CARDIOVASCULAR AND RESPIRATORY RESPONSES IN THOROUGHBRED HORSES DURING TREADMILL EXERCISE

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SUMMARY

Six detrained Thoroughbred horses performed incremental treadmill tests. Maximal oxygen uptake (\(\dot{V}_{O_2\text{max}}\)) was measured during exercise by analysis of expired gas. The relationships between oxygen consumption (\(\dot{V}_{O_2}\)) and work rate, heart rate (HR), cardiac output (\(Q\)), stroke volume (SV) and arteriovenous oxygen content difference [\(\Delta(C(a\!-\!v)O_2)\)] were examined during submaximal and maximal exercise. The relative contributions of blood flow and extraction of oxygen from muscle capillaries were assessed during exercise at \(\dot{V}_{O_2\text{max}}\). Mean \(\dot{V}_{O_2\text{max}}\) was 129.7 ± 2.9 (mean ± S.E.M.) ml kg\(^{-1}\) min\(^{-1}\), which occurred at a mean speed of 8.0 m s\(^{-1}\), with the treadmill set at a slope of 10%. At \(\dot{V}_{O_2\text{max}}\) the mean HR was 222 ± 7 beats min\(^{-1}\). Maximal \(Q\) was 534 ± 22 ml kg\(^{-1}\) min\(^{-1}\) and mean SV was 2.4 ± 0.1 ml kg\(^{-1}\). Mean \(\Delta(C(a\!-\!v)O_2)\) was 24.5 ± 1.2 ml 100 ml\(^{-1}\) blood. Linear relationships were found between \(V_{O_2}\) and work rate, HR, \(Q\) and \(\Delta(C(a\!-\!v)O_2)\). Stroke volume did not increase significantly during exercise. Increasing metabolic rate during exercise was associated with linear increases in arterial haemoglobin concentration and oxygen content, and decreases in arterial oxygen partial pressures and haemoglobin saturation. We conclude that the relatively high \(\dot{V}_{O_2\text{max}}\) in the detrained Thoroughbred racehorse is dependent on the generation of a large \(\Delta(C(a\!-\!v)O_2)\), despite development of hypoxaemia and haemoglobin desaturation, during strenuous exercise.

INTRODUCTION

The large increases in oxygen consumption (\(\dot{V}_{O_2}\)) during transition from rest to exercise in the horse and the linearity of the relationship between \(V_{O_2}\) and submaximal work effort have been well established (Hall & Brody, 1934; Orr et al. 1975; Thomas, Fregin, Gerber & Ailes, 1980; Waugh et al. 1980; Seeherman, Taylor, Maloiy & Armstrong, 1981; Thomas & Fregin, 1981; Thornton et al. 1983; Persson, 1983). However, the responses of the equine respiratory system during
maximal exercise have been measured infrequently. The pioneering work of Karlsen & Nadaljak (1964) is the only report of ventilation, oxygen usage and associated variables in racehorses during maximal exercise. They examined the respiratory responses of Standardbred trotters during an incremental exercise test on a racetrack.

Extrapolation to maximal heart rates of $V_{O_2}$ during submaximal exercise has been used to estimate maximal oxygen uptake ($V_{O_2max}$) in the horse (Gillespie, 1975; Meixner, Hornieke & Ehrlein, 1984; Foreman, 1984). These reports suggest that the horse has a relatively high maximum aerobic power. There have been no reports of directly measured maximum ventilation or $V_{O_2max}$ in Thoroughbred horses.

In this study, the relationship between $V_{O_2}$ and exercise intensity was examined in six Thoroughbred racehorses during submaximal and maximal exercise. In particular, the means by which oxygen transport mechanisms adjust to meet the demands of a wide range of metabolic rates during submaximal and maximal exercise were investigated. Changes in heart rate (HR), stroke volume (SV), cardiac output (Q) and arteriovenous oxygen content difference ($C(a-v)O_2$) were examined as $V_{O_2}$ increased during exercise.

