

Relationships between Arterial Oxygen Tensions and Cardiocirculatory Function during Submaximal Exercise in the Horse

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Summary

The relationship between arterial oxygen tension (PaO_2) and various markers for cardio-circulatory capacities were studied during submaximal exercise in 6 clinically healthy horses on 20 occasions. On each occasion the horse performed a standardized incremental exercise test on a high-speed treadmill. The PaO_2 was determined at rest (PaO_2 -B) and at each treadmill velocity. The exercise-induced hypoxemia was expressed as the oxygen tension measured at a speed of 9 m/sec on a 6.25% treadmill slope (PaO_2 -V9) and as the interpolated value at the treadmill velocity which produced a heart rate of 200 bpm (PaO_2 -200). A heart rate of 200 bpm approximately corresponds to the lactate threshold of the horse. The total red cell volume was determined post exercise from Evans blue dye dilution and the hematocrit.

The PaO_2 -V9 was inversely correlated with the total red cell volume ($r = -0.72$) and the post exercise hematocrit ($r = -0.65$). The PaO_2 -200 was also correlated with these variables, and was negatively correlated with V_{200} ($r = 0.85$). The PaO_2 -200 was predictable with a SD of ± 0.6 kPa.

Exercise-induced hypoxemia seems to be due mainly to the increased circulating red cell volume caused by mobilization of erythrocytes from the spleen during exercise. This increased red cell volume results in a shorter red cell transit time through the lungs. The oxygen deficit in the increased circulating red cell volume is partly met by an increased alveolar ventilation.

Index terms: Blood volume; heart rate; blood lactate; treadmill exercise.

Introduction

Arterial oxygen tension determined at rest and during and after exercise has been used extensively to assess impairment of pulmonary function in horses with obstructive pulmonary disease (Gillespie *et al.*, 1964; Sasse, 1973; Bergsten, 1974; McPherson and Lawson, 1974; McPherson *et al.*, 1978; Littlejohn and Bowles, 1981). The factors regulating arterial oxygen tension in the healthy horse at rest and during exercise are,

however, not well known. Thus, there is still some divergence of opinion regarding the effect of exercise on arterial oxygen tension. Although several studies imply an unchanged arterial oxygen tension during exercise (Bergsten, 1974; Pan *et al.*, 1983; Rose *et al.*, 1983b; Parks and Manohar, 1984), others indicate that exercise causes a significant hypoxemia of the arterial blood in the horse (Littlejohn and Bowles, 1981; Bayly *et al.*, 1983; Rose *et al.*, 1983a; Thornton *et al.*, 1983) and in human athletes exercising close to the limit of their capacity (Rowell *et al.*, 1964; Astrand and Rodahl, 1970). Recently, Dempsey and Fregosi (1985) suggested that the arterial hypoxemia observed during severe exercise in humans is due to an inadequate red cell transit time for equilibration of oxygen between end-capillary blood and alveolar gas. The mechanism of exercise-induced hypoxemia in the horse has not yet been established but several possibilities exist; alveolar hypoventilation, ventilation/perfusion inequalities, intrapulmonary right-to-left shunting, and/or changes in alveolar-capillary diffusion gradient.

The primary objective of this study was to elucidate possible relationships between arterial oxygen tension and cardio-circulatory functions during submaximal exercise in the normal horse. A second objective was to improve the precision in detection of abnormal hypoxemia caused by pulmonary disease avoiding misinterpretation due to apprehensive hyperventilation at rest.

Materials and Methods

Horses. The investigation included six Standardbred horses, two mares and four geldings, varying in age between 3 and 13 (mean = 8.5) years. Carotid arteries had been relocated to a subcutaneous site. These horses were normally used for research and student demonstrations and repeated careful clinical examinations had revealed no evidence of disease. Except for one young mare, the horses had been used as racehorses but had not been in training for several years. All horses were fed on a traditional regime of oats, hay and water. Each horse was studied on 3 or 4 occasions with time intervals between studies varying between 1 and 19 (mean = 5) months, the total number of studies amounting to 20.

