# The basic bones of calcium metabolism

## A three-part series by Susan Garlinghouse, DVM

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PART 1

**How calcium levels are regulated in the body, and their relationship to certain syndromes seen in endurance horses, including synchronous diaphragmatic flutter ("thumps"), tying up and fatigue**

Trends seem to come and go in the never-ending search to find better ways to manage our competition horses. Not surprisingly, conversations often center around the use, misuse and manipulation of electrolyte balance.

Although the primary electrolytes lost in sweat during sustained exercise are sodium, chloride and potassium, small amounts of calcium and magnesium are also excreted. Excessive derangements in any or all of these electrolytes can cause significant trouble, including contributions to colic, rhabdomyolysis ("tying up"), hyperthermia, synchronous diaphragmatic flutter ("thumps"), cardiac arrhythmias, exhausted horse syndrome and other related metabolic issues.

As such, there are numerous commercial and home-made formulations available to the endurance rider for the intent of replacing lost electrolytes during or following exercise. Some riders administer significant amounts through various means, while others prefer to simply offer good quality hay (itself a good source of potassium, with varying amounts of calcium and magnesium and generally fairly minimal amounts of sodium) and free-choice salt.

On the increase is the use of concentrated calcium solutions, gels or "drenches" formulated primarily for cattle for post-calving hypocalcemia (low plasma calcium levels, also referred to as "milk fever") or hypomagnesemia (low plasma magnesium levels, or "grass tetany"). Although the disease presentation and treatment is different in cattle, these products can be administered effectively to endurance horses under some circumstances.

However, as with many other supplements and practices, a good working knowledge of the physiology and potential for adverse effects is the key to using such supplements to best and safe effect.

### A primer on calcium

Before a discussion of calcium-related metabolic issues and corrective supplements can take place, let's start with an explanation of what calcium does in the body. Most people are aware that calcium is a main component in bones and teeth, and serves as the primary storage depot for this mineral. Less than 1% of the body's total calcium stores are outside of bones and teeth, yet their most critical functions occur outside of the dense tissues.

Within the circulation and soft tissues of the body, calcium plays a role in blood clotting after injury, activating enzymes, transporting ions across cellular membranes, maintaining normal heart rates and rhythm, and in muscle contraction. Calcium ions carry a small positive electrical charge and so play a key role in sending and receiving neurotransmitter signals between cells—thus the categorical term "electrolyte."

Without the presence of ongoing adequate calcium within the individual muscle cell (or without adequate levels of other key substrates), muscles cannot contract to perform work. As calcium availability drops, the force of each contraction decreases, accelerating the onset of fatigue. In extreme cases, the lack of available calcium, along with deficiencies or excess of other substrates and metabolic by-products, causes the muscle cell to freeze in the contracted state—conditions commonly described as cramp, charley horse, tying up or exertional rhabdomyolysis.

How is it that if there is sufficient calcium in the body to provide structure for 150 pounds of bone, and yet potentially be lacking in muscle cells? There is a difference between the molecular form of calcium in bone bonded to other molecules and the active ionized form of calcium circulating throughout the body available for uptake by cellular tissues for ongoing metabolism.

The supply of calcium mobilized from bone storage to active circulation is regulated by parathyroid hormone released from the parathyroid gland and through the action of osteoclast cells—not a rapid process. If unable to keep up with ongoing demand during prolonged exercise, circulating levels may drop to the extent of causing hypocalcemia-related conditions.

### Calcium and thumps

Aside from muscle cramps, what other abnormalities might low calcium cause in endurance horses? A common term is that of a fatigued horse showing signs of "thumps" or synchronous diaphragmatic flutter—an abnormal and consistent twitch of the diaphragm muscles in time with the heartbeat. While the "thumping" itself is not a problem, it is a symptom of underlying metabolic abnormalities of electrolyte imbalances (including, classically, calcium), dehydration and potentially low sodium, chloride, potassium and magnesium.

