Nasal strips do not affect pulmonary gas exchange, anaerobic metabolism, or EIPH in exercising Thoroughbreds

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In recent years, the use of an adhesive external nasal dilator strip (Breathe Right, CNS, Chanhassen, MN) has become widespread in human athletic competition as a mechanical means of lowering nasal airflow resistance and improving performance (32). It has been argued that a reduction in nasal airflow resistance would diminish the work of breathing during exertion, thus curtailing the metabolic \( \text{O}_2 \) requirements of respiratory muscles and allowing increased \( \text{O}_2 \) supply to the working musculature, resulting in enhanced athletic performance. However, studies examining the effects of a nasal dilator strip in resting normal human subjects have reported conflicting results, with some indicating a reduction in nasal resistance to airflow (11, 26), while such an effect could not be discerned in others (15, 33). In the same manner, whereas O’Kroy (24) failed to demonstrate any benefits of nasal strip application on maximum \( \text{O}_2 \) uptake, maximum ventilation, and indexes of perceived exertion/dyspnea in human subjects, it has also been reported that nasal strip application delayed the onset of oral breathing (9, 27) and decreased \( \text{O}_2 \) uptake and perceived exertion in human subjects (9). Similar to the use of the Breathe Right strip in human athletes, an equine nasal strip (Flair Equine Nasal Strips, CNS), a self-adhesive, thin, soft strip embedded with three flexible pieces whose springlike action is purported to hold the nasal passages open to maximize airflow, has been developed and is being marketed as a “drug-free way to help horses improve their breathing” (CNS). When applied according to manufacturer’s instructions, the rostral edge of the strip should be 3.81 cm (1.5 in.) from the nostril.

It is well known that strenuously exercising horses routinely exhibit significant arterial hypoxemia and hypercapnia (5, 6, 31, 34), pulmonary capillary hypertension (16–20), and a rather high incidence of exercise-induced pulmonary hemorrhage (EIPH) (12, 30). During heavy exertion, peak inspiratory and expiratory airflow of horses may approach (and even exceed) 65–89 l/s, and the work of breathing increases dramatically (2, 14). Should the equine nasal strip be effective in lowering nasal airflow resistance of exercising horses possibly via minimizing/preventing dynamic collapse of the lateral nasal wall, the ensuing significant reduction in the work of breathing may help improve exercise performance by increasing \( \text{O}_2 \) availability to the working muscles. Thus, at constant workload, curtailment of anaerobic metabolism may diminish lactate and ammonia production. Also, because EIPH results from stress failure of pulmonary capillaries brought about by the high transmural \( \text{O}_2 \) tension,
lary minus perivascular (alveolar) pulmonary capillary pressures (22, 35), the application of a nasal dilator strip may help diminish the incidence of EIPH by attenuating the pleural pressure swings during exertion. To our knowledge, there have been no scientific reports evaluating the use of an equine nasal dilator strip on blood gases, indexes of anaerobic metabolism, and/or the incidence of EIPH in racehorses. The present study was, therefore, undertaken to determine whether application of an external nasal dilator strip may help improve arterial hypoxemia and hypercapnia, diminish lactate and ammonia production, and/or affect the occurrence of EIPH in Thoroughbred horses performing strenuous exercise.

MATERIALS AND METHODS

Horses

Experiments were carried out on seven healthy, sound Thoroughbred horses (2 fillies, 5 geldings), 2.5–5 yr old and weighing 431–509 kg. They were exercise trained for 7 wk before blood-gas tension/pH and lactate/ammonia production studies were undertaken. The horses were housed in an air-conditioned building and were accustomed to being handled by people. They were fed a diet of alfalfa hay and oats, and free access to water was provided. The horses were dewormed periodically and were inoculated with tetanus toxoid and strangles vaccine. Our protocols and procedures were approved by the Institutional Laboratory Animal Care and Use Committees.

