = A\ (1 - e^{-t/\tau})$$

where y is the response of $\dot{V}O_2$ and $\dot{V}CO_2$, A is the amplitude of the response between 10 and 120 seconds, t is the time after work rate increase, and τ is the time constant of the response (63% of the total amplitude of the response to the final plateau value). Because τ is the only parameter evaluated in this single exponential model, it can be used to compare relative speeds of response.[12] The curve-fitting procedure was iterated until further changes in the parameters for the model did not result in a reduction in the mean square error between the curve drawn from the model and the mean of the data set for each horse. Normality of the data was tested by use of the Kolmogorov-Smirnov test. The homoscedasticity assumption was evaluated by performing a Spearman rank correlation between the absolute values of the residuals and the observed value of the dependent variable. Model fit was evaluated by examination of plots of standardized residuals. The model fit was accepted when 95% of the standardized residuals were between –2 and +2.

Accumulated O_2 deficit during the sprint exercise was calculated as the difference between the calculated $\dot{V}O_2$ demand and the actual $\dot{V}O_2$ during the 120 seconds of exercise, with the use of described assumptions.[6,13,14] Actual $\dot{V}O_2$ was calculated by use of the trapezoidal rule[15]; the calculated $\dot{V}O_2$ demand was calculated from the speed-$\dot{V}O_2$ relationship determined during the incremental exercise test and the speed of the horse during the exercise tests in the 3 trials (Fig 1). The speed-$\dot{V}O_2$ relationship was determined by use of $\dot{V}O_2$ rates that were < $\dot{V}O_2$max.

Statistical analyses—Data were analyzed in a 3-way crossover design by use of a 2-way repeated measures ANOVA (repeated measures on treatment [ie, NoWU, LoWU, or HiWU] and time factors) or as a 1-way repeated-measures analysis (repeated-measures on the treatment factor) depending on the data being analyzed.[16,17] Linear regression techniques[k] were used to evaluate the relationship between T_{bl} and time and T_{bl} and $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics. An ANCOVA was used to test for differences among slopes of the regression lines. The initial value was set as the covariate. Significance was defined as $P < 0.05$ for each of the main effects of treatment or time and as $P < 0.1$ for the interaction. When there was a significant main or interaction effect, differences were identified by use of the Student-Newman-Keuls test (to detect differences among warm-up treatments). Unless stated otherwise, results are recorded as mean ± SEM.

Results

$\dot{V}O_2$max and speed-$\dot{V}O_2$ relationship—Mean $\dot{V}O_2$max (st) of the 6 horses was 151.6 ± 5.4 ml/kg/min or 63.8 ± 1.8 L/min at a treadmill speed of 10.7 ± 0.2 m/s. Mean value of the correlation coefficients for the speed-$\dot{V}O_2$ regression was 0.995 ± 0.001, the slope of the regression line was 0.267 ± 0.01 ml of O_2/kg/min, and the ordinate intercept was 6.1 ± 0.6 ml of O_2/kg/min.

Exercise test—Work intensity during the high-speed run was 114 ± 2.2% $\dot{V}O_2$max. One horse failed to complete the 2-minute exercise period during both NoWU trials (run times for this horse for trials 1 and 2 in NoWU were 100 seconds and 110 seconds, respectively). Therefore, data for total $\dot{V}O_2$ and accumulated O_2 deficit, and for T_{bl}, $\dot{V}O_2$, $\dot{V}CO_2$, and RER at 120 seconds of exercise for the NoWU treatment are mean ± SEM values for 5 horses.

Blood temperature—Warm-up exercise resulted in a significant increase in T_{bl}. Five minutes after completion of the warm-up, T_{bl} in LoWU and HiWU was 0.9 ± 0.1 C and 1.9 ± 0.2 C higher, respectively, compared with that of NoWU (**Fig 2**). These between-treatment differences were maintained throughout the high-speed exercise test. However, a significant effect of warm-up on the net change in T_{bl} during sprint exercise (NoWU, 2.0 ± 0.1 C; LoWU, 2.3 ± 0.08 C; HiWU, 2.3 ± 0.1 C) was not detected ($P = 0.1$; power, 0.662). Similarly, the slope (increase in T_{bl} per second of exercise) of the linear relationship between time and T_{bl} (from the walk immediately preceding the exercise test to 120 seconds of exercise) was not different ($P = 0.56$) among trials (NoWU, 0.018 ± 0.0007; LoWU, 0.0185 ± 0.0004; HiWU, 0.0179 ± 0.0007).

