Ventilatory and arterial blood gas tension adjustments to strenuous exercise in Standardbreds

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Summary

Five healthy, fit Standardbreds (mean ± SEM, 490.4 ± 15.0 kg, 4.0 ± 0.5 years) were studied during a standardized test carried out on a treadmill. The test consisted of an 8-minute warm-up and a 9-minute exercise period (1 minute at 1.7, 4, 7, 8, 9, and 10 m/s; 2 minutes at 4 m/s; and a 1-minute walk at a 6% slope). Respiratory airflow, tidal volume (VT), and respiratory frequency (f) were continuously recorded, using 2 ultrasonic pneumotachographs connected to a face mask and mass spectrometer. Oxygen uptake, carbon dioxide output, and expired minute volume ($\dot{V}E$) were obtained on a breath-by-breath basis. Arterial blood was tested at the end of each step for O_2 and CO_2 partial pressures. Heart rate was continuously recorded, using a heart rate recording system. Stride frequency was measured at each step, and the stride frequency-to-f ratio was calculated. Venous blood was tested for plasma lactate concentration before and 2 minutes after completion of the test.

Some horses had a locomotion-respiration coupling (LRC), but this coupling was occasional and intermittent. The f was lower and VT was higher than values reported for Thoroughbreds working under similar experimental conditions. Nevertheless, maximal $\dot{V}E$ did not overshoot maximal $\dot{V}E$ reported in Thoroughbreds. All horses were hypoxemic and hypercapnic, but there was wide variability between subjects. The horses with the highest oxygen uptake and the lowest plasma lactate concentration were more hypoxemic and hypercapnic.

The Standardbreds, studied under our laboratory conditions, did not have constant LRC and had lower f with higher VT than did Thoroughbreds under similar experimental conditions. Despite these differences in breathing strategy, the Standardbreds did not have higher $\dot{V}E$ than did Thoroughbreds, and they were hypoxemic and hypercapnic. The fact that these Standardbreds, which obviously freely selected their breathing strategy, were unable or unwilling to adopt compensatory hyperventilation reinforces the hypothesis that, in strenuous exercising horses, there could be a physiologic limit to ventilation, most probably related to mechanical factors, but independent of any LRC.

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Arterial hypoxemia and hypercapnia have been documented during heavy exercise in horses.[1,2] Several studies have been performed to attempt to elucidate the mechanisms leading to the early development of this phenomenon in horses. Although right-to-left shunts and matching of ventilation and perfusion have been reported to be accessory factors, the shortening of the capillary transit time, reducing the time for O_2 equilibration, has been put forward to explain impairment of O_2 diffusion.[3] The same authors also suggested that development of relative alveolar hypoventilation could be partly responsible for the exercise-induced hypoxemia. This alveolar hypoventilation has been related to mechanical factors, such as the increase in the total pulmonary resistance and work of breathing during exercise.[4,5] However, most studies of arterial blood gas tension adjustments during exercise have been performed in Thoroughbreds,[1,3-8] in which the respiratory frequency (f) is tied to limb stride frequency (SF). It has, therefore, been suggested that, in galloping horses, the only way to increase minute expired volume ($\dot{V}E$) is to increase tidal volume (VT), a breathing strategy that could ultimately limit the ventilatory response to exercise.[9] If this is true, the ventilatory and blood gas tension changes induced by strenuous exercise in trotting horses, in which the coupling is not supposed to be compulsory,[10-12] should be different from those in galloping horses.

The aim of the study reported here was, therefore, to determine ventilation and breathing strategy, arterial blood gas tension modifications, and the possible relation between locomotion and respiration during heavy exercise in Standardbreds.

Materials and Methods

Horses—Five healthy, fit Standardbreds (1 stallion, 2 geldings, and 2 mares; mean ± SEM, 490.4 ± 15.0 kg, 4.0 ± 0.5 years) free of cardiac, respiratory, or locomotor disorders were studied. Careful clinical examination (including endoscopy of the airways, tracheobronchial lavage, pulmonary function tests, arterial blood gas analysis, and ECG) was performed to assess their soundness. Horses were well accustomed to the treadmill work and the laboratory procedures.

