Exercise-induced physiological adjustments to stressful conditions in sports horses

T. Art*, P. Lekeux

Equine Sports Medicine Center, Faculty of Veterinary Medicine, University of Liège, Bât. B42, Sart Tilman, B-4000 Liège, Belgium

Abstract

Among athletic/sports animals, the horse has a unique ability to increase its oxygen uptake by a factor of 60 during heavy exercise. This is achieved by physiological adaptations of all the links in the oxygen chain. Ventilation is increased by a factor of 30. Since the horse is a compulsory nasal breather, this hyperpnea necessitates high transmural pressure changes, which may be responsible for the dynamic collapse of the airways. Blood flow is increased by a factor of 10. Since the left ventricle is not very compliant, this increase necessitates a high filling pressure in the pulmonary circulation, which may induce capillary stress failure and exercise-induced pulmonary haemorrhage. Lastly, oxygen transport is improved by splenic contraction which increases haemoglobinemia by 50%.

Sports horses frequently suffer from several problems, which are related either to endogenous or exogenous stresses experienced during their career. These stresses, caused by the use of the horse as a competition animal, may lead to several medical problems.

At a systemic level, endogenous stresses include hyperkaliemia, lactacidemia, and hyperthermia; oxidative stress may induce problems at a general, and/or a pulmonary level.

External factors, e.g. poor quality of inspired air, transport, hot and humid ambient conditions, and microbiological agents, may also induce abnormal body attacks, and lead to health problems.

© 2004 Elsevier B.V. All rights reserved.

Keywords: Horse; Exercise; Stress

1. Introduction

Horses are unique athletes within the animal kingdom. Their survival in the wild rests partly on their capacity to provide an explosive effort, when necessary, to escape from predators. Consequently, they can increase, almost instantaneously, their oxygen consumption and ventilation by factors of more than 60 (Eaton, 1994) and 30 (Art et al., 1990; Art and Lekeux, 1995a), respectively.

This increase in oxygen consumption is provided by both the increase of oxygen extraction, as
well as cardiac output (by a factor of 10) (Eaton, 1994); the latter being related to the increase in heart rate (which changes from 25–30 bpm at rest, to 220–240 bpm during maximal exercise). Another important physiological adjustment is the horse’s ability to empty its spleen by an autonomic contraction reflex at the start of exercise (Persson, 1967; Evans, 1994); a characteristics shared with the dog. This offers the possibility of doubling the number of erythrocytes and, consequently, the blood oxygen transport capacity in the same proportion.

Among the multiple physiological characteristics for exercise, horses are known to support arterial hypoxemia and, sometime, hypercapnia, during strenuous effort. This is a strategy, which seems to avoid metabolic energy wastage, while maintaining an extremely high level of ventilation (Bayly et al., 1989; Lekeux and Art, 1994).

Consequently, horses are particularly well designed for intense exercise. However, during their athletic career, sports horses frequently suffer from several problems related to either endogenous or exogenous stress. These stresses are actually caused by the use of horses as sports’ or competition “tools”, and most of them would probably never occur (at least chronically) in the wild. Stress may also lead to medical problems in the systems involved in the realisation of effort.

This paper reviews the stress experienced by sports horses, namely, those which induce respiratory problems, but excluding those leading to osteo-articular injuries.

2. What are the endogenous stresses experienced by the sports horses?

Endogenous stresses are constraints attacking the body, which are induced by the exercise-induced physiological adjustments themselves. They include: (1) changes at the blood composition level related to potassium release, lactic acid, heat produced by the working muscular cells, and oxidative stress related to the oxygen metabolism; and (2) pressure change and airflow-induced mechanical stresses at the level of the pulmonary vessels and airways.

