A CLINICAL AND PATHOLOGICAL STUDY OF VITAMIN A DEFICIENCY IN CALVES

by

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INTRODUCTION

During the last thirty years a large number of papers has been written about the effects of vitamin A deficiency in cattle. Many of these reports were based on field cases that occurred during years of drought and poor plant growth while others were based on controlled experiments. The ration used in these latter experiments often did not contain hay or they contained a low quality roughage such as straw. It is known that the ruminal microflora plays a very important role in the synthesis of certain vitamins and proteins in the body of ruminants. It is known that normally hays and grasses play a very important role in the general physiology of the rumen. When hay is left out, or a poor quality straw is used, it is possible to develop multiple deficiencies.

The reported experiment in this thesis was undertaken with the express idea of providing a ration as near as possible like the one that an animal would receive on the farm, yet be deficient in carotene. This diet was to include a good grade of oat hay that was extremely low in carotene, yet would contain factors that would maintain the ruminal flora. It was assumed that this would aid in maintaining the appetite of the animals so that the manifestations of other deficiencies might be eliminated.

There has been very little study made of the histopathology of vitamin A deficiency in cattle. Another purpose of this investigation, therefore, was to study the effects of vitamin A deficiency upon some of the tissues and organs of the body.

REVIEW OF LITERATURE

For centuries the effects produced by the lack of vitamin A have been observed both in animals and man without anyone knowing the cause. It was discovered, many centuries before Christ, that liver and grass were beneficial to the eyes of man and cattle respectively. All of the important progress in the field of nutrition concerning carotene and vitamin A has been made in the last thirty years.

Sources of Carotene and Vitamin A.

Cattle are able to convert provitamins A into vitamin A.in the body. The main provitamins A are alpha, beta, and gamma carotene, and cryptoxanthin. Green pasture grasses, green leafy legume hay, alfalfa silage, alfalfa meal and carrots are really good sources of carotene while other green grass hays, yellow corn, and corn silage of good quality are fair sources. Booher and Marsh (1941) pointed out that beta carotene is the chief source of vitamin A in green leaves.

For years livestock owners have known about the importance of colostrum to the general health and welfare of new born animals. Interest has centered around the carotene and vitamin A content of colostrum and whether or not these substances could be increased by adequate feeding. Savage and McCay (1942) reviewed the literature concerning colostrum and pointed out its importance as a source of immune bodies. Aschoffenburg et al (1948) produced evidence that the protective value of colostrum is associated with its globulin content. Dann (1933) found the vitamin A concentration in colostrum to be 10-100 times

greater than in the later milk. Maynard (1947) and Sutton et al (1947) reported that vitamin A and carotene are from 6 to 20 times greater in colostrum than in later milk. This is somewhat below the concentration reported by Dann. Stewart and McCallum (1938) found that vitamin A in colostrum varies from 35 to 1181 I. U. per 100 ml. According to Hart and Guilbert (1941) and Barron (1942), the calf is not able to store much vitamin A during fetal life and therefore is dependent upon colostrum for it.

Moore and Berry (1944) determined the amount of carotene and vitamin A in calves before sucking and each day thereafter for 1 week. The vitamin A content of the blood before sucking varied from 2.4 to 4.2 ug. per 100 ml. and in 3 days increased to 18.8 to 57.7 ug. per 100 ml. The carotene content for the Jersey and Guernsey breeds is 3 to 4 times higher than for the Holstein breed.

Stewart and McCallum (1942) were unable to increase the vitamin A content of colostrum by feeding 3 pounds of carrots or 1/7 pint of cod liver oil per day to cows. Parrish et al (1948) could not find any significant difference in the levels of total protein, casein, and albumin-globulin fractions in colostrum when heifers and cows received a high or low protein diet. Eaton et al (1949) found that vitamin A of the colostrum could be increased by adding extra amounts of vitamin A to the diet of cattle. Spielman et al (1946) (1947) (1949) demonstrated that vitamin A supplementation before parturtion increased the amount of vitamin A in the blood and liver of the new born calf and in the colostrum of the dam. Vitamin A alcohol was found to be the most effective supplement. This increase of vitamin A resulted in superior performance on the part of calves. Parrish et al

(1949) reported similar results. They found that feeding tocopherols prepartum did not help to increase vitamin A or carotene of the colostrum. Thomas et al (1947) showed that the vitamin A content of colostrum in sows and dairy goats could be increased by supplementing their rations with vitamin A.

The Effects of Plant Growth and Storage upon Carotene Content of Hay and Grain.

