Vitamins: Their Role in Therapeutics, Prophylaxis, and Supportive Therapy in the Neonatal Calf

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Introduction

The neonatal period (first twenty-eight days)[23] stresses the bovine calf more than any other period of its life. Along with the hazzards of parturition and adaption, the calf is born with little or no bodily stores of vitamins and is dependent almost totally on the quantity and content of the milk of the dam. The rumen, which supplies many of the vitamins to the adult, does not start effective production until long after this period.[43] Anything that may interfere with proper absorption of needed vitamins may result in deficiency symptoms or more commonly a decreased resistance to disease.

Supplemental vitamins for prophylactic and supportive treatment of the bovine neonate is highly indicated. This practice, however, is not commonly exercised in treating the sick calf.

The purpose of this paper is to provide information so the practitioner can intelligently decide why and when administration of vitamins are indicated and how to properly use them. Vitamin sources and functions along with experimentally and clinically proven guidelines about vitamin usage for the neonatal calf will be given. Tables on minimal daily requirements, practical daily supplemental levels, suggested prophylactic or supportive therapeutic doses, and reported toxic doses are also presented.

Fat Soluble Vitamins

General Concepts

The fat soluble vitamins are vitamin A, D, E and K. Colostrum ususally contains adequate carotene (the precursor of vitamin A) and the other fat soluble vitamins except possibly vitamin E (See Table 1). The diet of the dam through pregnancy will significantly effect the vitamin concentration of the colostrum.[48] Therefore, it is essential that she receive an adequate plan of nutrition during pregnancy, especially during the last trimester, with a good supply of quality grass, hay or silage. Parenteral supplementation of A, D_3 and E to the dam during pregnancy will also boost the vitamin levels in the colostrum and significantly decrease calf mortality.[34]

Excess fat soluble vitamins ingested by the calf are stored. These stores can be readily depleted in stressful conditions such as disease, parasitism, environmental changes, and changes in the quality and quantity of the milk received. Care must be taken in the parenteral use of some of these vitamins in the calf as very high levels given daily may have pronounced toxic effects, even death.

Vitamin A

For supplemental use there are two main sources of vitamin A. One source are carotenes which are found in natural feed stuffs, beta carotene (provitamin A) being the most common. The other source of vitamin A used supplementally is preformed vitamin A, the commonest form being vitamin A alcohol (retinol).

The principal physiological functions of vitamin A in the calf are: 1) the formation of visual pigments in the retina of the eye; 2) the maintenance of lysosome stability; and 3) the promotion of growth and maintenance of epithelial tissues. Deficiency of vitamin A will cause a breakdown in any or all of these bodily functions, therefore, the clinical signs associated with vitamin A deficiency are many and are related to these functions.[19]

In field cases of acute vitamin A deficiency, the young calf frequently dies as a result of pneumonia or severe diarrhea because of a lowered resistance to many respiratory infections and enteric infections.[20,48] Deficiency of vitamin A in the dam during gestation may result in abortion, birth of dead calves, blind calves, and weak calves[33] with a high incidence of scours[43,33,20,20] There may be quite a variance in utilization of carotene[39] and minimal daily vitamin A requirements in winter is possibly twice as high as in summer.[29,40]

Reported total doses per calf for prophylaxtic and supportive therapy range from 250,000 to 1,000,000 IU.[3,21] Parenteral vitamin A is recommended in newborn calves within the first week of life to prevent scours and pneumonia, and provide an immediate high level of vitamin A to the liver to store.[21] It is also indicated in calves that are depressed, have pneumonia or enteritis, and it produces a specific prophylaxtic effect against coccidosis.[20] Injections

of vitamin A, D_3 and E to cows and heifers significantly decreased calf mortality and decreased the average interval until their next pregnancy.[34]

Overdoses of vitamin A may decrease vitamin C and E levels in the calf. Greater than 3 million IU/50 kg body weight per day will produce gross signs of hypervitaminosis A, where as, levels as low as 225,000 IU/50 kg body weight per day produces changes in the mineralization of bones.[18]

Vitamin D

There are two natural forms of vitamin D that can be supplied to the calf: Vitamin D_2 (ergocalciferol) and vitamin D_3 (cholecalciferol). Both forms seem to be equally potent for the calf.[48] D_2 is usually taken in by the calf in dietary forms such as sun cured roughages, whereas, D_3 is manufactured by the calf. Due to its availability, D_3 is the most commonly used form of vitamin D parenterally.