2.1. Muscular metabolism related stresses

2.1.1. Hyperkaliemia

During muscular contraction, plasma potassium (K+) is lost from the muscle cells, and this is reflected in an increase in K+ concentration. In the horse, the K+ concentration is related to the workload (dependent of the number of cells recruited, and the frequency of recruitment); high intensity effort may lead to concentration in excess of 10 mmol/l (Harris and Snow, 1992), a level greater than those reported for maximal exercise in man (Green et al., 2000). It has been suggested that hyperkaliemia induces muscle fatigue, by decreasing the membrane potential and causing neuro-muscular conduction disturbance. Surprisingly, while K+ infusion induces electrocardiogram abnormalities, once the threshold of 7–9 mmol/l is reached (Glazier et al., 1982; Epstein, 1984), exercise-induced hyperkaliemia has no influence on cardiac function. Training attenuates exercise-induced hyperkaliemia (McCutcheon et al., 1999).

2.1.2. Hyperlactacidemia and resulting acidosis

Once the intensity of exercise reach a certain level, energy becomes partly provided by anaerobic metabolism. Consequently, acid lactic efflux, from cells to the blood, occurs, and blood lactate increases. When exercise becomes supra-maximal, blood lactate increases sharply, and may reach levels as high as 30 mmol/l after a race (Snow and Valberg, 1994). This order of blood lactate concentration results in a decline in venous blood pH to 7.000 or less. The resulting muscular acidemia may be responsible for the occurrence of muscular fatigue and dysfunction due to disturbance in mitochondrial function, impairment of glycolysis and consequent decline in muscle ATP concentration, swelling of mitochondria and sarcoplasmic reticulum. When exercise is prolonged in such situations, muscle strain may result, leading to muscle damage and myopathy (Snow and Valberg, 1994). Training increases the oxidative metabolism potential of skeletal muscle and, consequently, induces a decrease in lactate production during sub-maximal exercise. It increases the rate of lactate removal after exercise. Lastly, it improves the buffering capacity of the cytoplasm, allowing higher lactate levels to be
reached during supra-maximal exercise (McCutcheon et al., 1987).

2.1.3. Hyperthermia and resulting dehydration and ionic disorders

Muscular contraction necessitates energy, which is provided by the degradation of ATP. The conversion of stored chemical energy into mechanical energy is relatively inefficient, considering that 80% of this energy is lost as heat. Furthermore, heat must be dissipated to avoid a life-threatening body temperature rise. The horse controls this rise mainly by evaporation of sweat, vasodilatation, and, by thermal polypnea (McConaghy, 1994). Generally speaking, evaporative cooling is efficient in preventing any major body temperature rise during any kind of exercise. Nevertheless, in hot and/or humid climatic conditions, exercise-induced heat stress may occur, because heat production exceeds heat dissipation (see Section 3.4).

During endurance efforts, the main mechanism for heat removal is the evaporation of sweat. In favourable weather conditions, sweat loss can be around 5 l/h, but in hot humid conditions, it may reach values as high as 10–15 l/h. Water loss after a race (estimated by weight loss) may be as high as 40 l. The water for sweating is derived from both extracellular and intracellular fluids, and represents a loss of over 15% of total body water. Sweat is hypertonic in comparison to plasma (e.g. plasma and sweat concentrations are, respectively, 139 and 249 mmol/l for Na⁺, 3.7 and 78 mmol/l for K⁺, and 100 and 301 mmol/l for Cl⁻; Rose et al., 1980), therefore, its production is accompanied by a loss of electrolytes, such as sodium, potassium calcium, and, especially, chloride, with a consequent reduction of these ions in plasma. For example, a deficit of 25 l in extracellular fluid will be accompanied by a chloride deficit of about 4000 mmol. In order to maintain electrical neutrality, hypochloremia is associated with an increase in plasma bicarbonate, inducing metabolic alkalosis. This alteration in fluid and electrolytes status impairs performance capacity, and may even be life-threatening (McConaghy, 1994).

Regular training (and/or exposure to hot ambient conditions) will result in a number of physiologic adaptations conferring improved thermoregulatory ability: increased plasma volume, greater stability of cardio-vascular function during exercise, and improved efficiency of evaporative heat loss as a result of changes in sweating response (Geor and McCutcheon, 1998; Lindinger et al., 2000).

