Blood-gas measurements adjusted for temperature at three sites during incremental exercise in the horse


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Taylor, L. E., D. S. Kronfeld, P. L. Ferrante, J. A. Wilson, and W. Tiegs. Blood-gas measurements adjusted for temperature at three sites during incremental exercise in the horse. J. Appl. Physiol. 85(3): 1030–1036, 1998.—Rectal temperature (Tre) is often used to adjust measurements of blood gases, but these adjusted measurements may not approximate temperatures during intense exercise at main sites of gas exchange: muscle and lung. To evaluate differences in blood gases between sites, temperatures (T) were measured with thermocouples in the rectum (re), in mixed venous blood (v), in gluteal muscle (mu), and on the skin (sk) in seven Arabian horses as they underwent an incremental exercise test on a treadmill. Blood samples were drawn from the carotid artery and pulmonary artery (mixed venous) 30 s before each increase in speed and during recovery. Blood gases and pH were measured at 37°C, and all variables were adjusted to Tre, T, and Tmu. Adjusted variables during exercise and recovery were significantly different from each other at the three sites. Linear and polynomial equations described the time course of venous temperature and T from Tre and Tsk during exercise and from Tsk during recovery. Interpretation of changes in muscle metabolism and gas exchanges based on blood-gas measurements is improved if they are adjusted appropriately to Tmu or T, which may be predicted from Tsk in addition to Tre during strenuous exercise and from Tsk during recovery.

Arabian horses; muscle temperature

Blood-gas and pH are often measured in studies of exercise. Adjustment of blood-gas data for changes in temperature during moderate and strenuous exercise is important for proper interpretation. These measures are dependent on dissociation constants that vary with temperature (4, 8), which rises during moderate and strenuous exercise. Rectal (Tre), blood, and muscle temperatures (Tmu) have been used previously in horses for adjustment factors during exercise (2, 6–8, 14); Tre has underestimated temperatures in working muscle and lung, which would lead to an overestimation of pH and an underestimation of Po2 and PacO2. No reports have been found concerning temperature changes during prolonged strenuous exercise. In addition, no reports have been found in Arabian horses, a breed adapted to endurance exercise that has certain characteristic responses (11, 17, 23, 25).

The present investigation had three objectives: 1) to determine simultaneously Tmu, temperature in mixed venous blood (Tv), Tre, and temperature on the skin surface (Tsk) during incremental exercise in conditioned Arabian horses, 2) to compare the differences in Tre, Tv, and Tmu with respect to mixed venous and arterial H+ concentration ([H+]i) and blood gases, and 3) to derive regression equations for prediction of Tmu and Tv from Tre and Tsk during exercise and from Tsk during recovery.

Materials and Methods

Experimental animals. Seven Arabian horses [4–5 yr, 410 ± 41 (SE) kg body wt] were conditioned for 20 wk on a treadmill (Mustang 2200, Kagra). The right carotid artery was surgically relocated to a subcutaneous position in each horse >6 mo before the study. Horses were fed a cracked corn and oat mix and grass hay to meet requirements for moderate exercise in horses (13). The protocol and procedures were approved by the university’s animal care committee.

Experimental protocol. Feed, but not water, was withheld overnight for ≈12 h before the morning of the exercise test. The average ambient temperature of the climate-controlled barn that housed the treadmill was 12°C, relative humidity was 40%. Horses were brought to the barn and allowed ≈1 h of acclimation before the study. A sterile 18-gauge catheter (Angiocath, Becton-Dickinson) was introduced into the carotid artery and kept patent with heparinized saline.

A sterile polyethylene catheter (PE-240, Intramedic) was introduced into the pulmonary artery. Placement of the tip of the catheter was determined by changes in blood pressure via water manometer. Subsequent studies with the same group of horses confirmed via pressure transducer that catheters had been placed in the pulmonary artery. Copper-constantan thermocouples (Physitemp Instruments) were used to measure temperatures. One thermocouple was passed through the tubing into the pulmonary artery for measurement of T (model IT-18EXLNC). Tmu was measured 8 cm deep in the left middle gluteal muscle (model MT 23/8). Tsk was measured 10 cm deep in the rectum (model ESO-1), and Tsk was measured on the dried surface of the skin over the right gluteal muscle (model SST-1). Thermocouples were calibrated initially in a water bath, starting with ice water and heating to 45°C using a mercury thermometer marked to 0.1°C (ERTCO Precision model 1033, Baxter). Subsequent calibration was tested in a water bath at 37°C during each experiment. Heart rates were recorded before measurement of Tmu, just before each change in speed, with a commercial digital heart monitor (Polar Pacer, Polar CIC).