The carotene content of cattle feed can change materially over a period of time by oxidative processes. It is of importance to be aware of the factors that hasten or retard these destructive processes.. Coward (1925) mentioned that when the leaves of plants turn brown and dry up, their vitamin A value is lost. According to Hartman (1931), this loss of carotene is brought about by oxidative processes in drying hay in sunlight, or during unusually long seasons of dryness, as mentioned by Hart and Guilbert (1933). Schmidt (1941) revealed that alfalfa hay when exposed to the sun for 48 hours loses about 98 per cent of its carotene. Artificial drying of freshly cut green alfalfa preserves much of the carotene. Savage et al (1942) pointed out that hay may lose carotene on storage without marked change in color, and conversely that lack of green color is not necessarily an indication of the absence of carotene. Virtanen (1938) found that maintaining the acidity of silage below pH. 4 prevented destruction of carotene. Fraps and Treichler (1933) reported that the vitamin A content of feeds decreased in accordance with the length of time in storage and that alfalfa leaf meal lost 50 per cent and yellow corn 30 per cent of the vitamin A potency in 5 months under

ordinary storage conditions. Guilbert (1935) found that variations in temperature play an important role in the rate of loss of carotene. Over a 15 day period the rate of loss of carotene was roughly doubled for each 10 degree rise in temperature. Baltzer et al (1942) made a survey of different grades of hay fed to dairy cattle in Michigan and found that the carotene content varied from 4.3 to 57.4 ug. per gram of dry matter. Mitchell and Hauge (1946) presented evidence that the carotene destroying enzyme in alfalfa is a lipoxidase. These workers in a later report (1946) pointed out that only a small amount of carotene was lost before wilting took place. The cell permeability appeared to limit carotene destruction. Enzymic destruction of carotene during field curing of hay appeared to be greater than destruction by light.

Absorption of Carotene and Vitamin A

Bicknell and Prescott (1947) reported that fat is necessary for the efficient absorption of carotene. The probable importance of fat is due to its dissolving the carotene and making it available for absorption. The transfer of carotene across the wall of the intestine probably depends on the formation of a water soluble diffusible complex with bile acids. After the carotene has passed into the lacteals it is transported to the liver in colloidal suspension.

In animals the presence of bile is unnecessary for the absorption of vitamin A because when the common bile duct is tied off, vitamin A is still absorbed. Fat and fatty acids are also necessary because when fat absorption is impaired the vitamin A absorption is interfered with. When vitamin A is transferred across the intestinal wall in the form of an alcohol, the naturally occurring vitamin A esters are first hydrolyzed,

(by the enzymes of the intestine), like the other esters of the fatty acids. Before passing into the lacteals, the alcohol is again combined with fatty acid. The maximum rise in the level of vitamin A in the blood occurred within 3 to 5 hours after the carotene is taken by mouth.

There are several factors which play an important role in the amount of carotene or vitamin A which can be absorbed by the intestine. Breece et al (1942) tested the absorption of vitamin A in 29 patients with severe pulmonary tuberculosis and intestinal symptoms of varying severity and found it to be only half that of normal patients. Reifman et al (1943) and Shaw et al (1944) observed that the rate of absorption of vitamin A and carotene was proportional to the dosage given. Greaves and Schmidt (1935) proved that bile salts, such as glycodesoxycholic or desoxycholic acid, were required for the absorption of provitamin A in vitamin A deficient choledochostomized rats as judged by the vaginal smear technique. Irvin et al (1941) made use of intestinal loops of dogs to show that very little carotene is absorbed in cottonseed oil solutions. When ox or hog bile or pancreatic lipase was administered, significant quantities of carotene were absorbed. Molander (1949) demonstrated that only 66 per cent of the carotene in an emulsion of mineral oil was absorbed by the gastrointestinal tract. This was true when the carotene was dissolved in corn oil. The fatty acids of corn oil are apparently not efficient carriers of carotene. The author studies the distribution of carotene when absorbed in the above vehicles and found that mineral oil and corn oil caused a systemic distribution, whereas the fatty acids of corn oil carried most of the carotene to the liver.

Conversion of Carotene to Vitamin A

Rosenberg (1942) stated that the mechanism of the conversion of provitamin A to vitamin A was not known. Moreover, exact site for the conversion of provitamin A was unknown although evidence pointed toward the liver. He claimed that the rat was the most efficient converter of provitamin A; chickens, guinea pigs, rabbits, pigs, and cattle were less efficient. Cats are apparently incapable of this conversion. Bicknell and Prescott (1947) reported that the conversion of carotene to vitamin A occurred in the liver by the action of an enzyme, carotenase. Incubation of carotene with fresh liver tissue or aqueous extracts changes the carotene to vitamin A; this conversion is stopped by heat, which suggests the action of an enzyme.