Therefore, although it is not possible to eliminate the effects of adverse environmental conditions on performance, it is evident that a thorough exercise training program, together with a subsequent period of acclimatization, will serve to minimize the environmental impact (Marlin et al., 1999; McCutcheon and Geor, 2000).

2.1.4. Oxidative stress

Exercise enhances the production of free radicals and, in humans, reactive oxygen species (ROS) are frequently implicated in exercise-induced tissue damage and muscle fatigue. The source of ROS is the partial reduction of oxygen in the mitochondria during oxidative phosphorylation, and increased activity of xanthine oxidase during anaerobic degradation of purine nucleotides (Sen, 1995).

On the other hand, free radicals are known to partly contribute to the development of some infectious and inflammatory disorders in the lungs (Repine et al., 1997).

Antioxidant defence mechanisms exist and can counteract the formation of oxygen radicals. Oxidative stress results from an imbalance of these defence mechanisms and the production of oxidant substances.

Because oxygen consumption and exercise capacities in the horse are superior to other species, they are very likely to experience oxidative stress (Mills and Higgins, 1997). In equine species, measurements of oxidative markers, either in blood (systemic markers) or in pulmonary epithelium lining fluid (pulmonary markers), have been conducted on healthy horses during exercise (Mills et al., 1996). Similar measurements have also been carried out on horses suffering from heaves, at different stage of their illness (either in remission or in crisis), either at rest (Art et al., 1999a), or during exercise (Art et al., 1999b). These experiments led to the conclusions that (1) horses experience oxidative stress during exercise, and that this stress is higher when exercise is performed in hot and humid environmental conditions (Mills et al., 1996), and (2) that the stress is more intense when heavy horses are in clinical crisis than in remission, both at rest and
during exercise (Art et al., 1999a,b; Kirschvink et al., 1999).

In humans, regular physical exercise is crucial to maintain and promote the natural capacity of the host to defend itself against the reactive oxygen-induced damage (Sen, 1995). In horses, the effect of training has not been reported previously. In man, several studies on antioxidant supplementation and deficiencies have been carried out (Sen, 1995). In horses, one study has reported some beneficial effects of an antioxidant cocktail on pulmonary inflammation in heavy exercising horses (Kirschvink et al., 2002a).

2.2. Mechanical stresses

Owing to several physiological and morphological characteristics, horses at exercise experience huge pressure changes in their airways, as well as in pulmonary vessels. These physical effects are so significant that they sometimes lead to rupture of the alveolar membrane.

In the horse, the morphological characteristics include the length of the airways (that induces a high airway resistance and inertance) (Art et al., 1989), and the low thoracic compliance. Physiological characteristics include compulsory nasal breathing (that lead to a high resistance to breathing during exercise) (Art et al., 1988), and locomotion–respiration coupling (that results in very high respiratory frequency during exercise). This induces dramatically high pressure changes in the airways and the intrapleural space during high intensity exercise, i.e. very negative pressure during inspiration and very high positive pressure during expiration (Art et al., 1990). These large pressures variations have two consequences: (1) in the airways, they may facilitate a partial collapse in the respiratory tree which is poorly supported by bones, cartilages or nothing; (2) at the capillary blood barrier level, where the combination of low alveolar pressure (during inspiration) and high vessel pressure (due to pulmonary hypertension) may result in rupture of the membrane and pulmonary haemorrhage.