There are two main functions of vitamin D: 1) it is necessary for the normal growth and mineralization of bones; and 2) it functions in the absorption of dietary calcium and phosphorus from the gastrointestinal tract.

Deficiency of vitamin D in the young calf produces the classical clinical case of rickets and associated clinical signs of poor growth, loss of appetite, arthritis, digestive disturbances, labored breathing, irritability, weakness, tetany and convulsions.[43] Calves born of dams deficient of vitamin D may be born dead, weak, or deformed.[43]

The minimum daily requirement for vitamin D in the calf is 330 IU/50 kg of body weight.[6] Normally, both exposure to sunlight and the feeding of good quality hay along with the dam's milk will furnish the required amount of vitamin D. However, in the case of a debilitated calf, anorexic, and confined, a vitamin D deficiency could easily arise and complicate his condition. When given at a supplemental level of 2,200 IU/50 kg of body weight per day it was found to significantly increase growth rate of calves.[30]

The body stores vitamin D to a much lesser extent than vitamin A, but it too may be toxic to the young calf if given in too high of quantities. Such toxicity has been produced by giving 1 million IU of vitamin D_3 daily to young calves.[8]

Vitamin E

Functionally, vitamin E and the mineral Selenium should always be considered together as a common vitamin or doublet[52], as their actions within the body are closely related.

The source of vitamin E is in a group of compounds called the tocopherols, of which alpha-tocopherol is the most biologically active. This can be provided to the calf naturally in feedstuffs or more commonly synthetic D,L-alpha-tocopherol acetate or D-alpha-tocopheryl acetate can be added to the diet or given parenterally. The D form, being better utilized than the L form, is usually used in injectable vitamin E and vitamin E-selenium compounds.

Vitamin E seems to be involved in a large number of body functions, some of them include reproduction, muscular development, immune stimulation, and regulation of various enzyme systems.[52,43,12,11] Its main role seems to be as an antioxidant in the prevention of peroxidation of lipids within cell membranes.

TABLE 1. Comparative Vitamin Composition of Colostrum (first 24 hours after calving) and of milking[a]

		Colostrum	Milk	
		Friesian	Friesian	Streight bred beef cow[b]
Fat	g/kg	36	35	28
Non-fatty solids	g/kg	185	86	88
Carotenoids	u/g fat	25-45	7	
Vitamin A	IU/g fat	140-160	26	
Vitamin D	IU/g fat	.9-1.8	.6	
Vitamin E	IU/g fat	.1-.15	.02	
Thiamine	mg/kg	.6-1.0	.4	
Riboflavin	mg/kg	4.5	1.5	
Nicotinic acid	mg/kg	.8-1.0	.8	
Pantothenic acid	mg/kg	2.0	3.5	
Pyridoxine	mg/kg	—	.35	
Biotin	u/kg	20-80	20	
Vitamin B_{12}	u/kg	10-50	5	
Folic acid	u/kg	1-8	1	
Ascorbic acid	mg/kg	25	20	
Choline	mg/kg	370-690	130	

a Modified from Roy J.H.B., The Calf, Fourth edition, 1980, Table 3.1, p. 58
b From Meiton 1967, J. Ani. Sci. 26:804
- Averge milk yield per day over 175 days for streight bred beef cows 3-10 yr. old was 4.22 kg/day (9.3 lbs/day)

TABLE 2. Vitamin A, D and E Equivalencies

Vitamin (IU)	Form (mg)	Equivalency
A	Beta carotene	1 mg = 400 IU[a]
	Retinol	3 mg = 1 IU
D	Cholecalciferol (D_3)	.025 mg = 1,000 IU
E	D,L-alpha-tocopherol acetate	1 mg = 1 IU
	D-alpha-tocopherol acetate	1 mg = 1.36 IU

a This may vary widely due to the type of feedstuff (N.R.C., 1976)