Exercise Training

After they were familiarized with walking, trotting, cantering, and galloping on the high-speed treadmill for 1 wk, all horses were exercised 3 days/wk in the following manner on the treadmill set at the flat, i.e., 0% grade: Beginning with a walk at 2 m/s for 60 s, belt speed was increased at 1 m/s every 60 s. After the horses trotted at 6 m/s for 60 s, the belt speed was raised to 8 m/s and then to 14 m/s for 60 s. Horses galloped at 14 m/s on a 3.5% uphill grade for 1 wk. All horses were exercised 3 days/wk in the following manner on the high-speed treadmill set at a 3.5% uphill grade.

Work Intensity Eliciting Maximal Heart Rate

Trials to ascertain work intensity needed to elicit maximal heart rate of the horses were undertaken on completion of exercise training (see above). It was observed that galloping at 14 m/s on a 3.5% uphill grade not only elicited maximal heart rate but induced EIPH in all horses as demonstrated by the presence of fresh blood in the trachea on airway endoscopic examination (12, 30). These trials also revealed that our horses could not sustain galloping at 14 m/s on a 3.5% uphill grade for >120 s, despite vigorous humane encouragement. Thus, for the present study, this workload, i.e., 14 m/s on a 3.5% uphill grade, was selected, as it represented a strenuous effort eliciting maximal heart rate and was capable of inducing EIPH consistently.

Experimental Procedures

On the day of the study, after local infiltration of 2% lidocaine hydrochloride in the 17th intercostal space, the abdominal aorta was percutaneously catheterized as described previously (21, 25). Thereafter, cardiac catheters (8-F) equipped with a tip manometer (Millar Instruments, Houston, TX), a fluid-filled lumen, and a thermistor (Edward Laboratories, Santa Clara, CA) were advanced into the pulmonary artery via introducers inserted into the left jugular vein. The locations of various catheters were confirmed by monitoring the characteristic phasic blood pressure waveforms on an oscillographic recorder (E for M, Lanexa, KS). These catheters permitted simultaneous sampling of the aortic and mixed venous blood as well as continuous monitoring of the core temperature (Cardiotherm, Columbus Instruments, Columbus, OH) during the experiments. After catheter placement, horses stood on the treadmill for ~50–55 min before blood-gas tension, pH, and lactate/ammonia studies were undertaken.

Blood-gas tensions, pH, Hb concentration, Hb-O2 saturation, and O2 content were determined using a carefully calibrated blood-gas analyzer/oximeter (model ABL520, Radiometer, Copenhagen, Denmark), and all blood-gas tension/pH data were corrected to the simultaneously measured pulmonary artery blood temperature. The calibration of the blood-gas tension/pH analyzer was checked at 30-min intervals and was verified using tonometered solutions of known blood-gas tensions, pH, Hb concentration, and O2 saturation.

For lactate determinations (21, 25), the mixed venous blood samples obtained at various intervals (see Experimental Design and Protocol) were immediately deproteinized with chilled perchloric acid (8% wt/vol), and the supernatant was harvested for lactate analysis (Sigma Diagnostics, Sigma Chemical, St. Louis, MO). All lactate assays were carried out in duplicate, and the values were within 5% of each other.

For determining plasma ammonia concentration (21) at various intervals (see Experimental Design and Protocol), mixed venous blood samples were collected in chilled tubes containing ammonia-free lithium heparin, and plasma was quickly separated. The plasma ammonia assays (Sigma Diagnostics) were also performed in duplicate, and the values were within 5% of each other.

Experimental Design and Protocol

All horses were studied in two sets of experiments: the control study and the nasal strip study. The sequence of these treatments was randomized for every horse, and 7 days were allowed between experiments on each horse. All experimentation was carried out in an air-conditioned laboratory, where the ambient temperature was maintained at 20°C.

