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The Effect of Cessation of Training on Cardiorespiratory Variables during Exercise

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ABSTRACT Six Thoroughbred horses were exercised at increasing speeds (in steps of 1 m s^{-1} after 1 min at each speed) from 6 m s^{-1} up to 13 m s^{-1} at 3° when fully fit, and to 11 m s^{-1} at 3° after 15 weeks out of training. There were no significant effects of detraining on maximum rate of O_2 consumption ($\dot{V}_{O2max}$), any of the measured cardiorespiratory variables, blood gases or pH at 11 m s^{-1}. There was, however, a significant doubling of blood lactate concentration. Thus, although the cardiorespiratory responses to high intensity exercise are not affected by 15 weeks of relative inactivity, there may be a reduction of the animals' endurance as they become more reliant on anaerobic metabolism.

Key words Horses; detraining; oxygen consumption; lactate.

INTRODUCTION

Lameness is the most common cause of days lost to injury in racehorses. The treatment often results in an enforced period of inactivity. Detailed studies have been performed on the effect of periods of inactivity (bed rest) and of cessation of training (detraining) on the cardiovascular and respiratory responses to exercise in man.[3,13] The common response is one of reduced maximum oxygen uptake ($\dot{V}_{O2max}$) accompanied by a reduction in cardiac stroke volume, and hence in cardiac output. A decline in arterial minus mixed venous oxygen content may also occur.

In the few studies that have been performed on horses, no significant effect of 5 weeks to 6 months of detraining was determined.[1,5,5] However, it appears that $\dot{V}_{O2max}$ was not reached in these studies. It was decided, therefore, to exercise thoroughbred racehorses up to $\dot{V}_{O2max}$ using an exercise protocol previously described[6,7] when they were fully fit and after 15 weeks of relative inactivity.

MATERIALS AND METHODS

Six Thoroughbred horses were used, ranging in age from 3 to 13 years with mean body masses that varied from 446 ± 13 to 457 ± 11 kg over the experimental period. They were stabled under the same conditions and fed hunter cubes (Dodson & Horrell Ltd) and hay, with water provided ad lib. A carotid artery was raised to a subcutaneous position in each horse in order to aid its subsequent catheterisation.

All horses had been in training for a period of 4 to 6 months. A 6 to 8 week period of slow long work periods of walking and trotting was followed by 2 days a week of work that included cantering at progressively increasing speed plus 4 slow work days. The horses then underwent galloping exercise for 2 days a week as well as 4 days per week of slow work for at least 3 weeks. Following this, and prior to the horses going into the period of relative inactivity, they were involved for at least one month in high speed exercise protocol on a treadmill. All the

horses were considered to be racing fit prior to the period of relative inactivity. Studies were performed on a treadmill (Säto, Sweden) housed in an air-conditioned building maintained at a temperature of 20°C and a humidity of 60%. Whilst on the treadmill, the horse wore a harness which was clipped onto a strap attached to the frame surrounding the treadmill. One air fan, placed in front of the horse, was used during exercise tests, to assist heat loss.

The exercise tests were performed on the treadmill set at an incline of 3°. The horses trotted at approximately 3.6 m s^{-1} for 15 min before being taken to a canter at 6 m s^{-1}. The speed was then raised in steps of 1 m s^{-1} after 1 min at each speed with no period of recovery in between, and attainment of $\dot{V}_{O_2max}$ was defined as a levelling of $\dot{V}_{O_2max}$ despite an increase in speed.[6,7] When fully fit, the horses were taken up to 13 m s^{-1}. Following the exercise test the animals walked at approximately 1.6 m s^{-1} for 30 min. The first test was performed when the horses were fully fit, and 3 subsequent tests were performed at 5 week intervals. Following the first test the horses were not exercised at all except for a 20 min walk each morning, when they were led rather than ridden. Only data from the first and fourth tests (i.e. fully fit and 15 weeks out of training) are presented in this report.

The horses were fitted with a lightweight, fibreglass mask which housed two flow tubes, one for each nostril, leaving the mouth free.[18] Velocity of the airflow in the tubes was detected by phase shifts in beams of ultrasound transmitted alternately in one and then the other direction.[17] Respiratory gas concentrations were monitored by a mass spectrometer (Airspec Ltd, MGA 2000) connected to one of the flow tubes. Respiratory airflow through both tubes and respiratory O_2 and CO_2 concentrations were recorded on a Gould 6 channel recorder with rectilinear coordinates. During analysis of these data, a period representing 5 to 8 respiratory cycles was selected from recordings at the end of each velocity. The traces of respiratory air flow and respiratory gas concentrations were digitized, using an Acorn Archimedes microcomputer and a GTCO digitizing pad (Digipad 5), thus enabling the following to be calculated: tidal volume (V_T), respiratory minute ventilation ($\dot{V}_I$), oxygen consumption ($\dot{V}_{O_2}$), carbon dioxide production ($\dot{V}_{CO_2}$), and respiratory exchange ratio (R). Values for V_T and $\dot{V}_I$ have been corrected to ATP, whereas those for $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ are at STPD. The characteristics of the mask have been published elsewhere.[18]

