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Electrolytes, Cations, and Anions in the Performance Horse

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Introduction

Few activities rival the thrill of equine competitions, whether it be a down to the wire stretch drive between Thoroughbreds, a Quarter Horse out manoeuvring a cow in a cutting competition, an Arabian endurance horse completing a 100 mile ride with seemingly little effort, or a European Warmblood completing a gruelling cross country course. The horse's capacity for work is truly amazing. The fundamental mechanisms of these highly developed and complex systems required to perform such feats are just beginning to be understood. Training and genetics are major contributors to performance, but nutrition has received wide recognition as a determining factor in the success or failure of equine athletes.

Of the nutrients, electrolytes, both cations and anions, are somewhat unique in their ability to affect performance. Not only do electrolytes have effects individually, because of their propensity to be ionized, they have the ability to interact with one another to create additional effects. Strictly defined, an electrolyte is a chemical compound that ionizes when dissolved or molten to produce an electrically conductive medium. Physiologically, electrolytes are required to regulate the electric charge across cell membranes and participate in a number of reactions necessary for life. Sodium, potassium, and chloride are the minerals most often thought of when the term electrolyte is used, although calcium, phosphorus, magnesium, and sulphur may also exist in ionized states in the body. Other compounds such as lactate, bicarbonate, and even proteins may act as electrolytes as well. A complete treatment of all of these compounds is beyond the scope of this paper and therefore the focus will be limited primarily to sodium, potassium, and chloride.

Maintenance and Exercise Requirements

The maintenance requirements for sodium and potassium have been relatively well-established [1] and are listed at 20 and 50 mg/kg of body weight per day (bw/d) respectively. Data to establish the maintenance requirement for chloride is somewhat scarce. One study suggested that 20 mg/kg BW/d is required to replace endogenous losses and that as much as 80 mg/kg BW/d is required to maintain acid base status[2]. The effects of varying amounts and ratios of electrolytes will be discussed in detail later. The requirements for exercise are largely dependent on the amount of each lost in sweat. Since roughly 90 percent of the weight lost during exercise is sweat[3], one could theoretically calculate the requirement for exercise by knowing the weight lost during exercise and multiplying by the concentration of each mineral in sweat. That is the approach most nutritionists employ in attempting to define the requirements for exercise in spite of the fact that the sweating rate and the concentration of minerals in sweat are not constant. Despite the shortcomings of that method, it is easy to see that under extreme conditions, the requirement for potassium, sodium, and chloride could easily be as much as three to four times maintenance. If a deficiency of one of the electrolytes occurs, the effects on acid-base status and performance can be dramatic.

Review of Acid Base Chemistry and Buffering Capacity

The body has several mechanisms that work in concert to maintain a homeostatic condition of parameters including temperature, osmolarity, and pH of body fluids. Electrolytes play a critical role in those mechanisms. The body maintains these parameters within a fairly narrow range and should any one of these remain outside of the "normal" range for an extended period of time, the horse becomes a very fragile creature fighting for survival. While temperature regulation is critical and does play a role in acid-base physiology, osmolarity and pH will be the focus of this discussion as both are important in understanding acid-base balance and mineral metabolism. Many of the concepts given here are derived from the work of Peter Stewart[4], a medical physicist, who developed the concept of strong ion difference as a way to explain changes in acid-base status in mammals.

Osmolarity. Sodium, potassium and chloride are the major ions involved in the regulation of osmotic pressure in body fluids. Osmolarity is a measure of the osmotic pressure and is determined by the number of particles dissolved in those fluids. The osmolarity of extra cellular fluid (ECF) is approximately 300 milliosmols/liter. That means that 300 milliequivalents (mEq) of solute (particles) are contained in each liter (1,000 cc) of ECF. Of that total, roughly 140 mEq are sodium, 110 mEq are chloride, 28 mEq are bicarbonate ions, 3 to 5 mEq are potassium, 2.5 mEq are calcium, and 5 to 6 mEq are glucose. The balance is a combination of plasma proteins, free amino acids, urea, sulphates, phosphates, and lactate. Obviously, sodium and chloride are the largest components affecting osmolarity, with the bicarbonate ion a distant third. The osmolarity of intracellular fluid is similar, except that potassium is the predominant particle within cells and sodium and chloride are at very low levels. The amounts of the various components of plasma can be affected by diet, exercise and management. However, it is important to remember that the total osmolarity of ECF will always be near 300 milliosmols/liter and the body continuously monitors and adjusts the concentrations of those components to maintain a homeostatic condition.

