Ventilatory dynamics and control of blood gases after maximal exercise in the Thoroughbred horse

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Padilla, Danielle J., Paul McDonough, Casey A. Kindig, Howard H. Erickson, and David C. Poole. Ventilatory dynamics and control of blood gases after maximal exercise in the Thoroughbred horse. J Appl Physiol 96: 2187–2193, 2004. First published February 6, 2004; 10.1152/japplphysiol.00998.2003.—Despite enormous rates of minute ventilation (V̇E) in the galloping Thoroughbred (TB) horse, the energetic demands of exercise conspire to raise arterial PCO2 (i.e., induce hypercapnia). If locomotory-respiratory coupling (LRC) is an obligatory facilitator of high VE in the horse such as those found during galloping (Bramble and Carrier. Science 219: 251–256, 1983), VE should drop precipitously when LRC ceases at the gallop-trot transition, thus exacerbating the hypercapnia. TB horses (n = 5) were run to volitional fatigue on a motor-driven treadmill (1 m/s increments; 14–15 m/s) to study the dynamic control of breath-by-breath VE; O2 uptake, and CO2 output at the transition from maximal exercise to active recovery (i.e., trotting at 3 m/s for 800 m). At the transition from the gallop to the trot, VE did not drop instantaneously. Rather, VE remained at the peak exercise levels (1.391 ± 88 l/min) for 13 ± 13 s via the combination of an increased tidal volume (12.6 ± 1.2 liters at gallop; 13.9 ± 1.6 liters over 13 s of trotting recovery; P < 0.05) and a reduced breathing frequency (71.3 ± 5.2 breaths/min (at gallop); 97.7 ± 5.9 breaths/min over 13 s of trotting recovery (P < 0.05)). Subsequently, VE declined in a biphasic fashion with a slower mean response time (85.4 ± 9.0 s) than that of the monoexponential decline of CO2 output (39.9 ± 4.7 s; P < 0.05), which rapidly reversed the postexercise arterial hypercapnia (arterial PCO2 at gallop: 52.8 ± 3.2 Torr; at 2 min of recovery: 25.0 ± 1.4 Torr; P < 0.05). We conclude that LRC is not a prerequisite for achievement of VE commensurate with maximal exercise or the pronounced hyperventilation during recovery.

exercise recovery; locomotory-respiratory coupling; blood acid-base; equine

The control of minute ventilation (VE) differs between Thoroughbred (TB) horses and humans during maximal exercise. For example, the horse displays a strict 1:1 coupling of breathing to stride frequency [i.e., locomotory-respiratory coupling (LRC)] while cantering and galloping (8), whereas humans are not limited to a particular coupling ratio (7). Notwithstanding this constraint, the exercising TB horse can achieve prodigious values for pulmonary VE (>1,800 l/min in extremely fit horses) and rates of gas exchange [i.e., O2 uptake (VO2) of >70 l/min; CO2 output (VCO2) of >80 l/min during maximal exercise (26, 32)]. In this regard, it may be that LRC may actually limit VE during brief, maximal exercise (26, 32).

In contrast to the above, LRC has been considered the key facilitator of VE in TB horses, with high ventilatory volumes being achieved with supposedly little respiratory muscle contribution to exercise hyperpnea (8). However, electromyographic studies demonstrate that the diaphragm is highly active in running horses (2). Other indicators of the increased diaphragmatic activity include substantial transdiaphragmatic pressures (43) and high diaphragm muscle blood flows (31). In addition, the horse diaphragm is highly oxidative and, therefore, suited to sustained high-intensity respiratory efforts (38). It has also been shown recently that rib cage expansion is very limited and out of phase with inspiration in the galloping horse, which will serve to place a greater reliance on diaphragmatic vs. intercostal or accessory muscle breathing (32).