Standardized exercise test (SET)—Exercise tests were carried out in horses on a treadmill[a] in a laboratory in which temperature was maintained at 15 C, with 55% relative humidity. Once the test started, 2 fans placed in front of the horses continuously blew air at a constant flow. All horses were investigated between 9 AM and 12 PM.

After an 8-minute warm-up period (3-minute walk and 5-minute slow trot), the horses were equipped with the mask-pneumotachograph assembly and the treadmill was inclined to 6%. The test consisted of 6 incremental exercise periods of 1 minute each at 1.7, 4, 7, 8, 9, and 10 m/s. The horses continued to trot during the test. After completion of the test, the treadmill was lowered to the level position and the horses trotted (4 m/s) for 2 minutes and walked for 1 minute before the end of the exercise. Two weeks prior to this study, a test to fatigue was conducted to determine the individual maximal oxygen uptake ($\dot{V}_{O_{2max}}$) of each horse.

Arterial blood gas tensions—Prior to the study, a 20-gauge catheter[b] was placed in the transverse facial artery of the horses under local anesthesia, and was secured to the skin by application of glue. A 110-cm plastic extension line was attached to the catheter to allow blood to be drawn at a distance from the horse. The catheter and extension were flushed with heparinized saline solution between sample collections to maintain patency. Immediately prior to collection of each sample, 12 ml of blood and saline solution were drawn and discarded. Arterial blood samples were withdrawn during the last 10 seconds of each step of the test into 2-ml syringes, the dead space (VD) of which was filled with sodium heparin (10,000 IU/ml). The syringes were capped and stored in crushed ice until analysis, which was performed within 10 minutes of sample collection.

Arterial partial pressure of O_2 (Pa_{O_2}) and CO_2 (Pa_{CO_2}), hemoglobin saturation with O_2, O_2 content, arterial pH, and HCO_3^- and base excess concentrations were measured, using a blood gas analyzer.[c] The data were temperature corrected. The arterial blood temperature was estimated according to its relation to rectal temperature. This relation was established during a preliminary experiment in which 3 horses performed the same SET while rectal and blood temperatures were simultaneously measured. The blood temperature was measured in the pulmonary artery, using the thermistor of a Swan-Ganz catheter[d] inserted in the left jugular vein through an 8.5-F inducer[e] and connected to a cardiac output computer.[f]

Stride frequency—The SF was measured, using the tip of a 3-m-long catheter (ID: 3 mm; OD: 4 mm) covered with a slightly inflated balloon and fixed between the right shoulder and a leather band passing on the shoulder. The other end of the catheter was connected to a pressure transducer.[g] The peak pressure signal at the end of the stance phase was used to calculate SF for each speed. The recording of each test by a video camera allowed control of the accuracy of the calculated SF.

Ventilation—Respiratory airflow from each nostril was measured, using a pneumotachometer composed of 2 ultrasonic flow transducers[h] mounted diagonally across a polyvinyl chloride tube. Each pneumotachometer had a full scale range of ± 60 L/s (giving a total peak flow range of ± 120 L/s). The linearity and symmetry of the output of both ultrasonic pneumotachometers have been verified for 1 to 55 L/s.

Both tubes were mounted in a lightweight mask and were positioned so as to be in line with the normal flow of air from the nostril. This reduced any extra dead space and, because flow was not subject to constriction or redirection, pressure loading on the ventilatory system was minimized. The mask was constructed from fiberglass and fitted closely to the muzzle of the horse, leaving the mouth free. Its lack of influence on arterial blood gas tension has been assessed.[5] Tidal volume was obtained by numerical integration of the flow signal and was electronically corrected to BTPS conditions. Before and after each SET, the flow tubes were individually calibrated by use of a high-flow source and an air velocity transducer.[i]