**MATERIALS AND METHODS**

Six detrained Thoroughbred geldings, 4-8 years of age with body masses from 446 to 520 kg, were used. Each horse was accustomed to exercise on a high-speed treadmill (Beltalong, Euroa, Victoria, Australia) at the trot, canter and gallop, wearing a face mask. However, the horses had received no regular physical conditioning during the 4 months prior to the exercise test.

A standardized treadmill exercise test was used. After a 2-min period of exercise at 3 m s$^{-1}$, the treadmill speed was increased by 1 m s$^{-1}$ every 60 s. Treadmill angle of inclination was constant at 6° (+10% slope). The test was terminated when it was obvious that the horse could not maintain power output, or when two stages of the exercise test were completed without an increase in HR. A 120-W fan was placed in front of the treadmill to direct air onto the horses during exercise. The treadmill was housed under cover but not isolated from environmental temperature or humidity.

A catheter, for arterial blood sampling, was placed in the left common carotid artery, which had been surgically relocated to a subcutaneous position. The pulmonary artery was catheterized using a Swan–Ganz balloon-tipped catheter, placed via the left jugular vein. Blood samples were collected anaerobically into heparinized syringes, simultaneously from the carotid and pulmonary arteries over the last 15 s of each exercise stage. The blood samples were used for measurement of blood gases, haemoglobin concentration and acid–base values using a blood gas analyser (ABL2, Radiometer, Copenhagen). Before capping and storage of the samples in an ice bath, care was taken to ensure that there was no air in the syringes. The samples were analysed within 1 h of collection. Blood samples taken at the conclusion of the first stage of an exercise test were processed at the resting rectal temperature. Subsequent blood samples were processed at the rectal temperature immediately post-exercise.
The $Q$ was calculated from rates of $\dot{V}_{O_2}$ and $C(a-v)O_2$ according to the Fick principle.

The gas collection mask contained two inspiratory and two expiratory unidirectional rubber valves, the areas of which were 115 and 125 cm$^2$, respectively. Total mask deadspace, estimated from water displacement, was 1-5 l. The expiratory gas was channelled into 9 cm diameter flexible tubing, 2-3 m in length, and from there into a mixing chamber constructed of 10 mm thick Perspex. The design of the chamber, which had a volume of 57·4 l, was similar to that reported for human use (Jones & Campbell, 1982). The expired gas then passed through 0-9 m of the flexible tubing to the inlet of a flowmeter (GD 101, Fluid Inventor, Sweden). Total volume of the tubing and mixing chamber was 77·8 l. The transit times for passage of expired gas from mask outlet to flowmeter ranged from 4 to 7 s during exercise. As the gas entered the flowmeter, its temperature was measured with an industrial thermometer. Sample tubes were placed downstream from the flowmeter for connection to a paramagnetic oxygen analyser (Servomex 570A, Sybron-Taylor, UK) and infrared carbon dioxide analyser (CD 102, Datex, Finland). An Aridus sampling tube (Gambro Engstom AB, Sweden) was used to limit the entry of water vapour into the oxygen analyser. The carbon dioxide analyser was calibrated daily with room air and two precision gas mixtures containing carbon dioxide concentrations between 2 and 6%. The oxygen analyser was calibrated daily, using a mixture of carbon dioxide and nitrogen, and room air.

The HR was recorded using a radiotelemetry electrocardiograph (Evans & Rose, 1986). Respiratory frequency was measured by counting the audible expirations over a 15-s period. Mixed expired gases were collected and the gas flow rate, temperature, %$O_2$ and %$CO_2$ recorded over the same period. Standard formulae were used for calculation of the respiratory variables (Jones, 1980; Jones & Campbell, 1982).

Regression and correlation analyses were used to examine relationships between variables during exercise. Only results from work efforts up to and including those at which $\dot{V}_{O_2,max}$ was achieved were used to examine the relationship between $\dot{V}_{O_2}$ and other variables. The $\dot{V}_{O_2,max}$ was identified as the point at which a levelling off in $\dot{V}_{O_2}$ occurred despite an increase in exercise intensity. Significance of the linear regression slope was determined by an $F$-test, and the correlation coefficient by a $t$-test. All mean values are expressed as ± standard error (s.e.m.).

