

Cardiopulmonary Response to Exercise and Detraining in the Quarter Horse

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Summary

Immediately following 6 months of combined endurance and interval training, five Quarter Horses began a 6 month period of detraining, followed by 18 weeks retraining. Every 6 weeks, each horse performed a standard exercise test on a treadmill. The exercise test consisted of four 5-min increments of increasing speed (1.0 to 2.8 m/s) at a 7° (12%) incline. We measured heart rate, arterial pressure, right ventricular pressure and dP/dt, and pulmonary artery pressure, flow velocity, and blood temperature. Arterial samples were obtained for lactate, hemoglobin, electrolytes, blood gases, and pH. There were no significant differences in the measured variables at either rest, exercise, or recovery during any of the detraining tests. The retraining program did not produce any significant changes in the measured variables during the test, either. However, there were small, nonstatistically significant changes similar to those observed in the initial training program, i.e., lower heart rate, dP/dt, lactate, and blood temperature during exercise. These results suggest that prolonged detraining does not significantly affect the cardiopulmonary responses to exercise in the previously trained horse.

Index terms: Standard exercise test; endurance training; interval training; pulmonary artery pressure, right ventricular pressure, dP/dt, pulmonary artery blood flow velocity; lactate; blood temperature.

Introduction

In recent years, the recognition of the horse as a natural athlete has led to increased scientific and veterinary interest in equine sports medicine, exercise physiology, new training methods, and factors improving the performance of horses (Engelhardt, 1977; Milne *et al.*, 1977; Bayly *et al.*, 1983; Persson, *et al.*, 1983). Detraining refers to the decrement in exercise performance that occurs when an athlete stops training. This is particularly important in the horse that is recovering from injury or illness. There are

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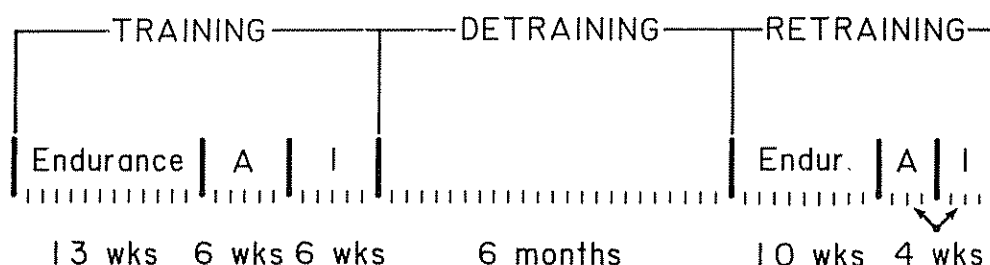
few studies describing the equine athlete's response to detraining, how soon it starts, how much occurs, and how rapidly a horse can be retrained and placed back in competition. The effects of training and detraining on skeletal muscle fiber type composition and enzyme activities in the horse have been studied by Guy and Snow (1977a, 1977b) and Snow and Guy (1979). They observed a decrease in enzyme activity after 5 weeks detraining; however, an increase or "rebound effect" occurred after 10 weeks detraining. Thornton *et al.* (1983) recently described the effects of 5 weeks of intensive training followed by 5 weeks of detraining on oxygen uptake, cardiac output, blood gas tensions, pH and blood lactate during and after exercise in Standardbred trotters and found no significant changes related to detraining. Foreman (1983) and Foreman *et al.* (1983) observed minimal changes in heart rate and lactate in fit Thoroughbreds during 10 weeks of detraining. The objective of this study was to examine the effects of an extended period of detraining and subsequent retraining on selected cardiopulmonary and metabolic responses to an exercise test on a treadmill. This study is a follow-up to the companion paper wherein the effects of initial training are described (Sexton *et al.*, 1987).

Materials and Methods

Experimental Animals. Five 2-year-old Quarter Horses were used in the study. The left carotid artery had been surgically elevated prior to the study to permit measurement of arterial blood pressure and collection of arterial blood samples (Tavernor, 1969). All horses were weighed weekly and fed a concentrate mixture formulated to meet NRC (1978) requirements and grass hay twice daily.