Whereas humans usually hyperventilate during intense exercise [e.g., arterial PCO2 (PaCO2) typically falls to values of <30 Torr; Refs. 10, 27] and defend arterial PO2 (PaO2) close to resting values, horses routinely become markedly hypercapnic and hypoxic (6, 25, 35, 36) during brief, incremental exercise (PaCO2 of ∼55–65 Torr; PaO2 of ∼60–80 Torr). This hypercapnic response is a direct consequence of the extraordinarily high metabolic rate coupled with an inadequate ventilatory response in the TB (6, 35).

Horses and other quadrupeds (8) are considered to rely on LRC primarily during the canter and gallop to facilitate VE, which would act to reduce the energetic cost of VE. This would be advantageous because it would serve to minimize the redistribution of cardiac output from the exercising limbs to the respiratory muscles (3, 20). Indeed, in humans, reducing respiratory muscle work elevates the proportion of cardiac output available to the working limb muscles (21, 22).

The purpose of the present investigation was to explore the notion that, if LRC is an obligatory requirement for achieving very high VE such as those present in the galloping horse, when LRC is removed abruptly at the onset of trotting, VE should drop immediately and precipitously. Specifically, we tested the hypothesis that, at the onset of recovery from galloping [i.e., across the transition from the gallop (LRC present) to the trot at 3 m/s (no LRC)], VE would fall precipitously and PaCO2 would increase transiently above end-exercise levels. By resolving, for the first time, the breath-by-breath responses of VE and gas exchange (VO2 and VCO2) across the gallop-trot transition, we sought to gain novel insights into the control of breathing in the horse.

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Methods

Animals. Five TB geldings (4–10 yr; 470–600 kg) were used in this investigation. Horses were housed in enclosed dry lots with a shaded area and fed alfalfa, grass hay, and concentrate twice daily, with water and salt available ad libitum. Deworming and vaccinations were administered at regular intervals. A high-speed treadmill (SATO, Uppsala, Sweden) was utilized to exercise horses at least twice weekly to maintain fitness and condition. All procedures were approved by the Kansas State University Institutional Animal Care and Use Committee.

Instrumentation. Before each trial, with the use of aseptic techniques, each horse was instrumented with a 7-Fr introducer catheter inserted into the right jugular vein, and an 18-gauge, 2.0-in. catheter was placed in a previously elevated carotid artery or transverse facial artery (20 gauge, 1.5 in. in 2 TB horses). Lidocaine (2%, i.e., 2 ml) was utilized subcutaneously for the insertion of the catheter in the carotid artery and jugular vein but not in the transverse facial artery (2 horses). To determine mean pulmonary artery temperature (for correction of arterial blood gases), a thermistor catheter was inserted through the 7-Fr introducer catheter and advanced into the pulmonary artery 8 cm past the pulmonary valve. Calibration of the thermistor catheter was conducted with the use of a Physitemp thermocouple thermometer (BAT-10, Physitemp, Clifton, NJ). To withdraw arterial blood, a cannula (1.6-mm inner diameter, 3.2-mm outer diameter) was attached to the arterial catheter.

Measurement of breath-by-breath gas exchange. To measure expired $V_e$, an ultrasonic phase-shift flowmeter (model FR-41 eq; Flowmetrics-BDRL, Birmingham, UK) was used as described previously (52). Briefly, hoses were outfitted with a lightweight fiberglass facemask (<1 kg). This mask was fitted internally with silicone rubber and foam gaskets to maintain an airtight seal. The flow tubes were then placed in the openings of the facemask opposite each nostril to allow measurement of airflow for each nostril. Each flow tube contained two ultrasonic transducers that quantified velocity of airflow at a resonant frequency of 40 kHz. Each ultrasonic phase-shift flowmeter (i.e., left and right) underwent a three-point calibration at −20, 0, and +20 l/s using a rotameter (KDG Flowmeters, Burgess Hill, UK) certified by the National Board of Standards. Because the system responded linearly to changes in airflow and the design characteristics of the flow probes negate the effects of temperature and humidity (52), the above calibration allowed for the measurement of the full extent of the exercise and recovery $V_e$ response.