Exercise test protocol. Exercise consisted of an incremental test, with moderate increases in speed at each step to elicit steady changes in temperature. Horses walked for 4 min at 1.5 m/s at 0% slope, the slope was then raised to 6% (3.6°), and the speed of the treadmill was increased every 4 min by 0.5 m/s. Total time of the exercise portion of the test was 52 min, with horses reaching a top speed of 7.5 m/s. Horses then completed a 16-min walking recovery period at 0% slope.

Sampling protocol. Resting arterial and mixed venous blood samples and resting temperatures were recorded simultaneously before exercise. Samples were drawn every 4 min, 30 s before the treadmill was stopped for 5 s to record Tmu, Tre, Tv, and Tsk were measured during blood sampling times. After measurement of Tmu, the treadmill speed was increased and
had returned to the desired speed within 10 s. Samples were also taken every 4 min for 16 min during the walking recovery period.

The arterial and mixed venous samples (2 ml) were drawn into plastic heparinized syringes (300 U lithium heparin; Sigma Chemical) and stored in an ice-water bath until analyzed for pH, PCO₂, and PO₂, at 37°C (Stat Profile 1, Nova Biomedical) within 30 min. All measurements were adjusted to T_re, T_v, and T_mu, with the following equations (12)

\[ \text{pH}_{\text{adjusted}} = \text{pH} + [-0.0147 + 0.0065(7.400 - \text{pH})(T - 37)] \]  
\[ \text{PCO}_2_{\text{adjusted}} = \text{PCO}_2 \times e^{0.04375(T - 37)} \]  
\[ \text{PO}_2_{\text{adjusted}} = \text{PO}_2 \times e^{2.930(T - 37) - (5.49 \times 10^{-3}T + 0.071 - (9.72 \times 10^{-9}T + 2.3) \ln(\text{PO}_2)} \]

where \( y = e^{0.88 \times \ln(P_{O2})} \). Changes in the dissociation constant of carbonic acid in plasma with varying pH and temperature were also taken into account (20). The [H⁺] was calculated from pH values.

Data are summarized as means ± SE and examined by ANOVA for repeated measures (19). Dunnett’s t-test was used for comparison of preexercise means with means during exercise and recovery. \( P < 0.01 \) was considered significant. Regression equations were based on mean values for the seven horses at each step and were obtained with a curve-fitting program (22).

RESULTS

Temperatures and heart rate. T_sk was lower than T_v, T_re, and T_mu at all times, and T_mu was higher than T_re and T_sk at all times (Fig. 1). T_v was lower than T_re during rest and recovery but higher during exercise. Temperature increased at all four sites during exercise (Fig. 1), with T_mu higher than the others for the walking sample (4 min). Temperatures were different from each other at all four sites by 20 min, and all four temperatures had increased above resting values by 20 min (3.5 m/s, medium trot). Increases from rest to the end of exercise were 33.5 ± 0.06 to 37.9 ± 0.1°C, 37.8 ± 0.05 to 40.4 ± 0.1, 37.4 ± 0.05 to 41.0 ± 0.1°C, and 37.8 ± 0.05 to 42.0 ± 0.1°C for T_sk, T_v, T_re, and T_mu, respectively.

T_re continued to rise for 5 min during the walking recovery period, but T_sk, T_v, and T_mu decreased immediately, T_mu at a relatively slower rate. All four temperatures remained significantly different from each other throughout the recovery period. Only T_re returned to the preexercise value by the completion of the 16-min recovery period.

Heart rate was 33 ± 0.7 beats/min at rest and increased linearly with exercise time to 173 ± 2.3 beats/min at the last step (52 min, 7.5 m/s) of the exercise test and returned to 80 ± 4.3 beats/min at 16 min of recovery.

[H⁺]. Changes in arterial and mixed venous [H⁺] are summarized in Fig. 2. The three adjusted arterial [H⁺] values were between 38.9 and 38.0 nM at rest and decreased during exercise, reaching the lowest value at 30 min (5.0 m/s) before returning to near resting values at the end of exercise. By 28 min the changes from rest in temperatures at the measured sites (Fig. 1) resulted in a difference between adjusted arterial [H⁺] values at the three sites. Arterial values were lowest for [H⁺] adjusted to T_re, followed by [H⁺] adjusted to T_v, with [H⁺] adjusted to T_mu being highest. The rapid decline in T_re resulted in [H⁺] adjusted to T_re being the lowest during recovery, with [H⁺] adjusted to T_mu remaining the highest.