Standard Exercise Test. A standard exercise test was performed on a treadmill built for horses prior to training and periodically during training, detraining and retraining (Erickson *et al.*, 1983; Sexton *et al.*, 1986). After the collection of data at rest on the treadmill, a continuous, 20-minute, four-stage treadmill test increasing in speed from 1.0–2.8 m/sec was used to collect data during exercise. All data were obtained with the treadmill set at a fixed grade of 7° (12%). During the exercise test, arterial pressure, right ventricular pressure and dP/dt, pulmonary artery pressure and flow velocity, the electrocardiogram, blood temperature, and heart rate were recorded continuously. Arterial blood samples were taken at the end of each work level for lactate, hemoglobin, and hematocrit analysis. Arterial samples for serum chemistries were taken at rest, 1.6, 2.8 m/sec, and at 10 min post-exercise. Each horse was tested before training; at the end of endurance, gallop, and interval training; at 6-week intervals during detraining; and after 10 and 18 weeks of retraining (Fig. 1)

Detraining and Retraining. The horses had just completed an intense 25-week combined endurance and interval training program (Sexton *et al.*, 1986). The total training program lasted 174 days, consisting of 90 days of endurance work and 84 days of combined endurance and interval training. The training program was followed by 6 months of detraining in order to simulate a period of prolonged inactivity, which is often associated with musculoskeletal injury. Each horse was placed in a paddock (30 x 80 m) and only subjected to exercise during the standard exercise test at 6-week intervals. The horses were relatively inactive during this period of time. Retraining was similar to the initial training program, but was only 18 weeks in length, including 10 weeks of endurance training and 8 weeks of combined endurance and interval training.



A = Accelerations

I = Intervals

FIGURE 1. Schedule used to train, detrain, and retrain Quarter Horses on a dirt track, using endurance, accelerations (A), and intervals (I)

Results

The average resting values and peak responses measured during the training, detraining, and retraining tests for heart rate, right ventricular dP/dt , pulmonary artery flow velocity, blood lactate, and temperature are shown in Fig. 2–6, respectively. There were no statistically significant differences in the measured variables at either rest, peak exercise, or recovery during any of the detraining tests. The performance of the exercise tests, however, appeared to be more difficult as detraining progressed, evidenced by increased sweating and more labored breathing. The retraining program, which consisted of additional endurance and interval training, did not produce any statistically significant changes in the measured variables during the exercise test, either. However, there were trends very similar to the training effects we observed in the initial training program, i.e., lower heart rate, dP/dt , blood lactate concentration, and blood temperature during exercise (Sexton *et al.*, 1987).

Discussion

Some of the changes in our studies may be the result of maturation of the horses, since we initiated our study with two-year-old horses and the training, detraining, and retraining studies took over a year. Heart rate varies with age, breed, body size, and the animal's excitability (Engelhardt, 1977; Evans, 1985; Persson, 1983; Physick-Sheard, 1985). The anticipation of exercise, psychosomatic factors, and changes in sympathetic and or parasympathetic activity often increase heart rate. We observed an average heart rate of 34–46 beats/min at rest; the highest rate occurred prior to the initial pretraining exercise test and the lowest prior to the last test at the end of retraining, suggesting less anticipation of the treadmill tests as the study progressed and a more highly trained state at the end of retraining. The maximal heart rate in the conditioned horse is 210–240 beats/min (Asheim *et al.*, 1970; Lindholm and Saltin, 1974). The heart rate during a standard submaximal exercise test generally decreases with training. The average peak heart rate attained in our studies ranged from 145–180 beats/min (Fig. 2) at 2.8 m/sec on a 12% grade. Although there was a significant decrease with initial training ($P \leq .05$), there was no change with 24 weeks detraining or retraining. Although published data on recovery heart rate are conflicting (Physick-Sheard, 1985), our results