The daily minimum requirement of vitamin E reported for the young calf is anywhere from 3 to 45 IU per 50 kg of body weight.[43,24,1] The exact requirement may vary according to other dietary constituents such as unsaturated fats, vitamin A, and mineral intake, especially selenium. If no polyunsaturated fatty acids are in the diet, 3 mg per day is sufficient.[3,4] The higher the level of circulating vitamin A, the higher the requirement of vitamin E.[48] Deficiencies in selenium will increase the requirement of vitamin E. High levels of sulfate in forges can decrease the utilizable selenium in plants.[54] It has also been found that high levels of dietary nitrite will increase the daily requirement of vitamin E.[43]

Clinical signs associated with vitamin E deficiency in calves are not rare as milk is often low in vitamin E. The most notable signs being those associated with white muscle disease or nutritional muscular distrophy. Other less specific signs are heart failure, paralysis, slow growth and unthriftiness.[43] On two North Dakota ranches, calves born to dams deficient in selenium and vitamin E were born dead, weak, and some with skeletal myopathies.[54] Other

TABLE 3. Suggested minimum daily allowances for vitamins in calves up to 100 kg[a].

Vitamin	Amount		Reference derived from
A[b]	4850	(IU/50 kg body weight)	Eaton et al., 1972
D	330[c]	(IU/50 kg body weight)	Bechdel et al., 1938 and N.R.C., 1976
E	3-30[d-e]	(IU/50 kg body weight)	Huber and Thomas 1967
	35-45[f]		Agricultural Research Council, 1965
Thiamine	2.1-2.9	(mg/50 kg body weight)	Zintzen, 1973
Riboflavin	1.75-2.25	(mg/50 kg body weight)	Brisson and Sutton, 1951
Niacin	13.0[g-h]	(mg/50 kg body weight)	Hopper and Johnson, 1955
Pyridoxine	3.25[h]	(mg/50 kg body weight)	Thomas and Okamoto, 1955
Biotin	.09[h]	(mg/50 kg body weight)	Thomas and Okamoto, 1955
Pantothenic acid	9.75[h]	(mg/50 kg body weight)	Thomas and Okamoto, 1955
B_{12}	22-44	(/50 kg body weight)	Lassiter et al., 1953
Choline	1300[h]	(mg/50 kg body weight)	Thomas and Okamoto, 1955

a Only vitamins where a definite dietary need has been established
b The minimum requirement in winter is possibly twice as high as in summer (Keener et al., 1942; Moore et al., 1943)
c Requirement increases in absence of sunlight or low calcium and phosphorus levels
d Depends on other dietary constituents such as unsatisfied fats, and certain minerals
e Deficiency in selenium will increase the requirement, .25 mg of Se per day is sufficient (Blaxter et al., 1961)
f Requirement of vitamin E for calves on low Se diets
g Only needed when tryptophane is low in diet
h Required level not established, but this amount successfully raised calves on synthetic milk diets

TABLE 4. Suggested Practical Dairy Supplemental Level of Vitamins for Calves up to 100 kg

Vitamin	Amount		Reference derived from
A	27,500	(IU/50 kg body weight)	Thomas and Okamoto, 1955
D	2,200	(IU/50 kg body weight)	Keys, 1944
E	44-150	(IU/50 kg body weight)	Huber and Thomas, 1967; MacDonald et al., 1981
Thiamine	3.0-3.6	(mg/50 kg body weight)	Pohlenz, 1973; Roy, 1980; Thomas and Okamoto, '55
Riboflavin	6.5-14.6	(mg/50 kg body weight)	Thomas and Okamoto, 1955; Roy, 1980
Niacin	13.0	(mg/50 kg body weight)	Thomas and Okamoto, 1955; Roy, 1980
Pyridoxine	3.25-3.5	(mg/50 kg body weight)	Thomas and Okamoto, 1955; Roy, 1980
Biotin	.1-.2	(mg/50 kg body weight)	Thomas and Okamoto, 1955; Roy, 1980
Pantothenic acid	10-28[b]	(mg/50 kg body weight)	Thomas and Okamoto, 1955; Roy, 1980
B_{12}	63	(/50 kg body weight)	Thomas and Okamoto, 1955;
Choline	1300	(mg/50 kg body weight)	Thomas and Okamoto, 1955;
C	65-136	(mg/50 kg body weight)	Roy, 1980

b In the form d-pantothenic acid

researchers state there is a relationship between bovine abortions, stillbirths, weak calves and other reproductive problems, with selenium-vitamin E deficiencies.[52]