Control study. Measurements were first made in duplicate (5 min apart) on quietly standing horses (rest 1 and rest 2) when heart rate and pulmonary vascular pressures had been stable for 10–15 min. Then exercise was performed in the following manner on the high-speed treadmill set at a 3.5% uphill grade. Beginning with a walk at 2 m/s for 60 s, belt speed was raised in increments of 1 m/s every 60 s until the speed was 6 m/s. After the horses had trotted for 60 s at 6 m/s, belt speed was raised to 8 m/s (canter) for 60 s and then to 14 m/s. Horses galloped at 14 m/s on a 3.5% uphill grade for 120 s. Thereafter, the belt speed was first decreased to 5 m/s (trot) for 60 s and then to 2 m/s (walk) for 5 min before the treadmill was stopped.

In the above-described incremental exercise protocol, along with core temperature measurement, simultaneous aortic and pulmonary arterial blood samples were obtained.
to determine blood-gas tensions, pH, Hb concentration, Hb-O₂ saturation, and O₂ content at 55 s of trotting at 6 m/s, 55 s of cantering at 8 m/s, 30, 60, 90, and 120 s of galloping at 14 m/s on a 3.5% uphill grade, 60 s of trotting at 5 m/s, and 2 min of walk at 2 m/s. Hereafter, the measurements at 5 and 2 m/s are also referred to as recovery data. Pulmonary arterial blood samples were also obtained before exercise (at rest) and at 2 min of walk at 2 m/s (during recovery) for lactate analysis as described above. For plasma ammonia assays, mixed venous blood samples were obtained before exercise (at rest), immediately on completion of exercise at 8 and 14 m/s on a 3.5% uphill grade, and at 2 min of walk at 2 m/s during recovery.

Nasal strip study. Measurements were first made on quietly standing horses (without the nasal strip) when heart rate and pulmonary vascular pressures had been stable for 10–15 min (hereafter, these pre-nasal strip measurements are referred to as rest 1). Then, with care taken to follow the manufacturer’s instructions supplied with the product, an equine nasal dilator strip (Flair Equine Nasal Strips) was applied. Five minutes later, resting measurements (rest 2) were completed and exercise was initiated. Exercise was performed on the treadmill set at a 3.5% uphill grade exactly as described for the control study (see above). Sampling intervals and procedures for handling the arterial and mixed venous blood for various measurements during exercise and recovery were identical to those in the control study.

Postexercise Airway Endoscopic Examination

In the control and nasal strip experiments, with the use of a flexible fiber-optic endoscope (Pentax Fiberscopes, Orangeburg, NY), careful endoscopic examination of the nasopharynx, larynx, and trachea (up to the carina) was undertaken 45–50 min after exercise (12, 30). The presence of fresh blood in the airway was regarded as indicative of the occurrence of EIPH (12, 30). In the present study, preexercise endoscopic examination of the airways was not performed. However, our extensive clinical and experimental experience indicates that fresh blood is not normally present in the airways of healthy horses.

Measurements and Data Analysis

In the present study, the O₂ extraction (%) was calculated as (arterial-mixed venous O₂ content gradient/arterial O₂ content) × 100. All data were subjected to repeated-measures, split-plot design ANOVA (29) using the SAS statistical software package (SAS version 6.12, SAS Institute, Cary, NC), and treatment comparisons were made using the least-squares significant difference method (29). Data for the control and nasal strip experiments were also individually subjected to ANOVA and then to Newman-Keuls multiple-range test (29) to determine the significant effects of work intensity within each treatment. The ANOVA procedure had revealed that the between-horse and horse × treatment (i.e., control vs. nasal strip) interaction effects were not statistically significant for any of the variables examined in this study. For all statistical analyses, the level of significance was set at $P < 0.05$, and the data are presented as means ± SE. The statistical power of comparisons for various variables in the present study was >80%.

RESULTS

Resting Data

Statistically significant differences were not observed between the preexercise values (i.e., rest 1 vs. rest 2) in the control or the nasal strip study for any of the variables examined in this study. Also, the preexercise data for the nasal strip study were not significantly different from those for the control study (Fig. 1; see Figs. 3–5).