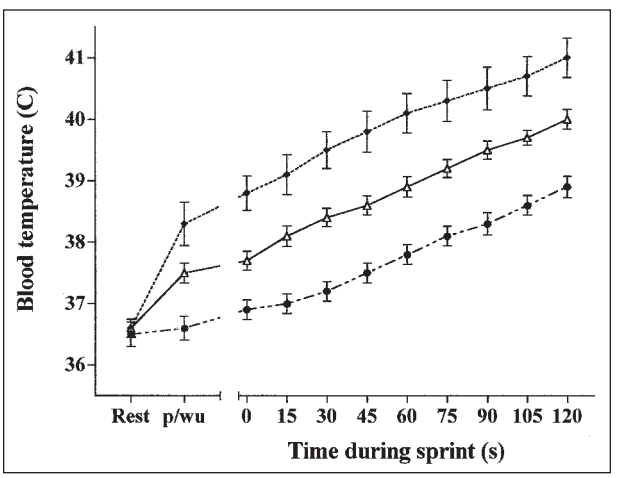

Figure 2—Mean (± SEM) blood temperature at the level of the right side of the heart of 6 horses at rest, 5 minutes after completion of 3 warm-up exercise protocols, and during 2 minutes of exercise at a speed equivalent to 115% of Vo_2max. Because 1 horse in the no warm-up trial did not complete the high-speed exercise, the values at 120 seconds for this group are the mean for 5 horses. ● = No warm-up. ▲ = Low-intensity warm-up. ◆ = High-intensity warm-up.

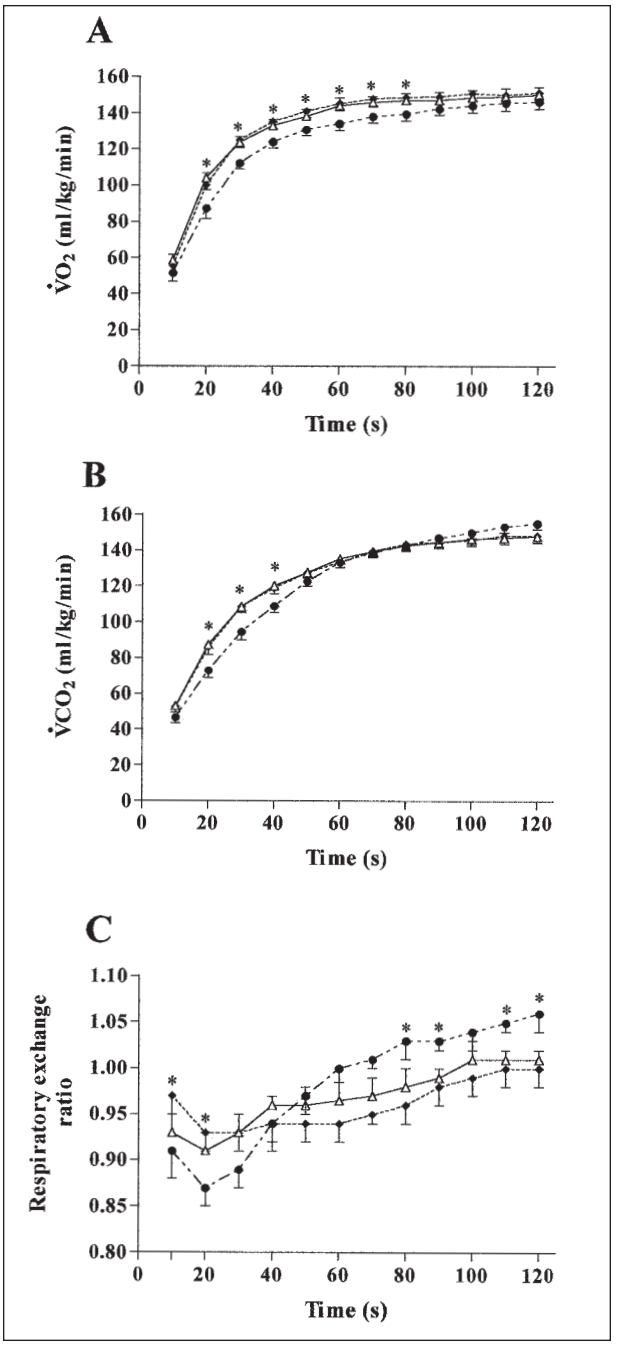

Figure 3—Mean (± SEM) values for respiratory gas exchange variables measured between 10 and 120 seconds after initiation of exercise at 115% of V_{O_2} max after 3 warm-up protocols in 6 horses. A—V_{O_2}. B—CO_2 production (V_{CO_2}). C—Respiratory exchange ratio. *Significant ($P < 0.05$) differences between results of trials with no warm-up and trials with low-intensity or high-intensity warm-up. *See* Figure 2 for key.