The work rate was calculated as follows:

\[
\text{Work rate (W)} = \text{speed (m min}^{-1}\text{)} \times \sin \text{treadmill angle} \times 6·12^{-1}.
\]

RESULTS

Mean rectal temperature immediately prior to exercise tests was 37·5 ± 0·17°C. Immediately after the completion of the exercise tests, mean rectal temperature was 39·6 ± 0·14°C. Mean environmental temperature on exercise test days was 16·5 ± 1·30°C and mean barometric pressure was 761·8 ± 1·97 mmHg (1 mmHg = 133·3 Pa).
Mean work rate increased from 1481 ± 95.5 W at the slowest speeds to 3877 ± 188.0 W at work rates which resulted in $\dot{V}_{O_2\text{max}}$. This 2.6-fold increase in work rate was associated with a 2.2-fold increase in mean rate of oxygen consumption.

Mean $V_{O_2\text{max}}$ was 129.7 ml kg$^{-1}$ min$^{-1}$ (range 120.8–142.2 ml kg$^{-1}$ min$^{-1}$). The $\dot{V}_{O_2\text{max}}$ was reached at treadmill speeds ranging from 6.4 to 9.2 ms$^{-1}$ (mean 8.0 ± 0.42 ms$^{-1}$), and at work rates ranging from 3016 to 4260 W. The mean metabolic rate at the slowest treadmill speed was approximately 45% of $\dot{V}_{O_2\text{max}}$.

Oxygen consumption during exercise was highly correlated with treadmill work rate (Fig. 1). The linear relationship between $\dot{V}_{O_2}$ and treadmill velocity was also very close; $\dot{V}_{O_2} = 23.08 + 13.36v$, $P < 0.01$ ($r = 0.948$, $P < 0.001$), where $v$ is the treadmill velocity in m s$^{-1}$.

The 2.2-fold increase in mean $\dot{V}_{O_2}$ was associated with a 1.4-fold increase in $\dot{Q}$ (from 388 to 534 ml kg$^{-1}$ min$^{-1}$) and a 1.6-fold increase in $\dot{C}(a-v)O_2$ (from 15.5 to 24.5 ml kg$^{-1}$ min$^{-1}$). Mean HR increased from 161 to 222 beats min$^{-1}$ at $\dot{V}_{O_2\text{max}}$, a 1.4-fold increase. The mean values (±S.D.) for HR, $\dot{Q}$, SV and $\dot{C}(a-v)O_2$ at $\dot{V}_{O_2\text{max}}$ are presented in Table 1. The $\dot{V}_{O_2\text{max}}$ occurred at maximal HR in five horses. In one horse $\dot{V}_{O_2\text{max}}$ was attained at a submaximal heart rate of 188 beats min$^{-1}$. However, there was a close relationship between percentage of maximal oxygen consumption and percentage of maximal heart rate. In 26 observations at exercise intensities up to and including $\dot{V}_{O_2\text{max}}$, the correlation coefficient was 0.829 ($P < 0.001$).

HR, $\dot{Q}$ and $\dot{C}(a-v)O_2$ all increased linearly with increases in $\dot{V}_{O_2}$ (Figs 2–4, respectively). However, there was no significant dependency of SV on metabolic rate during exercise to $\dot{V}_{O_2\text{max}}$. Mean SV at $\dot{V}_{O_2\text{max}}$ or less than $\dot{V}_{O_2\text{max}}$ was

![Figure 1](image-url)
2.45 ± 0.09 ml kg⁻¹. There was considerable variation of stroke volume among horses. At ˙V̇O₂max, mean SV was 2.43 ± 0.15 ml kg⁻¹, ranging from 1.95 to 2.97 ml kg⁻¹.