Exercise testing. The treadmill exercise test consisted of four increments in velocity as previously described (Persson, 1983; Persson *et al.*, 1983). Each horse was trotted for 2 min at treadmill velocities of 6, 7, 8 and 9 m/sec. The slope of the treadmill was kept throughout at 6.25%. Heart rate (HR) was monitored continuously by a bipolar ECG lead and recorded (Mingograph 804, Siemens-Elema, Stockholm) during the last 15 sec at each speed and also after exercise. The respiratory rate (RR) was recorded at the end of each speed and before and after exercise.

Arterial blood samples were collected anaerobically from a PVC cannula inserted in the relocated carotid artery and venous samples were drawn from a PVC cannula inserted into the jugular vein before the test. The cannulas were secured to the neck with adhesive tape and used to obtain arterial blood for gas tension determinations and venous blood for lactate determinations during the last 15 sec of each speed in the exercise test. Arterial and venous blood was also sampled immediately before (B = basal values) and 2 and 5 min after exercise. Blood volume determination was performed using the Evans blue (T-1824) dye dilution method immediately after the exercise test to ensure complete splenic emptying (Persson, 1967) and the total red cell volume (CV) was calculated as the difference between the total blood and plasma volumes. Rectal tem-

peratures were recorded before and after each test and body weight was recorded within 15 min of the completion of the test procedure.

Analytical procedures. After flushing with blood to remove the heparin/saline solution retained in the cannula, the arterial blood samples were withdrawn into heparinized disposable syringes which were capped and stored in crushed ice for less than 90 min until analyzed for arterial oxygen tension (PaO_2) (I.L. 613, Autocal pH/Blood Gas Analyzer). It was found that no changes of gas tensions occurred during the storage in crushed ice. The blood gas values were corrected for temperature changes assuming that the rise of the body temperature was proportional to the increase of the treadmill velocity.

Blood lactate concentration (LA) was determined enzymatically (Boehringer Test Combination No. 124842) on aliquots (0.1 ml) of venous blood deproteinized at the time of collection in ice-cold perchloric acid (1 ml) and stored at 4°C until analyzed within a one week period.

Derived parameters. From the regression of heart rate (HR) on treadmill velocity (V) during the work test, the velocity producing HR = 200 bpm (V_{200} , m/sec) was interpolated and the velocity producing a lactate concentration of 4 mmol/l ($V_{\text{LA}4}$, m/sec) was interpolated from the exponential regression of blood lactate concentration on speed (Persson, 1983).

As arterial oxygen tensions were closely correlated to both speed and heart rate during exercise (Fig. 2, 3; mean $r = 0.98$ for both relationships), PaO_2 at treadmill velocity of 9 m/sec ($\text{PaO}_2\text{-V9}$) and PaO_2 at a heart rate of 200 bpm ($\text{PaO}_2\text{-200}$, kPa) were also calculated from the respective linear regressions. The basal value for PaO_2 ($\text{PaO}_2\text{-B}$) was determined in arterial blood collected from the horse at rest on the treadmill before the start of the exercise test.

Statistical analyses. Standard statistical methods were used (Hald, 1952). Regression analyses were performed with a computer using the SAS program at the Uppsala University Datacentre. The results are presented as means with standard deviations (SD) unless otherwise indicated.

Results

The heart rate increased in proportion to the severity of exercise from an average of 34.5 ± 3.6 bpm at rest to 209.9 ± 6.6 at 9 m/sec (Fig. 1). After exercise HR decreased rapidly to below 100 bpm within a 5 min recovery period. The respiratory rate increased from an average of 30.2 ± 7.2 at rest to 117.2 ± 11.2 bpm at 9 m/sec, but, in contrast to HR this increment was not consistently proportional to the increase in speed in the individual horse. Immediately after exercise, i.e. when the treadmill had stopped, RR decreased significantly to 85.2 ± 15.5 ($P < 0.001$) and then increased again ($P < 0.001$) to 119.4 ± 15.3 and 125.4 ± 20.7 bpm 2 and 5 min after exercise, respectively (Fig. 1).