FIGURE 2. Effects of initial training, detraining, and retraining on heart rate during a standard exercise test at 2.8 m/sec, 12% grade on the treadmill. Control and peak values \pm SEM are shown.

indicate that recovery heart rate at five minutes post-exercise decreases with training from 88 to 67 b/min ($P \leq .05$) but there is very little loss of training during the detraining period. Persson (1983) discussed recovery heart rate and found that at five minutes post-exercise there was the best correlation with the exercise heart rate of about 200 beats/min. Foreman, *et al.* (1983) observed no significant differences in recovery heart rate in four Thoroughbreds on the racetrack during 10 weeks of pasture rest after 9 weeks of traditional American Thoroughbred race training. Recovery heart rate curves demonstrated only a 6% increase in area under the curve with detraining. They suggest that this lack of a significant increase in recovery heart rate during 10 weeks detraining implies that a fit Thoroughbred can be given a month off training without significant changes in the cardiopulmonary response to exercise.

The results in right ventricular dP/dt correlate with the findings in heart rate. Resting dP/dt progressively declined during the course of the study, also demonstrating less anticipation of exercise. RV dP/dt declined from 670 to 567 mm Hg/sec during initial training, ranged from 495–565 mm Hg/sec during detraining, and declined to 400–470 mm Hg/sec during detraining (Fig. 3). There was a progressive increase in dP/dt with exercise, increasing threefold during the exercise test. Maximum right ventricular dP/dt showed a significant decrease ($P \leq .05$) from 2120 to 1600 mm Hg/sec with initial training. After only 6 weeks of detraining, there was an increase in the maximum dP/dt to 1850 mm Hg/sec. Retraining resulted in a small additional

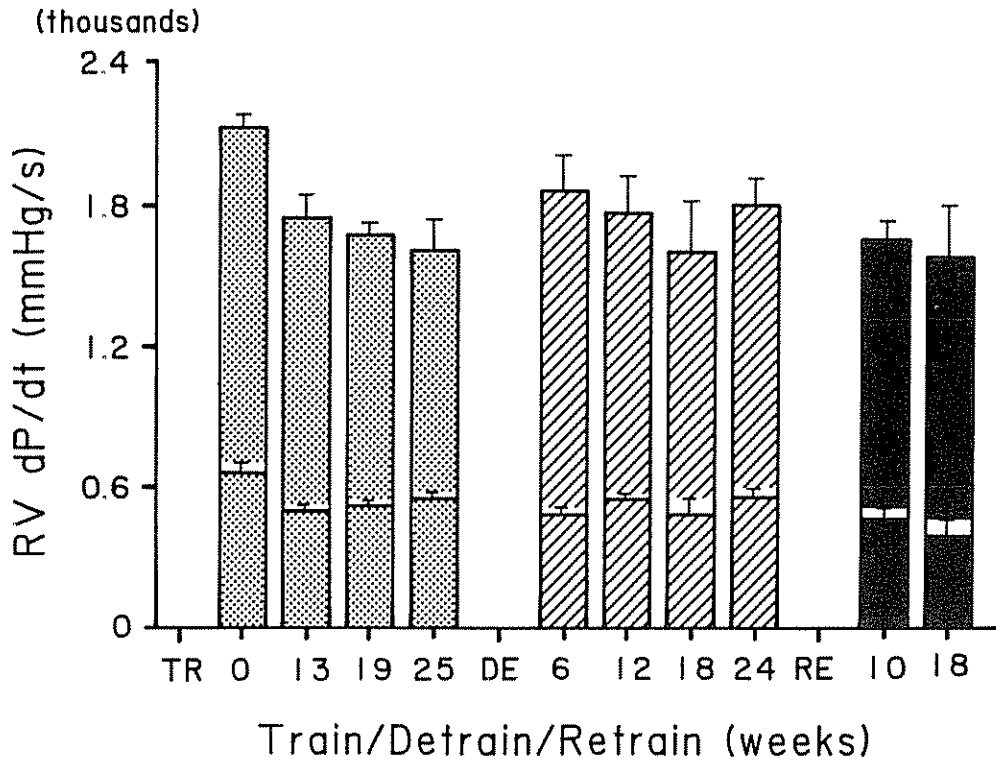


FIGURE 3 Effects of initial training, detraining, and retraining on right ventricular (RV) dP/dt during a standard exercise test at 2.8 m/sec, 12% grade on a treadmill. Control and peak values \pm SEM are shown.