2.2.1. Airway collapse

Equine airways are composed of the nostrils, nasal cavities, pharynx, larynx, trachea, bronchi, and bronchioles. These structures are supported, in some places, by bones (nasal cavities) or cartilages (trachea and bronchi), and in other places by muscles only (nostrils, pharynx and larynx), or are unsupported (bronchioles). Although a reduction of cross-section diameter is potentially possible anywhere in the respiratory tree, the fact that some parts are unsupported by rigid structures makes them more susceptible to collapse than other. They consequently represent a potential bottleneck that may decrease airway permeability. Studies of the inspiratory and expiratory components of the resistance values in trotting horses have shown that, during inspiration, the extra-thoracic airways account for more than 90% of the total pulmonary resistance, while, during expiration, the intra-thoracic airways are responsible for more than 50% of the pulmonary resistance (Art et al., 1988). This observation may be explained by the fact that, during exercise, a dynamic partial collapse may occur when the pressure surrounding the airways exceeds the pressure within the lumen. When a horse inhales, sub-atmospheric pressure in the extra-thoracic airways may be as low as minus 5 kPa, while the pressure in the surrounding tissues remains atmospheric. During expiration, the intra-thoracic pressure becomes greater than the pressure prevailing inside some of the intra-thoracic airways. When exposed to compressive pressures, these structures tend to collapse, with a consequent increase in their resistance to airflow.

If this collapse occurs normally in healthy horses, it can be expected to be dramatically worse in horses suffering from either upper or lower airway obstruction, a condition accompanied by substantial transmural pressure during exercise (Funkquist et al., 1988; Rehder et al., 1995; Art et al., 1998).

It has been shown that both the extra- and intra-thoracic parts of the trachea are sufficiently compliant to decrease their cross-sectional areas when subjected to high, but nevertheless physiological, compressive transmural pressures (Art and Lekeux, 1991a). Moreover, the shape of the cross-sectional area of the individual trachea significantly influences collapsibility: tracheae with a circular cross-sectional shape are less compressible than tracheae with a more ellipsoidal shape (Art and Lekeux, 1991b). This is particularly important in view of the variability observed in this shape among individual horses—some horses being, probably, more susceptible than others to dynamic tracheal collapse.
2.2.2. Exercise-induced pulmonary haemorrhages

Marked exercise-induced pulmonary hypertension during exercise is a specific feature in horses: the mean pulmonary arterial pressure rises about 3-fold, from 28 mm Hg at rest to about 84 mm Hg at a fast gallop, with maximal reported values of 100 mm Hg (Evans and Rose, 1988; Wagner et al., 1989; Erickson et al., 1990; Manohar, 1993). There is also a significant increase in pulmonary capillary, wedge and venous pressure (Manohar and Goetz, 1996). The factors that increase vascular pressure in the horse are not clear. The very high left atrial pressure, necessary to rapid ventricular filling,—in this species the heart rate may increase up to 240 bpm—could be partly responsible.

In man, blood flow distribution is mainly influenced by gravity and, consequently, shows a vertical gradient according to the relative magnitude of pulmonary arterial, venous and alveolar pressures (Amis et al., 1984; Fishman, 1985). It has been long thought that the same was true for large quadrupeds, but recent work has provided evidence that, in these animals, gravity is a minor factor determining blood flow distribution, even at rest, despite the large hydrostatic gradient imposed by the height of the lung. Blood flow is preferentially distributed in the dorsal regions of the lung (Hlastala et al., 1996), a phenomenon which is exacerbated during exercise (Bernard et al., 1996). Because most of the affected areas appear to be distributed in the dorso-basal broncho-pulmonary segments, blood perfusion distribution may be related to the preferential site for pulmonary bleeding.

During heavy exercise, alveolar pressure is likely to become highly negative (Art et al., 1990), while pressure in the vessels (pulmonary or bronchial) increases greatly. Consequently, transmural pressure, i.e. the pressure gradient between airways and blood vessels, is expected to reach high values. Moreover, because of poor collateral ventilation, the transmural pressure in asynchronous regions is suspected of becoming sufficiently high to cause capillary rupture.

Why capillary rupture occurs, rather than pulmonary edema, may be explained by the fact that both alveolar and pulmonary capillary blood pressures are subjected to frequent and cyclic variations, due to respiration and cardiac contraction, respectively. Consequently, transmural pressure (between pulmonary circulation and alveoli or between bronchial circulation and alveoli) is expected to display important, irregular and sudden variations, which could cause capillary rupture, rather than a continuous high value causing pulmonary edema.