[H⁺] adjusted to T_v values were between 41.7 and 40.7 nM at rest (Fig. 2); they reached a low point at 32 min and then increased steadily until the end of exercise. Estimates of mixed venous [H⁺] exhibited a pattern similar to the values of arterial [H⁺] when adjusted for T_re, T_v, and T_mu.
PCO\(_2\). Changes in arterial (Pa CO\(_2\)) and mixed venous (Pv CO\(_2\)) PcO\(_2\) are summarized in Fig. 3. The adjusted PaCO\(_2\) values were 41–42 Torr at rest. The increased T\(_{mu}\) resulted in a difference in Pa CO\(_2\) and Pv CO\(_2\) adjusted to T\(_{mu}\) at the first sampling time. Pa CO\(_2\) at the three sites declined steadily during exercise, with differences between the three adjusted values by 20 min of exercise. None of the values had returned to preexercise levels by the end of recovery. The rapid decline in T\(_{v}\) during recovery resulted in Pa CO\(_2\) adjusted to T\(_{v}\) being the lowest; Pa CO\(_2\) adjusted to T\(_{mu}\) was the highest.

The adjusted PvCO\(_2\) values were 47–48 Torr at rest (Fig. 3) and were increased by 10 Torr during exercise. The PvCO\(_2\) values were decreased by 20 Torr during the first 4 min of recovery and did not return to preexercise values by the end of recovery. PpvCO\(_2\) adjusted to T\(_{mu}\) was highest throughout exercise, followed by PpvCO\(_2\) adjusted for T\(_{v}\) and T\(_{re}\).

HCO\(_3\)\(^{-}\) concentration. Changes in HCO\(_3\)\(^{-}\) concentration ([HCO\(_3\)\(^{-}\)]) are summarized in Fig. 4. The adjusted arterial [HCO\(_3\)\(^{-}\)] values were 25.5–26.0 mM at rest, increased at the walk, and then decreased steadily during exercise. Arterial [HCO\(_3\)\(^{-}\)] adjusted to T\(_{mu}\) was always the highest value, followed by [HCO\(_3\)\(^{-}\)] adjusted to T\(_{v}\) and T\(_{re}\) respectively. All values increased during recovery and approached preexercise levels by 16 min of recovery.

Adjusted mixed venous [HOC\(_3\)\(^{-}\)] was 27.5–28 mM at rest and remained higher than arterial [HCO\(_3\)\(^{-}\)] at all times, reaching a peak value of 33 mM at 16 min (3 m/s) before returning to preexercise levels by the end of exercise (Fig. 4). Adjusted values were different at all three sites after 20 min of exercise. Mixed venous [HCO\(_3\)\(^{-}\)] adjusted to T\(_{mu}\) was higher than the other values at the first sampling, and all values increased during recovery but failed to reach preexercise levels.

PO\(_2\). Changes in arterial (Pa O\(_2\)) and mixed venous (Pv O\(_2\)) P\(_O2\) are summarized in Fig. 5. Adjusted PaO\(_2\) was 90–94 Torr at rest, and all values increased gradually during exercise; they were different from each other at 20 min of exercise. However, PaO\(_2\) adjusted to T\(_{mu}\) was greater than the other values at the first sampling during exercise (4 min, 1.5 m/s) when the horse was still walking. Also, PaO\(_2\) adjusted to T\(_{v}\) was lower than at the other sites at rest, partly because resting blood temperature was at its lowest level. During exercise, PaO\(_2\) adjusted to T\(_{mu}\) was always the highest, followed by PaO\(_2\) adjusted to T\(_{v}\) and T\(_{re}\) respectively. After the end of exercise, all three adjusted values peaked at the 4th min of recovery and then declined slightly during the remainder of the recovery period. None of the values had returned to preexercise levels by the end of recovery.