The thyroid gland apparently plays a role in conversion and storage of vitamin A and carotene in the body, although experimental evidence at times has been contradictory. Drill and Truant (1947) made a study of the effect of thyroidectomy on the conversion of carotene to vitamin A and found that the thyroid gland plays a major role in the conversion. Johnson and Baumann (1947) found that rats were able to store preformed vitamin A equally well whether they are hypothyroid or hyperthyroid. When carotene was fed to hyperthyroid animals they accumulated larger stores of vitamin A than normal rats receiving equivalent amounts of carotene. They hypothyroid rats stored very little vitamin A. The addition of thyroxin restored the conversion ability of these rats. Wiese et al (1948), however, demonstrated that rats made hypothyroid utilized carotene and vitamin A equally well.

Some research work has been done in the last few years which indicates that the intestine may be the site of conversion of carotene

to vitamin A. Mattson et al (1947) (1948) concluded that the site of conversion of carotene to vitamin A in the rat is the intestinal wall. This is based on the fact that the intestine is the only organ of the rat in which an increase of carotene is found following its administration, and vitamin A is found in the intestinal wall only during a period following the feeding carotene or vitamin A. The authors pointed out quite conlusively that vitamin A is the substance formed in the intestine as it exhibits fluorescence, possesses an absorption curve similar to that of vitamin A, and shows a single fluorescing band in a mixed chromatogram with vitamin A. Stallcup and Herman (1950) using isolated loops of intestine, sections of liver and blood plasma from dairy calves established that conversion of carotene to vitamin A took place in the intestine and liver but not in the blood plasma. Sexton et al (1946) demonstrated that the intestinal wall appears to act as a barrier beyond which beta-carotene can not pass. Beta-carotene injected intravenously, intraperitoneally, or intraspenically accumulated in the liver without an increase in the vitamin A content of the liver or plasma. Beta-carotene could be detected in livers for as long as 46 days after injection. When beta-carotene was given orally in cottonseed oil, the vitamin A levels of liver and plasma increased hourly.

Vitamin A and Carotene Requirements

Guilbert and Hart (1935), Moore (1939) and Ward et al (1940) reported that the daily minimum carotene requirement of the bovine is between 24 to 35 ug. per kg. of body weight. Hart et al (1940) summarized their data on the minimum vitamin A and carotene requirements

of various species.

TABLE I

Daily Intake per kg. of Body Weight

	Vitamin A		Carotene	
Species	Micrograms	I.U.	Micrograms	I.U.
Cattle	5.1 - 6.4	21-27	26 - 33	43-55
Sheep	4.3 - 6.3	17-26	25 - 35	42-53
Swine	4.4 - 5.7	18-24	25 - 39	42-65
Horse	4.2 - 5.3	17-22	20 - 30	33-50

Gallup et al (1941) stated that a plasma carotene value of less than 150 ug. per 100 ml. in pregnant Jersey cows indicates a borderline deficiency of vitamin A. Boyer et al (1942) reported that a blood plasma vitamin A level of 10 ug. or more per 100 ml. is necessary for adequate nutrition of growing calves. The daily carotene requirements necessary to maintain an adequate plasma vitamin A level and prevent deficiency symptoms are 75 ug. per kg. for Holstein yearlings and 125 ug. per kg. for Guernsey yearlings. The blood plasma carotene levels which would maintain an adequate blood plasma vitamin A are 50-70 ug. of carotene per 100 ml. for Holsteins and 110-140 ug. of carotene per 100 ml. for Guernseys. Sutton et al (1945) observed that Holsteins and Ayrshires require less carotene to maintain blood plasma levels of vitamin A than do Jerseys and Guernseys.

Animals require far more carotene and vitamin A for optimum growth and reproduction than for maintenance. Loosli et al (1945) re-

commended the following: calves weighing 100, 150, 200, 400, 500 and 800 pounds should receive 6, 10, 12, 25, and 35 mg. of carotene per day respectively for optimal growth. Lewis et al (1945) found that calves need from 64 to 512 I.U. of vitamin A daily per kg. of body weight for optimal growth and some storage in the liver. They found that a high concentration of vitamin A retarded growth. Payne and Kingman (1947) stated that for adequate health and reproduction, Herford heifers and cows should have blood plasma levels of carotene of at least 117.25 and 82.88 ug. per 100 ml. respectively.

