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The Effects of Maximal Exercise on Acid–Base Balance and Arterial Blood Gas Tension in Thoroughbred Horses

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Summary

Carotid artery blood samples were sequentially collected for acid–base and blood gas measurements from five fit Thoroughbred horses during maximal exercise. Each horse galloped 1.6 km, and samples were collected via a catheter when the horse was at rest, after 0.8 km and 1.6 km of galloping, immediately upon stopping and 5 minutes after exercise. The arterial oxygen tension (PaO_2) and pH decreased progressively during exercise in all horses and four of them developed an obvious hypercapnoea. Each animal became progressively more acidaemic in the time it took to pull it up and in the succeeding 5 minutes. The horses developed a respiratory alkalosis, however, and the PaO_2 returned to a level higher than that measured at rest. It was concluded that this phenomenon possibly limits performance in racing horses. The reasons for the development of hypoxaemia and hypercapnoea are not clear, although there are several possible causes: 1. the existence of marked ventilation–perfusion inequalities due to arteriovenous shunting; 2. an increase in the blood–alveolar diffusion distance; 3. alveolar hypoventilation. Further work is needed to assess the possible role of each of these factors in affecting performance during maximal exercise.

Introduction

Little information is available concerning the response of the equine respiratory system to exercise and the role it might play in limiting performance. On the basis of prediction formulae, Gillespie (1974) concluded that ventilatory factors were unlikely to limit oxygen consumption in horses exercising maximally. It is generally believed that man's ability to exercise is not limited by respiratory considerations, and the evidence for this opinion has been well reviewed (Wasserman *et al.*, 1981). Treadmill studies with horses working at submaximal levels have reinforced Gillespie's opinion and indicated that equidae and humans may be similar as regards the response of the respiratory system to exercise (Bergsten 1974; Bayly 1979; Thomas and Fregin, 1981).

The horse appears capable of greatly increasing the oxygen-carrying capacity of its blood when exercising, due primarily to its ability to increase haematocrit greatly via splenic contraction (Persson 1967, 1968; Milne 1974). This impression has been supported by blood gas studies performed during submaximal treadmill work and by analysis of carotid arterial samples obtained immediately after maximal exercise (Milne 1974; Krzywanek *et al.*, 1976). No data are available concerning acid-base or blood gas alterations which may occur during maximal or exhaustive exercise, because it is difficult to obtain samples under such exercise conditions. One potential technique for sampling arterial and mixed venous blood from working saddle horses has been described (Littlejohn and Kruger, 1976). However, no results have been forthcoming. There is evidence from human studies to suggest that some athletes, especially those who are highly trained and pushing themselves to the limit of their capacity, may undergo arterial desaturation, and that in these situations, ventilatory factors may limit oxygen transfer and thus performance level (Rowell *et al.*, 1964; Dempsey *et al.*, 1982). The purpose of the present study was to study acid-base and blood gas changes in Thoroughbred horses galloping maximally and to compare the findings to those found in humans under equivalent conditions.

Materials and Methods

Five healthy Thoroughbred horses (four geldings and one mare) were used. All had been in training for at least three weeks prior to the study, although none was considered sufficiently fit to race. Blood samples were collected from the carotid artery via a commercially available 15 gauge, 21-inch polyvinylchloride catheter (Intrafusor, Sorenson Research Co.) and a clear plastic extension tube which was attached to the mane in the middle third of the neck by adhesive surgical tape. Prior to placement of the catheter, an area in the lower third of the left side of the neck was clipped, shaved and surgically scrubbed. The subcutaneous tissue just dorsal to the jugular vein was infused with 15 to 20 ml of a local anaesthetic solution (2% lidocaine) over a length of 3 to 4 inches. The horse was physically restrained with a twitch and the carotid artery surgically approached by incising the skin and bluntly dissecting through the fascia and muscles until the artery could be palpated. The catheter was introduced through an attached 14 gauge needle, then the soft tissue and skin were sutured and a pressure bandage applied to the surgical site. The extension tube was connected to the catheter and both were allowed to fill with blood before being flushed with a heparin-saline solution (30 000 units heparin to 250 ml normal saline) through a three-way stopcock. The tube was then taped to the mane in such a way that the proximal 5 inches and stopcock were free. The catheterization procedure was conducted at least 60 minutes prior to the exercise test.

Samples (5 ml each) for blood gas analysis were collected in heparinized 10 ml syringes when the horse was in the normal resting state, while it was galloping, and 5 minutes after it had stopped. Rectal temperature and blood lactate concentrations were measured at rest and 5 minutes post exercise. The exercise load consisted of galloping 1.6 km as fast as possible on a 0.8 km track. The stopcock was opened by the rider after 0.4 km, to allow the heparin-saline solution to be cleared spontaneously. The rider withdrew blood after 0.8 km, 1.6 km and immediately after stopping the horse. The horse took 0.1 to 0.2 km to pull up after completing the 1.6 km work-out. Each sample was collected over

approximately 5 seconds and then dropped on to the track. An assistant picked it up, expressed all air bubbles, capped it with an airtight seal and placed it on ice. After pulling up, the horse was walked until the 5 minute post-exercise sample was taken.

