Hypocalcemia:

How to Recognize, Treat and Prevent It

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Introduction: The small segment of the goat population in which Hypocalcemia is primarily found is rarely seen by a veterinarian in large livestock practice, whose clients are more likely to be business-oriented raisers of hair or meat goats, or those that keep dairy goats for commercial milk production. Generally it is the small-animal practice veterinarian, not particularly familiar with or experienced in caprine management, that is called upon to treat the pet goats, family milkers, and show stock kept in relatively small, home-oriented herds.

Purpose: This article is presented to provide information for goat owners that will enable them to avoid hypocalcemia. It was also written to encourage veterinarians that read this information , when called upon to treat does with the symptoms outlined below to ask, before making a diagnosis, a very simple, fundamental question: "What are you currently feeding this animal, and how much of each substance is being fed?"

Goat Owners: Please feel free to give a copy of this article to your veterinarian.

How can I tell early on that my goat has hypocalcemia?

Ask yourself: In the early months of pregnancy (or during lactation, if she is in milk) have I been feeding my doe a regular, ample grain ration along with her hay? And especially, have I been feeding this ample grain ration along with grass hay instead of alfalfa?

Signs to watch for any time from the 12th week of the pregnancy on:

- Does the doe gradually or suddenly lose interest in her grain ration?
- And soon after that, does she also lose interest in her hay ration?

If this is the case, and if no corrective action is taken quickly, you can expect the next signs to be: She weakens fast, acts lethargic, is depressed. Her rear legs appear wobbly. If this situation is allowed to progress without intervention, she goes down and won't get up. Her body temperature will be normal (102.3) when these signs first appear, but will drop to sub-normal (below 102) as they progress. These symptoms are classic for hypocalcemia. Without fast and correct intervention she will die.

What is hypocalcemia?

Hypocalcemia (calcium deficiency) is a serious condition in which the calcium that a doe needs to support herself and provide for the needs of her developing fetuses (or to produce milk if she is lactating) is unavailable to her because of incorrect feeding.

What steps can I take to correct this?

(Before continuing, I want to point out that any dosages I mention herein are intended for full-sized dairy-type does, weighing $\sim 120\text{-}150$ lbs avg. If the doe your are concerned about is of a smaller variety you will need to adjust recommended doses accordingly.)

Immediately, and time is of the essence here, start her on Nutridrench or oral propylene glycol to provide her with the necessary energy so that she will not become ketotic. (Ketosis is a metabolic problem caused by the animal's having to live on its own body reserves because it has stopped eating food. If not corrected, this will result in coma and subsequent death.). I recommend either the appropriate dose of Nutridrench for her weight, or 60cc propylene glycol, 2x daily for 2 days to restore her, with 30cc daily thereafter until she is clearly eating properly again, to prevent/reverse ketosis.

Then, start her on calcium replacement therapy quickly. While calcium gluconate is commonly used for this, I prefer a product called CMPK (or a generic substitute thereof), because calcium gluconate contains only calcium, whereas the CMPK products contain magnesium, phosphorus, and potassium as well, all of which make the calcium more readily available for the body to use. Logic tells us that calcium that is in combination with these other elements that make it work better will be able to correct the deficiency and restore the doe to health much faster than can a product that contains the same amount of calcium alone, with no enhancements. One caveat: keep in mind that a single dose, or only a few doses, of this product will ONLY balance the doe's calcium level FOR THE MOMENT, but those babies will continue to grow and to drain her system of the needed calcium and phosphorus, so you will need to continue the doses daily until she freshens and is eating sufficient amounts of properly balanced nutrients to enable her to lactate properly.

The CMPK should be given at the rate of 30cc (1oz) every 2 hours or so until she appears bright and alert and willing to eat once more. The reason we give this specific amount every two hours, instead of giving a larger dose less often, is because calcium, necessary for muscle contractions, plays a vital role in the proper beating of the heart. But the downside of that is that to give a larger dose all at once, particularly at the later stages of the treatment, might actually cause the heart to beat too rapidly and create further difficulties to add to the ones the doe is already experiencing. We avoid this potential for overdose by giving smaller amounts more often, as I recommend here. I advise people who are administering calcium to check its effect upon the patient's heart by periodically checking the heart rate of another, normal doe (70 - 80 beats per minute), and comparing it with that of your hypocalcemic doe. Initially the hypocalcemic doe's heart rate will be considerably slower than that of the normal doe because she lacks the calcium to keep it beating normally. When the treated doe's heart rate is the same as, or a tiny bit faster than, the normal doe's heart rate, things are going well.