Oxygen consumption ($\dot{V}_{O_2}$) and CO_2 output ($\dot{V}_{CO_2}$)—A mass spectrometer[j] was used to sample air in 1 flow tube, and O_2 and CO_2 concentrations in the respiratory gases were continually measured on a breath-by-breath basis. Oxygen uptake and $\dot{V}_{CO_2}$ (STPD) were instantaneously calculated by use of on-line and breath-by-breath computer analysis.[k] Before and after each SET, the mass spectrometer was calibrated, using gas mixtures of known composition. More details about the method are given elsewhere.[5]

Breathing strategy—The instantaneous respiratory airflow, VT, and O_2 and CO_2 fractions in the respiratory gases were continuously recorded by use of a magnetic tape recorder.[1] These curves allowed evaluation of the possible occurrence of big respiratory cycle (BRC; ie, a respiratory maneuver frequently observed in galloping horses that transiently breaks the locomotion-respiration coupling (LRC) and allows the horse to breathe with a higher VT and a lower f during 1 cycle.[13]

Heart rate—Heart rate values were recorded, using a horse tester[m] throughout each investigation and recovery period. They were calculated and recorded during consecutive periods of 5 seconds. After each experiment, the stored data were displayed on a microcomputer.

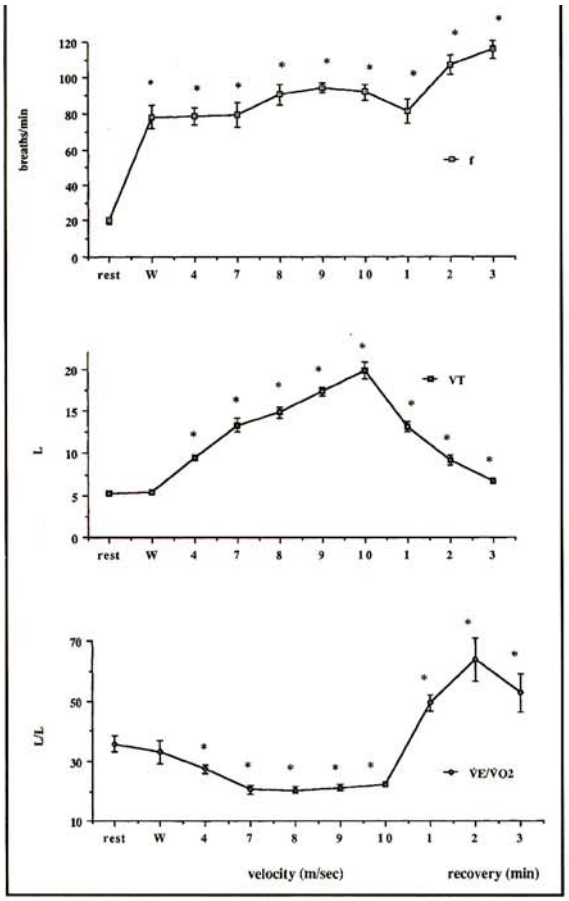

Figure 1—Respiratory frequency (f), tidal volume (VT), and ventilatory equivalent for O_2 ($\dot{V}E/\dot{V}_{O_2}$) in 5 Standardbred trotters at rest, during an incremental standardized treadmill test, and during a 3-minute recovery period after the test. * Significantly ($P \leq 0.05$) different from at rest values.