Acid-base and blood gas determinations were made within 10 minutes of the collection of the final sample, using an automated blood gas analyzer (Radiometer ABL2), with the appropriate corrections for body temperature and blood haemoglobin content. Samples for blood lactate determination were collected in oxalate tubes, placed in an ice bath and deproteinized with 0.6 M perchloric acid. Lactate concentration was measured by an enzymatic method (Sigma Chemical Co.).

Results

The mean values together with the standard error of the mean for pH, arterial oxygen (PaO_2) and carbon dioxide tensions (PaCO_2), bicarbonate concentration (HCO_3^-), base excess (BE), percentage saturation of haemoglobin (% sat) and lactate concentration are shown in Table 1. Although there was considerable individual variation, the observed changes were consistent for all five horses. After galloping 0.8 km, all horses had decreased PaO_2 values, and there was a marked drop in % sat. Four of the five were hypercapnoeic and acidaemic, the acidaemia apparently having both respiratory and metabolic components in those animals. All horses appeared very fatigued after 1.6 km and slowed quickly when pulled up. At 1.6 km all horses displayed a profound hypoxaemia and acidaemia. All but one were hypercapnoeic ($\text{PaCO}_2 < 48.0$ mm Hg/6.40 kPa); the exception had a PaCO_2 of 39.9 mm Hg (5.32 kPa).

In the 0.1 to 0.2 km (10–20 s) it took to pull each horse up, marked changes occurred. The acidaemia became more severe but was metabolic in origin. All animals were either normocapnoeic or hypocapnoeic, although two animals remained markedly hypoxaemic ($\text{PaO}_2 = 62.2$ mm Hg/8.29 kPa and 68.2 mm Hg/9.09 kPa, respectively). The others had a PaO_2 greater than 80 mm Hg (10.6 kPa). Five minutes after stopping, all horses were slightly more acidaemic although they exhibited marked respiratory alkalosis and a % sat greater than 90%. Lactate concentrations were all greater than 16.0 mmol/l, confirming that the horses had exercised in a maximal or near-maximal fashion.

Discussion

The progressively developing hypoxaemia (5/5), hypercapnoea (4/5) and acidaemia (5/5) which occurred in response to strenuous anaerobic work has not been previously reported. Similar findings are considered unusual in maximally exercising people, although hypoxaemia has been reported in some elite athletes (Rowell *et al.*, 1964; Dempsey *et al.*, 1982). These subjects did not however show evidence of CO_2 retention. It is not immediately clear whether hypoxaemia limits performance in any way. In our study, two horses appeared visibly more exhausted than the others. These two also exhibited the most severe hypoxaemia and acidaemia, but galloped the 1.6 km in the fastest times. Their lactate concentrations, however, were in the middle of the recorded range for the five horses. Therefore, it is possible that the degree of hypoxaemia and acidaemia which developed was a function of the effort expended, although this was not corroborated by the lactate measurements. It would appear, however, that the develop-

TABLE 1. Acid-base and blood gas data from five Thoroughbred horses performing a maximal exercise test.

	At rest	After 0.8 km	After 1.6 km	On stopping	5 minutes post exercise
pH	7.398 ± 0.011	7.207 ± 0.054	7.120 ± 0.020	7.069 ± 0.032	7.032 ± 0.034
P _a O ₂ (mm Hg)	96.2 ± 6.0	64.7 ± 2.5	61.5 ± 2.1	78.6 ± 8.4	106.3 ± 1.3
P _a O ₂ (kPa)	12.83 ± 0.80	8.63 ± 0.33	8.20 ± 0.28	10.5 ± 1.12	14.18 ± 0.18
P _a CO ₂ (mm Hg)	44.5 ± 1.6	49.8 ± 2.2	50.8 ± 4.3	40.4 ± 5.4	27.1 ± 1.0
P _a CO ₂ (kPa)	5.93 ± 0.21	6.63 ± 0.29	6.80 ± 0.57	5.38 ± 0.78	3.61 ± 0.13
HCO ₃ ⁻ (mmol/l)	26.6 ± 1.1	19.5 ± 2.9	15.4 ± 0.9	10.6 ± 0.9	6.6 ± 0.8
BE	1.8 ± 0.9	-9.7 ± 4.1	-15.4 ± 1.1	-20.9 ± 1.6	-25.7 ± 2.4
O ₂ Sat (%)	96.2 ± 0.5	82.5 ± 2.8	77.4 ± 1.6	82.7 ± 4.4	91.32 ± 0.5
Lactate (mmol/l)	0.90 ± 0.06	ND	ND	ND	18.9 ± 2.1

Data are expressed as the mean ± SEM.