While the high transmural pressure is the cause of the rupture, other factors may favour the occurrence of this phenomenon. Some factors are likely to decrease the stress resistance of the pulmonary tissues. On the one hand, it has been shown that lung injury and hemorrhage may result from inflammatory processes, mainly due to a local release of toxic products (heparin, oxygen metabolites) by phagocytic cells, like neutrophils or macrophages. On the other hand, it has been shown in rabbits that alveolar hypoventilation increases stress failure of pulmonary capillaries, and may cause disruption of the capillary endothelium, alveolar epithelium, or occasionally all layers of the wall (Fu et al., 1992). If the same is true in the horse, the alveoli of the dorso-caudal regions of the lung, which are more distended because of gravitational effects, should also be more fragile.

Therefore, the presence of chronic pulmonary inflammatory processes, the increase in lung volume during high exercise intensity, or both factors together, may be partly responsible for increased blood vessel fragility and, therefore, for the occurrence of “bleeding”.

There remain many unanswered questions regarding exercise-induced pulmonary haemorrhage. In particular, it is unclear whether they have functional consequences for respiration or performance. Training has probably no influence of the occurrence of pulmonary haemorrhage. Moreover, researchers are faced with the difficulty of experimentally reproducing pulmonary haemorrhage. This inability to specify the severity and frequency of their occurrence probably explains the discrepancies between the conclusions from field studies on the effect of bleeding on performance (Roberts, 1998), as well as on proposed preventive therapeutics (such as furosemide administration, Erickson et al., 1999), or tools (such as the nasal strip; Erickson et al., 2000).
3. What are the exogenous, i.e. environmentally related, stresses experienced by the sports horses?

These stresses are related to environmental hazards, either capable of inducing respiratory disorders after inhalation, or disturbing thermal homeostasis. It must be pointed out that these external assaults (?) will be facilitated by some of the previously cited endogenous stresses. For example, pulmonary hemorrhage, or lower airway oxidative stress, will facilitate the occurrence of pulmonary inflammation by inhalation of dust, or of pulmonary infection by inhalation of virus.

3.1. Biological agents

Exercise is recognized as a stress which can significantly alter the host’s immunity response and, therefore, increase its susceptibility to diseases (Hines et al., 1996). In horses, deleterious effects of exercise on systemic immunity are mainly observed either during strenuous effort or overtraining, while moderate training could have a rather beneficial effect on defence mechanisms. It seems, therefore, that exercise stress may have a dual effect on the immune system according to the intensity and chronicity of the work (Hines et al., 1996; Raidal et al., 2000, 2001). Young horses in training are less resistant than older ones to exercise-induced immunosuppression, perhaps, because their post-exercise cortisol concentration is higher (Horohov et al., 1999). Unfortunately for our sports horses, they are generally young, and light training is rarely used; most of them are submitted to a workload that would, most probably, decrease their resistance to infection rather than to increase it.

On the other hand, the functional capacity of pulmonary alveolar macrophages and bronchoalveolar lavage-derived lymphocytes are significantly affected by training, suggesting that pulmonary immune function may be impaired in sports horses (Folsom et al., 2001; Lunn et al., 2001).

The consequences of this relative exercise- and training-induced immunosuppression is that bacterial and viral agents found a favourable growth habitat in young sports horses. They (!means the biological agents!) are, therefore, frequently responsible for respiratory problems. International competitions, implying frequent transport over long distances, and concentration in the same location of horses coming from different countries contribute most probably to the propagation of viral infections.

The occurrence of an infectious pulmonary disease depends on the level of airborne bacteria, viruses, or mycoplasma, on the pathogenicity of a given agent, and on the resistance of the host. Prevention of infectious respiratory diseases is difficult, given that these diseases are highly contagious (especially equine influenza), the fact that many are endemic, their ability to be transmitted by routes other than aerosol, and the fact that asymptomatic carriers, or horses during the incubation phase, may transmit infections. Stable design and management is unlikely to reduce the incidence of highly infectious respiratory diseases, such as influenza; however, it may decrease the transmission of less contagious infections. Regular disinfection of stables is recommended to prevent the persistence of infectious agents. Viral infection may be prevented by the appropriate use of vaccines.