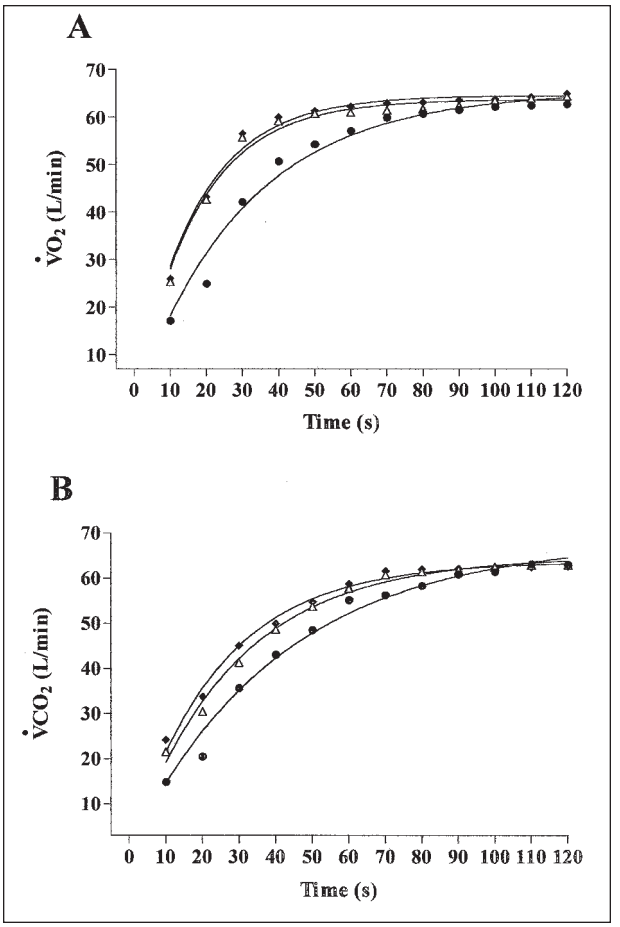

Figure 4—Changes in respiratory gas exchange variables during high-speed exercise after 3 warm-up protocols in a horse. A—V_{O_2}. B—V_{CO_2}. The best-fit monoexponential function is superimposed on each data set (solid lines). Notice acceleration of the kinetics of V_{O_2} and V_{CO_2} during exercise after low-intensity or high-intensity warm-up. *See* Figure 2 for key.

Table 1—Effect of warm-up exercise on the time constant (s) for the rapid increase in oxygen consumption during the period between 10 and 120 seconds after initiation of exercise at 115% of maximum oxygen consumption ($\dot{V}O_2$ max) in 6 horses

Horse No.	No warm-up	Low-intensity warm-up*	High-intensity warm-up*
1	22.52	18.86	17.80
2	19.91	17.68	15.42
3	24.33	18.54	15.58
4	24.25	13.43	14.38
5	24.30	14.50	17.54
6	21.93	12.54	13.23
Mean ± SD	22.87 ± 1.78	15.92 ± 2.76	15.66 ± 1.77

*All values significantly ($P < 0.001$) different from corresponding values in the no warm-up group.

Gas exchange measurements—During the first 80 seconds of exercise, $\dot{V}O_2$ was significantly higher in LoWU and HiWU than in NoWU (**Fig 3**). However, peak values (the highest value achieved during the exercise test) for $\dot{V}O_2$ achieved during the high-speed exercise test were not significantly different ($P = 0.45$; power, 0.41) among trials (LoWU, 150.2 ± 3.2 ml/kg/min; HiWU, 151.2 ± 4.2 ml/kg/min; NoWU, 145.1 ± 4.1 ml/kg/min). Characteristic responses of pulmonary gas exchange in a representative horse were depicted (**Fig 4**). Warm-up exercise accelerated the $\dot{V}O_2$ kinetics. For both warm-up trials, **τ for the rapid exponential increase in $\dot{V}O_2$** ($\tau_{\dot{V}O_2}$ [s]) was approximately 30% shorter ($P < 0.001$) relative to values for NoWU (**Table 1**, Fig 4). Regression analysis revealed a highly significant ($r^2 = 0.689$; $P < 0.001$) linear relationship between T_{bl} at the start of the high-speed run (values at the end of the 2 minute