Moore et al (1948) reported the normal cerebrospinal pressure of Guernseys and Jerseys to be around 75 to 120 mm. of water. Increased spinal pressure was not observed when the animals received 30 ug. of carotene or more per pound of body weight. Watkins and Knox (1950) studied the blood plasma levels of carotene and vitamin A in beef cattle and concluded that vitamin A deficiency is not apt to occur except in times of prolonged drought when plant growth is sparse. Bassett et al (1946) established that the vitamin A requirement necessary to prevent nervous symptoms in growing fox pups is between 15 to 25 I. U. per kg. of body weight per day. Storage of vitamin A does not occur in the liver until 50 to 100 I. U. of vitamin A per kg. are fed per day.

Storage of Vitamin A and Carotene

The amount of vitamin A and carotene in the blood and in the tissues is dependent on various factors. Popper and Steigmann (1943) list the following: 1. nutritional intake; 2. disturbance of intestinal

absorption; 3. increased demand; 4. disturbed interaction of liver and blood; 5. sparing action of vitamin E; 6. lipid concentration of blood. Barron (1942), Josephs (1943), and editors of Nutritional Reviews (1943) pointed out that in general diseases of the lungs, liver, stomach, and intestine reduce the amount of vitamin A and carotene in the body. However, in one disease, tuberculosis, the vitamin appears to be increased. Wendt (1941) reported that in dogs some vitamin A is normally destroyed when the blood passes through the lungs. Aron et al (1946) observed a depression of plasma vitamin A and carotene in man with an artificially induced fever. Pavcek et al (1945) noticed that livers of cattle with telangiectasis are toxic when fed to rats and stated that it is probably due to their abnormally high content of vitamin A. Kao and Sherman (1940), Lewis et al (1941), Bassett et al (1946), and Frey et al (1947) showed that the amount of vitamin A stored in tissues depended on its concentration in the diet.

Guilbert and Hart (1935) found that 93 per cent of the body stores of vitamin A in cattle is in the liver. The principal stores of carotene are in the body fat. Sakamoto (1940) determined that next to the liver the content of vitamin A is highest in the choroid and retina. In the other tissues very little vitamin A is present. Frey et al (1946) used 140 Herefords for experimentation. Of these, 22 animals were killed immediately and it was found that the livers contained 9.68 ug. of vitamin A for each 1 ug. of carotene. Another group of 98 animals was placed on a fattening ration for 166 days. At the end of this period their livers contained 1.06 ug. of vitamin A for

each 1 ug. of carotene. The remaining Herefords were kept on a maintenance ration for the same period as the fattening animals and their livers contained 2.5 ug. of vitamin A for each 1 ug. of carotene. Spielman et al (1946) showed that feeding large amounts of carotene to cows during gestation increased the content of carotene in the blood plasma and the liver of new born calves. The addition of 1 million I.U. of vitamin A daily to the normal ration of pregnant cows resulted in an average total fetal liver content of 97,177 I.U. of vitamin A. This work definitely showed that both vitamin A and carotene can be transferred from the mother to the fetus by way of the placenta. Johnson and Baumann (1947) working with rats found that at low levels of carotene intake, (35 I. U. daily), more vitamin A appears in the kidney than in the liver; at higher levels more vitamin A appears in the liver than in the kidney. Coombes et al (1940) stated that foxes do not store carotene in the liver, whereas storage of vitamin A on a good diet is between 150 and 200 ug. per gram.

Cox (1941) studied three human cases which had passed through a condition of massive necrosis of the liver and came to the conclusion that hepatic cells are not essential for storage of vitamin A. The altered liver tissue had more vitamin A than the normal tissue. He felt that vitamin A was stored in the Kupffer cells which were markedly increased in disease. Clayton and Baumann (1944) pointed out that hepatic storage of vitamin A appeared to be relatively independent of other biochemical processes taking place in the liver. Dimethylaminobenzene, hydrocoumarin, vitamin K, high or low fat diets, and severe choline deficiency did not affect the storage.

Manifestations of Vitamin A Deficiency.

In the Eyes of Cattle

The symptoms of vitamin A deficiency have been observed for many years, especially those symptoms involving the eyes. The Bible (601 B. C.) probably gave the first information indicating that the vitamin A deficiency may have existed in ancient times and involved the eyes. "And the wild asses did stand in the high places, they snuffed up the wind like dragons, their eyes did fail, because there was no grass." Connell and Carson (1896) described a condition called "fat sickness" which is often accompanied by inflammation of the eye or total loss of sight in cattle.