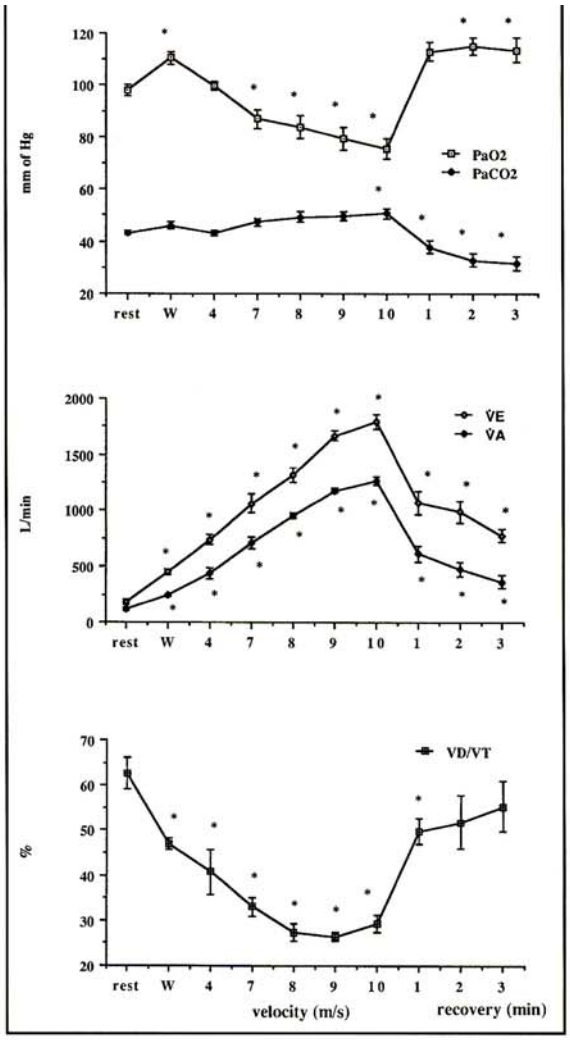

Figure 2—Arterial partial pressure of O_2 (Pa_{O_2}) and CO_2 (Pa_{CO_2}), expired minute volume ($\dot{V}E$), alveolar ventilation ($\dot{V}A$), and physiologic dead space-to-tidal volume ratio (VD/VT) in 5 Standardbred trotters at rest, during an incremental standardized treadmill test, and during a 3-minute recovery period after the test. * Significantly ($P \leq 0.05$) different from at rest values.

Other measurements—Rectal temperature was continuously measured by a rectal probe[n] during the SET. Venous blood was tested by jugular puncture just before and 2 minutes after completion of the test, using a vacuum tube containing sodium heparin and sodium monoiodoacetate. This sample was used for lactate (LA) determination by a colorimetric method.[o]

Calculations and analysis—Tidal volume, f , $\dot{V}E$, $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, SF, and heart rate were mean values collected at rest and during the last 15 seconds of each step of the incremental SET, as well as 1, 2, and 3 minutes after the most intense step (10 m/s). The inspiratory drive was also calculated as the VT-to-inspiratory time ratio. Arterial blood gas tensions were tested at rest, during the 10 last seconds of each step, and 1, 2, and 3 minutes after the last step of the SET. Alveolar ventilation was estimated from the ratio of $\dot{V}_{CO_2}$ to Pa_{CO_2} (expressed as a fractional gas concentration). As well, the physiologic VD-to-VT ratio (VD/VT) was calculated.

Data are reported as mean ± SEM. Analysis of variance was used to assess significant differences between the values at rest and during exercise and recovery. Correlation coefficients and their significance were computed to assess the relation between arterial blood gas tensions and $\dot{V}_{O_{2max}}$ and LA.

Results

Respiratory frequency was calculated over the last 30 seconds of each step, because it was not regular throughout a given step. It remained similar during walking and trotting at 4 and 7 m/s (approx 79 breaths/min; Fig 1), increased at 8 m/s, but did not increase further at 9 and 10 m/s. The highest values were recorded after 3 minutes' recovery (115.8 ± 5.1 breaths/min). Tidal volume and $\dot{V}E$ (Fig 2) increased progressively and significantly with each step of the test, reaching peak values (at 10 m/s) of 19.8 ± 1.0 L and 1,812 ± 65 L/min, respectively. No BRC were

Table 1—Cardiorespiratory variables, heart rate (HR), and stride frequency (SF) in 5 Standardbred trotters at rest, during an incremental standardized treadmill test, and during a 3-minute recovery period after the test (first and second minutes at 4 m/s and the third minute at walk)