Heart rate (HR) was recorded telemetrically (Hippocard) from electrodes on the mid- and lateral thorax. The accuracy of this system has been tested previously.[12] A veterinarian monitored the performance of the horse, and by pressing a button on a recording watch, marked the recording so that specific times during the exercise could be recognised.

A total of 3 catheters were inserted into the left jugular vein and the carotid while the horse was standing in stocks. The areas of insertion were clipped and scrubbed with povidone-iodine (Pevidine, BK Veterinary Products Ltd) and surgical spirit. Local anaesthetic, lignocaine hydrochloride (Astra Pharmaceuticals Ltd), was administered before incisions were made in the skin. A polythene catheter (7F) for sampling mixed venous blood and a thermistor temperature probe (8F) were inserted into the left jugular vein. They were advanced until the catheter tip lay in the right atrium. A Swan-Ganz catheter (7F) for measuring temperature and taking blood samples, was inserted into the raised carotid artery. The catheters were flushed regularly with heparinised saline (10–20 IU ml^{-1}) to keep them patent.

Before drawing a sample, the deadspace was removed from the catheter and discarded. Plastic blood gas syringes (Monoject) were prepared by flushing with heparin (5000 IU ml^{-1}), which was then removed to leave the deadspace filled with heparin. The syringes also contained a moveable stainless steel washer to aid mixing of the blood. A total of 5 ml of blood were removed at each

of 7 sample times. After withdrawal of the blood sample, any gas bubbles were removed, the end capped and the syringe placed on crushed ice and water for storage (< 1 h) before analysis. Before a sample was analysed for pH and blood gases, it was mixed thoroughly and the blood in the tip discarded.

Measurements were performed on an ABL 330 blood gas analyser (Radiometer Ltd) which has an automatic temperature correction facility, based on the characteristics of human blood. The accuracy of the correction for horse blood was tested by comparing the results from the analyser against values for tonometered blood at different temperatures which were determined at the appropriate temperature with a BMS3 Mk II/PHM 73 analyser (Radiometer Ltd). The values from the ABL 330 all fell within ±0.3 kPa or ±0.01 pH unit of the values determined directly with the BMS3 Mk2/PHM 73.

Oxygen content was measured by the method of Tucker.[16] A water-jacketed glass chamber housed a Clarke-type O_2 electrode. The chamber contained freshly prepared 0.69% (w/v) potassium ferricyanide solution, which also contained 0.3 g $(100\ ml)^{-1}$ saponin and a small amount of octan-1-ol, n-octanol. This served to release the O_2 from the blood. The solution was completely deoxygenated before being added to the chamber and both were kept at 37°C. The system was calibrated with distilled water at 0°C equilibrated with pure O_2. The accuracy, over 20 determinations, of measurements of O_2 content was ±1.08% (range) of the expected value. Cardiac output was calculated from O_2 uptake and arterial minus mixed venous O_2 content using the Fick equation. Blood lactate concentration [La] was assayed spectrophotometrically and haemoglobin concentration [Hb] was determined by a haemoglobinometer (Coulter Electronics Ltd).

All variables were calculated as means ± SE and differences between 2 mean values were determined by Student's *t*-test with $p<0.05$ taken as the level of significance. The number of animals (n) contributing to the mean values was 4, except for temperature of arterial blood, plasma lactate concentration and blood pH, when $n=6$.

RESULTS

Maximum oxygen uptake (V_{O2max}) was not significantly affected by 15 weeks out of training (Table 1). Oxygen consumption reached a plateau at 9 to 10 m s^{-1} both when the horses were fit (F) and after relative inactivity (RI). The respiratory exchange ratio was higher at lower speeds in the horses after 15 weeks of relative inactivity. At a speed of 7 m s^{-1} the value from the horses out of training was significantly higher than that obtained when they were fit (1.02±0.04 vs 0.88±0.01).

The respiratory and cardiovascular responses to exercise were also similar before and after a period of relative inactivity (120 breaths min^{-1} at 9–11 m s^{-1}). Minute ventilation reached maximum values at 10 m s^{-1} of 1564±60 l min^{-1} (F) and 1470±36 l min^{-1} (RI). At higher speeds $\dot{V}_I$ did not increase in proportion to the increase in $\dot{V}_{O_2}$ (Fig. 1).