Right and left airway flows, as well as inspired and expired $O_2$ and $CO_2$, were collected with a commercial data analysis system (DATAQ, Akron, OH) and stored for later analysis. The conversion of $V_e$ to STPD was conducted using standard equations, whereas $V_02$ and $VCO_2$ were calculated using the principle of mass balance [i.e., $V_02$ STPD = ([$V_e$ (STPD) × $F_{O_2}$] − [$V_e$ (STPD) × $F_{O_2,STPD}$]) and $VCO_2$ STPD = $V_e$ (STPD) × ($F_e$ STPD$CO_2$ − $F_i$ STPD$CO_2$)]. Inspired and expired gas fractions ($F_i$ and $F_e$, respectively) were measured using a mass spectrometer (Perkin-Elmer, model 1100, Pomona, CA), which was calibrated using gravimetrically determined gas concentrations that spanned the range of $O_2$ and $CO_2$ concentrations between the inspired air and that expired by the horse. Gas was sampled continuously via a sampling port attached between the two nostril openings of the fiberglass mask (i.e., between the nares of the horse).

Experimental protocol. An incremental exercise test was conducted with each TB horse on a level treadmill. The horses trotted (i.e., warmed up) at 3 m/s for 800 m, and the speed of the treadmill was rapidly increased to 7 m/s for 1 min, and then the speed was increased in 1 m/s increments until fatigue (i.e., the horse could no longer keep up with the speed of the treadmill despite humane encouragement). The treadmill was immediately slowed to 3 m/s for 800 m, and cardiovascular, $V_e$, and gas-exchange measurements ($V_02$ and $VCO_2$) were continuously recorded from the immediate offset of maximal exercise and throughout the 4-min recovery period. Arterial blood samples were collected, and pulmonary arterial temperature was recorded during the last 10 s of the final stage of exercise and at 2 and 4 min of the recovery period.

Blood analysis. Arterial blood samples were placed immediately on ice after anaerobic withdrawal (~5 ml) into plastic, heparinized syringes. Blood gases ($P_{O_2}$ and $P_{CO_2}$), $pH$, and plasma lactate concentrations were quantified after the exercise test (within 1–2 h) with a blood-gas analyzer (Nova Stat Profile, Waltham, MA). Blood gases and $pH$ were then corrected to the individual horse’s pulmonary arterial temperature (16). To ensure technical and internal consistency, one person conducted all blood analyses. Equipment was calibrated before and after each exercise test in accordance with manufacturers’ standards.

Modeling of $V_02$, $VCO_2$, and $V_e$. Four-breath rolling averages for $V_02$, $VCO_2$, and $V_e$ were used for modeling data. Curve fitting was accomplished with KaleidaGraph software (Synergy software, Reading, PA) and was performed on the gallop-trot transition data using a one-component model

$$V(t) = V(b) + A_1(1 - e^{-t/TD_1})$$

and a more complex, two-component model

$$V(t) = V(b) + A_1(1 - e^{-t/TD_1}) + A_2(1 - e^{-t/TD_2})$$

where $V$ indicates the gas-exchange variable of interest (i.e., $V_02$, $VCO_2$, or $V_e$), $t$ is a given time point, $b$ is baseline (end-exercise), $A_1$ and $A_2$ are the response amplitudes, $TD_1$ and $TD_2$ are the independent time delays, and $t_1$ and $t_2$ are the time constants.