The presence of night blindness or nyctalopia is one of the earliest signs of vitamin A deficiency. Hart and Guilbert (1933) observed that when cattle received dry feed for nine months, night blindness was quite common in the younger animals. Wald (1934) found that night blindness is brought about by the lack of vitamin A which maintains visual purple. There is a visual cycle in the eye and vitamin A plays an important part in it. In a normal animal the visual purple of the retinal rods is bleached out by bright sunlight. Vitamin A is the precursor of this substance which is essential to vision in dim light. Visual purple is bleached to retinene or visual yellow and this in turn is partly reconverted to vitamin A. Calves which Moore (1939) placed on a low carotene ration at 40-90 days of age usually developed nyctalopia in from 48 to 73 days. Schmidt (1941), Jones et al (1943), and Stubbs (1944) also reported night blindness as an early symptom. Some of the other symptoms reported by workers are dilatation of the pupils, lacrimation, protusion

of the eye-balls, xerophthalmia, papillary edema, fading of the tapetum lucidum, corneal opacity and ulceration, and permanent blindness. Jones et al (1943) observed that the pupils of the eyes did not close properly and that the eye-balls appeared to protrude from their sockets (fish-eyes). Hart and Guilbert (1933), and Jones et al (1943) reported a very marked lacrimation in vitamin A deficiency. Xerophthalmia has been described quite widely in man and rats but not in cattle. Mead and Ryan (1931) were the only workers to report this condition in cattle. De Schweinitz and De Long (1934), and Moore (1939) using an ophthalmoscope observed definite swelling and clouding of the optic disc due to papillary edema. The latter author though this edema was brought about by an increased intracranial pressure. Moore observed the fading of the tapetum lucidum from a yellow color (when adequate vitamin A was present) to a pale blue (lack of vitamin A). Hart and Guilbert (1933), Schmidt (1941), Jones et al (1943) and Alvarez (1947) reported inflammations of the eyes which led to corneal opacities and ulcerations. Moore et al (1934) and Moore (1939) did not find this true in their experimental animals. Crocker (1919), Moore et al (1934), McNutt and Wall (1938), and Moore (1939) showed that permanent blindness is brought about by stenosis of the optic nerve where it passes through the optic foramen. Wezel and Moore (1940) discussed some of the theories given for blindness such as inbreeding, a hereditary condition analogous to Leber's disease, basterial or protozoal infections, food poisoning, dietary factors, insidious rachitis, intracranial pressure, and pressure from overgrowth of bone.

In the Eyes of Other Mammals and Man

Hostetler et al (1935) fed pigs a diet deficient in vitamin A and noticed the following symptoms: lacrimation or watery discharge from the eye, slight pus, a protrusion of the eye-ball, clouding of the iris, temporary blindness in one or both eyes which later became permanent. Some of the eyes had a tendency to ulcerate and finally rupture. Hale (1935) placed gilts on a diet so that their vitamin A was depleted to a very low level before breeding and for 30 days after breeding which is the time during which the eyes develop in the pig embryo. The animals then received adequate amounts of vitamin A. Two gilts farrowed 21 pigs among none of which had eye-balls. Another gilt that received a single dose of cod liver oil two weeks before conception farrowed 14 pigs which had various eye defects - some without eyes, others with one eye, and still others with one large eye and one small eye. All were blind. Fairbanks (1940) and Anderson and Hart (1943) observed eye changes in the horse similar to those described for cattle. Mann et al (1946) showed that the eyes of rabbits develop metaplasia and keratinization of the cornea with the final result that this structure gradually became opaque.

Bicknell and Prescott (1947) stated that the first detectable sign of a lack of vitamin A in man was a slight impairment of dark adaptation. The change in the eye that could be observed clinically was a drying or xeransis of the eye known as xerophthalmia which is accompanied by photophobia. Moore (1945) reviewed some of the literature on the relationship of vitamin A, riboflavin, and ascorbic acid in dark adaptation. It was pointed out that when individuals with poor

adaptation do not respond to vitamin A therapy, often the addition of riboflavin produces immediate response. Pure ascorbic acid often brings about improvement in dark adaptation as does vitamin A. Spies et al (1945) mentioned that in humans with riboflavin deficiency, interstitial keratitis is seen in 60 per cent of the patients and corneal ulceration in 53 per cent. Patients responded to riboflavin therapy.