Values	Rest	Walk	4 m/s	7 m/s	8 m/s	9 m/s	10 m/s	1 min	2 min	3 min
$\dot{V}_{O_2}$ (ml/kg/min)	11.4 ± 0.8	28.8 ± 2.0	55.4 ± 3.8	103.6 ± 5.0	133.2 ± 3.1	157.4 ± 4.3	165.6 ± 7.3	44.3 ± 4.2	33.0 ± 3.5	26.0 ± 0.8
$\dot{V}_{CO_2}$ (ml/kg/min)	8.8 ± 0.4	23.4 ± 1.8	53.4 ± 4.6	95.4 ± 3.6	134.2 ± 3.2	167.6 ± 5.2	184.6 ± 8.8	66.0 ± 6.6	43.2 ± 4.2	31.0 ± 4.0
R (%/100)	0.77 ± 0.02	0.81 ± 0.02	0.96 ± 0.02	0.93 ± 0.04	1.01 ± 0.01	1.07 ± 0.02	1.11 ± 0.01	1.49 ± 0.08	1.37 ± 0.12	1.07 ± 0.13
HR (beats/min)	47.2 ± 3.8	98.0 ± 4.8	140.6 ± 6.1	189.6 ± 8.2	201.8 ± 5.8	210.4 ± 5.1	220.8 ± 3.8	140.4 ± 3.0	128.0 ± 4.3	104.6 ± 4.0
VT/IT (L/s)	3.1 ± 0.6	15.0 ± 1.5	24.2 ± 1.5	32.8 ± 1.9	44.1 ± 1.8	50.1 ± 2.2	56.9 ± 3.3	33.0 ± 2.8	31.4 ± 3.0	29.2 ± 1.8
SF (strides/min)	...	61.2 ± 1.2	93.8 ± 2.1	109.6 ± 1.8	113.8 ± 1.8	119.4 ± 2.1	123.2 ± 2.1	91.0 ± 1.7	91.4 ± 1.6	59.6 ± 0.4

All the values during exercise and recovery were significantly different from the rest values with $P \leq 0.05$.
$\dot{V}_{O_2}$ = oxygen consumption; $\dot{V}_{CO_2}$ = carbon dioxide output; R = respiratory quotient; and VT/IT = tidal volume-to-inspiratory time ratio.

Table 2—Individual values of SF-to-respiratory frequency ratio in 5 Standardbred trotters during an incremental standardized treadmill test

Horses	4 m/s	7 m/s	8 m/s	9 m/s	10 m/s
DY	1.29	1.39	1.08	1.24	1.43
CK	1.54	1.73	1.50*	1.46	1.44
AL	1.14	1.53	1.26	1.24	1.50*
CO	1.15	1.48	1.47	1.33*	1.23
DB	0.95	1.00*	1.08	1.10	1.16

* Locomotion-respiration coupling.
See Table 1 for key.

Table 3—Individual arterial partial pressure of O_2 (Pa_{O_2}) and CO_2 (Pa_{CO_2}) at 10 m/s, maximal oxygen uptake ($\dot{V}_{O_{2max}}$), plasma lactate concentration, after exercise, and VT/IT at 10 m/s in 5 Standardbred trotters

Horses	Pa_{O_2} (mm of Hg)	Pa_{CO_2} (mm of Hg)	$\dot{V}_{O_{2max}}$ (ml/kg/min)	Lactate (mmol/L)	VT/IT (L/s)
DY	83.9	48.6	147	9.2	45.8
CK	81.3	48.3	160	9.1	55.4
AL	68.2	54.8	186	5.9	64.3
CO	64.7	55.2	181	5.8	63.0
DB	85.1	46.0	154	10.2	56.1

See Table 1 for key.