As the levels of circulating electrolytes decrease, the transduction of electrical signals along neural pathways is disrupted between the brain, muscles and organs. One of these pathways is the phrenic nerve, which travels over the top and on both sides of the heart, which in turn generates its own electrical signal to drive cardiac contractions. When electrolytes levels are insufficient to appropriately conduct signals, the nerves become hyper-irritable and pick up the electrical signals produced by the heart. In the case of the phrenic nerve, these electrical signals are then abnormally

conducted to the diaphragm muscles, causing them to contract in time with the heart beat.

In all cases, thumps should be viewed as a symptom of underlying abnormalities in hydration and electrolyte balance including, but not limited to, calcium. In some mild cases, the condition can be resolved by allowing the horse to rest and providing a calcium-rich feed, such as alfalfa hay. If minor, the horse may be sufficiently recovered to continue onwards with caution. In more severe cases, veterinary intervention may be necessary in the form of IV fluids to restore hydration and electrolyte balance.

## Calcium supplementation

In recent years, it has become increasingly popular to administer (usually via oral syringe) concentrated calcium solutions or gels, including those liquids often used in intravenous therapy, such as 23% calcium gluconate or CMPK—a mixed solution providing calcium, magnesium, phosphorus and potassium. While formulated for IV therapy, it is also an acceptable practice to administer it orally.

Although additional calcium supplementation can help prevent or resolve those issues related to hypocalcemia, there are also significant risks involved which should be considered. Concentrated calcium administered in excess carries the potential to cause increasingly severe cardiac arrhythmias or even death in extreme cases (though, admittedly, this would be difficult to do via oral routes). In solutions, such as CMPK that also provide concentrated amounts of potassium and magnesium, such electrolytes in excess can additionally contribute to cardiac dysfunction.

The tricky question becomes, "How much is too much?" As with many other issues in managing horses, everything is relative. The amount of calcium supplementation that is either needed, if at all, or qualifies as excessive or even dangerous, is going to be entirely different for a well-conditioned Arabian exercising at a conservative pace in cool, dry weather than would be the case for a more heavily muscled breed competing at speed over difficult terrain in hot, humid weather. Likewise, the amount of calcium the more strenuously exercising individual might benefit from could easily become excessive if administered to the more moderately ridden horse under less stressful conditions.

As such, riders choosing to consider any form of calcium supplementation, aside from providing a moderate amount of alfalfa at control checks, should never assume that what works in the hot and humid Southeast will work equally well without risk for horses in the cooler and more arid Mountain Region. Better to consult with an experienced treatment veterinarian or certified head control judge from the specific region familiar with local conditions to discuss the type and amount of calcium supplementation.

Also carefully consider whether any additional supplementation at all is recommended or needed beyond providing simply good preparation, judgment and horsemanship on the day of competition.

## Avoiding calcium imbalance

Although it would seem logical that the way to increase calcium availability during exercise is to increase dietary calcium at all times (such as by feeding an increased amount of alfalfa or adding a calcium supplement), the opposite is actually true. In fact, the way to provide sufficient calcium during endurance exercise is to limit its supply to the body outside of the actual endurance event.

Part of the body's homeostasis mechanism is to closely control and regulate the amount of calcium (and other electrolytes) in plasma circulation at all times. As mentioned previously, calcium's mobilization from bone storage depots into circulation is controlled by parathyroid hormone. Additionally, excess calcium (such as that after eating a calcium-rich meal) is removed from circulation and moved into storage by the hormone calcitonin. While there are other substrates that also play a part of calcium metabolism, for this discussion, parathyroid hormone and calcitonin are the key players due to their antagonistic co-relationship.

When high amounts of calcium are routinely fed in the equine diet, increased calcitonin is produced and released by the thyroid gland to effectively reduce blood calcium levels. The effects of calcitonin are to increase the action of osteoblast cells (storing calcium in bone), inhibit osteoclasts (which release calcium from bone into circulation) and increase the kidney's excretion of calcium in urine.