After bringing her heart rate up to normal, she will need DAILY MAINTENANCE DOSES OF ~ 30cc (1oz), continuing UNTIL SHE FRESHENS, at which point she will no longer need to provide large amounts of calcium and other nutrients for the fast-growing fetuses within her. Should she start to deteriorate at any time before then, that amount should be increased appropriately, but only temporarily, until she is regulated once more. Hopefully, for future pregnancies this doe's diet will be corrected so that this disorder will not re-occur.

BTW: Once her calcium level has been regulated and she resumes eating, she will probably initially refuse any grain that is offered. That should not cause you concern because her instinct is still trying to

regulate her calcium-deficient condition and she is the best monitor of that. In short order she will probably resume eating the grain again, at which time she should be limited to just a small amount at each feeding.

If she is not eager at first to eat her hay (grass is OK initially if that's what you have access to... Alfalfa or alfalfa pellets would be a really good choice now) you would be wise to bring her some of her favorite browse... I feed Salal up here in the Northwest, and the wild huckleberry, both of which stay green all winter... In your area there must be something yummy that, of course, is not toxic. If you don't know her favorite, give her a variety and let her choose.

If possible, it would be better to use injectable CMPK instead of the oral form to treat your doe. This is because: (a) It's always risky to dose a seriously debilitated animal orally as there is a potential for part of the fluids to end up in the lungs of the struggling animal, resulting in aspiration pneumonia, and (b) Calcium is, in concentrated form, somewhat caustic, and it will often burn the tender membranes of your goat's throat. The problem with my telling you this is that in their infinite wisdom the powers that be have decreed that injectable CMPK, while relatively inexpensive (about \$4/1000ml in the catalogs) should be a prescription-only item, available therefore only under the guidance of a veterinarian. The down side of this is that in addition to an increased per-dose cost, many veterinarians, especially those that are not goat-oriented, may not understand hypocalcemia, or why continued doses are necessary. As a result they might be overly-cautious about its use and generally will only want to prescribe/provide a single dose, or perhaps two. This is tantamount to trying to fix a leaking dam by putting your finger in the hole to stop the water flow.

One additional, important note regarding treatment of your hypocalcemic doe:

While you are treating her, keep in mind that if this very weak and debilitated doe has been down for 3 or more days, it is essential that you get her back up on her feet ASAP. Otherwise her legs will quickly lose their muscle tone and be unable to support the heavy weight of her body if she tries to get up on her own. If she is allowed to remain down for too long a period, her leg joints may begin to 'ankylose', or freeze permanently in the bent position. This is irreversible. To prevent it you may have to create a makeshift 'sling', attaching it to a pulley that is fastened to an overhead beam in the barn. About every 2 hours the sling should be raised up so that she can touch the ground comfortably with her feet and move around, and then lowered again so she can rest for a while... The process should be repeated continuously, 2 hours up and 2 hours down, until she can once again support her body's weight with her own legs. This generally takes but a few days, though her pregnant condition may place an added burden upon her as she tries to get her strength back. If the reader wants a picture of a sling I have one in my archives, at sreith@qwest.net...

How could my pregnant goat get hypocalcemia? And how can I prevent it?

If your doe is still milking when she is bred, she will need to continue the ration you have been giving her to support her milk production. But if she is 'dry' (not lactating) when you breed her she will need little or no grain for the first 3 months of her pregnancy, as calcium is not yet required for support of fetal growth. You see, at 3 months the fetus is no bigger than a newborn baby kitten.

But once that first 90 days or so has passed, the now completely formed fetus starts to grow rapidly. It will continue to do so, making increasingly greater demands for calcium to achieve that, over the next

8 weeks. So it's appropriate at that point to begin giving a small amount of alfalfa with the grass hay, increasing it gradually until at the time of freshening she is getting all alfalfa, which in my view should be continued for the length of time she remains in milk. The feeding of alfalfa should taper off only as her milk production tapers off, until she reaches the end of that lactation, at which time she can once again be given all grass hay.

At that same 90 day (3-month) point when you start giving the pregnant doe alfalfa, you should also start offering a small amount of grain, no more than a handful at a feeding. It should be increased slowly over the next 60 days (2 months) so that by the time the animal freshens she is getting maybe a cup in the morning and a cup at night. Then, depending on the amount of milk the doe is giving per milking, you should increase the grain so that she is getting enough to help produce the milk but not make her fat. A pound of grain is usually recommended for 8 lbs (~ a gallon) of milk. I add alfalfa pellets to a doe's grain to keep her busy while I milk her out.