observed. The inspiratory drive increased from 3.1 ± 0.6 L/s at rest up to 56.9 ± 3.3 L/s at 10 m/s. The ventilatory equivalent for O_2 ($\dot{V}E$-to-$\dot{V}_{O_2}$ ratio) decreased from 35.8 ± 2.6 L/L at rest to 20.5 ± 1.4 L/L at 7 m/s, remained steady during the SET, then increased to 63.8 ± 9.1 L/L during the recovery period. The SF increased linearly with the increase in speed, changing from 93.8 ± 2.1 strides/min at 4 m/s 123.2 ± 2.1 strides/min at 10 m/s (Table 1). Although the pooled data did not indicate any LRC, coupling between SF and f occurred in some horses during short periods (ie, from 20 to 35 seconds; Table 2). The LRC ratio was 1.00 (1 stride/1 breath), 1.50 (3 strides/2 breaths), or 1.33 (4 strides/3 breaths). When LRC occurred, f became momentarily regular. Arterial partial pressure of O_2 decreased from 110.5 ± 2.5 mm of Hg at rest to 75.4 ± 3.7 mm of Hg at 10 m/s, whereas Pa_{CO_2} decreased from 44.9 ± 0.6 mm of Hg at rest to 42.9 ± 1.1 mm of Hg at 4 m/s, then increased to 50.6 ± 1.9 mm of Hg at 10 m/s. Consideration of individual data pointed out a wide interindividual variation (eg, at 10 m/s, P_{O_2} ranged from 64.7 to 82.1 mm of Hg, and Pa_{CO_2} ranged from 46.0 to 55.2 mm of Hg; Table 3). One minute after the end of the test, both variables returned to rest values. Alveolar ventilation increased from 113.3 ± 10.1 L/min at rest to 1,264.8 ± 37.7 L/min at 10 m/s while horses were under similar conditions; the VD/VT decreased from 62.6 ± 3.4% to 29.3 ± 2.0%. Other respiratory and arterial blood values also were determined (Tables 1 and 4).

Each horse reached $\dot{V}_{O_{2max}}$ during the test. The correlation between Pa_{O_2} at 10 m/s and $\dot{V}_{O_{2max}}$ (negative correlation; $r = -0.90$; $P < 0.01$) and LA concentration (positive correlation; $r = 0.97$; $P < 0.001$) was strong and significant. The same was true for the correlation between Pa_{CO_2} and $\dot{V}_{O_{2max}}$ (positive correlation; $r = 0.85$; $P < 0.01$) and LA concentration (negative correlation; $r = -0.99$; $P < 0.001$).

Discussion

In light of the numerous studies of equine respiratory physiology during exercise, it appears that the equine respiratory system seems unable to respond to the metabolic demand resulting from high muscular aerobic capacity. These animals develop CO_2 retention and hypoxemia during strenuous exercise. Shortening of the capillary transit time, reducing the time for O_2 equilibration, is partly responsible for the impairment of O_2 diffusion.[3] But, in contrast to other mammals with higher $\dot{V}_{O_{2max}}$ and shorter capillary transit time, horses fail to compensate for this

Table 4—Arterial blood gas values in 5 Standardbred trotters at rest, during an incremental standardized treadmill test, and during a 3-minute recovery period after the test (first and second minutes at 4 m/s and the third minute at walk)

Values	Rest	Walk	4 m/s	7 m/s	8 m/s	9 m/s	10 m/s	1 min	2 min	3 min
PCV (%)	35.1 ± 1.6	42.5* ± 0.8	43.7* ± 0.9	49.2* ± 1.2	54.1* ± 1.0	56.8* ± 0.9	57.8* ± 0.9	57.2* ± 0.9	56.4* ± 1.2	55.9* ± 1.1
Sa_{O_2} (%)	97.9 ± 0.2	98.3 ± 0.2	97.5 ± 0.1	95.2* ± 0.9	91.0* ± 2.7	90.2* ± 2.6	86.2* ± 3.3	95.9* ± 0.9	96.6 ± 0.8	96.9 ± 0.6
Ca_{O_2} (ml/100 ml)	19.2 ± 0.5	26.4* ± 1.1	23.1 ± 1.4	25.5* ± 1.2	25.9* ± 0.8	26.9* ± 0.7	26.4* ± 0.9	28.3* ± 0.7	28.1* ± 1.1	28.3* ± 0.9
pHa	7.372 ± 0.015	7.423* ± 0.016	7.406* ± 0.012	7.365 ± 0.014	7.320 ± 0.024	7.237 ± 0.032	7.220* ± 0.036	7.243* ± 0.044	7.283 ± 0.046	7.307 ± 0.047
HCO_3^- (mmol/L)	26.1 ± 0.6	26.3 ± 0.05	25.9 ± 0.5	25.8 ± 0.7	24.2 ± 1.1	21.9* ± 1.4	19.5* ± 1.6	15.7* ± 1.6	15.1* ± 1.8	15.4* ± 1.9
BE (mmol/L)	1.2 ± 0.7	1.9 ± 0.5	1.9 ± 0.6	0.9 ± 0.8	−1.4* ± 1.3	−4.1* ± 1.8	−7.1* ± 2.1	−9.6* ± 2.4	−8.7* ± 2.5	−8.2* ± 2.6