The use of injectable vitamin E-selenium compounds, for prophylaxis and supportive therapy, have gained much acclaim by some practitioners. Poisoning due to mercury, cadmium, cobalt, and lead have been prevented or alleviated by the use of vitamin E.[55] Injections of selenium-E may be especially indicated in the weak, sick, or debilitated neonatal calf. Supplemental vitamin E will improve the humoral immune response of various species of animals when exposed to different antigens, especially *E. coli* in chickens and turkeys and Chlamydia in sheep.[42] In a recent study on vitamin E and the immune system in calves, calves given 1000 IU/day of vitamin E showed a trend towards earlier stimulation of IgG1 and an increase in the lymphocyte

TABLE 5. Suggested Parenteral Vitamin Doses for Therapy, Prophylaxis, or Supportive Therapeutics in Calves up to 100 kg.

Vitamin	Dose	Frequency	Reference derived from
A	500,000-1,00,000 IU	Once during 1st week after birth then every 2-4 months	N.R.C., 1976; Herrick, 1974
D	75,000-150,000 IU	Once during 1st week after birth then every 2-3 months	Pfizer Inc.
E[a/c]	75[b]-300 IU/50 kg	Repeat every 5-10 days	Schering Vet Lab; MacDonald et al., 1981
	300 IU/50 kg	On onset of clinical sign of deficiency every day for 2 weeks	Thomas & Okamoto, 1955
K_1[d]	0.5-2.5 mg/kg	Daily for 4-6 days	Mount et al., 1982
K_3	2.2-11 mg/kg	As indicated	Professional Vet Lab Minneapolis, MN
Thiamine	100-400 mg	Daily for 3 days	Zintzen, 1973
Riboflavin	50 mg	Daily as indicated	Med Tech Inc, Elwood, KS
Niacin	1,000 mg	Daily as indicated	Med Tech Inc, Elwood, KS
Pyridoxine	100 mg	Daily as indicated	Med Tech Inc, Elwood, KS
Biotin	0.1-1.0 mg	Daily as indicated	Weise et al., 1946
Pantothenic acid	100 mg	Daily as indicated	Med Tech Inc, Elwood, KS
B_{12}	1 mg	Daily as indicated	White, 1955
C	200 mg/kg	Daily as indicated	Dvorak, 1965
	1,000-2,500	Daily for 3 days	Beaman, 1982

a Excesses or deficiencies in dietary or supplementary selenium may alter the levels of vitamin E needed
b Myosel-E, Schering Vet Laboratories, Schering Corp., Kenilworth, N.J. 07033
c Higher levels of vitamin E may stimulate Ab response, lymphocytic activity, and increase the calf's resistance to pathogens as has been shown to do in other species (Cipriano, 1982)
d IV use not recommended

TABLE 6. Reported Toxic Doses of the Fat Soluble Vitamins for Calves up to 100 kg.

Vitamin	Dose		Reference derived from
A	3,000,000[a]	IU/50 kg body weight/day	Hazzard et al., 1964
	225,000[b]	IU/50 kg body weight/day	Roy, 1980
D	1,000,000	IU/day	Blackburn et al., 1957
E	2,300-4,600[c]	IU/50 kg body weight	MacDonald et al., 1981
K_1[e]	>250[d]	mg/50 kg body weight	Mount, Feldman and Buffington, 1982

a Level required to develop gross clinical signs
b Level required to develop changes in the mineralization of bones
c When combined with Selenium 1-2 mg/kg body weight it produced a single lethal dose
d Toxicity manifested as hypersensitivity reactions
e Large amounts of K_3 may produce hemolysis, methemoglobinuria and porphyrinuria

stimulation index over control calves and vitamin E deficient calves.[12] The immune response in calves may be more effectively stimulated by lower levels of vitamin E, as was found to be the case in the guinea pig.[2]

Vitamin E-selenium can also be toxic to calves in very high doses. MacDonald et al., (1981) reported a single lethal dose of vitamin E-selenium that was between 2300 IU vit E/50 mg Se and 4600 IU vit E/100 mg Se per 50 kg body weight. In certain areas of the U.S., particularly areas of Nebraska, grazing animals may suffer from selenium toxicity, therefore, prophylactic treatment should be given in the form of vitamin E only.[17] There are also selenium deficient areas of the U.S., these include areas in the southeast, northeast, midwest, and far northwest.