HR was not significantly lower at 11 m s^{-1} after 15 weeks out of training. Coupled with a slightly lower mass-specific cardiac stroke volume in the horses out of training, there was a statistically insignificant reduction in mass-specific cardiac output at $\dot{V}_{O2max}$ (Table 1).

Arterial PO_2 (PaO_2) fell to exactly the same value (8.4 kPa, 63 mmHg) at 11 m s^{-1} in the horses when fit and after a period of relative inactivity. None of the other blood gas values were significantly affected by the horses being out of training either. The reduction in PaO_2, compared to resting values, was more than compensated by a 47% increase in [Hb] at 10 m s^{-1}, which maintained arterial oxygen content (CaO_2) significantly above the resting values [8.6±0.5 mmol l^{-1} (F), 9.3±0.1 mmol l^{-1} (RI)] in the horses when fit and detrained. In fact, after

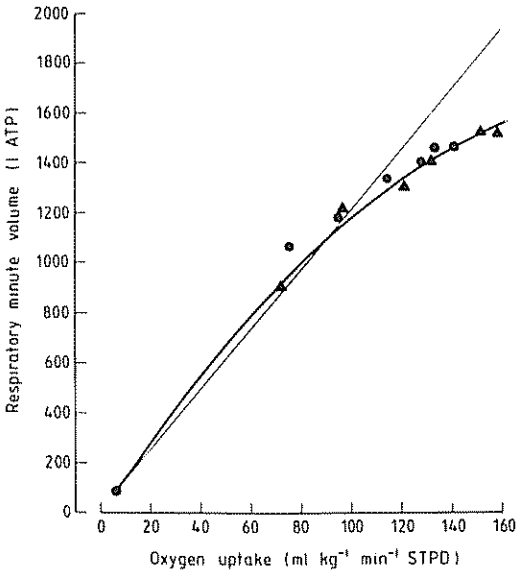

Fig 1 The relationship between respiratory minute volume and oxygen uptake in 4 Thoroughbred racehorses when fully fit (▲) and after 15 weeks out of training (●).

an initial increase at 6 m s^{-1}, CaO_2 remained constant throughout the exercise period at approximately 11 mmol l^{-1}, as PaO_2 declined and [Hb] increased. However, mixed venous O_2 content declined progressively during the exercise protocol [from 7.0±0.4 mmol l^{-1} (F), 7.5±0.3 m mol l^{-1} (RI) at rest to 2.1±0.4 mmol l^{-1} (F), 2.5±0.7 m mol l^{-1} (RI) at 10 m s^{-1}].

Temperature of arterial blood increased from 37.2°C at rest to 41.2°C at 11 m s^{-1} in the horses when fit and after a period of relative inactivity. The only measured variable which showed a statistically significant change after 15 weeks out of training was [La] (Table 1), in which there were significant differences between the two sets of data at 10 and 11 m s^{-1}. When the horses were fit, [La] increased from 0.6±0.03 mmol l^{-1} at rest to 5.4±0.96 mmol l^{-1} at the conclusion of the 11 m s^{-1} run. After 15 weeks out of training, the values were 0.9±0.31 mmol l^{-1} and 11.1±2.49 mmol l^{-1} respectively. Despite the higher [La] during exercise after 15 weeks out of training, there were no significant effects on either arterial or mixed venous pH.

Table 1. *Mean (± SD) of respiratory, cardiovascular, blood gas and acid/base variables during exercise at $\dot{V}_{O_2max}$ in 4 Thoroughbred horses when fit and after 15 weeks out of training*

Treadmill speed for blood gas determinations was 10 m s^{-1} and for all other variables speed was 11 m s^{-1}

	Fit	15 weeks out of training
$\dot{V}_{O_2max}$ (ml kg^{-1} min^{-1} STPD)	158±16	141±0.34
$\dot{V}_I$ (l min^{-1} ATP)	1 526±16	1 458±68
$\dot{V}_{CO_2}$ (ml kg^{-1} min^{-1} STPD)	160±15	157±4.1
$\frac{\dot{V}_{CO_2}}{\dot{V}_{O_2}}$	1.0±0.1	1.12±0.03
Heart rate (beats min^{-1})	210±5	203±2
Cardiac output (ml kg^{-1} min^{-1})	789±102	622±24
Cardiac stroke volume (ml kg^{-1})	3.8±0.4	3.2±0.13
Haemoglobin concentration (g dl^{-1})	22.8±0.65	21.8±0.33
PaO_2 (kPa)	8.4±0.5	8.4±0.6
$PaCO_2$ (kPa)	8.2±0.6	6.8±0.2
CaO_2 (m mol l^{-1})	10.9±0.7	11.7±0.7
$C\bar{v}O_2$ (m mol l^{-1})	2.1±0.4	2.5±0.7
pHa (n=6)	7.198±0.035	7.176±0.095
Arterial temperature (°C) (n=6)	41.2±0.7	41.1±0.8
Blood lactate concentration (m mol l^{-1}) (n=6)	5.45±0.96	11.15±2.5