At the same time, parathyroid hormone production is suppressed. Parathyroid hormone's action is to increase the release of calcium from storage into circulation to increase circulating calcium levels and make it more easily available for ongoing metabolism.

The production and changes in calcitonin and parathyroid hormone (PTH) levels are relatively slow processes, with hormonal concentrations taking several weeks to fully adapt to changes in dietary calcium levels. As such, when a horse is routinely fed significantly more calcium in its diet than needed to meet basic requirements, the body's hormonal pattern is set to primarily remove the excess calcium, not make it readily available.

When the horse then subsequently attends a strenuous endurance ride, that hormonal pattern may not allow for rapid enough mobilization of calcium to meet the demands of exercise—resulting in problems associated with low blood calcium levels.

How to avoid this hormonal imbalance? Paradoxically, the way to make calcium more available during exercise is to not feed calcium in excess at home. Feeding a sufficient, but not significantly excessive, amount of calcium in the maintenance diet results in relatively lower calcitonin levels, and higher PTH levels—the hormonal pattern optimized to make calcium more rapidly and readily available from storage depots when needed, such as during endurance exercise. Although calcium content of forages varies, most grass hays with a few pounds of additional alfalfa, or a diet providing a few pounds of beet pulp, provide sufficient but not excessive dietary calcium.

During the ride itself, calcium-rich feeds can then be provided immediately before and during the event with the expectation of better calcium absorption and availability during strenuous and prolonged exercise.

PART 2

## The relationship between hyperthermia, calcium and blood pH on metabolism and its role in avoiding associated performance problems

In Part 1, we started a discussion of how calcium levels are regulated in the body, and their relationship to certain syndromes seen in endurance horses, including synchronous diaphragmatic flutter ("thumps"), tying up and fatigue.

In this article, let's look at the relationship between calcium and acid-base physiology—essentially, how the body regulates the pH of blood and why it matters when considering calcium issues. More importantly, how to manipulate these essential mechanisms to improve the health and performance of your horse during competition.

Let's consider a typical scenario at an endurance ride. It's a warm day, maybe a little (or a lot) humid and there's still some winter fuzz that hasn't been shed out yet. The horses are working hard for hours on end, and with all that muscular exertion, core body temperatures are rising. Even with an expected rectal temperature of 103°F, the body must work efficiently and constantly to dispel excess heat and avoid hyperthermia—without doing so, the core body temperature rises 0.5° per minute and reaches lethal levels in as little as 15 minutes of moderate trotting in ambient conditions.

The fact that we are able to exercise our horses at conservative speeds, often without more than a moderate increase in rectal temperature—let alone lethal levels—attests to the efficiency of the body's cooling mechanisms.

How does the body dissipate excess heat? The primary pathway is evaporative losses through sweating, although radiation, conduction and convection all play a role. However, as every rider in the humid regions knows, panting is also a common mechanism by which the body can dispel excess heat when sweat production alone isn't enough, or is compromised by high humidity impeding efficient evaporation.

In most conversations, when discussing acidity, it usually has to do with either lactic acid production in exercising muscles or gastric acid in the stomach contributing to ulcers. However, the relative acidity of blood (or its converse, alkalinity) has a far greater overall impact on normal metabolism and health—to the extent that an uncorrected change of as little as 10% in either direction outside of the normal range of 7.35 to 7.45 rapidly becomes incompatible with life.

Even within normal ranges, a multitude of enzymes, transport proteins and cellular pathways are influenced and function differently with tiny shifts in the pH of the circulatory system.

### Equines and pH regulation

As such, the body is constantly and exquisitely tuned to regulate blood pH primarily via the kidneys and respiratory system. If the pH of the body becomes too acidic, the respiratory rate increases to rid the body of carbon dioxide ($CO_2$) and shift the balance back to a more alkaline balance. If the body is too alkaline, then the kidneys will increase the release of bicarbonate ($HCO_3$-) to bring the pH back to a more acidic balance—the key word being balance.