Blood pH. The pH of the blood is affected by acids and bases. An acid is any compound capable of donating a hydrogen ion to the solution and a base is any compound capable of accepting a hydrogen ion from the solution. The strength of an acid or base is measured by its ability to donate or accept hydrogen ions at the pH of body fluids. Hydrochloric acid, for example, is a strong acid because it is completely dissociated into a hydrogen ion (H^+) and a chloride ion (Cl^-) at a pH of 7.0. In other words, it is a strong hydrogen ion donor. Other strong acids normally found in body fluids include lactic acid, phosphoric acid, sulphuric acid and acetic acid. On the other hand, carbonic acid (H_2CO_3) is a weak acid because it tends to stay in that form at normal body pH and not dissociate as easily into a hydrogen ion (H^+) and bicarbonate ion (HCO_3^-). An ion with a positive charge is referred to as a cation and an ion with a negative charge is referred to as an anion. The sum of all the charges theoretically must be zero. In other words, for each cation in the body, there must be an associated anion. They may not be in the same place, i.e. the cation in the plasma and the anion inside of a cell, but the body must be electrically "neutral".

The normal pH of venous blood is 7.4. If the pH is higher than 7.4 then a state of alkalosis is said to exist and if the pH is lower than 7.4, a state of acidosis is said to exist. Certainly, there are varying degrees of each of these conditions and for a given individual, "normal" might be slightly above or below 7.4. Since pH is defined as the negative logarithm of the hydrogen ion concentration, a small change in the pH represents a significant change in the number of free hydrogen ions present. For example, if the pH changed from 7.4 down to 6.4, that would represent a 1000% increase in the hydrogen ion concentration and an acidic condition that certainly would be life threatening. The magnitude of pH change normally seen in sedentary horses is on the order of ± 0.1. In comparison, the exercising horse might experience a pH as low as 6.8 shortly after an extremely intense work, due to the accumulation of metabolic acids, primarily lactate. Even in this extreme condition the body quickly takes steps to reduce the concentration of free hydrogen ions and return the pH back to normal.

The pH of body fluids is tightly regulated. There are two primary mechanisms that accomplish this task. The first mechanism, which is relatively quick, is the use of a buffer to "capture" the free hydrogen ions. Buffers or more correctly buffer pairs serve to accept hydrogen ions and thereby help maintain pH in a narrow range when acids from metabolic processes are added to the system. Buffer pairs consist of a weak acid and the salt of that weak acid. There are four major buffer pairs and of these, the most important in plasma is the carbonic acid-sodium bicarbonate pair. As was stated earlier, plasma contains about 28 mEq of bicarbonate, six times the amount of the phosphate buffer pair, which is next highest in concentration.

The second mechanism is to convert the compound or compounds that are responsible for the free hydrogen ions into something less acidic that can either be metabolized or excreted from the body. This process is much slower and usually requires oxygen. For example, an anaerobically exercised horse produces lactic acid, a very strong acid that must be converted back into pyruvic acid. Pyruvic acid, a much weaker acid, can then be metabolized with oxygen to produce energy. While this process occurs, the hydrogen ions associated with lactate can be partially buffered by sodium bicarbonate. However, a significant number of free hydrogen ions remain free because the amount of sodium bicarbonate in the blood is limited and the level of lactate that can be accumulated during anaerobic metabolism is quite high. As a result, pH of blood can decrease to as low as 6.8. Therefore, anything that can be done to increase the amount of sodium bicarbonate in the blood has the potential to minimize changes in blood pH and potentially improve the performance of the horse. Both mechanisms are very important, but the buffer system has received the most attention in recent research because of the reported improvements to the anaerobic performance of horses orally dosed with sodium bicarbonate.