Strenuous exercise resulted in arterial hypoxaemia and haemoglobin desaturation linearly related to metabolic rate (Figs 5, 6). However, despite hypoxaemia and haemoglobin desaturation, there was no significant linear relationship between ˙V̇O₂ and arterial oxygen content (CaO₂). Mean CaO₂ during exercise at work rates of ˙V̇O₂max or less than ˙V̇O₂max was 28.9 ± 0.36 ml 100 ml⁻¹. The constancy of CaO₂ was

|                      | Horse* | Pony†  | Man‡  | Rat§  | Dog¶  | Possum||
|----------------------|--------|--------|-------|-------|-------|-------|
| V̇O₂max              | 130 ± 7| 122    | 43    | 83    | 87    | 64    |
| HR                   | 222 ± 18| 225    | 197   | 595   | 300   | 306   |
| Q                    | 534 ± 54| 621    | 268   | 547   | 558   | 618   |
| SV                   | 2.4 ± 0.4| 2.9    | 1.4   | 0.9   | 1.9   | 2.0   |
| C(α–v)O₂             | 24.5 ± 2.9| 16.2   | 15.5  | 15.6  | 10.5  |

* This study; † Parks & Manohar (1983); ‡ Saltin et al. (1968); § Gleeson & Baldwin (1981); ¶ Horstman et al. (1974); || Baudinette, Seymour & Orbach (1978).

Values are mean ± S.D.

HR = 106.4 + 0.934 ˙V̇O₂ (P < 0.01)

r = 0.874 (P < 0.001)

Fig. 2. Heart rate and oxygen consumption (˙V̇O₂) in six Thoroughbred horses during an incremental exercise test. 95% confidence intervals for mean values.
Fig. 3. Cardiac output ($\dot{Q}$) and oxygen consumption ($\dot{V}_{O_2}$) in six Thoroughbred horses during an incremental exercise test. 95% confidence intervals for mean values.

$$\dot{Q} = 261.1 + 2.254 \dot{V}_{O_2} \quad (P < 0.01)$$
$$r = 0.658 \quad (P < 0.001)$$

Fig. 4. Arteriovenous oxygen content difference [$C(a-v)_{O_2}$] and oxygen consumption ($\dot{V}_{O_2}$) in six Thoroughbred horses during an incremental exercise test. 95% confidence intervals for mean values.

$$C(a-v)_{O_2} = 8.395 + 0.122 \dot{V}_{O_2} \quad (P < 0.01)$$
$$r = 0.769 \quad (P < 0.001)$$
Exercise in horses

\[ \text{Pa}_2O_2 = 111.5 - 0.280\dot{V}_O_2 \quad (P < 0.01) \]
\[ r = -0.679 \quad (P < 0.001) \]

Fig. 5. Arterial oxygen tension (\(\text{Pa}_2O_2\)) and oxygen consumption (\(\dot{V}_O_2\)) in six Thoroughbred horses during an incremental exercise test. 95% confidence intervals for mean values.

\[ \text{Sa}_2O_2 = 104.3 - 0.142\dot{V}_O_2 \quad (P < 0.01) \]
\[ r = -0.759 \quad (P < 0.001) \]

Fig. 6. Arterial haemoglobin oxygen saturation (\(\text{Sa}_2O_2\)) and oxygen consumption (\(\dot{V}_O_2\)) in six Thoroughbred horses during an incremental exercise test. 95% confidence intervals for mean values.
concomitant with a linear increase in arterial haemoglobin concentration during exercise (Fig. 7). At \( \dot{V}_{O_2}^{\text{max}} \), mean arterial haemoglobin concentration was 239.0 ± 5.5 g l\(^{-1} \), an 11% increase over the value during exercise at the slowest treadmill speed.

Mixed venous oxygen content (\( C_{V\dot{O}_2} \)) fell linearly with increasing metabolic rate. Mean \( C_{V\dot{O}_2} \) decreased from 13.8 ± 0.6 ml 100 ml\(^{-1} \) at the lowest work rate to 2.8 ± 1.0 ml 100 ml\(^{-1} \) at \( \dot{V}_{O_2}^{\text{max}} \). The associated falls in mixed venous oxygen partial pressures (\( P_{V\dot{O}_2} \)) and haemoglobin saturation (\( S_{V\dot{O}_2} \)) were both highly significant. At \( \dot{V}_{O_2}^{\text{max}} \), mean \( P_{V\dot{O}_2} \) was 19.2 ± 0.95 mm Hg, and mean \( S_{V\dot{O}_2} \) was 87 ± 31.5%.