Respiration is one of the primary mechanisms by which the body can regulate acidity and alkalinity, and that as carbon dioxide is blown off during exhalation, the blood pH becomes more alkaline (the pH rises). The increased respiration can be due to excess acids needing correction (such as lactic acid during long sprints, or acids from different disease processes), or can be due to the body needing to dissipate excess heat. In any case, the results are the same in that blood pH increases towards the upper end of the normal range or slightly beyond. When pH levels rise above 7.45 due to panting, it is described as a *respiratory alkalosis*.

What does all this have to do with calcium metabolism?

Remember that there is a myriad of cellular pathways in the body that function for better or worse based on blood pH. One of these pathways is that of albumin, the primary protein in blood that binds and transports calcium through circulation, and is particularly sensitive to changes in pH.

In last month's article, it was discussed that only 1% of the body's calcium is outside of the large storage depots in bones and teeth. Of this 1%, approximately 50% is bound to various proteins, organic acids (such as citrate or lactate) or other minor molecules. Only the remaining 50% (or 0.5% of total body calcium) is in the biologically active form of ionized calcium (iCa).

When standard blood work panels measure total calcium levels, it is this total pool of both bound and "free" (unbound or ionized) calcium outside the bone that is quantified, but does not differentiate between the fraction components.

A useful analogy is thinking of a person's net worth—they might own hundreds of thousands of dollars in total assets, but that tells you nothing about how much is tied up in home, vehicles (and horses), nor anything about how much cash they have in their pocket right now.

A better lab test to assess an endurance horse's status, especially in blood samples drawn immediately after completing a hot and strenuous event, is the assay specifically quantifying the fractional component of unbound ionized calcium (the "cash in your pocket") that really makes the difference in a working horse during competition. That's opposed to the total calcium assay that is analogous to assets tied up in property, belongings and vehicles.

### Alkalosis explained

When the pH of the blood drifts towards alkalosis due to panting and excess heat accumulation, the effect on the albumin transport proteins is that they hold onto an even larger share of the ionized calcium, further decreasing the pool of biologically active ionized calcium, possibly to the point of producing calcium-depletion issues such as muscle cramps, tying up and thumps.

The analogy here I like to use is to visualize a train full of workers being transported to a job site. The train arrives at the appropriate station, and is traveling slowly enough for everyone to jump off, but because the doors won't open, all the workers are stuck inside the compartment and none are able to disembark to get to work.

Even worse, as the train is passing, conductors reach out to

the few workers already standing on the platform and pull them on board, too. Nobody can get to work and the train (still full of workers) just sails on by the job site.

This analogy should give a fairly good idea of how the biologically active form of calcium is adversely affected when a high respiratory rate increases blood pH to cause alkalosis. Even though sufficient calcium might be in circulation and storage depots, if it remains firmly bound to the transport protein because of high pH and alkalosis, it is effectively unavailable.

The alkalosis, and accompanying decrease in available ionized calcium, will continue until a more neutral pH can be restored by decreasing the respiratory rate, or allowing the renal (kidney) and adrenal system a chance to catch up and store balance to the system. Though an effective system, the renal end of the regulatory pathway is primarily driven by a variety of endocrine messengers, and is not rapid.

Several hours (or more) may pass before the kidneys have sufficient time to adapt and restore pH, by which time calcium-depletion syndromes have potentially long since appeared and ruined your ride day.

While the respiratory rate is the primary, and fastest, mechanism within the body to change blood pH, that pathway becomes temporarily hijacked during strenuous exercise during hot weather. Especially when humidity is adding to the heat stress, and the horse is breathing rapidly to service oxygen demands and help dissipate excess core heat, the high respiratory rate acts as a double sword by blowing off large amounts of the carbon dioxide that would otherwise help stabilize blood pH.

Again, as blood pH rises (becomes more alkalotic), less ionized calcium is available for the demands of strenuous and sustained exercise, to the extent that a rise in pH of only 0.1 means a 5% decrease in available calcium. And so the risks of calcium depletion issues such as tying up or thumps becomes ever more likely to occur.