Role of respiration. The sodium bicarbonate pair also serves as a major transport mechanism for the carbon dioxide (CO_2) generated during energy metabolism. The production of CO_2 from energy metabolism accounts for most of the metabolic acid generation in the body. There are three routes by which CO_2 can be transported from the tissues to the lungs: 1) attached to hemoglobin, 2) dissolved in plasma or 3) in the form of sodium bicarbonate. The latter is by far the most important mechanism. As energy is metabolized into CO_2 and water (H_2O), the two combine to form carbonic acid (H_2CO_3), a weak acid, which then partially dissociates into H+ and bicarbonate (HCO_3^-). Since this results in some unbuffered hydrogen ions, pH decreases. As such, this process is said to be acidogenic. The HCO_3^- then combines with a sodium ion (Na^+) in the plasma to form sodium bicarbonate ($NaHCO_3$). The sodium bicarbonate is then transported by the blood to the lungs where the process is reversed. The $NaHCO_3$ combines with H^+ to form $H2CO_3$ and Na^+. The carbonic acid then dissociates, resulting in the reformation of CO_2 and H_2O. The CO_2 is then ventilated from the lungs and the net result is a decrease in the hydrogen ion concentration and an increase in pH. Therefore, respiration is another important aspect of the control of acid base balance.

Role of the kidneys. The kidneys also play a role in the regulation of acid-base balance and osmolarity. They help maintain the concentrations of sodium, potassium and chloride in the blood by reabsorbing or filtering out those minerals as necessary. The primary route of elimination of dietary excesses of these three minerals is through the kidney. It is noteworthy that the availability of these minerals from the diet is very high. Chloride, potassium and sodium availability from the diet may be as high as 100 percent, 85 percent and 70 percent, respectively.

Methods Used to Manipulate the Acid Base Status

One relatively new area of research with horses is that of dietary cation-anion difference (DCAD) and its effects on acid-base status, buffering capacity of the blood and metabolism of minerals such as calcium. The impetus for these investigations has come partially from work on sodium bicarbonate dosing in Standardbreds [5,6] and from investigations on the effects of dietary cation-anion changes in other species [7,8,9].

Sodium bicarbonate dosing. As was previously mentioned, sodium bicarbonate dosing has been shown to improve the anaerobic performance of some horses. The effects are not consistent and all horses may not benefit. Any improvement is likely due to increased buffering capacity of the blood because of elevated bicarbonate levels. Normal bicarbonate concentrations are in the range of 28 mEq/liter. Horses that have been dosed with 1000 mg/kg of body weight may have bicarbonate concentrations approaching 40 mEq/liter[6]. Plasma concentrations of sodium also increase significantly. However, as the osmolarity of the blood must be maintained, the increase in sodium and bicarbonate are compensated for by decreases in potassium, calcium and chloride. These effects are potentially harmful, particularly for horses that are marginally dehydrated or on diuretics such as furosemide (Lasix)[10]. These horses can potentially experience muscle tremors, synchronous diaphragmatic flutter (thumps) and in severe cases, stoppage of the heart. Because of the potential for altering the performance if a horse and influencing the outcome of races or other competitions, many racing jurisdictions and sporting organizations have banned the pre-performance administration of sodium bicarbonate. It is evident that the disadvantages of sodium bicarbonate dosing are numerous. It is not recommended even in situations where it is permitted, particularly when one considers that there may be a safer way to increase the buffering capacity of the blood.

Dietary Cation Anion Difference. Levels of bicarbonate in the blood are closely related to the amount of sodium bicarbonate given. It would appear that the increase in bicarbonate levels in the blood after sodium bicarbonate dosing could be directly attributed to absorption of the bicarbonate contained in the sodium bicarbonate. However, the sodium bicarbonate given by naso-gastric tube is immediately titrated by HCl in the stomach to produce sodium chloride and carbonic acid: $NaHCO_3 + HCl$? $NaCl + H2CO_3$. The carbonic acid then dissociates into H_2O and CO_2. Sodium chloride is then absorbed from the gastrointestinal tract and dissociates in the luminal epithelium into sodium and chloride. The chloride may be recycled back into the parietal cells of the stomach in exchange for a bicarbonate ion. The bicarbonate ion then combines with the sodium ion to form sodium bicarbonate once again. The net result is an increase in the sodium bicarbonate concentrations; however it is not the same bicarbonate that was fed initially. This implies that it is the absorption of sodium that results in the increased buffering capacity rather than the bicarbonate per se and the ingestion of other sodium salts may have the same effect. A similar situation exists with potassium. Therefore, the absorption of sodium or potassium, whether it be from a single dosing or fed in the diet, should raise pH and increase the buffering capacity of the total system. A number of trials conducted with sedentary and exercising horses investigating various salts of sodium and potassium to test this hypothesis have been conducted and will be discussed in a following section.