The PaO_2 decreased during exercise in proportion to increasing treadmill velocity from a resting level of 13.5 ± 1.3 kPa down to 9.0 ± 1.0 kPa at 9 m/sec (Fig. 2) and in proportion to increasing exercise heart rate down to 9.8 ± 1.1 kPa at HR = 200 bpm (Fig. 3). In addition to these highly significant inverse relationships between PaO_2 and both treadmill speed and exercise heart rate, the arterial oxygen tension during exercise ($\text{PaO}_2\text{-V9}$ and $\text{PaO}_2\text{-200}$) also was correlated with various circulatory and lactate parameters (Table 1, 2). Thus, the total red cell volume (CV) and to a lesser degree



FIGURE 1. Heart rates (HR beats/min, mean \pm SD, closed symbols) and respiratory rates (RR breaths/min, open symbols) before (B), during and after gradually increasing treadmill exercise at a 6.25% slope. B = baseline, V = treadmill velocity.

the post exercise hematocrit (Hct) were inversely associated with $\text{PaO}_2\text{-V}_9$ and $\text{PaO}_2\text{-200}$ and the latter was also inversely correlated with V_{200} and V_{LA4} . Consequently, predictions of normal values for exercise arterial oxygen tensions can be made in the individual horse using the regression equations presented in Table 3.

The arterial carbon dioxide tension decreased significantly ($P < 0.001$) at the start of the work test from 5.8 ± 0.3 at rest to 5.4 ± 0.4 kPa at 6 m/sec and this hypocapnia prevailed throughout the work test and was further accentuated after cessation of exercise (Fig. 2).

The blood lactate concentration rose exponentially during exercise to an average of 5.8 ± 1.9 mmol/l at 9 m/sec and prevailed above this level throughout the five minute post exercise observation period (Fig. 4). Mirroring the LA response to the work test the bicarbonate concentration decreased from an average of 27.1 ± 3.2 at rest to 19.0 ± 4.0 mmol/l immediately after exercise and the arterial blood pH concomitantly fell from 7.414 ± 0.039 to 7.295 ± 0.064 ($P < 0.001$), (Fig. 4).

Discussion

The conflicting observations regarding the effect of exercise on the arterial oxygen tension has yet to be explained. In studies on ponies trotting and galloping on a treadmill