In the Reproductive System

Male

Reproductive failure, either permanent or temporary, is one of the most serious problems confronting dairy herd owners. In recent years more attention has been given to the reproductive performance of the male and its relationship to an adequate diet. Jones et al (1945) reported the characteristics of 67 normal bull semen samples without consideration of breed or age. The samples showed excellent motility, averaging about 5.4 ml. per sample; revealed a sperm concentration of 1,120,000 per cmm.; had 11 per cent abnormal spermatozoa, and in 70 per cent of the samples gave an initial pH. reading between 6.40 and 6.80.

The following effects have been observed in bulls when the vitamin A or carotene was low in the diet: 1. inability of bulls to mount and deliver semen although retaining an unusual degree of libido; 2. complete or partial failure of spermatogenesis; 3. a low concentration of motile spermatozoa; 4. a high percentage of abnormal spermatozoa; 5. a high pH of the semen; 6. variation in the number of sperm from the normal amount to none. Guilbert and Hart (1935) reported that spermatogenesis did not take place in the testes of one of their

experimental bulls fed a diet low in vitamin A. They mentioned, however, that this sterility was not permanent because adequate amounts of vitamin A restored the spermatogenesis. Hodgson et al (1946), Bratton et al (1948), and Madsen et al (1948) pointed out the inability of bulls to copulate although retaining some libido. These authors cited changes in the semen such as a low concentration of motile sperm, a high percentage of abnormal spermatozoa and an increase in pH. Hodgson et al (1946) and Erb et al (1947) stated that the formation of cysts in the anterior pituitary glands might be the cause of reproductive failure in vitamin A deficient bulls. Chevrel and Cormier (1948) found testicular changes in male rabbits on a vitamin A deficient diet although the animals received 10-20 mg. of tocopherol daily.

Female

It has been known for some time that cows suffering from vitamin A deficiency may show poor reproduction, premature births, may retain their placentae, and give birth to weak or dead calves. In rats and pigs resorption and malformations of the fetuses have been reported.

Hart and Guilbert (1933) made a study of vitamin A deficiency in a herd of 250 head of cattle of which 100 died. In 25 to 30 cows which calved at the height of the deficiency, all of the offspring were weak when born, had severe diarrheas, and died in one to five days. Hart and Guilbert (1947) stated that cows may abort if sufficient carotene and vitamin A are not present in their diet. Cunningham and Addington (1938) observed abortions in cows on vitamin A deficient rations which were explained on the basis of edema developing in the placentae and causing the fetal villi to separate from the maternal crypts of the cotyledons,

thus cutting off the food supply to the fetus. Hart (1940) cited some of his early work in 1911 where animals were placed on restricted rations of a single plant such as wheat, oats, or corn, in which it was shown that failure of reproduction could be nutritional in origin. Davis and Madsen (1941) found that heifers receiving 30-45 ug. of carotene per kg. produced calves that were either blind and weak, or dead at delivery. Braun (1945) and Sutton et al (1945) discovered that blood plasma vitamin A levels in cattle from two to four weeks before parturition or abortion take a significant sharp drop, and reach their lowest level a few days after parturition or abortion.

Hughes et al (1928) and Hughes (1934) mentioned that gilts and sows do not reproduce if the vitamin A intake is not adequate. The animals not only abort but often the fetuses are partially resorbed. Hade (1935) reported that in addition to pigs being farrowed with no eye-balls, they had hare lips, cleft palates, accessory earlike growths at base of ear, malformed hind legs, and kidneys failing to ascend from their embryonic position.

Mason (1935) stated that fetal death in the rat takes place because of pathological alterations in the epithelial cells of the maternal placenta consisting of focal necrosis in the maternal decidua adjacent to the fetal trophoblast. This is followed by alterations in the fetal tissue. Browman (1938) found that reproductive performance is much lower in rats twice depleted of vitamin A. No eye, limb, or other microscopic abnormalities were observed in the 260 young rats born to females on low levels of vitamin A supplement. Wilson and Warkany (1948) discovered malformation in 42 fetal and newborn rats from mothers deficient in vitamin A. The malformations consisted of failure of parts to develop,