reduction in dP/dt during exercise, decreasing from 1785 mm Hg/sec at the end of detraining to 1575 mm Hg/sec at the end of retraining. These results suggest very little decrement in ventricular function during detraining and a small additional training effect during the retraining period.

Cardiac output has been measured by other investigators during treadmill studies and is reported to increase six- to eightfold over resting values (Thomas and Fregin 1981, Thomas *et al.*, 1983; Thornton, *et al.*, 1983). The average mean flow velocity (Fig. 4) in our study increased five- to sixfold during the exercise test. Studies by Thornton, *et al.* (1983) demonstrated little change in cardiac output with 5 weeks of training followed by 5 weeks of detraining. The lower peak flows at 12, 18, and 24 weeks of detraining which we observed during the exercise test compared to 19 weeks of training may be the result of a decrease in blood volume associated with detraining.

The significant increase ($P \leq .05$) in hemoglobin which occurred during the exercise test from 10.2–14.8 g/dl is attributed to increased sympathetic activity which increases circulating catecholamines and results in splenic contraction. The hemoglobin concentration is also the result of the interaction between plasma volume and total hemoglobin. There was little change in the hemoglobin values during initial training or detraining. However, hemoglobin was 11.1 and 11.6 g/dl at rest prior to the tests during retrain-

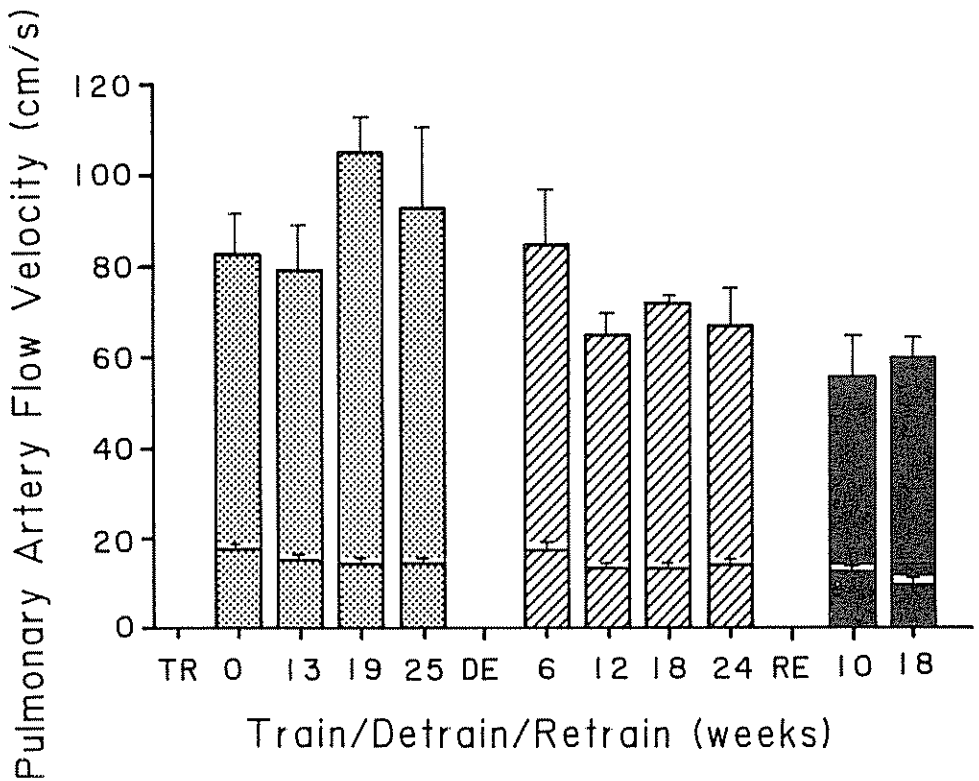


FIGURE 4 Effects of initial training, detraining, and retraining on mean pulmonary artery flow velocity during a standard exercise test at 2.8 m/sec, 12% grade on a treadmill. Control and peak values \pm SEM are shown.

ing. The hemoglobin at peak exercise during the final exercise test was 16.7 g/dl, much higher than other exercise values.