* Significantly different from the rest values with $P \leq 0.05$.
Sa_{O_2} = hemoglobin saturation with O_2; Ca_{O_2} = O_2 content; and BE = base excess.

phenomenon by hyperventilatory response during heavy exercise.[4,14] This lack of a compensatory hyperventilation is probably associated with mechanical limitations (ie, marked increase in the mechanical work of breathing and in respiratory muscles oxygen uptake, force-velocity characteristics and limitation of the respiratory muscles, and fatigue of the respiratory muscles).[15]

With the $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ that running horses can reach and the 1:1 LRC existing in galloping horses, VT has been suggested to be limited because of the short time available to achieve it.[4] Consequently, ventilatory limitation during exercise in galloping horses could be partly attributable to LRC.[9] If this is true, ventilation in our heavily exercising Standardbred trotters, in which LRC was intermittent and not compulsory, should have been less limited than that in galloping horses. Obviously, because of lack of LRC, the horses' breathing strategy was different from the strategy observed in galloping Thoroughbreds working under similar experimental conditions[16]: trotters breathed with a lower f, but greater VT to achieve approximately the same $\dot{V}E$. However, at $\dot{V}_{O_{2max}}$, the $\dot{V}E$ in the Standardbred trotters was not higher than $\dot{V}E$ in Thoroughbreds. Moreover, such as reported in Thoroughbreds,[4] Standardbreds became hypoxemic at approximately 80% of $\dot{V}_{O_{2max}}$ and hypercapnic at approximately 95% of $\dot{V}_{O_{2max}}$. These results point out the fact that, although trotters seem to be able to freely select their breathing strategy, they are not more able or willing than Thoroughbreds to adopt a compensatory hyperventilation. This adds evidence to the hypothesis that, because of their high metabolic needs on the one hand, and their respiratory physiologic and morphologic characteristics on the other hand, the horses, whatever their breathing strategy, tend to optimize, rather than maximize, the gas exchange at the cost of blood homeostasis but at the profit of the energetic cost of breathing.[5]

The Standardbreds did not have any BRC. In a previous study, Jolly et al[13] suggested that in galloping horses, these BRC could be attributable to chemoreflexes secondary to the changes in arterial blood gas tensions; to mechanoreflexes provided by the upper airways, the lungs, the thorax, or the respiratory muscles; to readjust the end-expiratory lung volume (EELV); or to reflexes originating from higher brain centers or induced by the severe exercise-induced capillary hypertension in the exercising horses. The fact that the Standardbreds did not have any BRC rules out the possibility that arterial blood gas tensions and capillary hypertension were factors inducing the BRC maneuver, but could confirm the theory that the readjustment of the EELV is the reason for occurrence of the BRC. Indeed, because of the lack of compulsory LRC, these horses are probably able to adjust their EELV breath-by-breath, and consequently, do not need to make BRC.