The adjusted PvO\(_2\) values were 37–38 Torr at rest (Fig. 5) and remained lower than PaO\(_2\) values at rest, exercise, and recovery. All three values decreased to 20–25 Torr by 20 min and remained low until the end of exercise. PpvO\(_2\) adjusted for T\(_{mu}\) was different at the first sampling, and all three values were different by
20 min of exercise. \( P V_{O_2} \) adjusted for \( T_{mu} \) was highest, followed by \( P V_{O_2} \) adjusted for \( T_v \) and \( T_r \), respectively. Only \( P V_{O_2} \) adjusted for \( T_v \) had returned to the pre-exercise level by 16 min of recovery.

Arterial and venous values and arteriovenous differences of \( P O_2 \), \( P C O_2 \), and \([H^+]\) are compared as measured at standard temperature (37.00°C) and adjusted to the mean \( T_{mu} \) (41.96°C) at the last step of the exercise test in Table 1. The magnitude of the differences between the standard and adjusted values increased with increasing \( T_{mu} \). The arteriovenous differences also increased with the rise in \( T_{mu} \) during exercise. Mean arteriovenous \( P O_2 \) was linearly related to mean \( T_{mu} \) during incremental exercise \((r^2 = 0.80, n = 15, P < 0.001)\) and recovery \((r^2 = 0.97, n = 5, P = 0.002)\).

### Table 1. Arteriovenous differences measured at standard temperature and adjusted to the highest muscle temperature observed during incremental exercise

<table>
<thead>
<tr>
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<th>Standard Temperature (37.00°C)</th>
<th>Muscle Temperature (41.96°C)</th>
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<tbody>
<tr>
<td></td>
<td>Arterial</td>
<td>Venous</td>
</tr>
<tr>
<td>( P O_2 ), Torr</td>
<td>88</td>
<td>16</td>
</tr>
<tr>
<td>( P C O_2 ), Torr</td>
<td>24</td>
<td>45</td>
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<tr>
<td>([H^+]), nM</td>
<td>32</td>
<td>42</td>
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Values are means. \([H^+]\), \( H^+ \) concentration; \( \Delta \), arteriovenous difference.

Prediction equations. Mean \( T_{mu} \) and \( T_v \) values were related linearly \((r^2 > 0.94)\) to \( T_{re} \) and \( T_{sk} \) during exercise over the range 37–42°C and to \( T_{sk} \) but not \( T_{re} \) during recovery. Quadratic equations yielded better fits \((r^2 > 0.97)\) for the same relationships (Figs. 6 and 7).

### DISCUSSION

This study extends the range of increases in temperature during exercise found previously in limited studies.

![Fig. 5. \( P O_2 \) in arterial (\( P A O_2 \), A) and mixed venous (\( P V_{O_2} \), B) blood in 7 horses at a standard temperature of 37°C and adjusted to rectal, blood, and muscle temperatures during incremental exercise and recovery. Values are means ± SE.](image)

![Fig. 6. Prediction of muscle (\( T_{mu} \)) and blood (\( T_v \)) temperatures by quadratic equation during incremental exercise from mean rectal temperature (\( T_{re} \)): \( T_{mu} = 32.7107T_{re} - 0.399646T_{re}^2 - 0.399646T_{re}^2 - 627.358, r^2 = 0.994; T_v = 18.5858T_{re} - 0.221481T_{re}^2 - 348.412, r^2 = 0.991; A \) and mean skin temperature (\( T_{sk} \)): \( T_{mu} = 259.524488 - 13.241223T_{sk} + 0.197739T_{sk}^2, r^2 = 0.991; T_v = 244.826453 - 12.288956T_{sk} + 0.182112T_{sk}^2, r^2 = 0.989; B \) in 7 horses.](image)

![Fig. 7. Prediction of \( T_{mu} \) and \( T_v \) by quadratic equation during walking recovery from mean \( T_{sk} \): \( T_{mu} = 47.782332T_{sk} - 0.640598T_{sk}^2 - 849.415829, r^2 = 0.974; T_v = 199.388706 - 9.787845T_{sk} + 0.147774T_{sk}^2, r^2 = 0.980) in 7 horses.](image)
allowing for the first time the derivation of quantitative relationships between temperatures at different sites, including rectum, blood, muscle, and skin. In addition, inclusion of Tsk in the experiment has allowed derivation of quantitative relationships and the prediction of Tmu and Tre from Tsk during recovery. The temperature differences between sites are reflected significantly in adjusted parameters, especially PaO2. Previous reports of hypoxemia during intense exercise may have included errors in PaO2 measurement of as much as 11 Torr. The predictive equations derived in this study could be used to avoid or reduce such errors in evaluation of acid-base balance and blood-gas variables during exercise.