organs fusing, and parts being out of position in the body. Warkany and Roth (1948) fed 260 female rats a basal ration and varied the amount of carotene in this manner: 28 rats received 25 mg. of carotene the first month and twice as much the last 2.5 weeks; 165 were given 4 mg. of carotene, 34 received 12 mg. and 33 received 25 mg. every 10 days. The results were as follows: 22 had heat cycles but did not mate; 124 mated but did not have litters; 114 had litters but only 30 carried young to term. Eighty-nine litters were abnormal and only 25 litters normal. When these figures were broken down further, 612 rats were abnormal and only 208 normal. Wilson and Warkany (1947) mentioned that the earliest sign of metaplastic keratinization in rats is seen in the genitourinary tract of the fetus on the 18th day of gestation in vitamin A deprived mothers. Truscott (1947) revealed that female rats placed on a vitamin A deficient ration showed a gradual decline in weight of their ovaries. Fraps (1947) reported that 38.2 mg. of carotene per rat daily is best for breeding.

In Gastrointestinal Tract

Enteritis in calves is quite generally a serious problem and incites interest, whether its cause be nutritional, infectious, or otherwise. Hart and Guilbert (1933), Keener et al (1942), and Thorp et al (1942) reported scours in calves fed a diet deficient in vitamin A. Stewart and McCallum (1938) made a study of the correlation between the incidence of white scours in calves and the amount of vitamin A in the colostrum. In 83 calves which received colostrum containing more than 250 blue units of vitamin A., 10.8 per cent developed scours, whereas in

28 calves which received colostrum containing less than 250 blue units, 25 per cent developed white scours. Phillips et al (1941) and Phillips (1945) stated that approximately 90 per cent of all scours and allied diseases of calves were directly related to nutritional causes. They recommended feeding with high potency shark liver oil and some of the members of the vitamin B complex group. Krauss et al of Ohio, and Huffman et al of Michigan (1945) and Spielman (1946) showed that scours is just as prevalent in calves which receive vitamin capsules as those which receive placebos. Blakemore et al (1948) observed that the administration of vitamin A concentrate fails to protect calves from white scours. Spielman et al (1949), however, found that calves fed a vitamin A supplement had less scours than those on the basal ration.

Tilden and Millar (1930) and Verder and Petran (1937) revealed that diarrhea (as a symptom of vitamin A deficiency) is more constant than the eye lesions in the monkey. Ramalingaswami (1949) described 20 cases of vitamin A deficiency in children associated with diarrhea which responded to vitamin A concentrates. Wheeler (1945) demonstrated that a high vitamin A content in the diet of domestic and non-domestic animals can accelerate the metabolism to the point where it causes diarrhea. Seelig (1940-41) selected 13 human patients with gastric ulcers which had been resistant to medical treatment for more than a year without success. These patients received daily doses of 120,000 to 780,000 I.U. of vitamin A. Radiological, gastroscopic and clinical evidence indicated favorable results in 3 to 5 weeks in all except 2 cases.

In the Nervous System

Moore and Sykes (1940) placed calves on a low vitamin A diet and observed papilledema, syncope, and incoordination. Cerebrospinal fluid pressures were high and there appeared to be a relationship between these pressures and the above symptoms. When these calves were placed on a normal diet the cerebrospinal pressures returned to normal. Boyer et al (1942) found that normally the intracranial pressure measures from 100 to 200 mm. of water and the vitamin C content of the spinal fluid is 2.3 mg. percent. They noticed that as the cerebrospinal pressure became higher there was a definite drop in the vitamin C levels. The administration of vitamin C to vitamin A deficient calves resulted in a rise in the vitamin C content of the cerebrospinal fluid. A reduction of the cerebrospinal pressure occurred in 3 out of 5 cases. Moore et al (1943) found that an intake of 62 ug. of carotene per kg. of body weight was not sufficient to prevent an increased spinal pressure but a minimum of 66 ug. of carotene prevented it in Holstein and and Ayrshire calves. Moore (1946) concluded that vitamin C does not play a role in the mechanism of increased spinal pressure.

In the Connective Tissues

Bechtel et al (1928), Guilbert and Hart (1935), Cunningham and Addington (1938) Schmidt (1941), and Hastings (1942) found edema associated with vitamin A deficiency. The edema appeared to be more extensive in the extremities. Creech and Seibold (1943) revealed that there was extensive edema in the subcutaneous tissues and muscles accompanied by an inflammatory process. Madsen and Earle (1947) stated

651 beef carcasses were condemned for generalized edema or anasarca by federal meat inspectors during July, 1941 to December, 1946. This edematous condition occurred in cattle after a long fattening period in dry lot when fed either stored or new corn in combination with a roughage of low carotene content such as oat, hay, and straw. Alfalfa was highly effective in curing the condition. They were of the opinion that the edema was not due to lowered colloidal osmotic pressure.