Despite the difference in the breathing strategy to reach a given $\dot{V}E$, alveolar ventilation seemed to reach a maximal value similar to the values already reported in Thoroughbreds during intense exercise.[4,15] However, the calculated measurements of alveolar ventilation and VD/VT are usually accurate when performed in horses at rest under steady-state conditions, which could not be the case during the incremental SET. This fact may be taken into account in interpretation of the absolute data. The VD/VT decreased significantly during this short intense exercise. During the recovery period, the opposite trend was found because of the simultaneous increase in f and decrease in VT. The same physiologic adjustments also have been reported in galloping Thoroughbreds.[15,17]

The effect of exercise on arterial blood gas tensions in trotting Standardbreds are conflicting. Fast trot has been documented to induce hypoxemia and hypocapnia,[18] hypoxemia and normocapnia,[7,19] or hypoxemia and hypercapnia,[2,14] the latter adjustment also being observed by us. Several explanations can be put forward to explain the discrepancy between the results (ie, state of training of the horses, their intrinsic aerobic capability, and duration of the exercise).

The horses of this study were hypoxemic and hypercapnic at the highest intensity of work, but there was wide interindividual variability in these exercise-induced modifications: some horses had only mild changes, whereas others had marked modifications in arterial blood gas tensions (Table 3). This variability has already been reported by Thornton et al[2] after a training period. According to results of this study, it is interesting that the horses with the highest $\dot{V}_{O_{2max}}$ and the lowest LA concentration after the SET were also more hypoxemic and hypercapnic during exercise. Although the number of subjects was limited, this suggests that horses with a high muscular aerobic potential could become hypoxemic and hypercapnic earlier than others. Further studies, including more horses and the simultaneous measurements of the mixed venous and arterial blood gas tensions, would be necessary to confirm this hypothesis.

Another factor that could explain the discrepancy between the results of the arterial blood gas tensions studies in trotters is duration of exercise. Indeed, Bayly et al[4] and Hodgson et al[8] reported that, in galloping horses, hypercapnia developed at high intensity of exercise but, if the effort was prolonged more than 2 minutes, this hypercapnia was progressively corrected. This suggests that, in Thoroughbreds, there is a respiratory lag at the beginning of exercise that may progressively be compensated for afterward. The same phenomenon is also probably true in Standardbreds. Consequently, the fact that the reported changes in Pa_{CO_2} differ from one study to another may be partly explained by the respective duration of the exercise steps used.

Lastly, the influence of wearing a mask on arterial blood gas tension during exercise has been documented in several studies.[7,20,21] However, the degree of influence is highly variable from one mask to another. The mask used in this study was light, did not hinder nostril dilatation, had a minimal VD, and was tested for its lack of influence on blood gas tension.[5]

In conclusion, the Standardbreds studied under

these laboratory conditions had a lack of compulsory coupling between respiration and locomotion. They consequently had another breathing strategy (ie, lower f with higher VT) than Thoroughbreds under similar experimental conditions. However, despite this difference in breathing strategy, Standardbreds did not reach a higher $\dot{V}E$ than did Thoroughbreds, and were hypoxemic and hypercapnic. These results suggest that, in heavily exercising horses, independent of the gait, there is a physiologic limit to ventilation, most probably related to mechanical factors that impede development of compensatory hyperventilation. When this critical level is reached, the energetic cost of breathing probably becomes so important that the horses can set ventilation at a lower level, at the cost of arterial hypoxemia and hypercapnia and at the benefit of the skeletal muscles.

[a] Equispeed, Versailles, Mich.
[b] Baxter, Brussels, Belgium.
[c] AVL 995, VEL, Louvain, Belgium.
[d] Elecath 73-4067 7F, Columbus Instruments, Columbus, Ohio.
[e] No. SI-09875-E, Arrow, Redding, Pa.
[f] CardiomaxII, Columbus Instruments, Columbus, Ohio.
[g] Statham-PD 23, Siemens, Solna, Sweden.
[h] BRDL Flowmetrics, Birmingham, United Kingdom.
[i] AVT model 8450/60/70, BTW, Eupen, Belgium.
[j] MGA 2000, Case Biggin Hill, Kent, United Kingdom.
[k] Case Biggin Hill, Kent, United Kingdom.
[l] MTR 3968A, Hewlett Packard, Brussels, Belgium.
[m] AMG, Dinant, Belgium.
[n] Ellab, Copenhagen, Denmark.
[o] Boehringer Ingelheim, Ingelheim, Germany.

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