Core Temperature During Exercise

The incremental exercise protocol caused a progressive significant rise in the core temperature of the horses in the control and nasal strip experiments, but statistically significant differences between the two treatments were not found at any point during the experimental protocol. The peak rise in core temperature in both experiments (mean increase = 3.4°C in both treatments) occurred at 120 s of galloping at 14 m/s on a 3.5% uphill grade.

Arterial and Mixed Venous Blood O₂ Tension During Exercise

Exercise at 6 and 8 m/s in either treatment did not cause statistically significant changes in the arterial O₂ tension, but in both treatments, a similar significant decrease was observed in the mixed venous blood O₂ tension (Figs. 1 and 2). Galloping at 14 m/s on a 3.5% uphill grade caused a significant reduction in arterial blood O₂ tension in the control and nasal strip experiments, but statistically significant differences between them could not be discerned. At 120 s of galloping at 14 m/s in the control and nasal strip studies, arterial blood O₂ tension was $72.9 ± 1.6$ and $73.4 ± 2.1$ Torr, respectively. The arterial hypoxemia of exercise was also accompanied by a significant drop in mixed venous blood O₂ tension in both studies. After exertion at 14 m/s on a 3.5% uphill grade, arterial and...
mixed venous blood O₂ tension recovered quickly; during the walk at 2 m/s, the values exceeded those at rest. Statistically significant differences between the control and nasal strip experiments were not found at any step of the exercise protocol.

Arterial and mixed venous O₂ tension data for individual horses at 120 s of galloping at 14 m/s on a 3.5% uphill grade in the control and nasal strip experiments are depicted in Fig. 2.

Arterial and Mixed Venous Blood Hb-O₂ Saturation During Exercise

Whereas during submaximal exertion at 6 and 8 m/s the arterial Hb-O₂ saturation was well maintained in both treatments, a progressive statistically significant reduction was observed during galloping at 14 m/s on a 3.5% uphill grade (Fig. 3). Arterial Hb-O₂ saturation at 120 s of exercise at 14 m/s on a 3.5% uphill grade in the control and nasal strip experiments approached 85.0 ± 1.7 and 86.5 ± 2.2%, respectively. However, statistically significant differences between the control and nasal strip experiments were not found at any step of the exercise protocol.

As work intensity increased in both treatments, the mixed venous blood Hb-O₂ saturation exhibited progressive significant reductions; these, however, quickly reversed during recovery. Statistically significant differences between the control and nasal strip experiments were also not found at any step of the exercise protocol.

Arterial Blood CO₂ Tension During Exercise

Compared with resting values (44.6 ± 0.7 and 45.1 ± 1.0 Torr in control and nasal strip experiments, respectively), exercise at 6 m/s caused a statistically significant reduction in arterial blood CO₂ tension (39.8 ± 1.0 and 39.7 ± 1.2 Torr in control and nasal strip experiments, respectively). However, starting with exercise at 8 m/s and continuing into galloping at 14 m/s in both treatments, arterial CO₂ tension increased, reaching 50.8 ± 1.8 and 50.2 ± 1.7 Torr at 120 s in the control and nasal strip experiments, respectively. During recovery, horses exhibited a dramatic hyperventilation and arterial CO₂ tension decreased to 23.8 ± 1.0 and 24.9 ± 1.7 Torr at 2 min of walk at 2 m/s in the control and nasal strip experiments, respectively. Statistically significant differences between the control and nasal strip experiments were also not found at any step of the exercise protocol.
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Arterial CO₂ tension data for individual horses at 120 s of galloping at 14 m/s on a 3.5% uphill grade in the control and nasal strip experiments are depicted in Fig. 2.