Goodness of model fit was determined via three criteria: 1) the coefficient of determination (i.e., $r^2$), 2) the sum of the squared residuals term (i.e., $\chi^2$), and 3) visual inspection of the model. The time delay from end exercise until the beginning of the response for $V_e$ was determined independent of model estimates because the response varied considerably between horses (i.e., in some, $V_e$ increased before falling ($n = 3$), whereas $V_e$ remained stable in others ($n = 2$)). Mean response times (MRTs) were calculated from the model parameters using the following equation for the one-component model

$$MRT = TD + \tau$$

and for the two-component model (see Ref. 30)

$$MRT = A_1/4(TD_1 + \tau_1) + A_2/4(TD_2 + \tau_2)$$

Statistics. A repeated-measures ANOVA was used for each variable compared over time. If significance was revealed, a Student-Newman-Keuls post hoc test was utilized to determine the point of significance. Paired $t$-tests were used to determine whether the two-component model provided a statistically better fit to the data than the one-component model. Between-variable comparisons were made by unpaired $t$-tests. Where a directional hypothesis was tested, a one-tailed test was utilized. Statistical significance was accepted at a $P$ value of ≤0.05.

Results

$V_e$. In the absence of overt pathology, the flow profiles for the left and right nostrils are almost superimposable. For the horses studied in the present investigation, the flow profiles from each nostril were remarkably similar throughout both exercise and recovery. Determination of inspired and expired volumes and $V_e$ were made using the total flow through both the left and right nostrils.

After the gallop-trot transition, $V_e$ remained elevated at or above end-exercise values for ~13 s, after which $V_e$ fell biphasically (Fig. 1). Quantitatively, $V_e$ was best characterized by a dual-exponential model with a fast and slow phase (Table
response was confirmed via a significantly higher correlation coefficient \([0.96 \text{ (two-component)} \text{ vs. } 0.93 \text{ (one-component); } P < 0.05]\) and a lower sum of squares residual term \([1.0 \times 10^5 \text{ (two-component)} \text{ vs. } 1.78 \times 10^5 \text{ (one-component); } P < 0.05]\. The prolongation of the exercise \(\dot{V}E\) into recovery and the biphasic nature of the subsequent \(\dot{V}E\) decrease in trotting recovery were due, in part, to a radical change in breathing strategy (Table 2; Fig. 3). Specifically, over the first 7–13 s of recovery, tidal volume (\(\dot{V}T\)) rose and breathing frequency (\(f\)) fell significantly (Table 2). After this period, \(f\) and \(\dot{V}T\) remained at end-exercise levels for 30 s. After 30 s, \(\dot{V}T\) fell progressively with time, but \(f\) did not differ from end-exercise values (Fig. 3; Table 2). At the end of the 4-min recovery period, \(\dot{V}E\) was still \(44.5 \pm 18.5\%\) (e.g., Figs. 1 and 3) above trotting baseline values.

\(\dot{V}O_2\) and \(\dot{V}CO_2\). \(\dot{V}CO_2\) and \(\dot{V}O_2\) both fell with a monoexponential profile after the gallop-trot transition (Figs. 1 and 2, respectively; Table 1) with MRTs of \(39.9 \pm 4.7\) and \(28.9 \pm 3.2\) s, respectively \((P < 0.05)\. Both \(\dot{V}O_2\) and \(\dot{V}CO_2\) apparently resolved to an elevated baseline (\(9.8 \pm 36.0\) and \(17.5 \pm 30.9\%\) above the pregallop, trotting baseline for \(\dot{V}O_2\) and \(\dot{V}CO_2\), respectively; e.g., Figs. 2 and 1, respectively). However, compared with \(\dot{V}E\), \(\dot{V}CO_2\) and \(\dot{V}O_2\) were relatively closer to trotting baseline values after the 4-min recovery period (Table 1; Figs. 1 and 2, respectively).