Vitamin K

Vitamin K is found in two natural forms (K_1 and K_2) and it can also be produced synthetically as menadione (K_3) which is then converted to K_2 in animals. K_3 is more biologically active than the natural compounds, but it is

poorly stored in the liver compared to K_1, so higher amounts are required to achieve the same physiological effect. For this reason vitamin K_1 is the preferred veterinary therapeutic agent.[41]

Vitamin K is involved in the blood clotting mechanism of all animals. The function it has is to stimulate the production of prothrombin (factor II) as well as coagulation factors VII, IX and X.

Deficiencies of vitamin K in the calf are extremely rare.[39] Severe pancreatic or liver disease, prolonged oral antibiotic therapy, and intestinal malabsorption may result or predispose to a vitamin K deficiency[41], as will exposure to dicoumarin from moldy sweet clover or the rodenticide dicoumarol.

Effective therapeutic doses specifically for the calf are not properly worked out, however, .5 to 2.5 mg/kg body weight of vitamin K_1 is recommended in large animals that have been exposed to warfarin related compounds.[41]

The natural forms are relatively nontoxic, but large amounts of menadione may produce hemolysis, methemoglobinuria, and prophyrinuria.[39] Also the intravenous route of administration is not recommended in clinically ill animals due to the potential of anaphylaxis.[41]

Water Soluble Vitamins

The B-complex vitamins

There are eight vitamins that makeup the so-called B-vitamin complex: thiamine (B_1), riboflavin (B_2), niacin or nicotinic acid (B_5), pyroxidine (B_6), pantothenic acid, biotin, folic acid, and cyanocobalamine (B_{12}). Of these eight, dietary requirements for all but folic acid have been experimentally determined for the preruminant calf (N.R.C., 1976).

In the adult, rumen bacteria synthesizes all of the daily needed B-vitamins. However, for the first eight weeks of life, depending on the diet of the calf, the rumen is still developing and it has little effect on the supply of dietary B-vitamins to the calf.[43]

The suggested minimum daily allowances for the seven dietary required B-vitamins are usually met by the milk supplied by the cow during early lactation *(See Table 1).* However, the neonatal calf that is weak, anorectic, scouring, orphaned, or just out of a poor milking heifer can easily be deficient in any or all of the B-vitamins. Certain antimicrobial drugs also decrease the intestinal synthesis of various B-complex vitamins, or reduce their activity.[45] In these instances B-vitamin therapy is indicated. Roy (1980), found that once the rumen begins to function, the incidence of diarrhea of calves given a balanced dry ration was negligible.

Functionally, the B-vitamins are all interrelated. When trying to decide which of the B-vitamins are needed for supportive therapy it is best to visualize the individual B-vitamins as links making up a chain that is being stretched by the stresses on the calf. Deficiency of any one may allow the chain to break. This can be repaired by individual vitamin replacement, but often the chain will only break again at its next weakest link. The use of B-complex compounds are therefore recommended and often are the most effective and economical way of treating the B-vitamin deficient calf. High levels of B-vitamins are relatively nontoxic to the neonatal calf because they are not stored by the body and are readily excreted in the urine. Therefore, daily parental administrations of B-complex is a common practice for the sick calf.

Thiamine functions in carbohydrate utilization, and the synthesis of steroids which directly stimulate gluconeogenesis, the extremely important pathway in providing energy to ruminants.[60] Thiamine is also involved in the synthesis of acetylcholine, the sodium transport system, and the maintenance of myelin.