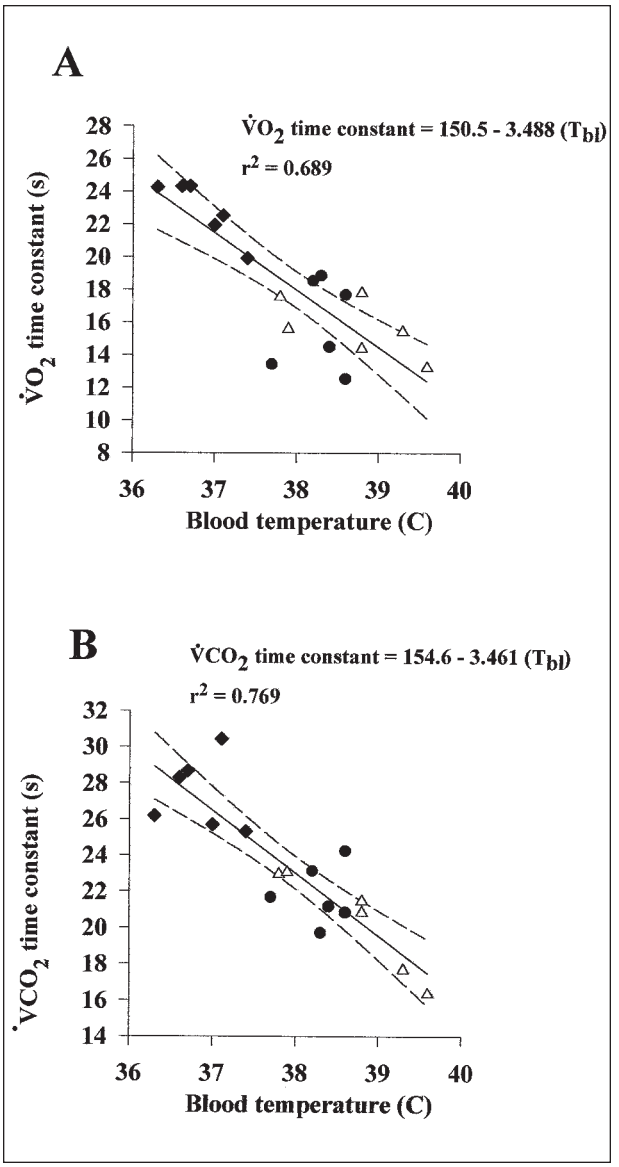

Figure 5—Relationship between blood temperature (T_{bl}) at the start of a high-speed exercise test and the time constant describing the rate of increase in respiratory gas exchange variables measured during the period between 10 and 120 seconds after initiation of exercise in 6 horses. A—$\dot{V}O_2$. B—$\dot{V}CO_2$. The line of identity (solid line) and 95% confidence limits (dashed lines) are depicted. Symbols represent the values for each horse for 3 warm-up conditions. *See* Figure 2 for key.

Table 2—Effect of warm-up exercise on the time constant (s) for the increase in carbon dioxide production during the period between 10 and 120 seconds after initiation of exercise at 115% of $\dot{V}O_2$max in 6 horses

Horse No.	No warm-up	Low-intensity warm-up*	High-intensity warm-up*
1	30.44	19.73	17.68
2	25.32	24.26	21.48
3	28.69	23.15	22.97
4	26.19	21.69	21.78
5	28.29	21.69	20.86
6	25.70	16.35	23.07
Mean ± SD	27.43 ± 2.02	21.06 ± 2.79	21.31 ± 1.97

See Table 1 for key.