The increase in blood lactate during exercise in the horse is well documented (Engelhardt, 1977) and it has been demonstrated that it is a good indicator of work intensity in the exercising horse (Lindholm and Saltin, 1974). Blood lactate accumulation is exponentially related to both exercise heart rate and work load expressed as velocity (Engelhardt, 1977; Persson, 1983; Wilson *et al.*, 1983). A significant increase ($P \leq .05$) in blood lactate occurred during the exercise test in our studies, particularly prior to initial training (Fig. 5). Detraining resulted in a non-significant increase in the blood lactate during the highest level of exercise, increasing from 2.2 mmol/l at the end of training to 3.0 mmol/l after 6 weeks of detraining. Retraining resulted in an additional non-significant reduction in blood lactate to 1.6 and 1.4 mmol/l after 10 and 18 weeks of retraining, respectively. The changes in blood lactate accumulation may have been larger during exercise during the detraining period if the horses could have been subjected to a more strenuous test. Thornton, *et al.* (1983) observed a significant decrease in blood lactate with 5 weeks of training in Standardbred trotters, but no change with 5 weeks of detraining. They also plotted blood lactate versus speed and observed

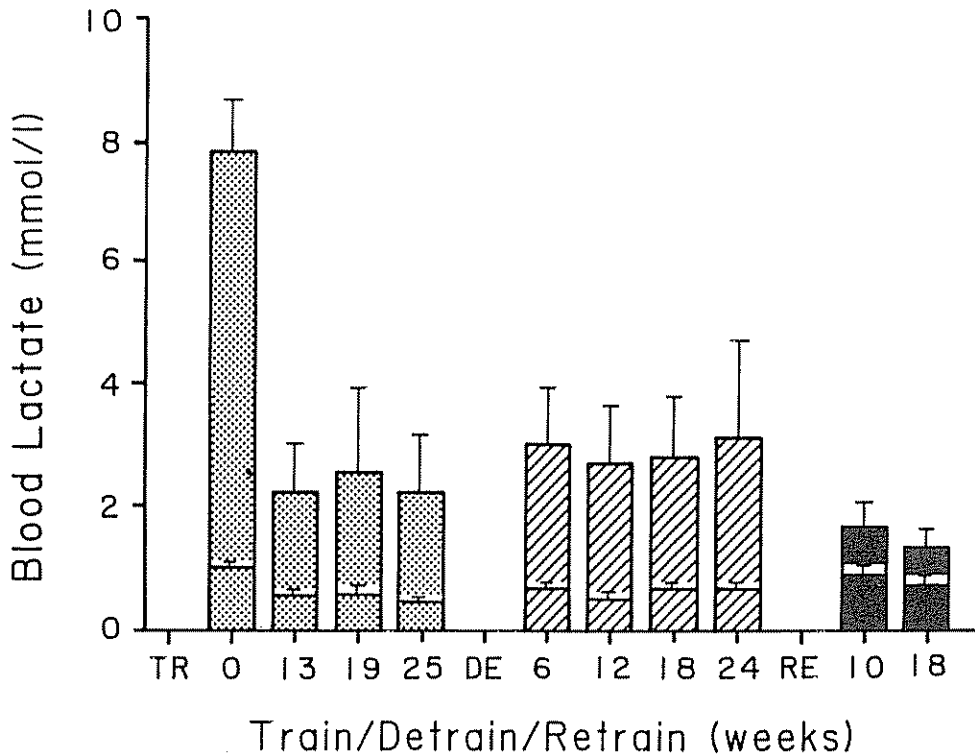


FIGURE 5. Effects of initial training, detraining, and retraining on lactate concentration during a standard exercise test at 2.8 m/sec, 12% grade on a treadmill. Control and peak values \pm SEM are shown.

no significant differences in the slope of the curve before and after training and detraining. The intercept increased with training, but did not change with detraining.