Assuming a normal pH at rest in the neighborhood of 7.35 and a potential pH rise to 7.55 (or more) during a strenuous, hot competition, this means that of the already very tiny pool of circulating and biologically available calcium, a further 10% of those calcium stores now became unavailable—the workers that can't get off the train, *plus* the workers already there that got pulled onto the train and away from the job site.

## Other factors to consider

Are there any other relevant factors that can add to alkalosis and thus increase problems with calcium availability? Of course there are! Both alkalosis and depleted calcium issues can be further exacerbated by decreased potassium. Usually, in medical situations, the body is depleted by potassium through vomiting or diarrhea, but can also be the result of potassium losses through prolonged exercise and potassium lost through sweating.

In order to save as much potassium as possible, the kidneys will retain more sodium, but does so at the expense of wasting and excreting more hydrogen (H+), the ions that are at the very heart of maintaining acid-base balance. As hydrogen ions are lost in the urine, the overall blood pH again tends towards alkalosis.

Dehydration (does *that* ever rear its ugly head in the endurance?) further exacerbates the pH complications. Termed *contraction alkalosis*, as dehydration progresses, fluid is initially lost from the extracellular space—literally, the fluid compartments outside of and surrounding cells, including fluid circulating in blood stores, the reserve stores within the GI tract, saliva, gastric juices, and urine.

Excessive depletion of these fluids can predispose endurance horses to secondary problems such as impaction colic, "choke" from insufficient saliva production, decreased ability to sweat (and therefore increasing the need to dissipate heat by panting), and issues contributing to exhausted horse syndrome.

In addition, the endocrine pathways regulating the kidneys conserve further fluid loss by concentrating urine to retain water, but do so at a cost of spending further hydrogen ions (H+) and conserving bicarbonate ($HCO_{3^-}$)—again, increasing pH and alkalosis.

Finally, alkalosis and calcium depletion can be exacerbated by overall electrolyte depletion. The primary ions lost in sweat are sodium, chloride and potassium. Sweat production is a biologically active process—water doesn't just haphazardly leak out through the skin, it follows electrolyte ions that are actively pumped outward through sweat glands, pulling water along with it. As overall dehydration increases, and electrolytes become depleted, sweat production is paradoxically (and frustratingly) one of the very first body systems to be adversely affected.

Without adequate sweat production to help cool the body and dissipate heat, the horse must cool itself by other methods—such as increased panting, and thus loss of carbon dioxide and increasingly alkaline pH, again leading us back to calcium becoming increasingly unavailable to the strenuously exercising horse.

## Calcium management concerns

It becomes increasingly apparent at this point that ensuring there is adequate and biologically active calcium during endurance exercise is a far, far more complicated issue than simply throwing down a flake of alfalfa or adding a handful of ground-up Tums or a splash of calcium drench to the feed pan. Knowledgeable and savvy calcium management then becomes not a game of just adding more calcium on ride day, but a constant awareness of how multiple, seemingly unrelated factors affect the overall acid-base status of the healthy endurance horse.

***Getting out of the ivory towers and back to the real world, how does the average rider do this?*** First and foremost, it becomes a matter of heat management. Every strategy undertaken to keep hot horses cooled (without getting chilled) reduces the need to pant excessively, and helps the body maintain neutral pH.

**Clip as needed.** Body clipping horses, including during the winter with at least a trace clip, removes a significant insulating layer and helps the horse stay cooler during exercise. The more hair removed, to the extent of even body clipping a slick summer coat before known hot and difficult events such as Tevis, Old Dominion and other predictably hot rides, the more you are allowing your horse to dissipate excess heat through pathways other than increased panting.

**Plan your ride day accordingly.** Use knowledge and research of the trail and weather forecast to plan when to make time while it's cooler, and when to back down when heat and humidity are at their worst.