Arterial Blood pH During Exercise

Except for a statistically insignificant rise in arterial blood pH at 6 m/s in both treatments, a progressive significant fall in arterial blood pH was observed with incremental exercise. At 120 s of galloping at 14 m/s on a 3.5% uphill grade in the control and nasal strip experiments, arterial blood pH approached 7.150 ± 0.031 and 7.146 ± 0.041, respectively. However, statistically significant differences between the control and nasal strip experiments were not found at any step of the exercise protocol.

Arterial blood pH data for individual horses at 120 s of galloping at 14 m/s on a 3.5% uphill grade in the control and nasal strip experiments are depicted in Fig. 2.

Arterial and Mixed Venous Blood O₂ Content During Exercise

Arterial blood O₂ content increased significantly during exercise in the control and nasal strip experiments primarily because of a 50% rise in Hb concentration (owing to the splenic release of the erythrocyte reservoir). At 90 and 120 s of galloping at 14 m/s on a 3.5% uphill grade, arterial blood O₂ content was reduced by 10.2 ± 0.33 mL/dl at 120 s of galloping at 14 m/s on a 3.5% uphill grade in the control and nasal strip experiments, respectively. Statistically significant differences between the control and nasal strip experiments were not found at any step of the exercise protocol.

Starting with exertion at 8 m/s, the mixed venous blood O₂ content decreased significantly as work intensity increased, reaching 2.2 ± 0.2 and 2.2 ± 0.3 mL/dl at 120 s of galloping at 14 m/s on a 3.5% uphill grade in the control and nasal strip experiments, respectively. Statistically significant differences between the control and nasal strip experiments were not found at any step of the experimental protocol.

O₂ Extraction During Exercise

In both treatments, incremental exercise was attended by progressive significant increments in arterial-mixed venous blood O₂ content gradient (Fig. 4) and O₂ extraction, but statistically significant differences between the control and nasal strip experiments were not found at any step of the protocol. At 120 s of galloping at 14 m/s on a 3.5% uphill grade in the control and nasal strip experiments, the corresponding values of O₂ extraction were 91.4 ± 0.8 and 91.4 ± 1.0%, respectively.
concluded that the external nasal dilator strip did not enhance exercise performance (24). However, a reduction in \(O_2\) uptake during submaximal exercise with a nasal dilator strip (9) and a delay in the onset of oral breathing in human athletes have been reported (9, 27). This discrepancy in the human data may be related to observations being made in so-called “responders” vs. “nonresponders.” A substantial between-subject variation in the compliance of the lateral nasal vestibule wall has been reported in the human population, which may account for the reported differences between responders and nonresponders (1). Whether a similar situation exists in the Thoroughbred horse population is not known. However, as discussed below, the morphology/physiology of the equine nostril is quite different from that of the human nose.

In the present study, the highest workload was 14 m/s on a 3.5% uphill grade. That this workload indeed represented a strenuous effort for our horses was indicated by the following facts: 1) this workload elicited maximal heart rate of the horses, 2) this workload could not be sustained for >120 s, despite vigorous humane encouragement, and 3) this workload induced EIPH in all horses in both treatments.