Clinical signs of thiamine deficiency in the calf are anorexia, emaciation, tetenic convulsions, and polyneurosis.[26] Cerebrocortical necrosis is the most important disease arising from B_1 deficiency in ruminants, and is usually seen in cattle between three to eighteen months of age.[60] Prolonged oral treatment with coccidiostats, eg. Amprolium (Merck, Sharp, and Dohme Ltd.) may produce cerebrocortical necrosis in young calves.[60] This condition and the previous clinical signs respond quickly to thiamine injections if given at an early stage. Relationships between supply of B_1 and resistance to disease have been demonstrated.[60]

Riboflavin functions as two coenzymes involved in a large number of enzyme systems, FMN and FAD. Functions affected by these two coenzymes are tissue respiration, the Krebs Cycle, fatty acid metabolism, amino acid metabolism, and purine metabolism. Another function of riboflavin is its role in maintaining the integrity of mucous membranes.

Clinical signs of deficiency are nonspecific and include anorexia, lesions on the gums, abnormal salivary secretions, excessive lacrimation (Weise et al., 1947), diarrhea, and excess shedding of the hair.[57] Decreased growth rate and lowered feed efficiency are common symptoms due to its involvement in the production of energy for the body. Deficient calves respond promptly to riboflavin administrations.[57]

Niacin functions in nearly all the cells of the body, performing a variety of metabolic activities. It's most important function is its involvement in the release of energy in carbohydrate metabolism. It is a component of two very important coenzymes of the body; NAD and NADP. Nicotinic acid can be formed by the preruminant calf if proteins containing appreciable quantities of tryptophan, such as milk, are included.[25] Nicotinic acid, is then converted to nicotinamide, the physiologically active form. Supplementally, nicotinamide is used more than nicotinic acid due to its activity and safety. Scouring and loss of appetite were observed in calves with acute nicotinic acid deficiencies.[22]

Pyroxidine is necessary for the proper function of the cell and it also plays a major role in many enzyme systems

concerned with protein metabolism. It is essential for the complete metabolism of tryptophan, and fat. Deficiency in the calf usually results in anorexia, diarrhea, convulsions, and death occurring at 3-4 weeks of age.[25]

Pantothenic acid has one of the most diverse functions of any of the B-vitamins because it is the major component of coenzyme A, the coenzyme required for acetylation of fats, carbohydrates, and amino acids so they can be metabolized.[35] This function also aids in the detoxification processes of the body. It also serves roles in the synthesis of fats, steroids, and ketone bodies.

In view of its diverse functions the deficiency signs of this vitamin are many. Most commonly in the calf you will see diarrhea, decreased growth, weakness of the legs and the inability to stand.[25]

Dietary supplementation of pantothenic acid is usually in the form of calcium pantothenate which is composed of both D and L isomeric forms, however, only the D form is biologically active. Therefore, the amount of supplementation should be based on it. Most injectable B-complex preparations use D-panthenol.

Biotin and our knowledge of its functions are limited. Biotin is used as a coenzyme in carboxylation reactions, particularly in the major pathway for fatty acid synthesis by the cells.

It seems quite difficult to cause a deficiency in biotin in the calf, however, it has been done.[48] Deficiency signs were characterized by paralysis of the hind quarters. This condition was cured by daily injections of biotin.[56]

Cyanocobalamin (B_{12}) was the last of the B-complex vitamins to be isolated (1948), and could be the most important B-vitamin supplemented to the neonatal calf. B_{12} acts as a coenzyme in reproduction of cells, maturation of red blood cells, and it helps maintain neural function. Most importantly to the ruminant it is needed to utilize propionic acid.

B_{12} is a metabolic essential and a dietary requirement for the preruminant calf[31], and it can be deficient in the young ruminant when fed a low cobalt diet, as cobalt is necessary for the synthesis of B_{12}.[39] Deficiency signs in the calf include cessation of growth, anorexia, and incoordination.[13] Injectable B_{12} has been used for supportive therapy with success in many diseased and debilitating conditions that cause inadequate intake or failure of rumen synthesis of B_{12}.[59]

Other Water Soluble Vitamins

Vitamin C

Ascorbic acid, vitamin C, is a metabolic need among all species, but a dietary need for vitamin C is not present in the calf.[58] There have been reports with cattle and rats on vitamin A deficient diets that ascorbic acid content of the tissues and blood plasma may be low resulting in an atypical vitamin C deficiency for those species.[39] Calves with enteric infections have been shown to have much less vitamin C in their blood than healthy calves.[49]

The three main physiological roles of vitamin C in the calf are its need for the proper formation of tissue collagen, its role in the metabolism of tyrosine by the body, and its role as a catalyst in hydroxylation reactions in the liver and especially the adrenals where it catalizes the activation of the adrenocorticosteroids.