**Use active cooling strategies throughout the day.** Many ride managers must restrict the use of on-trail water troughs to drinking only, but many may also be generous enough to put out tubs available for sponging or judicious scooping. Don't be wasteful by haphazardly flinging and wasting water, but apply water to areas of the body where the major blood vessels (and thus transported heat) travel close to the skin surfaces—generally on the underside and inner regions. The underside of the neck, chest, belly and between the hind legs are all especially good places to apply cooling water.

Don't forget to scrape off the water after 30 to 60 seconds, after it has had a chance to absorb skin heat—otherwise the water itself becomes an additional insulating layer holding in heat. Scrape and repeat as necessary.

**Get heavy manes *off* the neck.** The underlying skin does not benefit from the "shade" of an overlying layer of hair, nor does a wet mane act to cool—it's just another insulating layer trapping heat. Braid it or roach the mane entirely.

**Never ignore opportunities to use natural water on the trail**, both for drinking and cooling purposes. Master the art of throwing a sponge on a string. If you know there is natural water ahead of you on the trail, ride with the sponge standing by in your hand ready at a moment's notice to fling at a puddle as you pass by. Scoops attached to the saddle are fine when you can dismount, but sponges can be thrown into puddles, streams and rivers without having to slow down or stop. Do *not* ignore this strategy, and spend plenty of time at home mastering this simple but highly useful trick so your horse is used to flying sponges.

**Review your strategies for maintaining optimum hydration** before, during and after an event, especially when attending multi-day competitions. Starting with a well-hydrated horse, and minimizing net fluid losses throughout the day, are the key factors in not only maintaining an appropriate acid-base balance, but in preventing virtually every other metabolic challenge facing the endurance horse.

**Finally, review your electrolyting protocols.** While many horses can be carefully managed to compete successfully with just good basic nutrition, excellent horsemanship and savvy judgment, others may benefit from additional access to maintaining higher electrolyte levels during competition.

## PART 3

### A review some of the nutritional management strategies to optimize calcium balance during competition, and avoid some of the pitfalls associated with balancing the healthy performance ration

In previous issues, we started and continued discussions of how calcium levels are regulated in the body, how blood pH affects calcium availability, and their relationship to certain syndromes seen in endurance horses, including synchronous diaphragmatic flutter ("thumps"), tying up and fatigue.

In this final article, let's look at best practices of how to supply calcium (and other closely associated minerals, particularly phosphorus) in the daily ration to optimize overall health and performance during competition.

As previously discussed, 99% of the calcium within the body is stored in the bones and teeth, with only 1% in circulation in both protein-bound or ionized (bioactively available) forms. Likewise, approximately 80% of phosphorus in the body is stored in the bones and teeth, with the remainder in soft tissue and circulation.

### Calcium/phosphorus relationship

What does phosphorus have to do with calcium? Like calcium, phosphorus is a necessary macromineral required for energy production and cellular function, but both must be provided in the diet not only in *sufficient* amounts, but in the correct *ratio* to each other. The minimum ratio for the overall diet is a minimum of 1 part calcium to 1 part phosphorus, but the ideal ratio is generally considered to be closer to 2 parts calcium to 1 part phosphorus.

In theory, the upper range of tolerable ratio is 7 parts calcium to 1 part phosphorus. However, due to a variety of drawbacks, a better acceptable upper cap is 3 parts calcium to 1 part phosphorus.

If overall phosphorus levels in the ration are higher than calcium, it is referred to as being "inverted." During digestion and movement through the small intestine, both minerals compete for absorption sites and if there is insufficient calcium in relationship to phosphorus, the body will pull the deficit from storage depots in the bone.

While not dangerous short-term, long-term imbalances can lead to serious calcium depletion and increased risk of chronic lameness and even life-threatening fractures.

Note that it is important to monitor calcium-phosphorus ratios in the overall ration, not just within specific feedstuffs provided. For example, the Ca:P ratio in alfalfa and beet pulp are 5.5:1 and 6:1, respectively, which at face value would seem to be excessively high in calcium. At the other end of the spectrum, barley, corn and oats (referring to grain, not cereal hay) are 1:5, 1:15 and 1:4, respectively, making them all seem unacceptably high in phosphorus.