Blood gas response across the gallop-trot transition. The hypercapnia found during exercise was completely reversed

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Table 1. Model parameters of \(\dot{V}E\), \(\dot{V}CO_2\), and \(\dot{V}O_2\) responses during the trotting recovery period from maximal exercise

<table>
<thead>
<tr>
<th>Variable</th>
<th>(\dot{V}E)</th>
<th>(\dot{V}CO_2)</th>
<th>(\dot{V}O_2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>End-exercise baseline, l/min</td>
<td>1411.5 ± 90.1</td>
<td>68.7 ± 6.2</td>
<td>64.2 ± 6.3</td>
</tr>
<tr>
<td>(A_1), l/min</td>
<td>-301.0 ± 67.4</td>
<td>-48.6 ± 4.3</td>
<td>-46.0 ± 4.3</td>
</tr>
<tr>
<td>(A_2), l/min</td>
<td>-172.5 ± 34.8</td>
<td>-172.5 ± 34.8</td>
<td></td>
</tr>
<tr>
<td>End-recovery value, l/min</td>
<td>937.9 ± 125.3</td>
<td>20.2 ± 2.1</td>
<td>18.2 ± 2.1</td>
</tr>
<tr>
<td>TD1, s</td>
<td>6.3 ± 1.8</td>
<td>9.6 ± 1.0</td>
<td>11.1 ± 1.9</td>
</tr>
<tr>
<td>TD2, s</td>
<td>88.5 ± 25.3</td>
<td>112.2 ± 15.6</td>
<td></td>
</tr>
<tr>
<td>(\tau_1), s</td>
<td>15.0 ± 5.6</td>
<td>30.3 ± 4.2*</td>
<td>17.8 ± 3.1</td>
</tr>
<tr>
<td>(\tau_2), s</td>
<td>112.2 ± 15.6</td>
<td>39.9 ± 4.7†</td>
<td>28.9 ± 3.2††</td>
</tr>
<tr>
<td>MRT, s</td>
<td>85.4 ± 9.0</td>
<td>39.9 ± 4.7†</td>
<td>28.9 ± 3.2††</td>
</tr>
</tbody>
</table>

Values are means ± SE (\(n = 5\)). \(A_1\); primary component; \(A_2\); secondary component; TD, time delay; \(\tau\), time constant; MRT, mean response time; \(\dot{V}E\), minute ventilation; \(\dot{V}CO_2\), CO2 output; \(\dot{V}O_2\), O2 uptake. *\(P < 0.05\) between \(\dot{V}CO_2\) and \(\dot{V}O_2\). †\(P < 0.05\) between \(\dot{V}E\) and \(\dot{V}CO_2\) or \(\dot{V}O_2\). ††\(P < 0.05\) between \(\dot{V}O_2\) and \(\dot{V}CO_2\).

Table 2. Recovery values at specified times for \(f\), \(\dot{V}T\), and \(\dot{V}E\)

<table>
<thead>
<tr>
<th>Time</th>
<th>(f)</th>
<th>(\dot{V}T)</th>
<th>(\dot{V}E)</th>
</tr>
</thead>
<tbody>
<tr>
<td>13 s</td>
<td>113.8 ± 5.2</td>
<td>126.6 ± 1.2</td>
<td>1391 ± 88</td>
</tr>
<tr>
<td>30 s</td>
<td>100.5 ± 6.1</td>
<td>119.9 ± 1.5†</td>
<td>1171 ± 104††</td>
</tr>
<tr>
<td>2 min</td>
<td>108.7 ± 8.1</td>
<td>101.1 ± 1.5†</td>
<td>1061 ± 103†††</td>
</tr>
<tr>
<td>4 min</td>
<td>110.4 ± 5.6</td>
<td>8.9 ± 1.5*‡‡</td>
<td>963 ± 127†††</td>
</tr>
</tbody>
</table>