Pulmonary artery blood temperature progressively increased during the standard exercise test as the work load increased, declined rapidly during the first minute of recovery, and then slowly returned toward resting values during the next 20 minutes of recovery. The average maximum blood temperature attained during the exercise tests declined significantly ($P \leq .05$) during initial training from 40.5–39.6°C (Fig. 6), suggesting more efficient cooling by the lungs and through the skin resulting from an increase in plasma volume. Following 6 weeks of detraining, there was a nonsignificant increase in the peak blood temperature from 39.6°C at the end of training to 40.0°C, suggesting that cooling was less efficient. Retraining resulted in an additional reduction in blood temperature during exercise, decreasing from 39.7°C at the end of detraining to 39.1°C during the exercise tests at 10 and 18 weeks of retraining. The difference between the temperature at rest and the temperature at the peak of the exercise test declined from 2.1–1.6°C with initial training, increased to 1.6–1.9°C with detraining, and declined to 1.3–1.5°C with retraining. These results suggest that the trained horse has a larger plasma volume, is better able to dissipate heat during exercise, and can



FIGURE 6. Effects of initial training, detraining, and retraining on pulmonary artery blood temperature during a standard exercise test at 2.8 m/sec, 12% grade on a treadmill. Control and peak values \pm SEM are shown.

possibly run farther and work harder and more efficiently before heat exhaustion occurs or before heat limits performance.

Human studies. Saltin and co-workers (1968) studied the cardiopulmonary and hematological changes in normal men after 20 days of bed rest and then after 53–55 days of retraining. The most striking effects of inactivity were pronounced decreases in maximal oxygen uptake, stroke volume, and cardiac output. They observed a 28% decrease in maximal oxygen consumption with detraining and a subsequent 33% increase with retraining. During upright exercise at submaximal loads, stroke volume and cardiac output decreased 30% and 15%, respectively, with detraining, whereas submaximal heart rate increased. The arterio-venous oxygen difference was higher after detraining for any given level of oxygen uptake. These trends were reversed with retraining. These decreases in stroke volume and cardiac output were attributed to an impairment in venous return and a direct effect on myocardial function. Henriksson and Reitman (1977) observed that the activity of the oxidative enzymes decreased in human skeletal muscle much more rapidly with detraining than maximal oxygen consumption.

Nadel (1985) recently reviewed the physiological adaptations to aerobic training. Expansion of blood volume during training provides a higher central circulating blood volume during exercise and contributes to higher stroke volume and cardiac output. Increased blood volume also enhances the body's ability to maintain a high skin blood flow in potentially compromising conditions, such as during heavy exercise in the heat,

thereby providing for a greater heat transport from core to skin and for a relatively lower storage of heat. The decline in cardiovascular function following a few weeks of detraining may be largely due to a reduction in blood volume which appears to limit ventricular filling (Coyle *et al.*, 1986).

Conclusions

In conclusion, our study suggests that 6 months of detraining in the horse causes only minimal decrement in the cardiopulmonary and blood lactate responses to a submaximal standard exercise test. These observations may be very important in the retraining and rehabilitation of a horse following extended injury, lameness, or illness. The exercise test which we used was not maximal and a more strenuous test may reveal more pronounced decrements in the measured cardiopulmonary parameters and blood lactate concentration. The deconditioning or inactivity in an injured horse may not be as severe and may not compare to that in human studies where bed rest is used. However, as in human studies, the oxidative enzyme systems in the horse may decrease much more rapidly with detraining than the cardiopulmonary system.

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