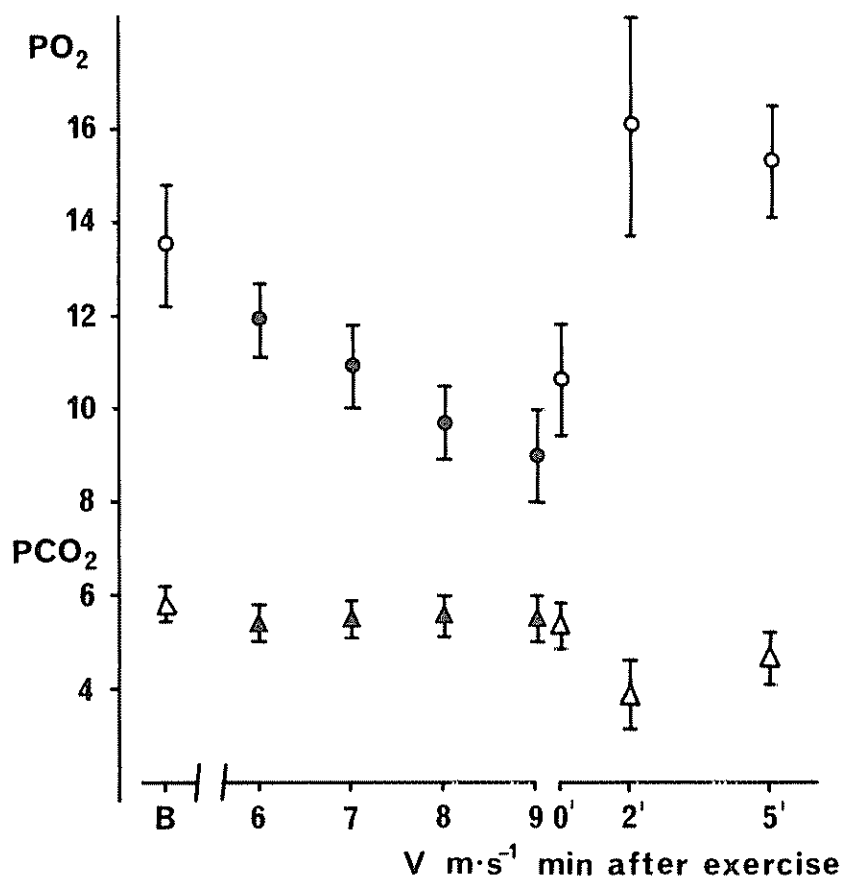


FIGURE 2. Arterial oxygen tension (PO_2 , kPa, mean \pm SD, circles) and arterial carbon dioxide tension (PCO_2 , triangles) before (open symbols), during (closed symbols), and after (open symbols) treadmill exercise as in Fig. 1. B = baseline, V = treadmill velocity.

up to a near maximal speed, no decrease of arterial PO_2 was detected (Parks and Manohar, 1984). Other studies on Standardbreds during submaximal exercise on a horizontal treadmill ($HR \leq 170$) and on a tilted treadmill (19% gradient, $HR \leq 178$ bpm) seem to corroborate this observation (Bergsten, 1974; Rose *et al.*, 1983b). On the other hand evidence of an exercise-induced arterial hypoxemia has also been presented (Littlejohn and Bowles, 1981; Bayly *et al.*, 1983; Rose *et al.*, 1983a; Thornton *et al.*, 1983) and these observations are corroborated by the results of the present investigation. The consistent and close relationship between the decrease of PaO_2 and the work load in these apparently healthy horses seems to imply that exercise-induced hypoxemia should be regarded as physiological in the horse.

One possible cause of the decrease of PaO_2 is an inadequate alveolar ventilation during exercise as there is a phase locking between limb movement and respiratory frequency (Bramble and Carrier, 1983; Hörnicke *et al.*, 1983; Garlando *et al.*, 1985). It appears, however, that the healthy horse has a very large ventilatory reserve (Gil-

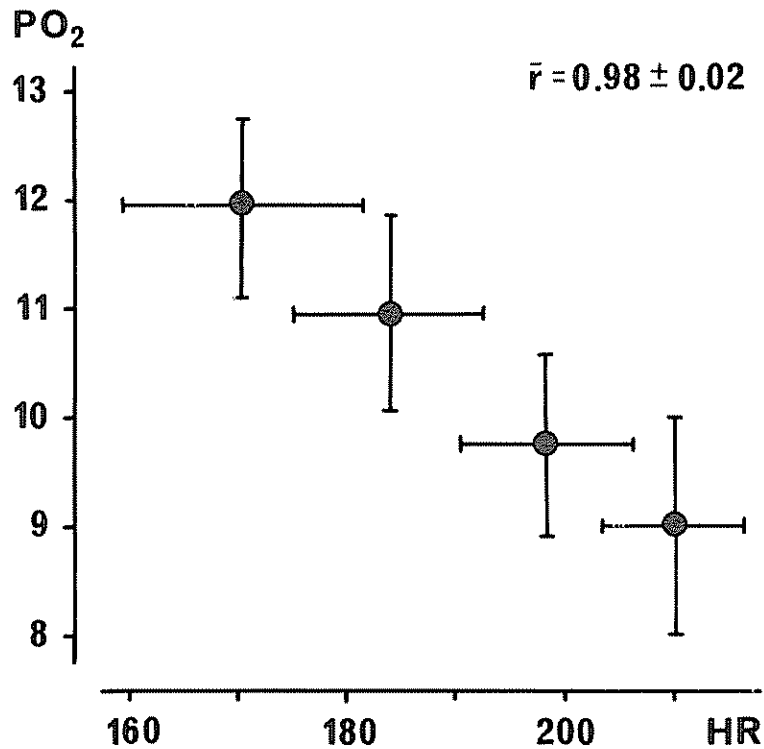


FIGURE 3 Arterial oxygen tension (PO₂, kPa, mean ± SD) in relation to heart rate (HR, beats/min, mean ± SD) during treadmill exercise at 6, 7, 8 and 9 m/sec and on a 6.25% slope \bar{r} = mean correlation coefficient ± SD of 20 determinations

lespie, 1974) implying that it is unlikely that restricted alveolar ventilation would cause arterial hypoxemia during submaximal work. The fact that the horses remained hypocapnic during the work test corroborates this view.