Values are means ± SE (\(n = 5\)). \(f\), Breathing frequency; \(\dot{V}T\), tidal volume. *\(P < 0.05\) from end-exercise. †\(P < 0.05\) from 13 s. ‡‡\(P < 0.05\) from 30 s. †††\(P < 0.05\) from 2 min.
within 2 min after the gallop-trot transition, and the ensuing hypocapnia was maintained through 4 min of trotting (Fig. 4; PaCO$_2$ at gallop: 52.8 ± 3.2 Torr; 2 min of trot: 25.0 ± 1.4 Torr; 4 min of trot: 24.6 ± 1.5 Torr; both $P<0.05$ vs. gallop). This hyperventilation is further evidenced by the pronounced increase in the ratio of V$\dot{E}$ to V$\dot{CO}_2$ (from 20 at the gallop to 60 within 1–2 min of trotting recovery; e.g., Fig. 4). Plasma lactate was elevated to 25.5 ± 4.0 mM at the gallop and remained unchanged throughout the recovery period (2 min of trot: 24.9 ± 3.6 mM; 4 min of trot: 24.5 ± 3.7 mM). Arterial pH increased from the gallop (7.217 ± 0.107) to 2 min of trot (7.301 ± 0.093; $P<0.05$) but did not differ between 2 and 4 min (7.307 ± 0.098) of the recovery period.

**DISCUSSION**

To the best of our knowledge, this is the first study to provide a breath-by-breath analysis of V$\dot{E}$, gas exchange (i.e., V$\dot{O}_2$ and V$\dot{CO}_2$), and related metabolic and blood-gas measurements across the gallop-trot transition at the cessation of maximal exercise in the TB horse. The finding that V$\dot{E}$ did not decrease abruptly at the gallop-trot transition suggests that any mechanical link between locomotion and respiration (i.e., LRC) does not constitute an obligatory component of extraordinary V$\dot{E}$ commensurate with those found during exercise. Specifically, an abrupt reduction in stride length and frequency, as seen at the transition from galloping to trotting (i.e., 3 m/s), and the removal of the synchrony between stride and breathing frequency (LRC) did not reduce V$\dot{E}$ instantaneously. Furthermore, given the finite V$\dot{CO}_2$ dynamics, had a precipitous fall in V$\dot{E}$ occurred, it would be expected to sustain or even exacerbate the exercise-induced hypercapnia (i.e., elevate PaCO$_2$ further). In contrast to this notion, our results show that the hypercapnia and hypoxemia of exercise are quickly reversed (at least within
trot, compensatory hyperventilation was evident. One putative endothermic vertebrates general requirement for sustained aerobic activity among "during postmaximal exercise trotting recovery. Rapid reductions of \( V_\text{E} \) of the postgallop trot (due to an altered breathing strategy) addition, the results of the present study, where \( V_\text{E} \) during brief, maximal exercise, suggestive of some sort of functional ventilatory restraint. That this restraint is not the result of an absolute limitation to ventilation per se is supported by studies using heliox inspirates (14) and hypoxia (37). In addition, the results of the present study, where \( V_\text{E} \) remained at or rose above (3 of 5 horses) end-exercise values for the first 7–13 s of recovery also argues against any purely mechanical limitation to airflow generation.

Marlin et al. (via respiratory inductance plethysmography; Ref. 32) have shown that (during the canter and gallop) \( V_\text{T} \) is increased by abdominal elongation and expansion consistent with a major diaphragmatic contribution to inspiration, whereas Ainsworth et al. (2) determined (via electromyogram analysis) that diaphragmatic contractions are always in phase with esophageal pressure changes and inspiratory flow generation as exercise intensity increases. These results suggest that inspiratory tidal airflow generation during running in the horse may be the sole province of the diaphragm, a contention that is supported by the extraordinary thickness, oxidative capacity, and blood flow capacity of the equine diaphragm (31, 38).

Although LRC may limit airflow generation during running in the horse, one potential benefit of a LRC-derived constraint on exercise ventilation can be gleaned from the work of Harms et al. (21, 22). These authors reported that the work of breathing can affect performance and time to fatigue in humans by redistributing flow away from the locomotory and toward the respiratory muscles. As the work of breathing increases to a much greater degree in the horse than the human (cf. Refs. 1 and 3), this mechanism has perhaps an even greater fatigue-generation potential in the horse. This is particularly true because the TB horse is thought to rely primarily on diaphragmatic breathing (2, 32) during the gallop; thus any attempt to overcome the constraint resulting from LRC could well lead to both diaphragmatic and locomotory muscle fatigue.