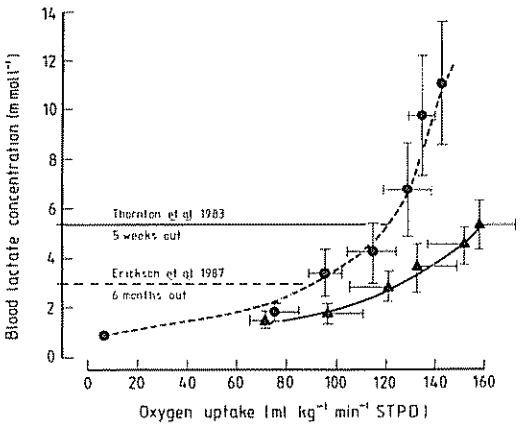

Fig. 2. The relationship between mean blood lactate concentration ([La]) and mean oxygen uptake ($\dot{V}_{O_2}$) ± SE, in Thoroughbred racehorses when fully fit (▲) and after 15 weeks out of training (●). Also included are data for Standardbred horses after 5 weeks out of training ([La] 5.4 mmol l^{-1}, $\dot{V}_{O_2}$ 111 ml kg^{-1} min^{-1})[15] and for Quarter horses after 6 months out of training ([La], 3.0 mmol l^{-1}).[5]

DISCUSSION

The increase in R at lower speeds after 15 weeks without training is the reverse of what happens with training in both horses and humans.[8,9,14] There were no signs of hyperventilation during exercise in the horses after the period of relative inactivity, compared with when they were fully fit, as blood gases were virtually identical, at least up to 8 m s^{-1}. This would suggest that metabolic substrates shifted from a mixture of fats and carbohydrate when the horses were fit towards pure carbohydrate after 15 weeks of relative inactivity, thus leading to a greater rate of depletion of muscle glycogen.[14]

The values obtained in the present study for V_{O_2max}, and for $\dot{V}_I$, cardiac output, cardiac stroke volume, HR and blood gases at $\dot{V}_{O_2max}$ were similar to those previously obtained for fit Thoroughbred horses.[6,8] When walking and trotting, horses may show signs of hyperventilation and increases in arterial and end-tidal P_{O_2}[1], but at higher levels of exercise (>6 m s^{-1}), arterial hypoxaemia develops.[1,2] It was reported that submaximal exercise training gave rise to a 23% increase in $\dot{V}_{O_2max}$ to 160.0 ± 11.0 ml kg^{-1} min^{-1} in Thoroughbred horses[8]. There was a significant increase in cardiac stroke volume, with no change in HR. There was also a significant decline in arterial minus mixed venous oxygen content. There was no significant effect on $\dot{V}_I$ nor on [Hb]. Training also caused a significant reduction in [La] and an increase in O_2 carrying capacity during exercise in Standardbred horses.[11,15] A 15 week period of inactivity might be expected to reverse these changes.

Although there were trends toward reduced cardiac stroke volume and cardiac output at $\dot{V}_{O_2max}$ after detraining, these were not significant. In humans, the reduction in cardiac stroke volume during exercise following a period of detraining appears to be the result of a decline in blood volume, which in turn could limit ventricular filling.[4] The only variable which showed a significant change in the present study after a period of relative inactivity was [La]. This is contrary to previous findings. In standardbred trotters detrained for 5 weeks [La] values of 5.4 mmol l^{-1} (at $\dot{V}_{O_2}$ of 111 ml min^{-1} kg^{-1}) were measured.[15] While in Quarter horses detrained for 6 months [La] values of approximately 3 mmol l^{-1} were recorded during exercise.[5] This apparent discrepancy may be because these investigations did not involve exercising the horses at sufficiently high levels to expose the effects of detraining.

In the present study, such values during exercise after 15 weeks out of training were also not significantly different from those obtained at the same (submaximal) values of $\dot{V}_{O_2}$ when the horses were fit (Fig. 2). These findings relate well to those in which an increase in the M subunit of LDH after 10 weeks of detraining was reported.[10]

Even when taken to $\dot{V}_{O_2max}$, it appears that a period of relative inactivity of 15 weeks does not significantly affect the cardiorespiratory responses to exercise in the previously trained horses.[15] However, the more rapid accumulation of lactate after such a period of relative inactivity may well affect the en-

durance of the animals as they apparently become more reliant on net anaerobic metabolism.

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