TABLE 1 Correlation coefficients for the relationships between arterial oxygen tensions (PaO₂-B = baseline value before exercise; PaO₂-V9 = value at 9 m/sec; PaO₂-200 = value at V₂₀₀) and age, total red cell volume (CV), total red cell volume divided by body weight (CV/Bwt), post exercise hematocrit (Hct), interpolated treadmill velocity producing a heart rate of 200 bpm (V₂₀₀), and interpolated velocity at a blood lactate concentration of 4 mmol/l (V_{LA4}).

	Age	CV	CV/Bwt	Hct	V ₂₀₀	V _{LA4}
PaO ₂ -B	n.s.	n.s.	-0.44*	n.s.	n.s.	n.s.
PaO ₂ -V9	n.s.	-0.72†	-0.73†	-0.65**	n.s.	n.s.
PaO ₂ -200	-0.54*	-0.70†	-0.66**	-0.49*	-0.74†	-0.64**

Degrees of significance for correlation coefficients: * = P < 0.05, ** = P < 0.01, † = P < 0.001, n.s. = non significant.

TABLE 2 Determinants of arterial oxygen tensions assessed with regression analysis.

Y	X	r	F _B	Mean	±SD
PaO ₂ -B				13.5	1.3
PaO ₂ -V9				9.0	1.0
	CV/Bwt	0.73	-20.58†		0.7
	Hct	0.65	-12.95**		0.8
	Hct } V _{LA4} }	0.76	-16.02† -6.43*		0.7
PaO ₂ -200				9.8	1.1
	V ₂₀₀ } CV/Bwt }	0.85	-17.34† -11.09**		0.6
	V ₂₀₀ } Hct }	0.85	-29.31† -11.08**		0.6

Y = dependent variable, X = independent variable, r = correlation coefficient, F_B = F-value for regression coefficient. Other abbreviations as in Table 1

The seemingly paradoxical inverse relationship between PaO₂-200 and aerobic power, as represented by V₂₀₀ and V_{LA4} (Persson, 1983), is chiefly a reflection of the negative relationship between PaO₂ and work intensity. Treadmill velocity resulting in HR = 200 corresponds to the work load at the onset of blood lactate accumulation which also can be defined as V_{LA4} in the horse (Persson, 1983). The fact that PaO₂-V9 did not correlate with V₂₀₀ and V_{LA4} seems to support this view as these treadmill velocities were not 9 m/sec. The advantage with assessment of PaO₂ at V₂₀₀ is that this represents a comparable, near maximal work load attainable for all horses. Comparisons of PaO₂-200 are also possible between horses regardless of the design of the stepwise test and the kind of treadmill used. Further, there was no indication that this work load was severe enough to cause a leveling off of PaO₂ at a minimum value which would be essentially equal in all horses.

The highly significant negative influence of CV (and to a lesser degree Hct) on the arterial PO₂ during exercise suggests that the exercise-induced hypoxemia results in part from mobilization of the splenic red cell reservoir during exercise (Persson, 1967). As the size of the splenic reservoir is dependent on the total red cell mass (Persson *et al.*, 1973) a relationship between CV or the post exercise Hct and the degree of exercise-induced arterial hypoxemia seems plausible. The increase of the circulating red cell

TABLE 3 Regression equations for predictions of reference values for arterial oxygen tensions during submaximal treadmill exercise at a 6.25% slope.