It is suggested that supplemental vitamin C may prevent infection of young calves through the navel and may also help control pneumonia.[47] Young feedlot calves given 2.5 gm of vitamin C daily for three days produced a significant increase in resistance to respiratory infection.[7] It was postulated that immune stimulation was the mechanism as is thought to be the case in man also.

Storage of ascorbic acid is very limited. Excess ascorbic acid is excreted in the urine, therefore, the possibility of toxicity is remote.[14]

Choline

The roles of choline are to prevent abnormal accumulation of fat (fatty liver), and to act in forming acetylcholine, the main neurotransmitter of the body.

Johnson et al., (1951) described a choline deficiency syndrome occurring at seven days of age in the calf. The signs included anorexia, labored breathing, and slight fatty livers. After 16 days of age the deficiency could not be induced, probably due to a relative increase in synthesis compared to the requirement.

Recommendations

Therapeutically there are only a few clinically distinguishable disease conditions in the calf, where the administration of a specific vitamin will be beneficial. These conditions are: vitamin A to treat specific signs of hypovitaminosis A; vitamin D to treat rickets; vitamin E and selenium to treat white muscle disease or nutritional muscular distrophy; vitamin K to treat sweat clover poisoning; and thiamine (B_1) in treating cerebrocortical necrosis. Most clinical signs associated with deficiency of vitamins are nonspecific and may simply result in enhancing the establishment of a disease, or causing the calf to be weak, depressed, and anorectic.

The content of an injectable vitamin preparation should vary to fit the purpose intended. For use in specific therapeutic purposes, individual preparations of vitamins A, D, E-Se, K, and thiamine should be kept on hand as this is usually the most economical way to treat these metabolic disorders. For general prophylaxis and supportive therapy a multiple vitamin compound, whether commercially or individually prepared, is preferred. Keeping in mind that daily administration of high levels of fat soluble vitamins, especially vitamin A, is not needed as the calf can store excesses of these vitamins. Whereas, daily administration of water soluble vitamins may be indicated, because of the calf's inability to store excesses of these vitamins.

The following is a short listing of guidelines and indications for supplemental vitamin usage.

1. B-complex compounds are recommended over individual B-vitamin solutions.
2. Vitamin E and selenium should be used together except in high selenium areas.
3. Two or three injections of a vitamin A, D and E compound given to pregnant cows and heifers may significantly decrease calf mortality and decrease calving interval.
4. The newborn calf should receive an injection of vitamin A and other fat soluble vitamins within the first week of life to provide adequate body stores of the vitamins.
5. Vitamins A, D, E, C, and B_{12} levels of the calf effect the resistance of the calf to enteritis, pneumonia, and coccidiosis.
6. Administration of vitamin(s) E and/or C may stimulate the immune system of the calf.
7. Calves from heifers are often deficient in dietary vitamins.
8. Calves do not effectively synthesize B-vitamins in the rumen until four to eight weeks of age, therefore, administration of B-complex is indicated in the anorectic calf.
9. B-complex injections may stimulate the appetite of depressed, anorectic calves.
10. Prolonged antibiotic or coccidiostate therapy may require thiamine or B-complex, and vitamin K supplementation.
11. Weak or debilitated calves should receive an initial injection of vitamins A, D, and E, and then daily administration of a B-complex solution, or these vitamins may be incorporated with commonly needed fluid therapy.
12. The supplemental diet of an orphan calf should contain the essential vitamins in sufficient quantities to meet the total requirements of the calf.
13. Thiamine, B_{12}, and choline may be indicated in calves showing central nervous system disturbance signs.
14. Vitamin K is used in cases involving blood clotting problems.
15. Debilitated calves housed inside may require administration of vitamin D.

Setting up an effective vitamin regimen is like a successful antibiotic regimen. The practitioner must know which ones to use, why to use them, and how much to give. The information found in tables 2 through 6, as well as previous recommendations, should give you the necessary tools to decide why, when, and how to use vitamins in the treatment, prophylaxis and supportive therapy of the neonatal calf.

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