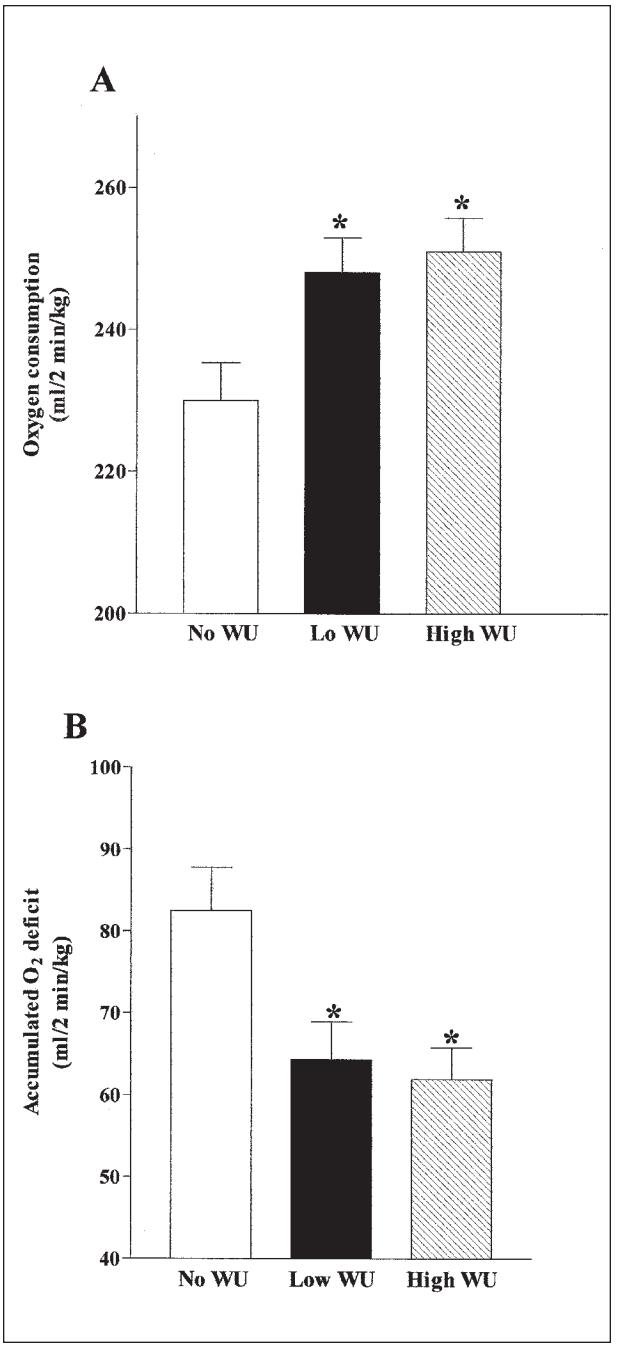

Figure 6—Mean (± SEM) values for respiratory gas exchange variables measured during a high-speed exercise test after no warm-up (No WU), a low-intensity warm-up (Low WU), or a high-intensity warm-up (High WU) in 6 horses. A—Total $\dot{V}O_2$. B—Accumulated O_2 deficit. *Significant ($P < 0.01$) difference from the trial with no warm-up.

walk) and $\tau_{\dot{V}O_2}$ (Fig 5). This relationship was described by the equation:

$$\tau_{\dot{V}O_2} = 150.5 - 3.488 \bullet T_{bl}$$

The ANCOVA did not reveal differences ($P = 0.81$) between the slopes of the linear regressions for T_{bl} versus $\tau_{\dot{V}O_2}$ and T_{bl} versus **mean τ for the rise in** $\dot{V}CO_2$ ($\tau_{\dot{V}CO_2}$).

Warm-up exercise also accelerated the kinetics of

$\dot{V}CO_2$. The $\tau_{\dot{V}CO_2}$ was 23% shorter ($P < 0.001$) in LoWU and HiWU than in NoWU (**Table 2**, Fig 4). As for $\dot{V}O_2$, a significant ($r^2 = 0.769$; $P < 0.001$) linear relationship was detected between T_{bl} at the start of the high-speed run and $\tau_{\dot{V}CO_2}$ (Fig 5). The $\dot{V}CO_2$ was significantly higher in LoWU and HiWU than in NoWU for the initial 40 seconds of the sprint exercise (Fig 3). The RER was significantly higher in LoWU and HiWU in the initial 20 seconds of the sprint. In contrast, mean RER during the last 40 seconds of the high-speed run was greater in NoWU, compared with results of trials preceded by warm-up (Fig 3). There were no differences between LoWU and HiWU for $\dot{V}O_2$, $\dot{V}CO_2$, RER, $\tau_{\dot{V}O_2}$, and $\tau_{\dot{V}CO_2}$.

The accelerated $\dot{V}O_2$ kinetics after a warm-up were also reflected in the values for total $\dot{V}O_2$ and accumulated O_2 deficit during the high-speed run. Mean values for total $\dot{V}O_2$ were approximately 15% higher ($P < 0.02$) in LoWU and HiWU than in NoWU (**Fig 6**). Conversely, accumulated O_2 deficit was significantly ($P < 0.001$) greater in NoWU, compared with that of LoWU and HiWU.