Unlike TB horses, Standardbred horses can achieve their maximum \( V_\text{O}_2 \) while trotting or pacing when the breathing and stride frequencies are not coupled. The maximum \( V_\text{O}_2 \) in competitive Standardbred horses (i.e., ~165 ml · kg\(^{-1}\) · min\(^{-1}\); see Refs. 4 and 18) is close to that found in the TB horse. However, \( V_\text{T} \) is far higher (20–26 vs. 12–14 l/breath) and \( f \) is lower (70–80 vs. 130 breaths/min) in the Standardbred vs. the TB horse. Therefore, for Standardbred horses at the transition from the maximum speed to either resting or a lower speed, one would not expect the same response as is found in the TB horse (i.e., increasing \( V_\text{T} \), lowering of \( f \)). Indeed, Standardbred horses increase \( f \) and decrease \( V_\text{T} \) across this transition. Thus, although the strategy is different, one common feature across these two breeds is that both hyperventilate during intense exercise (i.e., \( V_\text{E}/V_\text{O}_2 \), \( V_\text{E}/V_\text{CO}_2 \), and \( \text{PaO}_2 \) fall, and \( \text{PaCO}_2 \) rises) and hyperventilate in recovery (4, 35).

Mechanistic basis for slow \( V_\text{E} \) recovery in TB horse. In the TB horse, the presence of hyperventilation during maximal exercise and noticeable hyperventilation (see Fig. 4) during recovery suggests strongly that, in considerable contrast to humans (51), \( \text{PaCO}_2 \) is not a precisely controlled variable in equids (35, 39). In addition, the observation that \( V_\text{E} \) remains at or above end-exercise values during the early recovery period (Figs. 1 and 3) constitutes strong evidence that LRC may actually provide an impedance to \( V_\text{E} \) during exercise, whereas the slow, prolonged recovery profile for \( V_\text{E} \) suggests multiple sources of ventilatory control during recovery from maximal exercise.

Initial ("fast") component of \( V_\text{E} \). Throughout high-intensity exercise, \( \text{PaCO}_2 \) is markedly elevated in the TB horse (35, 36). As TB horses exhibit a normal ventilatory response to \( \text{CO}_2 \) at rest (in contrast to the situation during exercise; Ref. 28), it is likely that \( \text{CO}_2 \) is providing at least some of the initial stimulus responsible for the elevation of \( V_\text{E} \) noted in the present study. Indeed, \( V_\text{T} \) increased rapidly, whereas \( f \) fell (Fig. 3 and Table 1), indicative of increased alveolar ventilation and enhanced \( \text{CO}_2 \) elimination (51). However, by 2 min of recovery, \( \text{PaCO}_2 \) had fallen to hypocapnic levels (Fig. 4), whereas \( V_\text{E} \) was still elevated, which strongly suggests that the ventilatory response was being maintained at this point by other stimuli.

Secondary ("slow") component of \( V_\text{E} \). The secondary component of \( V_\text{E} \) during recovery started at ~90 s of recovery, likely at a time point when \( \text{PaCO}_2 \) was already well below 40 Torr (see \( V_\text{E}/V_\text{CO}_2 \) profile in Fig. 4). Although \( pH \) at end exercise (7.217 ± 0.107) and during recovery (2 min: 7.301 ± 0.093; 4 min: 7.307 ± 0.098) is reduced below resting baseline, these values are not different from those reported in humans (46) or in the pony (39), which, unlike its more fit relative, exhibits a more rapid \( V_\text{E} \) recovery-response profile. In addition, the changes in \( pH \) and \( V_\text{E} \) were not correlated.