3.2. Transport stress

The study of the effects of transportation on stress indices, lower respiratory tract contamination, and on peripheral blood neutrophil function, have shown that the physiological responses, occurring during long-term transport, include changes in muscle metabolism, stress indices, immune parameters, and body weight (Smith et al., 1996; Friend et al., 1998; Friend, 2000). These reactions most probably increase disease susceptibility, and influence energy available for athletic performance (Stull and Rodiek, 2000). They undoubtedly favour bacterial contamination of the lower respiratory tract, being, consequently, an important determinant in the development of respiratory diseases. Reduction in peripheral neutrophil phagocytic activity is evident up to 36 h after transportation (Raidal et al., 1997), pointing to the fact that horses may require a number of day to recover from the stress of transportation.

3.3. Airborne particles

Of all the organs, the respiratory tract presents the largest and most delicate surface for contact with
potentially deleterious airborne material. Indeed, the 
alveolar and bronchiolar areas, which in the adult 
horse equal approximately 100 m² at end expiration 
and 300 m² at end inspiration, are exposed to 
~30 x 10⁶ l of air annually. Consequently, breathing 
exposes the lungs to the potentially adverse effects of 
a complex mixture of gaseous and particulate pollu-
tants. The potential noxious consequences are diverse, 
and the resulting effects on health may be immediate 
or delayed.

Respiratory tract defences are, therefore, of para-
mount importance in the maintenance of respiratory 
health. Consequently, several specific (primarily for 
biological agents) or nonspecific (including defences 
against inorganic agents) mechanisms have evolved to 
remove inhaled agents and prevent their deleterious 
consequences. Unfortunately, the efficiency of these 
defences may be reduced, namely, by chronic realisa-
tion of exercise, as mentioned previously. They may 
be overwhelmed by extreme levels of exposure, or 
impaired by airway diseases.

Several equine respiratory disorders are directly 
caused by, or exacerbated by, inhalation of airborne 
dust. The most widely recognised disorder affecting 
adult horses is heaves (previously termed chronic 
obstructive pulmonary disease or recurrent airway 
obstruction). Heaves is caused by inhalation of 
agents in airborne organic dust, including moulds 
and bacterial endotoxins. Inflammatory airway dis-
eedisease, another chronic pulmonary disease mainly 
encountered in young race horses subjected to 
strenuous exercise, may also be associated with 
inhalation of airborne pollutants, including micro-
organisms and organic dusts (Burrell, 1985; Clarke 
et al., 1987). Additionally, pharyngeal lymphoid 
hyperplasia has been linked with inhalation of 
airborne dust in some studies, but not in others 
(Clarke et al., 1987). Acute respiratory diseases, 
including those caused by infectious agents, may be 
exacerbated and prolonged by concomitant dust 
inhalation.

Keeping horses permanently on pasture, with a 
shelter against inclement weather and no supplemen-
tary hay feeding, is the ideal dust-free regime. 
Unfortunately, for numerous practical reasons, sport 
horses are often stabled indoors. Therefore, a strict 
control of stable dust levels, by use of dust-free food 
and bedding, is essential if heaves horses are to 
remain in clinical remission (Vandenput et al., 
1998a,b).

Some degree of fungal contamination is present in 
all batches of hay, regardless of their quality. Hay that 
has heated and is very dusty may contain very high 
levels of many different pro-inflammatory agents, 
including mould spores, bacteria, endotoxins, protei-
nases, and forage mites (Clarke and Madelin, 1987; 
Woods et al., 1993). For example, a horse consuming 
heated hay may inhale 10¹⁰ dust particles per breath 
(Clarke and Madelin, 1987).