Discussion

Results of the study reported here indicated that kinetics of $\dot{V}O_2$ in moderately conditioned horses during intense exercise were accelerated appreciably by 2 specific warm-up protocols. After the low- or high-intensity warm-up, $\tau_{\dot{V}O_2}$ was shortened by approximately 30% and accumulated O_2 deficit during the 2-minute period of high-speed exercise was reduced by 20%. Therefore, because there were no differences between the low- and high-intensity warm-up treatments for any of the measured variables, the effects of these warm-up protocols on gas exchange during the subsequent intense exertion were not dependent on the intensity of prior exercise.

The study reported here was not designed to examine the components of and factors affecting the kinetics of respiratory gas exchange per se. Rather, it was an investigation of the effects of 2 specific warm-up protocols on the $\dot{V}O_2$ and $\dot{V}CO_2$ response measured in the same manner for each treatment. However, it should be recognized that the kinetic calculations were limited by the system used for measurement of $\dot{V}O_2$ and $\dot{V}CO_2$. Although our open-flow system continuously measured changes in the O_2 and CO_2 contents of expired air, the data were reported at 10-second intervals, and each data point represented the arithmetic mean of all measurements made within the previous 10-second interval. Results of a recent study clearly indicate that the $\dot{V}O_2$ response in horses during the rest-to-exercise transition is extremely rapid.[10] Specifically, during heavy exercise (approx 85% of $\dot{V}O_2$max) the τ (ie, time to attainment of 63% of the total amplitude of the response), and the time delay for the rapid exponential increase in $\dot{V}O_2$ were 20.7 ± 3.4 seconds and 20.3 ± 1.2 seconds, respectively.[10] Therefore, the 10-second measurement interval used in the study reported here was not optimal for complete characterization of the kinetics of respiratory gas exchange during the onset of exercise.

Results reported by Langsetmo et al[10] indicate that, during the early part (< 2 minutes) of heavy exercise, $\dot{V}O_2$ kinetics for horses can be partitioned into 2 phases.[18] The phase-1 response occurs during the rest-exercise or exercise-exercise transition (for a step change in work rate) and is attributed to increases in cardiac output and pulmonary and muscle blood flow as well as the early increase in O_2 extraction from blood.[18,19] The phase-2 response increases $\dot{V}O_2$ to a new steady state and is best described by an exponential function.[10] This rapid increase in $\dot{V}O_2$ is thought to reflect changes in oxidative metabolism in working skeletal muscle.[20,21] Because of aforementioned limitations of the gas exchange measurement system, we did not attempt to partition the $\dot{V}O_2$ response into these 2 kinetic components. Instead, we applied a single exponential model to best describe the $\dot{V}O_2$ response between 10 and 120 seconds of exercise by use of data collected after attainment of the target running speed. For each horse, the running speed was identical for all 6 exercise trials and the time to attain the target speed was similar for all trials (5 to 6 seconds).

Of further concern are the methods used for estimation of O_2 demand and O_2 deficit. The O_2 demand during the sprint exercise protocol was calculated from the relationship between $\dot{V}O_2$ and speed developed for work intensities < $\dot{V}O_2$max. The O_2 deficit was then calculated from the difference between the O_2 demand and the $\dot{V}O_2$ measured during supramaximal exertion. This approach has been established for estimation of accumulated O_2 deficit of horses and human athletes.[6,14] Nevertheless, it must be emphasized that the calculations and interpretations were based on 2 key assumptions. The first assumption was that the linear relationship between $\dot{V}O_2$ and speed, determined at work rates < $\dot{V}O_2$max, was also applicable for work intensities > $\dot{V}O_2$max such that the total energy input during the supramaximal exercise was equivalent to the calculated O_2 demand. However, this relationship was determined during an incremental exercise test, whereas the sprint protocol involved rapid acceleration to a supramaximal running speed. Therefore, it is possible that the actual O_2 demand during the sprint protocol differed from the O_2 demand calculated on the basis of data obtained during the incremental exercise test. The second assumption was that differences in core body temperature at the start of the sprint protocol did not affect O_2 demand. Verification of this assumption would require knowledge of the effects of body temperature on the $\dot{V}O_2$-speed relationship during incremental exercise. Finally, it must be acknowledged that interpretation of the data for the accumulated O_2 deficit would have been strengthened by measurement of the rate of appearance of lactate in blood; these measurements were not made.