It is also generally acknowledged that there is an extensive array of ventilatory stimuli present during and after intense exercise. Thus the control of the exercise (and postexercise) hyperpnea is complex and demonstrates considerable redundancy (for review, see Ref. 50). In early recovery after the gallop-trot transition, potential sources of ventilatory stimulation include arterial acidosis (lactic acidosis and hypercapnia), elevated catecholamines, temperature, potassium ions (\( K^+ \)), osmolality, venous distension, and/or some form of short-term potentiation (STP). This STP was originally termed "respiratory afterdischarge" by Gesell and White (19) and has subsequently been described as an exponential ventilatory response that decays relatively slowly after removal of the stimulus. Specifically, STP decays with a time constant ranging from 36 to 101 s after carotid sinus nerve stimulation in cats (12, 13) and 18 to 39 s after hypoxic exercise in humans (17). Thus, if
present in the TB horse, STP may have potentially played a role in the prolonged ventilatory response that was found during trotting recovery described herein.

The present investigation was not designed to determine which of these above mediators induced the postgalloping elevated ventilatory response. However, the temporal profile of some of these mediators decreases the likelihood that they played a major role in the extended hyperventilation seen in the trotting recovery. For example, the exercise-induced hypercapnia was resolved at least by 2 min, at which time the PaCO₂ had been driven substantially below resting values. Whereas catecholamines were not measured in the present investigation, Snow et al. (44) observed that blood catecholamine levels returned close to resting levels within 60 s postexercise, which is much faster that the Ve response. In addition, K⁺ recovers to baseline values within ~2 min of recovery (23).

How are ventilation and hyperthermia linked? Respiratory heat exchange occurs in many mammalian species that primarily employ nasal breathing (42). In large mammals such as the camel (41) and the horse (24, 34), ventilation during heat stress serves to maintain brain temperature as much as several degrees Celsius below core body temperature (34, 49). Although horses employ a robust sweating response (rates of >30 l/h) that works in concert with the respiratory system to dissipate heat during exercise (24), these responses are not adequate to prevent heat storage during exercise, in part due to a relatively low surface area-to-body mass ratio in the racing horse. Thus, during exercise, the TB horse routinely achieves core body temperatures up to ~42–43°C (24, 34, 49). During trotting recovery, with the locomotory muscles operating at a much reduced metabolic rate compared with the gallop, evaporative heat loss from the respiratory tract will augment heat dissipation and provides one possible explanation for the prolonged elevation in Ve seen postgalloping. This strategy may be affected, in part, by the adoption of an altered breathing pattern during the recovery from high-intensity exercise.

Core temperature follows a slow recovery time course after incremental exercise (33). In this investigation, core temperature was markedly elevated throughout recovery (~40.5°C at 4-min postgallop), and the rate of change in Ve during the secondary recovery component was highly correlated with that of core temperature (r² = 0.998; P < 0.05). Given that hyperthermia does constitute a powerful Ve stimulus in the horse (33) and that the temporal profile of other potential Ve stimuli (i.e., K⁺, catecholamines, PaCO₂) during recovery from exercise in the horse does not cohere with that of Ve, the possibility must be acknowledged that the hyperthermia, which attends maximal exercise and recovery in TB horses, may potentially contribute to the prolonged hyperventilatory response found during recovery.

In conclusion, rather than exhibiting the precipitous fall in Ve such as that observed in humans after intense exercise (29, 40, 46), horses sustain the full magnitude of the exercise hyperventilation for several seconds into recovery followed by a prolonged biphasic decrease in Ve. This sustained hyperventilation is the product of an immediate increase in Ve combined with a fall in f (Table 2 and Fig. 3). Because an immediate fall in Ve was not found during the off-transition from the gallop (LRC present) to the trot (no LRC), we conclude that LRC does not appear requisite to achieve a prodigiously high Ve equivalent to that seen during maximal exercise.

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REFERENCES