Artificial drying of hay, using a drying kiln with air 
blowers, or rapid high-temperature drying immedi-
ately after cutting, may significantly decrease micro-
bial growth. Soaking hay reduces the respirable dust 
challenge, although it does not always reduce the dust 
challenge to a level that results in complete resolution 
of symptoms of heaves. Soaking is efficient only 
when it is done for several hours and when the hay is 
completely soaked (with the bale strings cut). How-
ever, prolonged soaking of hay reduces its nutritive 
content due to leaching of soluble nutrients. The water 
used to soak the hay should be changed daily to 
prevent build-up of endotoxins and microbes. The 
dust content of hay may alternatively be reduced by 
mechanical agitation in conjunction with vacuum 
removal of liberated airborne particles.

Complete pelleted diets are a suitable alternative to 
forages since they contain all the required nutrients, 
have a very low dust content, and contain few mould 
spores (Raymond et al., 1994; Vandenput et al., 1997). 
Unfortunately, some horses dislike pellets, and since 
they are consumed very quickly, they may promote 
the development of stable vices.

Silage is currently the best alternative to hay 
(Vandenput et al., 1997, 1998a,b). Increasing numbers 
of horses are being fed on silage to minimise airborne 
dust, regardless of whether they suffer from heaves or 
not. Silage should not be fed to horses if it has a smell 
of ammonia, if it comes from a damaged bag, or if the 
bale was opened more than 5 days previously. The
risk of botulism may be minimised by feeding commercial silage (e.g. Horshage™), which is carefully harvested, often treated with preservatives, and ensiled at high dry matter (please clarify).

Bedding is also an important source of airborne fungal spores, endotoxins, and other pro-inflammatory agents. Wood shavings, paper bedding, peat moss and sawdust are suitable alternatives to straw (Vandenput et al., 1997; Ward et al., 2001). However, as with straw, they can become a source of moulds and endotoxins when used in deep-litter systems, or in warm and poorly ventilated stables (Clarke, 1987). Flax straw has a very low dust content, but, unfortunately, it may cause intestinal obstruction when eaten. Cardboard bedding (e.g. Ecobed™), and large wood shavings produced especially for horse bedding (e.g. Cleanbox™), are almost free of contaminants and are, thus, useful low-dust bedding materials (Kirschvink et al., 2002b). Non-biological beddings are very clean, thermally efficient and, unlike the aforementioned organic beddings, do not provide a medium for fungal growth. They are, however, expensive and some of them, such as plastic bedding, need to be cleaned, thus requiring additional labour (Thompson, 1995).

3.4. Climatic conditions

The risk of serious hyperthermia occurs when exercise is undertaken in hot and humid ambient conditions, and/or when the horse is inadequately trained for the performed exercise level, and/or when there is an impairment in the thermoregulatory mechanisms (dehydration, etc.). When body temperature rises excessively, the demands of muscle metabolism and skin blood flow for heat dissipation arise concurrently (Art and Lekeux, 1995b), at the risk of muscle perfusion, with deleterious consequences. Water and electrolytes losses are considerable, especially during long-term effort, and a number of clinical problems may result: exhausting syndrome, synchronous diaphragmatic flutter, heat stroke, rhabdomyolysis, and dry-coated. Good physical preparation, heat acclimatization, reduction of exercise intensity or duration, electrolyte supplementation, and appropriate watering and cooling during competition, may help to prevent the occurrence of serious health problems.

4. Conclusions

Horses are well “designed” to perform high level physical exercise. The chronic realisation of effort, especially those that are exhausting, either in speed or in endurance, may induce endogenous stresses that may, in some circumstances, have deleterious effects. These endogenous stresses may impair the body’s defences, and favour or facilitate attack by exogenous agents, such as viruses and bacteria. Moreover, the modus vivendi, related to the use of the horse as a sports and competition animal, i.e. living in closed boxes and eating dry concentrates and forages, transported for long periods, is responsible for exogenous attacks.

A thorough knowledge of these stresses, their origins and mechanisms, may help owners and trainers to minimize their occurrence, at least in some cases, by appropriate training and competition programs, progressive acclimatization, strict environmental management, or by appropriate preventive treatments (vaccines, diet supplementation, etc.).

References


modify the cardiorespiratory and ventilatory adjustments to exercise in horses? J. Appl. Physiol. 84, 845–852.