Chloride absorbed from the gastrointestinal tract, on the other hand, tends to lower pH because of decreased bicarbonate concentrations and increased acid production. This occurs because of a phenomenon known as anion gap. The sum of the charges of $(Na^+ + K^+) - (Cl^- + HCO_3^-)$ in plasma is always positive and tends to remain relatively constant. In other words there are always more cations than anions. If the concentration of one of the anions increases, the other must decrease in order to maintain the anion gap. So, as chloride is absorbed, bicarbonate must decrease. Because there is less bicarbonate to combine with free hydrogen ions to form carbonic acid, the hydrogen ion concentration increases, resulting in a decline of pH. Sulphates in the diet tend to have the same effect although not to as great a degree, mainly because they are not as readily absorbed from the gastrointestinal tract.

The overall acid or base generating power of the diet can then be calculated as the milliequivalents of sodium and potassium minus the milliequivalents of chloride plus sulfur ((Na +K) - (Cl +S)). This is referred to as Dietary Cation-Anion Difference (DCAD) and is calculated by multiplying the percent of each of those four minerals by the gram equivalent weight of each. That is then expressed per unit of dietary dry matter. A higher number indicates a diet has more base generating power, the lower the number the more acid generating power. Some nutritionists express DCAD per kilogram of diet dry matter while others prefer to express the total per 100 grams of dietary dry matter. Diets in the range of

200 to 250 mEq/kg diet dry matter are considered to be "neutral". Unsupplemented high concentrate diets tend to be below 150 and are therefore acidogenic. High forage diets tend to be above 250 and are generally base producing.

Research on the Effects of Dietary Cation Difference of Equine Diets

The effects of DCAD on production variables in other species have been widely investigated. Diets with a high DCAD have been shown to increase eggshell quality[7], improve milk production[8], and increase feed efficiency in swine[9]. In all these species, diets with a low DCAD have been show to decrease blood pH, decrease blood bicarbonate concentrations and increase the amount of calcium lost in the urine. In addition, significant increases in the incidence of tibial dischondroplasia, a metabolic bone disorder, have been demonstrated in broilers fed highly anionic (low DCAD) diets[7]. From these and other studies, it is obvious that DCAD is a highly significant factor in the efficiency of production, the skeletal soundness and longevity of other species. It is logical to assume that horses would be affected in a similar manner.

Investigations of DCAD focused on the horse are relatively recent. The data from these trials clearly indicate that horses are affected by changes in the DCAD in the same manner as other species[11-20]. Blood pH, blood bicarbonate concentrations and urine pH tend to increase linearly with increasing DCAD across the range of balances investigated to this point (-50 to 400 mEq/kg diet dry matter). Dry matter digestibility is also adversely affected by diets with a low DCAD[16, 17]. Calcium balance follows a similar trend and approaches a negative value even though diets used in these trails were designed with 50 to 75 percent excess calcium intakes. The calcium losses are primarily in the urine and are thought to be the result of an increase in the ratio of ionized to protein bound calcium in the blood due to lowered blood pH. This allows the kidney to more easily filter calcium out of the blood and eliminate it in the urine. Low DCAD may also increase osteoclastic activity in the bone, resulting in mobilization of skeletal reserves in an effort to maintain circulating blood calcium concentrations[19].

One of the most important questions answered in these trials was that the effects are caused by the strong cations and anions and not by the salts or the other dietary components. Several trials investigated both the citrate and bicarbonate salts sodium and potassium and demonstrated that regardless of the salt, effects on blood pH, bicarbonate concentration in the blood, and urine pH were identical. Additionally, various sources of chloride and sulphur were tested for acidogenic effects and again, the results were similar regardless of source. Sulphur was determined to have approximately 60% of the effect of chloride in lowering urine and blood pH and therefore the formula for DCAD is often modified to account for this difference[18].

The effects of DCAD have clearly been shown to be independent of the rest of the diet. In a trial using corn, oats, and alfalfa hay fed at either a high (330) or low (130) DCAD, blood and urine pH, and bicarbonate concentration were determined by DCAD and not the dietary constituents[20]. As a result, it appears that the supposed acidogenic effects of diets that are high in starch are due to a low DCAD and not the production of lactic acid in the hindgut. Obviously, if starch is fed in amounts sufficient enough to result in large quantities escaping digestion in the small intestine, the buffering effects of a high DCAD could be overwhelmed. Most of the studies to date have investigated the short-term effects of DCAD, which may be transitory. Long-term studies are currently underway with mature horses in an effort to study any compensatory mechanisms the horse may possess. Long-term studies in growing horses suggest that horses can compensate for DCADs that are very low or very high[19].