Critique of methods. The collection of blood samples into iced plastic syringes with subsequent analysis performed within 30 min may have slightly affected the present results. The PaO2 of human blood samples collected in plastic syringes and stored for 30 min in ice water was overestimated by 1–3% (10). The solubility of O2 increases with cooling, and plastic syringes do not form an impenetrable barrier to ambient air. Equine blood differs somewhat from human blood, but small increases in PaO2 and PCO2 have been found after 1 h of storage in iced plastic syringes (1).

Temperature variation. The present data cover a wider range of temperatures during exercise than any reported previously in horses (2, 5–8, 14, 21). The pattern of responses, however, at different measurement sites has been seen previously in different breeds of horses at various exercise intensities (6, 8). Tmu was always highest during exercise, followed by Tre, Tsk, and Tre, respectively. Tsk has a characteristic lag period during early recovery as it continues to rise before gradually decreasing. This study emphasizes that the use of Tre for the adjustment of blood-gas data after exercise may not be appropriate, even for submaximal exercise in moderate environmental conditions. Tre and Tsk declined rapidly during recovery, with Tmu decreasing at a slower rate. At lower exercise intensities (5–15 min, 1.5–3.0 m/s), Tsk and Tre were not different, and this has been observed previously in horses (5). Little cooling of the blood occurs across the lung; differences approximate 0.4°C between the pulmonary and carotid arteries during exercise in horses (6). Increasing the exercise intensity in an incremental fashion in the present study resulted in a significant difference in temperature at the different sites after 28 min (4.5 m/s). During this study, the highest mean temperature of 42.02°C was recorded 8 cm deep in the middle gluteal muscle at the end of exercise. The horses used in this study had been in training for 5 mo, and training has been shown to attenuate the rise in Tmu during exercise (21, 26).

Consequences of temperature differences. All the variables studied showed differences between measurement at standard temperature (37°C) and each of the three adjusted values, with the most dramatic differences found in the arterial measurements adjusted to Tre and Tmu.

[\(H^+\)]. The pattern of the [\(H^+\)] response to incremental exercise has been observed previously in ponies and Thoroughbred horses during moderate exercise (14, 15). In this study the difference in Tmu at the first sampling time resulted in adjusted arterial and mixed venous [\(H^+\)] values being different from corresponding values at the other two sites. Near the end of exercise, in arterial and mixed venous [\(H^+\)], there was a difference between [\(H^+\)] adjusted to Tre and [\(H^+\)] adjusted to Tmu of 1.8 nM. During exercise [\(H^+\)] in the working muscle was actually 1.1–1.8 nM higher than would have been estimated with Tre and Tmu, respectively. These differences may be important for the interpretation of muscle metabolism during exercise.

PCO2. The use of Tre or Tsk compared with Tmu after exercise at 3.5 m/s would have underestimated PaCO2 and PvCO2 during exercise and recovery. The overall response of PaCO2 to exercise has been seen previously (6, 24) in other breeds of horses exercising at similar intensities for 8–15 min compared with 52 min in the present study. The steady decrease in all three PaCO2 values throughout incremental exercise has not been reported previously and demonstrates the Arabian horse’s ability to hyperventilate, hence avoiding an exercise-induced hypercapnia and acidosis during this type of exercise. Increased PaCO2 values have been observed during short, high-intensity exercise in other breeds and lead to an arterial acidosis (2, 8). The Arabian horses were able to maintain a high level of alveolar ventilation in addition to avoiding acidosis. Whether the Arabian horse can avoid this arterial hypercapnia at maximal-intensity exercise has not been investigated.

PvCO2 increased slightly at the onset of exercise but never changed >5 Torr in either direction during exercise. The lack of a large or steady increase in CO2 in the mixed venous blood may be due to the submaximal exercise intensity. The arteriovenous difference averaged −6 Torr at rest, increased to −20 Torr after 28 min of exercise, and was −25 Torr at the end of exercise. The steady decline in PaCO2 helped maintain PvCO2, offsetting any tendency for it to increase, by presenting the working muscle with arterial blood that had a relatively low PCO2.