Does this mean that there is no place in in a healthy equine ration for alfalfa, beet pulp or grain? Of course not—the trick is to balance the ration so that adequate amounts of both calcium and phosphorus are provided, and at the correct balance within the overall ration.

Simply trying to balance calcium and phosphorus isn't enough—other minerals can be adversely affected by improper ratios and amounts as well. Although a full discussion is beyond the scope of this article, data provided by Joe Pagan, PhD, of Kentucky Equine

**Calcium and phosphorus in various forages**

| Forage type | Calcium % | Phosphorus % | Ca:P Ratio |
|---|---|---|---|
| Alfalfa | 1.5 | .27 | 5.5:1 |
| Bermuda hay | .49 | .20 | 2.5:1 |
| Oat hay | .30 | .20 | 1.5:1 |
| Barley hay | .36 | .23 | 1.6:1 |
| Timothy | .41 | .20 | 2.2:1 |
| Beet pulp | .62 | .09 | 6:1 |

Research, indicated that while calcium digestibility (absorption) is adversely affected by excess phosphorus, the opposite is not true. Phosphorus digestibility is not significantly suppressed by higher dietary calcium levels.

Why does this matter? A trend in recent years among non-university online equine nutrition sources has been to recommend that the best strategy to "correct" a high calcium-phosphorus ratio (i.e., a ration which provides significantly more calcium than phosphorus) is to add yet more phosphorus to "balance" the ratio.

This is an example of solving a problem on paper, but not in real life. The analogy I like to use is my preference for using a ratio of 2 parts salt to 1 part pepper on my food. Let's say that I get distracted and accidentally dump an absurd amount of salt on my dinner, so that the "ratio" is now 20 parts salt to 1 part pepper. Is the correct solution to save the steak to dump an also absurd amount of pepper just to make the numbers add up? The math works—so why is my meal still inedible? Instead, wouldn't it be better to just remove the excess salt so that an adequate amount remains without burying it in pepper?

As with salt and pepper ratios on dinner, the correct and common sense approach to balancing a high calcium-phosphorus ratio in the equine diet is not to add more phosphorus, but to decrease calcium content so as to provide sufficient, but not excessive, amounts in the total ration.

Sarah Ralston, PhD, VMD, Dipl. ACVN, commented, "As long as a horse is not affected by chronic renal disease and decreased kidney function, moderately excessive calcium (150% to 250% of recommended intake) is not an issue. Of more importance is to ensure and provide adequate minimum intake amounts of both calcium and phosphorus in the ration."

## Figuring adequate amounts of Ca, P

How best to do that without resorting to extensive spreadsheets and number-crunching? While calcium and phosphorus content can vary somewhat by region, the chart shown on page 6 demonstrates average calcium and phosphorus content of common forages.

For more details, also refer to Common Feed Profiles at www.equi-analytical.com. With the exception of calcium-rich alfalfa, notice that every forage listed provides a calcium-phosphorus ratio within acceptable ratios, without additional supplementation.

Further, sufficient amounts of both calcium and phosphorus for an adult horse at maintenance or light work is provided within correct ratios by feeding 20 lbs. of virtually any forage, other than alfalfa. Adequate additional calcium and phosphorus are supplied for harder-working horses by simply feeding another five pounds of hay—as energy requirements increase, so does appetite and voluntary intake.

Although a diet of hay alone is often deficient in other nutrients (primarily, energy/calories) for endurance horses in intense training and competition, supplying adequate calcium and phosphorus is accomplished simply by feeding a sufficient amount of quality forage.

A useful mathematical tool for working with nutrient profiles and mineral requirements can be found at www.nrc88.nas.edu/nrh, the site for the National Research Council Nutrient Requirements for Horses, as organized by a blue-ribbon committee of equine nutritionists and academics.