Values for blood pH and standard bicarbonate were not obtained for one horse. The results of these acid–base values during exercise in arterial and mixed venous blood of five horses are presented in Table 2. The fall in pH during exercise to \( \dot{V}_{O_2}^{\text{max}} \) was linear: \( \text{pH} = 7.4703 - 0.0018 \dot{V}_{O_2} \text{ml kg}^{-1} \text{min}^{-1} \), \( P < 0.01 \).

**DISCUSSION**

Combined data for \( \dot{V}_{O_2}^{\text{max}} \) in 45 wild and domestic species have demonstrated a close relationship between body mass and \( \dot{V}_{O_2}^{\text{max}} \) (Taylor et al. 1981). This relationship was calculated as:

\[
\dot{V}_{O_2}^{\text{max}} = 1.67M^{0.845},
\]
Table 2. pH and standard bicarbonate (SBC, mmol·l⁻¹) in arterial and mixed venous blood during a rapid incremental treadmill exercise test to $\dot{V}_{O_2}^{\text{max}}$ in five Thoroughbred horses

<table>
<thead>
<tr>
<th>Work rate (W)</th>
<th>pH</th>
<th>SBC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Arterial</td>
<td>Mixed venous</td>
</tr>
<tr>
<td>1439 ± 51 ($N = 4$)</td>
<td>7.377 ± 0.015</td>
<td>7.317 ± 0.014</td>
</tr>
<tr>
<td>1913 ± 29</td>
<td>7.335 ± 0.021</td>
<td>7.232 ± 0.035</td>
</tr>
<tr>
<td>2640 ± 155</td>
<td>7.295 ± 0.019</td>
<td>7.233 ± 0.017</td>
</tr>
<tr>
<td>3311 ± 180</td>
<td>7.258 ± 0.020</td>
<td>7.053 ± 0.083</td>
</tr>
<tr>
<td>4087 ± 109 ($N = 3$)</td>
<td>7.193 ± 0.056</td>
<td>6.701 ± 0.216</td>
</tr>
</tbody>
</table>

Values are mean ± S.E.M.

where $\dot{V}_{O_2}^{\text{max}}$ is in ml·s⁻¹ and Mb is body mass in kg. This equation predicts a $\dot{V}_{O_2}^{\text{max}}$ of 291 ml·s⁻¹ in 450-kg horses. The result in this study was approximately 1000 ml·s⁻¹, far greater than predicted by the above equation. The Thoroughbred horse, therefore, has a much higher $\dot{V}_{O_2}^{\text{max}}$ relative to body mass than that found in most other mammals, despite the finding that the resting metabolic rate in the horse is similar to that in other mammals (Bartels, 1964).

Assuming a resting mean $\dot{V}_{O_2}$ of 3 ml·kg⁻¹·min⁻¹ based on results from several reports (Orr et al. 1975; Thomas & Fregin, 1981; Meixner et al. 1984; Thornton et al. 1983), in the present study mean $\dot{V}_{O_2}$ at $\dot{V}_{O_2}^{\text{max}}$ increased by over 40 times the resting metabolic rate. This is a much greater increase than found in other mammals (Taylor et al. 1981). The remarkable increase in aerobic energy release in the horse is dependent on the large increase in the rate of oxygen delivery to the exercising skeletal muscles. The elevation in $\dot{V}_{O_2}$ is largely due to tachycardia, as SV during submaximal exercise in the horse is little different from SV at rest (Bergsten, 1974; Persson & Bergsten, 1975; Bayly, Gabel & Barr, 1983a; Parks & Manohar, 1983; Thornton et al. 1983). There was no significant change in SV with increasing $\dot{V}_{O_2}$ from approximately 45% $\dot{V}_{O_2}^{\text{max}}$ up to 100% $\dot{V}_{O_2}^{\text{max}}$ in this study. This result confirms those in Standardbred horses during incremental treadmill exercise test to near maximal heart rates (Thornton et al. 1983). Thomas & Fregin (1981) reported that SV increased with treadmill speed during submaximal exercise. However, the increase in SV in that study was minor when compared with the increase in $\dot{Q}$.