The cause of this hypocalcemia (calcium deficiency) problem that can show up in a doe anytime in the last 6-8 weeks of pregnancy is very basic. How best to explain it? Let's see... Most of us know that water is made up of a ratio of 2 parts Hydrogen to 1 part oxygen (H2:0). If you don't have that ratio, you don't have water. And some of us, particularly those among us that are raising goats in copperdeficient areas, understand that a ratio of 10 parts copper to 1 part molybdenum (10:1) is essential in order for copper to be available for our goats. Following that line of thinking, a ratio of 2 parts calcium to 1 part phosphorus (2:1) is needed to make calcium available to us, AND to our pregnant/lactating goats. If we don't have 2 parts of calcium for every 1 part of phosphorus, calcium isn't available. So you can see now how important it is that all of these ratios remain in balance. When they are not, the substance we need will not be available to us. In line with this, the goat must be provided with a ratio of at least 2 parts calcium (abundant in alfalfa) to 1 part phosphorus (abundant in grain) to make the calcium available to her that she must have to support herself as well as the rapid fetal development within her uterus (or a lactating udder). If her owners do not provide the correct balance for her in the feed she is given, she will become hypocalcemic (calcium-deficient).

At the beginning of the gestation, before the babies start demanding a lot of calcium so they can grow within her, a mature doe (not a yearling, however, that is herself still growing) can survive on an unbalanced diet heavy in phosphorus (in grain) and virtually devoid in calcium (in alfalfa), generally without serious consequences. But when suddenly at 3 months into her gestation her body starts needing lots of calcium for the babies' growth, with that same unbalanced diet she's been getting all along the calcium will not be available! Even if she were getting lots of good alfalfa along with that ample grain ration, she simply would not have the rumen capacity to eat enough alfalfa to achieve the 2:1 balance necessary to release sufficient calcium to meet the demands of her own body AND the rapidly growing fetuses inside of her. Her amazing instinct tells her to cut back on the grain to free up calcium from the hay. Well, when she stops eating that large amount of high-energy grain she has become accustomed to, she quickly becomes nutritionally deprived, in addition to the calcium deficiency she is already experiencing. She weakens fast, becomes lethargic and wobbly, and goes down, and owners and consulting vets stand around scratching their heads, not realizing what led up to this, and try to figure out what's wrong. They (might) offer her Nutridrench or propylene glycol to correct the ketotic situation she finds herself headed for (or in), and then they wonder why she continues to get weaker and weaker, not realizing her problem is that the mismanagement of her feeding program has deprived her of much needed calcium, vital not only for the babies' development, but for her own muscle tone as well. No calcium, no muscle tone, no heart pumping, dead goat.

I can't find anything in the goat books about this disease. Where can I get more information?

Hypocalcemia is a correctable metabolic disorder (condition), and not a disease. Regarding the lack of resource material covering this disorder, I will say that I have been making a valiant effort to encourage the veterinarians who write these books to update their work to include it, but progress is slow. While most of goat management coverage in our current resource books is quite helpful, in this particular area I see the following deficiencies:

In the Merck Veterinary Manual *a single sentence* does address this disorder that appears at times in later gestation. In the 8th Edition it is found on P.744, the 'Pregnancy Toxemia in Ewes' section, in the paragraph called 'Diagnosis'. The single sentence reads: "Hypocalcemia, uncomplicated by pregnancy toxemia, should always be considered for recumbent late-gestation sheep." That's it! There is no discussion of either causation or treatment to be found anywhere for this briefly referred-to hypocalcemia. I can only speculate that it is because it is not well understood by the author.

Smith and Sherman's 'Goat Medicine' book does a fine job of pointing out in the section on goats' dietary needs that a diet containing 2 parts calcium for every 1 part phosphorus is important. Unfortunately, it appears there was a lapse in transferring that information over into other areas of the book where it is needed. For example, in a discussion of 'metabolic disorders appearing in late gestation' a reference is made to hypocalcemia, which is then followed up by a vague comment about some magical but unexplained chemical imbalance within the hypocalcemic goat that makes calcium unavailable to her. The author fails to make use of this golden opportunity to explain to the reader (many of whom do not understand the nutritional needs of a pregnant/ lactating goat) the importance of providing a diet that contains 2 parts calcium for every 1 part phosphorus to free up calcium for her fetuses/milk production in order to prevent this hypocalcemia. Once again, I am left to assume that the authors are not making this connection? Oddly, there is another comment in that same section on metabolic disorders about goats needing "2 parts 'forage' to 1 part 'concentrate'", a misleading statement at best, and a recipe for disaster at worst, since vast numbers of goat owners have only grass for forage, and grass contains very little calcium at all.