The magnitude of the effect of warm-up exercise on $\dot{V}O_2$ kinetics was similar to that reported by Tyler et al,[5] who reported that 5 minutes of low-intensity exercise (50% of $\dot{V}O_2$max) reduced by 40% the time required to achieve 95% of $\dot{V}O_2$max during a bout of sprint exercise. As in the study reported here, the warm-up exercise was completed 5 minutes before the start of the high-speed run. However, whereas the horses in the study by Tyler et al[5] ran at a speed equivalent to 115% $\dot{V}O_2$max for approximately 60 seconds,

the horses in our study exercised at this workload for 2 minutes. We chose the longer exercise duration in order to examine respiratory gas exchange during a timeframe equivalent to that required of racehorses to complete a 1- to 1.25-mile race and to allow the horses to achieve a steady-state $\dot{V}O_2$.

Contrary to our second hypothesis, compared with a low-intensity warm-up, prior high-intensity exercise did not further augment $\dot{V}O_2$ kinetics during subsequent intense exertion. This hypothesis was of interest because of the substantial differences in warm-up strategies used by trainers of Thoroughbred and Standardbred racehorses. Furthermore, results of studies in human athletes indicate significant acceleration of $\dot{V}O_2$ kinetics during intense exertion after a high-intensity warm-up (sufficient to result in a blood lactate concentration > 4 mmol/L), whereas prior low-intensity exercise has minimal effects on respiratory gas exchange during subsequent exercise.[1,2,a] It has been hypothesized that metabolic changes induced by prior high-intensity exercise underlie the faster $\dot{V}O_2$ kinetics of the second exercise bout.[2] Specifically, intense warm-up that increases the concentrations of blood and muscle lactate and decreases the pH in working muscle may increase O_2 delivery by 2 mechanisms: vasodilation and increased muscle blood flow at the start of the second work bout,[2] and an acidemia-induced right shift of the oxyhemoglobin equilibrium curve that improves the diffusional gradient for O_2 between the capillary blood and the mitochondria of the exercising muscles.[22] Both of these mechanisms imply that the slower $\dot{V}O_2$ kinetics in human subjects during exercise without a high-intensity warm-up are largely the consequence of limitations to blood flow and O_2 delivery in working muscles.[23] In a companion study, there was no change in blood lactate concentration associated with low intensity warm-up in horses, whereas high intensity warm-up resulted in an approximately seven-fold increase in blood lactate concentration.[24] Therefore, in contrast to human athletes, our results suggest that even a low-intensity warm-up can augment $\dot{V}O_2$ kinetics in horses and that more intense warm-up exercise provides no further advantage. Alternatively, it is possible that more sensitive methods for measurement of $\dot{V}O_2$ kinetics, such as breath-by-breath analysis, would allow detection of an effect of warm-up intensity on respiratory gas exchange.

Some authors have suggested that increases in blood and muscle temperature as a result of warm-up underlie the changes in respiratory gas exchange during subsequent exercise.[25,26] In the study reported here, T_{bl} was increased by approximately 1 and 2 C after the low- and high-intensity warm-up protocols, respectively. From these data, we may infer that muscle temperature was increased by a similar magnitude after these warm-up protocols. Increased body temperature may enhance the $\dot{V}O_2$ response by a Q_{10} effect on muscle mitochondrial respiration (the change in $\dot{V}O_2$ upon a 10 C increase in temperature). Results of studies of the kinetics of skeletal muscle mitochondrial oxidative phosphorylation indicate that increased temperature increases $\dot{V}O_2$ by decreasing the efficiency of phosphorylation.[27] However, although mean values for peak $\dot{V}O_2$ achieved during the sprint exercise protocol were higher in the trials preceded by warm-up, this difference was small (3.5 to 4%) and not significant. Estimated on the basis of a Q_{10} of 2.3, the expected temperature effect on peak $\dot{V}O_2$ would be approximately 8 to 9% per degree of temperature. Such an increase in $\dot{V}O_2$ is far in excess of the changes measured in the study reported here. Furthermore, the $\dot{V}O_2$ responses (kinetics and peak values) were similar in the 2 warm-up experiments, although T_{bl} was approximately 1 C higher in HiWU than in LoWU throughout exercise. Similarly, in humans, $\dot{V}O_2$ kinetics and the amplitude of the $\dot{V}O_2$ response are unaffected by passive body heating that increases muscle and core body temperature, and there is little evidence in exercising humans that increased muscle temperature is associated with an increase in respiratory $\dot{V}O_2$.[4,26,28] Therefore, although there was a linear relationship between T_{bl} at the start of the high-speed exercise test and $\tau_{\dot{V}O_2}$, increased body temperature per se may not underlie the alterations in respiratory gas exchange. It is possible that the changes in $\dot{V}O_2$ kinetics are more closely related to 1 or more concurrent physiologic changes (eg, decreases in muscle and blood pH or an increase in PCO_2) that parallel the increase in blood temperature.