Similar to previous reports, at submaximal workloads, our horses exhibited hyperventilation, and the arterial blood \(O_2\) tension as well as Hb-\(O_2\) saturation were well maintained in the control and nasal strip studies (Figs. 1 and 3). Several investigators have reported on the arterial hypoxemia and hypercapnia during strenuous exercise in horses (5, 6, 31, 34). In agreement with these reports, we also observed a significant reduction in arterial \(O_2\) tension and desaturation of Hb during galloping at 14 m/s on a 3.5% uphill grade in both studies (Figs. 1 and 3), but statistically significant differences between the control and nasal strip experiments were not found. Although “relative” hypoventilation (as evidenced by the increased arterial \(CO_2\) tension during galloping at 14 m/s on a 3.5% uphill grade) and ventilation-perfusion inequality also contribute, the exercise-induced arterial hypoxemia in horses is believed to be due primarily to the diffusion limitation caused by a dramatically shortened transit time for blood in the pulmonary capillaries as cardiac output increases approximately seven- to eightfold (2, 5, 6, 14, 31, 34). Because we did not observe significant benefits to gas exchange in the lungs of exercising horses on application of the nasal strip (Figs. 1–4), the question remains whether application of an external nasal dilator strip indeed decreases nasal resistance to airflow in horses. Our knowledge regarding changes in total as well as regional pulmonary resistance in exercising horses is limited and somewhat controversial. For example, whereas Art et al. (2, 3) observed a significant increase in total pulmonary resistance of galloping horses, Slocombe et al. (28) reported that significant changes in total pulmonary resistance did not occur during exercise, even though it was reported that upper airway resistance had decreased significantly during galloping. By contrast, Art and Lekeux and co-workers (2, 3, 14) presented data indicating a significant increment in the overall equine upper airway resistance as well as in its components, i.e., the nasopharyngeal and laryngeal-tracheal resistances, during heavy exercise. It should also be pointed out that catheter placement via the nares [to measure airway pressure(s) for resistance calculations] significantly affects the airflow [note: a reduction in airflow approaching ~18% in the affected nostril of galloping horses was reported by Art and Lekeux and co-workers (2, 14)] and may, therefore, not permit an accurate assessment of the nasal resistance to airflow in strenuously exercising horses. Despite some disagreement (13), it is generally believed that, during quiet breathing in resting horses, 50% of the total pulmonary resistance results from the nasal passages, 30% from the remaining upper airways, and 20% from the intrathoracic airways (14). In strenuously exercising horses, despite physiological adjustments, e.g., a dramatic dilation of the external nares, full abduction of the larynx, and bronchodilation, which increase the cross-sectional area of the respiratory tree, Art and Lekeux and colleagues (2, 3, 14) showed that the relative contribution of the various airway segments to the total pulmonary resistance does not change. However, data are not available to assess the individual contribution of the segments of the nasal passage (i.e., nostril vs. the
nasal meatus) to resistance to airflow through them in exercising horses. Because the application of an adhesive nasal strip did not significantly affect the various parameters examined in our study (Figs. 1–5), it may be argued that the application of the nasal strip may not have made a significant contribution to lowering the total pulmonary resistance of galloping horses. However, further work is warranted to document whether this is indeed the case.

The morphology of the equine nose is quite different from that of the human nose. The skeleton of the lateral wall of the horse’s nose is incomplete rostral to the nasoincisive notch, and this so-called “soft” portion of the equine nose contains a unique structure known as the false nostril. The latter is a diverticulum of the nasal passage that opens into the dorsal lateral aspect of the true nostril. In an exercising horse, the effacement of the false nostril is brought about by the action of several muscles: the dorsal and ventral levator nasi, the dilator naris lateralis, the dilator naris apicalis, and the levator nasolabialis. As the lateral nasal wall is pulled taut on contraction of these muscles in an exercising horse, the anterior nasal cavity becomes almost circular (from being comma shaped at rest). The equine nasal strip covers a portion of the false nostril, and its beneficial effect on nasal resistance to airflow during strenuous exertion may result from its ability to minimize/prevent dynamic collapse of the lateral wall of the nostrils. Although it is difficult to discern the exact reasons for the lack of a beneficial effect of the nasal strip on the aerobic (gas exchange) and anaerobic (lactate and ammonia production) variables in strenuously exercising horses in the present study, the following possibilities may be considered. 1) Because active contraction of above-mentioned muscles tightly stretches the lateral nasal wall in healthy horses performing strenuous exercise, a significant dynamic collapse of the lateral nasal wall may not occur. In fact, there have been no direct reports documenting dynamic collapse of the lateral nasal wall in strenuously exercising horses. 2) In a maximally exercising horse, the lateral nasal wall may be maximally stretched on contraction of the above-mentioned muscles, such that the application of a passive device (adhesive nasal strip) is unable to cause a further stretching of the nasal wall. 3) It is also plausible that although application of the nasal strip may help lower the nasal resistance to airflow in exercising horses, the change may be of an insufficient magnitude to significantly affect the physiological parameters examined in the present study.