HCO₃⁻ = bicarbonate; TCO₂ = total carbon dioxide; BE = base excess; O₂ sat = oxygen saturation; ND = not done.

ment of exercise-induced arterial hypoxaemia and hypercapnoea is closely related to exertion level. Previous studies of PaO₂ and PaCO₂ during submaximal exercise recorded little or no change during exercise (Bergsten 1974; Bayly 1979; Thomas and Fregin, 1981). In one case, the PaO₂ did decrease from 12.0 to 10.8 kPa when the treadmill speed was increased from 100 m/min to 154 m/min (Bayly 1979), but there was essentially little change in % sat and oxygen carrying capacity because of compensatory alterations in temperature and pH. If this and other treadmill studies had involved faster speeds, a trend towards hypoxaemia and significant lowering of % sat may have been found.

To what extent the oxygen carrying capacity of the blood was affected by the hypoxaemia is unclear. Haemoglobin concentrations were automatically determined with the blood-gas analyser but not separately recorded. It has been well established that both oxygen carrying capacity and haemoglobin levels increase significantly in response to strenuous exercise (Persson 1967, 1968) and there is no reason to assume that this did not occur in this study. It is, therefore, possible to theorize about the effect of hypoxaemia on the oxygen carrying capacity of the blood. If mean haemoglobin concentrations of 130 g/l, 96% sat, at rest, and 200 g/l, 78% sat, after 1.6 km, are arbitrarily chosen, the oxygen carrying capacity of the blood would rise from 16.7 ml O₂/100 ml blood to 20.9 ml O₂/100 ml blood, which represents a 25% increase. While this increase is significant when compared to the resting oxygen carrying capacity, it is markedly lower than that possible for the same haemoglobin concentration at 96% sat. Therefore, in theory, arterial hypoxaemia may result in a significant reduction in maximal oxygen consumption and performance.

In practice, this may not occur. In discussing the consequences of this hypoxaemia in humans, Dempsey *et al.* (1982) ascertained that unless the arteriovenous oxygen content difference is widened to a maximal level, the working skeletal muscles should still be able to function without an increase in glycolysis, due to a very high muscle blood flow, maximal perfusion of muscle capillaries and the ability of the cells to continue to extract oxygen at a very low PO₂. Venous samples were not collected in this study, so it is not known whether P \bar{v} O₂ fell below a level at which muscle metabolism might have been impaired. It may be that the function of specific muscles, such as the myocardium or those of respiration, particularly the diaphragm, may be primarily affected by the drop in arterial oxygen content, imposing a limitation on exercise in this manner.

Whether such a limitation develops may depend on the magnitude of the PaO₂ decrease. The increases in PaCO₂ and temperature and the drop in pH serve to push the oxyhaemoglobin dissociation curve to the right. This increases the chance that the lowered PaO₂ will fall on the steep section of the curve, which would result in marked reductions in % sat and arterial oxygen carrying capacity for relatively small additional drops in PaO₂. In humans, oxygen transport is substantially reduced as PaO₂ falls below 10 kPa (Dempsey *et al.*, 1982).

Hypercapnoea may further influence the level of exertion of which a horse is capable by hindering the regulation of H⁺, particularly that generated in the working muscles. The elevated PaCO₂ greatly reduces the diffusion gradient needed for rapid removal of acid radicals. Impairment of this diffusion process may result in marked lowering of muscle cell pH and in this way reduce the efficiency of muscle metabolism. The importance of maintaining a significant H⁺ gradient was seen in the rapid changes which occurred in the brief time it took for the horses to pull up. Despite the removal of any

respiratory contribution to the acidaemia, the pH fell noticeably in a brief time (10–20 s). This was probably due to a marked increase in the $[H^+]$ or metabolic component, as indicated by the decrease in $[HCO_3^-]$.

The reason for the development of hypoxaemia and hypercapnoea during maximal exercise is unclear. It would seem likely that the development of both is due to the same pulmonary physiological occurrence at this level of exertion. Several possible explanations exist: 1. arteriovenous shunts develop which result in ventilation–perfusion inequalities; 2. marked increases in the blood–alveolar diffusion distance occur, possibly because of the development of interstitial pulmonary oedema; 3. inadequate ventilation occurs. In theory, any one or any combination of these could result in the observed acid–base and blood gas changes.