The time course for the rapid increase in $\dot{V}CO_2$ was also well described by a single exponential function. However, regardless of warm-up treatment, $\tau_{\dot{V}CO_2}$ was slower than the $\dot{V}O_2$ response and was associated with a delay in the increase in RER. The method of gas sampling could affect $\tau_{\dot{V}CO_2}$ because the $CaSO_4$ absorbent used in the gas sampling line can buffer CO_2 response times by adsorbing and releasing CO_2 with H_2O. In humans, the $\dot{V}CO_2$ response during the exercise transition also is slower, compared with the kinetics of $\dot{V}O_2$,[29] although the magnitude of the difference is greater than that observed in the horses of the study reported here. Whipp et al[29] have suggested that storage of CO_2 within the muscle is the primary mechanism responsible for this difference in $\tau_{\dot{V}O_2}$ and $\tau_{\dot{V}CO_2}$ during the early part of exercise.

Warm-up exercise accelerated the kinetics of $\dot{V}CO_2$, a finding consistent with that observed in a previous investigation of the effects of warm-up exercise on respiratory gas exchange in horses. Although Tyler et al[5] did not report actual $\dot{V}CO_2$ kinetics, mean $\dot{V}CO_2$ was significantly higher during the early part of intense exercise in horses that had received prior warm-up exercise, which was similar to findings in our study. Results of studies in humans also indicate an acceleration of $\dot{V}CO_2$ kinetics after warm-up exercise.[23,29] Those authors suggest that prior exercise results in an increase in the storage of CO_2 in muscle. In this circumstance, there will be a smaller increment in the transient CO_2 storage during the subsequent exercise bout and the kinetics of the output of CO_2 from working muscle will be closer to those of its production rate within muscle.[29] Importantly, given that CO_2 production is proportional to $\dot{V}O_2$, the faster $\dot{V}CO_2$ response could also reflect the augmentation in aerobic metabolism after warm-up. In this context, it is perhaps not surprising that, similar to $\tau_{\dot{V}O_2}$, there was a linear relationship between T_{bl} at the start of the intense exercise and $\tau_{\dot{V}CO_2}$. It is also possi-

ble that warm-up exercise altered the ventilatory response to subsequent sprint exercise. A more rapid increase in minute ventilation could also contribute to the faster $\dot{V}_{CO_2}$ response.

A consequence of the alterations in $\dot{V}_{O_2}$ during intense exercise after warm-up was a substantial reduction in the estimated accumulated O_2 deficit. At work intensities greater than those that induced $\dot{V}_{O_2}$max, measurement of accumulated O_2 deficit is one means for estimation of anaerobic energy transduction.[13,14] Given the assumptions underlying this calculation, the lower accumulated O_2 deficit in the LoWU and HiWU trials suggests a reduction in the requirement for energy transduction by anaerobic mechanisms. However, evaluation of whether there truly was an effect of warm-up on the anaerobic contribution to energy expenditure will, at the least, require measurement of rates of lactate accumulation in blood together with examination of changes in muscle metabolites.

[a]Gausche MA, Harmon T, Lammarra N, et al. Pulmonary O_2 uptake kinetics in humans are speeded by a bout of prior exercise above, but not below, the lactate threshold. *J Physiol (Lond)* 1989;417:138.
[b]Sato, BIAB Industrial, Uppsala, Sweden.
[c]IT-14, Physitemp Instruments, Clifton, NJ.
[d]PE 240, Becton Dickinson, Rutherford, NJ.
[e]BAT-10 thermometer, Physitemp Instruments, Clifton, NJ.
[f]Absco Scales, New Albany, Ohio.
[g]Model 3309-60, Cole-Palmer Instruments, Chicago, Ill.
[h]Oxymax-XL, Columbus Instruments, Columbus, Ohio.
[i]Electrochemical cell, Columbus Instruments, Columbus, Ohio.
[j]Single-beam nondispersive infrared sensor, Columbus Instruments, Columbus, Ohio.
[k]Fisher Scientific, Mississauga, ON, Canada.
[l]Systat 7.0, SPSS Inc, Chicago, Ill.

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