\(\left[HCO_3^-\right]\). Differences in adjusted plasma [\(HCO_3^-\)] between the three sites were small but significant after 20 min of exercise. The decline in all three adjusted [\(HCO_3^-\)] values (Fig. 4) suggests the development of acidosis during exercise in arterial and mixed venous blood. Increases in blood lactate during exercise will decrease the strong ion difference and, combined with a rise in the total concentration of plasma proteins, will contribute to a decrease in [\(HCO_3^-\)]. However, this decrease may be exaggerated during short, maximal-intensity exercise if \([HCO_3^-]\) values are not adjusted for temperature changes.

PO2. Differences in the adjusted PaO2 at the three sites during exercise and recovery were the most dramatic among the measured variables. At 40 min of exercise, there was an 11-Torr difference between PaO2.
adjusted for $T_{re}$ and $P_{aO_2}$, adjusted for $T_{nu}$ and an 8-Torr difference between $P_{aO_2}$, adjusted for $T_{v}$ and $P_{aO_2}$, adjusted for $T_{nu}$. These differences could lead to substantial error when $O_2$ extraction by the working muscle or alveolar diffusion in the lung is interpreted. They are important when variables, such as $O_2$ saturation, are calculated from $P_{aO_2}$.

A small, transient increase in $P_{aO_2}$ at the onset of exercise and a concomitant decrease in $P_{vO_2}$ during exercise have been seen previously in ponies and Thoroughbred horses exercising at moderate intensities (15, 18). However, the magnitude of the steady increase in $P_{aO_2}$ throughout exercise (Fig. 5) has not been previously reported. This may represent a breed characteristic: the Arabian horse is very well suited for long-distance aerobic exercise, as demonstrated by the predominance of slow-twitch, high-oxidative muscle fibers and by the high activity of aerobic enzymes (16). Our unpublished data have revealed that the Arabian horse can maintain a $P_{aO_2}$ between 90 and 100 Torr during exercise at heart rates > 210 beats/min.

Adjustment of arterial and venous $P_{O_2}$, $P_{CO_2}$, and [H+] to $T_{nu}$ during exercise reveals substantial differences from values at 37°C (Table 1). The 36% increase in arteriovenous $P_{O_2}$ would support a corresponding increase in aerobic metabolism, and increased $T_{nu}$ per se during exercise has been shown previously to alter muscle glycogenolysis and glycolysis in humans (3). This increase in arteriovenous $P_{O_2}$ would tend to slow any increase in intracellular acidity due to lactate accumulation. However, the 25% increase in arteriovenous $P_{CO_2}$ probably reflects a higher intracellular $P_{CO_2}$, which would tend to increase intracellular [H+].

Prediction equations. The quadratic equations found in this study (Figs. 6 and 7) could most likely be used to predict $T_{nu}$ and $T_{v}$ from $T_{re}$ and $T_{sk}$ in incremental tests of similar design. The use of $T_{sk}$ during recovery as well as exercise would be convenient. The $T_{sk}$ under the saddle during exercise and recovery has been used consistently during 50- to 100-mile endurance competitions. Unpublished reports show that it correlates closely (0.5–1.0°F) with $T_{re}$ during exercise under normal-to-hot environmental conditions but may have limited use when the relative humidity is low and the ambient temperature is < 70°C (P. Semmler, personal communication). One study suggests that changes in $T_{sk}$ may help provide a signal via cutaneous thermoreceptors for changes in breathing patterns during thermal stress (9).

Data comparable to our results have been found in five reports (2, 6–8, 14). Our equations had little predictive value for rest or light and moderate exercise when differences between $T_{re}$ and $T_{nu}$ or $T_{v}$ were < 1.0°C. During hard work, however, good agreement (±0.2°C) was found in constant-speed and incremental test protocols between actual measurements of $T_{v}$ and predicted estimates of $T_{v}$ from $T_{re}$ or $T_{sk}$ by our equations in some studies (2, 6, 14) but not others (7, 8). The use of these predictive equations may be limited by environmental conditions, as well as the breed and fitness level of the horses. Exercise intensity and duration are modifying factors that may affect the magnitude and speed of the increase in temperatures. The results suggest that the differences between $T_{nu}$ and $T_{re}$ or $T_{sk}$ and the differences between $T_{v}$ and $T_{re}$ or $T_{sk}$ need to be > 1.0°C for these equations to be useful in predicting $T_{nu}$ and $T_{v}$. The changes in temperature during exercise and recovery have sufficient impact on [H+] and blood gases to warrant prediction of $T_{nu}$ and $T_{v}$, if these values cannot be measured directly.

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REFERENCES