John Matthews, in his 'Diseases of the Goat', talks about 'hypocalcaemia', noting that it may appear in late pregnancy... AND in any stage of lactation (an important bit of information!). Sadly, he also misses the simple cause, a dietary imbalance that prevents the uptake of calcium from the feed, and relies on that magical but unexplained "failure in the homeostatic mechanisms to meet the increased demand for calcium". However, in his discussion of 'Hypocalcaemia' he does redeem himself admirably with the accuracy of the following statement that: "All recumbent or comatose goats should be treated as potentially hypocalcaemic and given calcium."

Disappointingly, in addition to failing to explain the actual cause of hypocalcemia (unbalanced diet), none of these popular reference books offer any suggestion that continued calcium replacement during this owner-created crisis be started as a treatment for it. In fact, all excepting John Matthews' book actually make very little of hypocalcemia, despite the fact that (while admittedly it remains unrecognized due to lack of text reference information on the subject) it shows up quite frequently in the 'down' pregnant/lactating does managed by inexperienced goat owners. As a result, its potential for use as a diagnosis is overlooked entirely by veterinarians seeking guidance from these books in their efforts to come up with a proper diagnosis and treatment for the animal. The huge down side of this is that when the hypocalcemic condition is not recognized, the veterinarian that is inexperienced in

diagnosing 'down' pregnant goats will, using these books for reference, almost always opt for a diagnosis of Pregnancy Toxemia, or Pregnancy Ketosis, or any combination or variation of those words. When asked directly about the possibility of a calcium deficiency, he/she frequently responds, "This goat's problem has nothing to do with calcium." The predictable treatment regimen will then be: "Treat with propylene glycol for ketosis, and get the babies out fast (C-section or, even worse, suggestion of Lutalyse for abortion, which will not work because the uterus has no muscle tone with which to expel the fetuses when put into the labor mode) to save the life of the doe." In instituting calcium therapy in lieu of such drastic measures I have never experienced loss of either the doe or the kids, and does thus properly treated to correct the condition invariably go on to freshen normally. Were it my goat down with this problem I would most surely engage the vet as a partner in instituting this treatment prior to embarking on anything so drastic as C-section or abortion!

A number of our foremost dairy goat nutritionists and veterinarians have made reference to the existence of hypocalcemia in their writings over the years, although none seem to address the actual cause of it, which is critical to its treatment. As actual livestock management is not their field, perhaps they assume that all dairy goat people instinctively understand how to correctly feed their pregnant stock? They apparently are unaware that it is the mistakes in the feeding programs made by those of us who do not understand this essential 2:1 nutritional need that cause this hypocalcemic (too little calcium) condition.

Here are a couple of contributions made by respected individuals that have helped us in our understanding of goats' dietary needs, and have suggested potential corrective measures to take when the needs are not met:

Dr M.E. Ensminger, a renowned livestock nutritionist from whose work a vast body of our experts draw today in order to determine the content of livestock feeds and make nutrition recommendations, says in his 'bible' of livestock nutrition called Feeds & Nutrition - Complete, published in 1978, that Alfalfa (lucerne), a legume, "is high in calcium, protein, and carotene, and in many other minerals and vitamins". He notes that "legumes are excellent calcium sources, while grasses and silages tend to be substantially lower in calcium content". He points out clearly that both bone growth and lactation (and muscle tone as well, BTW) require substantial quantities of these minerals. He says, "If there is a severe imbalance of them during pregnancy and early lactation, 'milk fever' <smile> may occur." And he continues, "Therefore, in order to prevent these problems, the calcium:phosphorus ratio should be at least 2:1." (2 parts calcium: 1 part phosphorus.) (FYI: He also states: "In males an imbalance of calcium to phosphorus often leads to the development of urinary calculi.")... Finally, as an addendum to those among us who rely on grass hay to feed our goats, Dr Ensminger suggests that where additional calcium is needed, ground limestone is generally the mineral of choice, but if the animals are in need of both calcium and phosphorus the best choices for provision of these 2 essential minerals are di-calcium phosphate or steamed bone meal.

Another knowledgeable person, a man with whom I had the good fortune to come into contact when I was living in So. California where I first began to raise dairy goats, was a veterinarian named Dr Robert A. Jackson. He was what you could call a goat vet's goat vet, and he and a dairy goat breeder/judge named Alice Gaye Hall frequently co-wrote articles on dairy goat management. In one such article, printed in the July '82 Dairy Goat Guide and entitled 'What to Know about Medications', the readers were advised that calcium is an important substance for goat owners to keep in their cupboards because goats often come down with 'eclampsia, which is much like milk fever...'. As do others, they called it milk fever when it is actually just one of the elements one finds in that disease,

and while they don't address its dynamics, they do make the observation that a calcium deficiency (hypocalcemia) sometimes exists in the pregnant/lactating goat, and that the owner should be prepared to treat it when it shows up.

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