Already feeding good hay and don't want to spend hours crunching numbers trying to mix a scoop of this and a pound of that? Good choice! Properly balanced rations require a significant investment in time and knowledge, and are most often adversely affected, rather than improved, by the well-intentioned but underinformed haphazard addition of multiple commodity feeds and supplements.

A better strategy, and often most cost-effective, is to choose a quality commercial feed that has already been formulated and balanced in terms of protein, minerals and vitamins. When chosen for the appropriate class of horse (maintenance, performance, growth, etc.) and fed in amounts according to label directions, it is rare that the overall nutrient profile is not well balanced and without significant holes and imbalances.

Can horses "balance" their own mineral profiles by supplying a free-choice mineral supplement? Independent and empirical studies performed over decades have failed to demonstrate that horses or other species can or will develop a specific appetite for any mineral deficiency other than salt.

While there are many commercial sources available to supply "free-choice" minerals, notice that the primary ingredients will always be a highly palatable substance such as alfalfa meal, grain or molasses. Horses eat the supplement because it tastes good, and therefore ingest the accompanying minerals, but not because they are able to discern deficiencies in their own ration and search out the specific required nutrient.

## Ride day calcium considerations

Back to calcium on ride day—it was previously discussed in prior issues that ensuring adequate calcium bioavailability during exercise is primarily a matter of supplying sufficient calcium in the daily ration, but not over-feeding calcium-rich feeds (such as alfalfa) habitually.

While there is a place for alfalfa in the endurance horse's nutrition program, thinking of it as a "supplement" rather than "mainstay" is a good strategy to help optimize calcium absorption and bioavailability during strenuous exercise. (For more details, refer to Parts 1 and 2.)

A common feed at vet checks has been a wet mash of wheat bran, with the belief that the laxative qualities of bran provided to humans are also applicable to horses and will help prevent colic. Sadly, such is not the case. Data published by Hintz, *et al* at Cornell University demonstrated conclusively that even when fed in amounts up to 50% of the total diet, wheat bran does not increase either the volume, moisture content or frequency of bowel movements in horses.

Additionally, wheat bran contains a high phosphorus content, primarily in the form of phytates that bind calcium, thereby rendering the calcium unavailable. The end result is that a pound of wheat bran (1.2% phosphorus) fed with a pound of alfalfa (1.5% calcium content) will result in virtually zero calcium being absorbed in the small intestine and available for ongoing exercise needs.

An oft-heard comment is from riders who opt to provide crushed Tums tablets in mash during the day as a calcium source. However, just one pound of alfalfa hay provides the same amount (and chemical form) of calcium as in nine Tums Extra Strength tablets—and pro-

vides some protein, gut fill and calories as well in a palatable form.

**Ride day tip:** Rather than adding a powder to mash that can only be administered at vet checks or via syringe, consider the trick of leaving vet checks on foot and carrying a flake of wet alfalfa with you to hand out to your horse as you stretch your legs.

Alternatively, consider carrying a baggie of soaked alfalfa pellets or beet-pulp-based performance feeds to provide a boost of calcium, along with calories and forage, to benefit gut motility.

If the choice is made to administer one of the calcium fluids, including CMPK or calcium gluconate, be aware of the relative pH difference in each. CMPK "drenches" available from feed suppliers catering to cattle are pH neutral and generally provided in a relatively innocuous flavored base.

Liquid calcium gluconate, however, is intended for addition to isotonic fluids in a highly diluted dose, and administered intravenously. The pH of such solutions not intended for oral administration can often be quite acidic, and thus unpalatable to horses unless mixed with a buffer or sweetener.

In conclusion, the take-home lesson for calcium during strenuous exercise is simple—provide enough, but not too much. Consider calcium-rich feeds at rides over drenches unless advised to do so by an experienced veterinarian familiar with conditions in your ride. Be aware of mineral interactions, but remember that adding more of the wrong mineral is not necessarily better.

Protect acid-base status and calcium availability by managing your horse and ride to avoid overheating and panting and, above all, have a great ride.

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