PaO ₂ -V9	= 15.4 - 0.093 · CV/Bwt;	SD = ±0.71
	= 19.7 - 0.192 · Hct;	SD = ±0.79
	= 23.1 - 0.188 · Hct - 0.434 · V _{LA4} ;	SD = ±0.70
PaO ₂ -200	= 23.6 - 1.15 · V ₂₀₀ - 0.064 · CV/Bwt;	SD = ±0.62
	= 29.1 - 1.40 · V ₂₀₀ - 0.141 · Hct;	SD = ±0.62

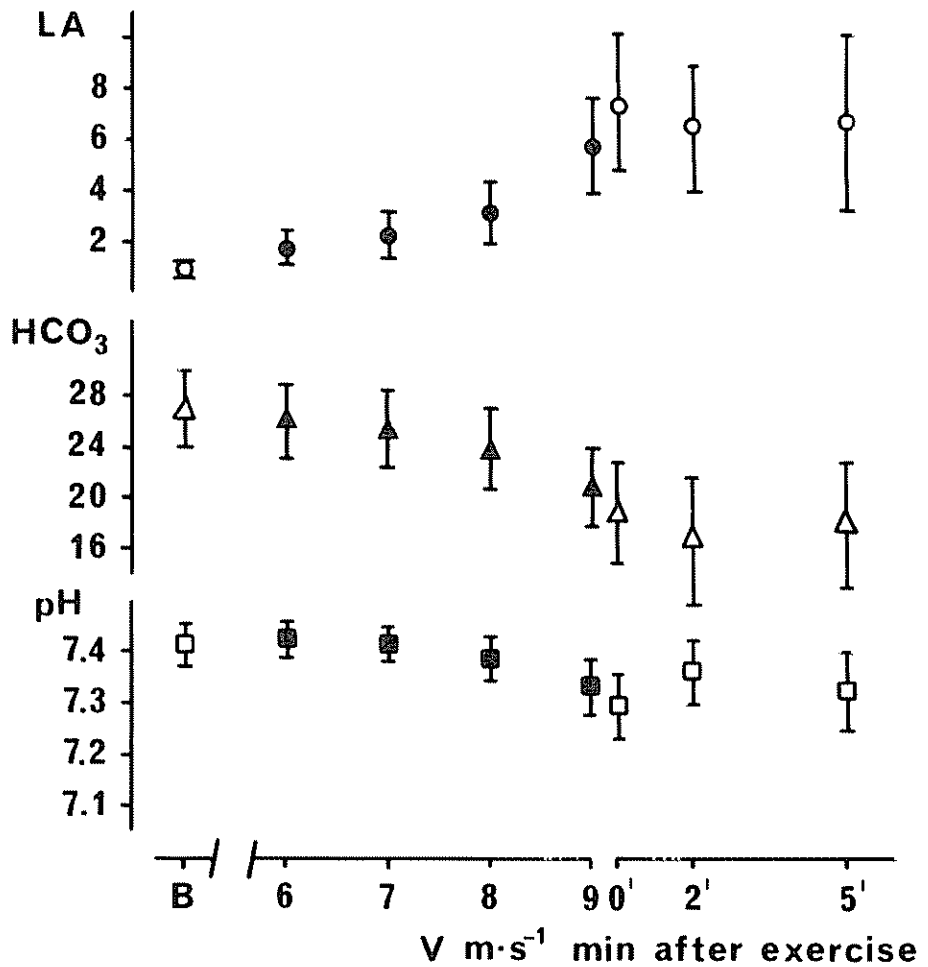


FIGURE 4. Mean values \pm SD of blood lactate (LA, mmol/l, circles), arterial bicarbonate (HCO_3 , mmol/l, triangles), and arterial blood pH (squares) before (open symbols), during (closed symbols), and after (open symbols) treadmill exercise as in Fig. 1.

volume would bring about a rise in the impedance of the pulmonary capillary bed unless met by right-to-left shunting of part of the blood flow or a shortening of the blood transit time engineered by the exercise-induced reduction in pulmonary vascular resistance. Both factors would lead to a restricted oxygen saturation of the blood due to a reduced equilibration of red cells with alveolar PO_2 . For oxygen saturation to occur, the decreased red cell transit time would need to be met by increased alveolar ventilation. Although hypocapnia during exercise indicates increased alveolar ventilation, it does not appear to be sufficient to prevent hypoxemia. It can only be speculated which one of these possible mechanisms is predominant, but, evidence is lacking for the opening of right-to-left shunts during severe exercise.