It is unlikely that pulmonary arteriovenous shunts play a significant role in the development of the observed blood gas changes. Such an occurrence has been largely discounted in man because it would be virtually impossible to achieve the size of shunt needed to produce the observed arterial desaturation (Rowell, personal communication) and because the degree of hypoxaemia developing during maximal exercise can be increased or decreased by lowering or raising the O_2 concentration of the inspired air (Dempsey *et al.*, 1982). Hypoxaemia can also be diminished or abolished by stimulating a greater ventilatory response by replacing nitrogen in the air with helium (Dempsey *et al.*, 1982). Therefore it would appear most likely that the observed hypoxaemia and hypercapnoea are due to diffusion limitations and/or ventilatory inadequacies. In the event that alveolar PO_2 is reduced, the alveolar–pulmonary capillary diffusion gradient would also be decreased. This factor, when combined with the very low % sat of the mixed venous blood being presented in the capillary and its extremely rapid flow rate, may result in inadequate time for equilibration of red cells with alveolar PO_2 and incomplete oxygen saturation. If this is the case, the cause of the observed blood gas changes would be most directly due to inadequate alveolar ventilation. This concept is supported by the observation in humans that voluntary hyperventilation during short-term maximal exercise will increase alveolar PO_2 and prevent or reverse the hypoxaemia (Dempsey *et al.*, 1982). Several possible causes of ventilation restrictions exist: 1. the ventilatory requirements of the horse may exceed peak airflow velocities; 2. physical constraints may arise in the mechanics of lung and chest wall movement; 3. high dead space ventilation may occur at rapid respiratory rates, which could also explain the occurrence of the hypercapnoea. The role of any of these factors in the genesis of relative alveolar hypoventilation is strictly hypothetical at present.

It is obvious from the range of values which existed among the small number of horses sampled in this study that considerable variation must be present among horses with respect to the acid–base and blood gas alterations which occur during maximal exercise. The observed trends, however, were consistent, although it remains to be determined whether most or all horses will become hypoxaemic and hypercapnoeic under similar conditions. Although the consequences of exercise-induced arterial hypoxaemia with regard to performance are not clear, and the possible causes require evaluation, it seems likely that maximizing pulmonary gas exchange during maximal exercise could result in improved performance. It would be interesting to investigate the effect of a long warm-up period and the possible induction of hyperventilation on changes in PaO_2 and $PaCO_2$ occurring during maximal exercise.

The described technique for blood collection is not without problems or dangers. It requires a skilled rider who is able to collect the samples with one hand while holding the reins in the other. Opening the three-way stopcock after 0.4 km and allowing the tube to be cleared spontaneously by the pressure of the arterial blood allows undiluted samples to be obtained. Leaving the stopcock open from the 0.4 km mark until stopping avoids the need to flush the catheter, reduces the hazard of introducing an air embolus while galloping, and minimizes potential blood clotting in the catheter and tube. Although the catheter and extension tube are of relatively large gauge, the three-way stopcock has a small port and this restricts the rate of blood loss during exercise to a regular drip of an inconsequential total volume. An apparatus similar to that previously described by Littlejohn and Kruger (1976) was tried and discarded in preliminary trials, the main problem being that the rate of syringe withdrawal was inconsistent and generally too slow for our purposes. Using the technique described here, we were able to draw samples from all horses in a consistent manner, which meant that the data from individual horses could be pooled.

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Biochemical Changes in Thoroughbred Racehorses Following Submaximal and Maximal Exercise

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Summary

Sixteen plasma biochemical variables and blood lactate were measured in Thoroughbred racehorses prior to and during the first hour after exercise. When horses were exercised at a canter, changes occurred in a number of blood constituents. With the exception of creatinine, phosphorus and potassium, all variables returned to their pre-exercise values within an hour. Phosphorus and potassium, however, were below their pre-exercise value after an hour. Exercise at a gallop resulted in changes in blood lactate and in all plasma constituents assayed, except in chloride and conjugated bilirubin. Of the constituents altered, only sodium, potassium, cholesterol and alkaline phosphatase returned to their respective pre-exercise values after one hour. At this time, bicarbonate, phosphorus and total bilirubin were below their resting values, whereas glucose, urea, creatinine, urate, calcium, lactate dehydrogenase, creatine kinase and blood lactate were above their pre-exercise values.

Introduction

The purpose of the present study was to compare and contrast the changes in blood constituents in mature Thoroughbred racehorses. Changes in blood constituents with increasing fitness and maturity in Thoroughbred horses have been documented (Judson *et al.*, 1983). Therefore, evaluation of changes due to different exercise levels is best achieved with horses at comparable levels of fitness. By using horses in racing condition we minimized the changes which have been reported with the progression of training programmes.

Materials and Methods

Studies were carried out on 16 clinically healthy, mature Thoroughbred horses in racing condition. The horses were housed in loose-boxes and maintained on a diet consisting of wheaten or oaten chaff, oats, bran and lucerne hay.