In our study, $\dot{V}_{O_2}$ was determined using a mask with unidirectional valves. Such a system may impose some limitations to ventilation at higher exercise intensities (Bisgard et al. 1978). Thus, the $\dot{V}_{O_2}^{\text{max}}$ may have been underestimated using the present system. However, during submaximal exercise, Persson (1983) found no differences in $\dot{V}_{O_2}$ when masks with different valve areas were used.

It has been suggested that the very high heart rates of maximal exercise in the horse could be associated with a fall in SV (Sporri, 1962; von Engelhardt, 1977).
There was no evidence of declining SV at high rates in this study. It is possible, therefore, that SV during maximal exercise in the Thoroughbred horse may be predicted from results of submaximal exercise tests. It would also be of interest to investigate the predictability of SV during maximal exercise in racehorses from assessments of SV or heart size in the resting horse. Such studies could help determine if there was a physiological basis for assessment of performance potential using indices of heart size in the resting horse (Steel & Stewart, 1972). Increases in the C(a−v)O₂ contributed greatly to increases in \( \dot{V}_\text{O}_2 \) during exercise. The maximal C(a−v)O₂ of 24.5 ml 100 ml⁻¹ during maximal exercise in this study is at least 50% greater than that found in man, rats, dogs and brush-tail possums (Table 1). The very high \( \dot{V}_\text{O}_{\text{max}} \) in the detrained Thoroughbred horse in comparison with other mammals is therefore dependent on generation of a large C(a−v)O₂. The \( \dot{Q} \) at \( \dot{V}_\text{O}_{\text{max}} \) in the horses in this study was greater than in man, but is similar to or less than maximal cardiac output in rats, dogs and possums. The most likely explanation for the high maximal C(a−v)O₂ in Thoroughbred horses is the increase in arterial haemoglobin concentration and oxygen content, coupled with a capacity for extraction of a large proportion of the delivered oxygen by the working skeletal muscles. The arterial oxygen content during exercise in this study was considerably greater than reported for resting horses (Milne, 1974; Bayly et al. 1983a; Thornton et al. 1983). Splenic contraction during exercise and the resultant large increase in blood haemoglobin concentration (Persson, 1967) are therefore central to the superior ability of the Thoroughbred horse to use oxygen during maximal exercise.

Use of the direct Fick technique for measurement of \( \dot{Q} \) during non steady-state exercise is subject to error from several sources (Guyton, Jones & Coleman, 1973). We have found that at the onset of exercise at work rates between 50 and 100% of \( \dot{V}_\text{O}_{\text{max}} \) in the racehorse, steady-state \( \dot{V}_\text{O}_2 \) is reached in less than 60s (D. L. Evans & R. J. Rose, unpublished data). Some error could be due to asynchrony between collection of arterial and mixed venous blood samples, and between blood sampling and analysis of mixed expired gas. During strenuous exercise with accumulation of lactate and hydrogen ions in the blood, the position of the oxyhaemoglobin dissociation curve is likely to be inconstant. There have been no reports of the temporal nature of changes in \( \text{PaCO}_2 \) and \( \text{PaO}_2 \) during work to work transitions in the racehorse. Inaccuracies may also arise from phasic variations in the composition of arterial blood with respiration (Kelman, 1977).

The results of this study confirm that hypoxaemia occurs in Thoroughbred horses during strenuous exercise (Bayly, Grant, Breeze & Kramer, 1983b). However, the degree of hypoxaemia may have been overestimated in this study, due to rectal temperatures being used for correction of arterial blood gases. Sexton, Erickson, DeBowes & Sigler (1985) have demonstrated that, during exercise, arterial blood temperatures are consistently higher than rectal temperatures. In the detrained Thoroughbred horse, hypoxaemia and haemoglobin desaturation are unlikely to limit performance at work rates at or below maximal oxygen uptake, as arterial oxygen content is maintained at the high concentrations which are found during submaximal exercise. The relatively high maximal aerobic power of the detrained
Thoroughbred horse is therefore related to a high arterial haemoglobin concentration, oxygen content and \( C(a-v)O_2 \) difference during exercise at \( VO_{2\text{max}} \). Further investigations are necessary to determine if \( VO_{2\text{max}} \) can be increased in the horse by physical training, and by which mechanisms oxygen transport might be so increased.

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