Perhaps the most significant finding, however, is that increasing the DCAD of the diet increases the buffering capacity of the blood sufficiently to improve anaerobic performance and speed recovery from exercise in horses running a mile at heart rates above 200 beats per minute[15]. Interestingly, horses fed the highly cationic diets (high DCAD) had higher concentrations of lactate in the blood, yet the pH of blood was unaffected. The higher lactate concentrations may be due to increased work effort. However it

is more likely due to an improved ability to remove lactate from muscle cells because of a higher concentration of hydrogen ion acceptors such as bicarbonate. In either case, the result was significantly faster times, higher lactate levels, unchanged blood pH and surprisingly a quicker recovery of heart rate. At a minimum, these data indicate that diets with a low DCAD certainly depress anaerobic performance. It further appears that anaerobic performance may be enhanced without the previously mentioned deleterious effects of sodium bicarbonate dosing.

The effects of DCAD on aerobic or endurance types of events are less clear. Since endurance horses tend to blow off excess amounts of CO_2 and suffer from a metabolic alkalosis, one could speculate that high DCAD diets are contra-indicated. That does not mean that electrolytes should be limited, it simply means that the balance of cations and anions needs to be monitored. Fortunately, the uses of chloride salts of sodium and potassium have no effect on the DCAD equation. Table 1 summarizes the effects of high and low DCAD on selected variables.

Table 1. Summary of Effects of DCAD on Selected Variables in Horses

Variable	Low DCAB (<100)	High DCAB (>250)
Blood pH	Decreased (7.35)	Increased (7.44)
Blood bicarbonate	Decreased (26 mEq/l)	Increased (32-36 mEq/l)
Urinary calcium	Very high	Decreased
Calcium balance	Very Low (near 0)	Increased (>15 g/d)
Dry matter digestibility	Decreased (60%)	Increased (66%)
Lactate Concentration (After strenuous exercise)	Lowered (60 mg/dl)	Elevated (80 mg/dl)
Recovery heart rate (After strenuous exercise)	Slowed	Quickened

DCAD of Commonly Fed Feeds in the USA

DCAD of sampled feedstuffs. Samples of forages and grains commonly fed to horses in the central United States were analyzed for mineral content in 1991[21]. The DCAD was calculated on individual feedstuffs and are shown in Table 2.

Table 2. Dietary Cation Anion Difference of Selected Feedstuffs[1]

Feedstuff	DCAD (mEq/kg diet dry matter)
Grains and grain mixes	
Oats	80
Corn	53
Soybean meal	466
Alfalfa (pelleted)	513
Grain mix (regionally milled)	172
Grain mix (national milled)	160
Pelleted concentrate	173
Complete mix	292
Hays (warm season)	
Native Prairie grass	50-150
Bermudagrass	200-250
Alfalfa	200-400

[1]DCAD calculated as mEq/kg diet dry matter (Na + K) - (Cl + S)

Based on the evidence from the sampled feedstuffs:

- DCADs of total rations can not be predicted with a large amount of accuracy because of the variability of mineral content of hays and grains.
- Commercially prepared grain mixes with added minerals will increase the DCAD of rations above values using only whole grains such as oats.
- Protein supplements with large potassium concentrations such as alfalfa pellets and soybean meal will increase the DCAD of most diets.
- Many rations found in practice are in the range of DCADs which have been shown to cause high calcium losses an negative effects on performance.
- The possibility of increased calcium loss in the rations with DCADs below 200 to 250 may have implications on the incidence of bone growth disorders and performance of horses.

In summary, electrolytes are necessary to maintain acid-base homeostasis and manage the conduction of actions potentials in nerve and muscle cells. They are also important as co-factors in many metabolic reactions necessary for life. A deficiency or excess of many of the electrolytes can be life threatening. Their effects on performance are not well understood because of the complex interactions between electrolytes and other diet constituents. Nevertheless, diet formulation that considers DCAD and at least provides a balance of cations and anions in a reasonable range is essential for performance horses.

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