The decrease in RR immediately after exercise, followed by a later increase, probably reflects a change in the regulation of breathing. During exercise the control of breathing

frequency is engineered by either central neurogenic or peripheral reflex mechanisms initiated by muscle and joint receptors (Agostoni and D'Angelo, 1976; Iscoe and Polosa, 1976; Panda *et al.*, 1979). The neurogenic factors associated with exercise hyperpnea do not act as regulatory stimuli, but as activators; chemical stimuli are important for finer adjustment of the ventilation (Åstrand and Rodahl, 1970). Cessation of limb movement means discontinuation of the neurogenic reflex stimulation and a temporary decrease in breathing frequency ensues. Other regulatory factors then cause breathing frequency to increase. Although a number of factors are known to stimulate ventilation, the predominant causes of post exercise hyperpnea are usually considered to be metabolic acidosis and elevated arterial blood temperature (Åstrand and Rodahl, 1970). In this study blood lactate increased with a concomitant decrease of HCO_3^- and pH and the rectal temperature rose by $1.7 \pm 0.4^\circ\text{C}$ during the work test. Thus, it is likely that the post exercise increase of PaO_2 and decrease of PaCO_2 were associated with a relative hyperventilation accompanying the metabolic acidosis and hyperthermia (Krzywanek *et al.*, 1976).

In conclusion, it seems plausible to assume that the exercise-induced hypoxemia seems to be caused mainly by the increase in circulating red cell volume due to mobilization of the splenic erythrocyte reservoir during exercise. This increase in red cell volume is accompanied by a reduction of the red cell transit time through the pulmonary capillaries. This enhanced perfusion rate seems to be only partly matched by an increased alveolar ventilation. Further, predictability of the normal PaO_2 in an individual horse seems to be improved by considering its total red cell volume or post exercise hematocrit and response to submaximal exercise.

Acknowledgments

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Ventilatory Response to Exercise in Horses with Exercise-Induced Hypoxemia

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Summary

Experiments were conducted to investigate the ventilatory response to strenuous exercise in six Thoroughbred horses. The horses performed 4 standard exercise tests (SET) on a treadmill at +10%. On 3 occasions they wore respiratory masks and breathed room air. On the other occasion $F_I O_2$ was increased in the range of 0.242 to 0.252. The SET consisted of a 2 min warm-up followed by 1 min galloping at each of 8, 9, and 10 m/sec. Arterial and mixed venous blood gas tensions, alveolar oxygen tensions ($P_A O_2$), alveolar-arterial oxygen differences $[(A - a)DO_2]$, minute ventilation (\dot{V}_E), and tidal volume were determined at each speed. The $P_A O_2$ increased by about 10 torr with the onset of exercise, but did not increase further despite increasing workload, \dot{V}_E , \dot{V}_A , and $P_a CO_2$, and decreasing pH. The $P_a O_2$ decreased and $[(A - a)DO_2]$ increased at each speed, the latter under normal conditions being 41.2 ± 2.9 torr (mean \pm SD) at 10 m/sec. The $[(A - a)DO_2]$ was not different under hyperoxic conditions, although $P_A O_2$ increased by 24.1 ± 1.0 torr and hypoxemia was largely abolished. The results of the studies suggest that exercise-induced hypoxemia is partially due to an inadequate ventilatory response. The possibility that diffusion disequilibrium and local \dot{V}_A : perfusion inequalities play a role cannot be discounted, whereas the existence of major pulmonary shunts seems unlikely.

Index terms: Blood gases, oxygen consumption, hyperoxia, hypercapnia

Introduction

Although hypoxemia frequently occurs in Thoroughbred and Standardbred horses during heavy exercise (Bayly *et al.*, 1983; Thornton *et al.*, 1983; Bayly and Grant, 1986), its origin is unknown. Elucidation of the mechanism involved in this exercise-induced hypoxemia has not been forthcoming and its resolution will probably require estimates or measures of alveolar ventilation (\dot{V}_A), alveolar-pulmonary capillary oxygen differences $[(A - a)DO_2]$, studies with multiple inert gases and isotopically labeled gases,