In exercising horses, the significant rise in the blood temperature, hypercarbia, and marked acidosis shift the Hb-O2 dissociation curve to the right, thereby facilitating increased O2 unloading from Hb at the working muscles. The extent of hyperthermia, hypercarbia, and acidosis observed in the nasal strip study was not different from that in the control study. In the control and nasal strip studies, there was a progressive significant exercise-induced reduction of a similar magnitude in the mixed venous blood O2 tension and Hb-O2 saturation (Figs. 1 and 3). The reduction in mixed venous blood O2 tension associated with the increased O2 delivery to the working muscles helps expand the partial pressure gradient for O2 diffusion across the blood-gas barrier, thereby facilitating pulmonary O2 transfer.

In exercising horses, arterial blood O2 content is known to increase dramatically because of the significant increase in Hb concentration caused on release of the splenic erythrocyte reservoir (5, 31). The extent of the increment in arterial O2 content of horses in the present study was also similar between the control and nasal strip experiments and was accompanied by a large reduction in the mixed venous blood O2 content (Fig. 4) in both sets of experiments as O2 extraction approached 91.4%. At constant workload, the similarity of the arterial-mixed venous O2 content gradient and O2 extraction between the control and nasal strip experiments suggests that the aerobic metabolic O2 requirements were probably similar for the two sets of experiments.

In the present study, we also determined mixed venous blood lactate and plasma ammonia concentrations (Fig. 5) as indexes of anaerobic metabolism (4, 7, 8, 10, 23). This was done in the context that the reduced work of breathing (caused by lowered nasal resistance to airflow) in the nasal strip experiments would diminish the metabolic O2 requirements of the respiratory muscles, thus allowing increased O2 availability to the working muscles, which, at constant workload, should decrease reliance on anaerobic metabolism. However, this appears to have not been the case in the nasal strip experiments in the present study (Fig. 5). It is well known that strenuous exercise causes blood lactate and ammonia concentrations to increase sharply (4, 7, 8, 10, 23). It is generally agreed that increased plasma ammonia concentration during exercise results from deamination of AMP into IMP and ammonia in the working skeletal muscles and the diffusion of the latter into the blood. Ammonia production is greatest when the rate of ATP utilization exceeds the rate of ATP resynthesis (4, 7, 23). Under such circumstances, the deamination of AMP is suggested to serve an important role, in that it favors ATP production through the myokinase reaction: 2ADP → ATP + AMP. In addition, AMP deamination may contribute to the control of glycolysis (4, 23). It is also noteworthy that a significant linear relationship between blood lactate and ammonia concentrations has been demonstrated in exercising human subjects (7), and it was suggested that a connection/link between ammonia production in muscles and glycolytic energy metabolism probably exists (7, 23). In the present study, although large significant increments in blood lactate and plasma ammonia concentrations were observed in the control and nasal strip studies, statistically significant differences were not observed between the two treatments at any step of the protocol (Fig. 5). Finally, in the present study, airway endoscopy revealed that the occurrence of EIPH was also unaffected by the application of an external nasal dilator strip. All horses
had experienced EIPH in both treatments as demonstrated by the presence of fresh blood in the trachea, larynx, and/or pharynx (12, 30). The fact that statistically significant differences in the above-described parameters were not observed in strenuously exercising horses after application of the external nasal dilator strip raises doubts regarding meaningful benefits to its use in racehorses.

In conclusion, our data demonstrated that application of an external nasal dilator strip neither improved the exercise-induced arterial hypoxemia and hypercapnia nor diminished the lactate and ammonia production or the incidence of EIPH in Thoroughbreds performing strenuous exercise.

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