In the Urinary System

There has been much controversy for several years as to whether or not the lack of vitamin A played an important role in the formation of urinary calculi. Osborn *et al* (1917) found a high incidence of calculi in their vitamin A deficient rats. Newson (1938) and Schmidt (1941) observed urinary calculi in cattle and sheep on vitamin A deficient rations. Newsom *et al* (1943) observed that lambs were more likely to develop urinary calculi on a ration composed of cane fodder, bran, and white corn than on one containing alfalfa, beet tops and yellow corn. Bassett *et al* (1946) placed fox pups on diets deficient in vitamin A and 21 animals out of 49 developed urinary calculi. In a study of 98 human cases of clinical urolithiasis, Jewett *et al* (1942) came to the conclusion that vitamin A deficiency does not play a role in it. Jones *et al* (1943) did not report urinary calculi present in their vitamin A deficient calves and yearlings. Beeson *et al* (1943) noticed that urinary calculi developed in the kidneys of sheep that received ample amounts of vitamin A.

Other Manifestations

Hughes et al (1929) stated that incoordination is the most outstanding symptom of avitaminosis A in pigs, whereas the eye lesions are not prominent. Patton (1944) (1945) observed a relàtionship between acetonemia and vitamin A deficiency in cattle, and pointed out that in uncomplicated cases animals often responded to vitamin A therapy. Burt (1944) and Scott (1945) reported that they obtained good results with vitamin A in the treatment of acetonemia in cattle. Shaw et al (1945) and Hendershot (1946) found that vitamin A did not play an important role in ketosis. Langham et al (1944) reported that pneumonia occurred in 33 calves out of 44 on a vitamin A deficient ration. After Mayer and Krehl (1948) placed rats on a diet deficient in vitamin A the animals developed symptoms resembling those of acute scurvy. The level of ascorbic acid in the blood was 50 to 66 per cent below normal. These scurvy symptoms disappeared when injections of ascorbic acid were given. Hassan et al (1948) considered that a deficiency of vitamin A in man causes a physiological leukocytosis, and that the vitamin is, therefore, directly concerned in the protective mechanism against infection.

In addition to the symptoms that have been mentioned previously in the thesis numerous workers have reported rough hair coats, scaliness of the skins, slowness of growth, and difficulty in getting up.

Histopathology

The microscopic study of vitamin A deficiency in cattle has been rather meager and confined to a very limited number of organs such as the eyes, pituitaries, kidneys, livers, and intestines. The most

accurate and thorough work has been done on the rat. Everett (1942) listed the four most important functions of vitamin A as follows: 1. Maintenance of normal epithelia of the skin, the eye, the upper respiratory, gastro-intestinal and genito-urinary tracts, and also of the ducts, and acinar tissues of secretory glands. 2. Maintenance of normal nerve tissue. 3. Promotion of normal growth of bone and of tooth enamel. 4. Participation in the visual cycle of dark adaptation.

Moore et al (1934) in the study of nutritional blindness in cattle observed pressure atrophy of the optic nerve where it passes through the optic foramen but sections from several cases did not show any exostosis of the canal. The canal gave more of the appearance of having had pressure applied from above which caused it to become smaller as growth proceeded. The authors suggested that intracranial pressure might produce this condition. Moore et al (1939) (1940) after studying more cases of permanent blindness in cattle stated that this condition is probably brought about by an increased intracranial pressure. Mellanby (1947) showed that there is extensive nerve degeneration especially in the cranial nerves such as the olfactory, optic, trigeminal (1st and 2nd divisions) and auditory in growing puppies in vitamin A deficient rations. The nerve degeneration is brought about by overgrowth of bone which puts pressure on the nerves. When vitamin A is low the osteoclasts fail to absorb bone normally but the osteoblasts continue to lay down new bone thus resulting in the overgrowth of bone. Wolbach and Bessey (1942) concluded from their studies of rats that the nerve lesions are wholly mechanical in origin. This condition of degeneration of the nerves, they reasoned, is brought about by stoppage of growth of the skeleton while the nerves continue to grow. These investigators confirmed the

formation of excess periosteal bone in realtion to the bony labyrinth of the ear in dogs, as reported by Mellanby, and have recorded similar findings in rats and guinea pigs. They did not find excess bone formation in any other part of the skeleton of the rat. Copp and Greenburg (1945) found that in vitamin A deficient rats the callus is much smaller in the healing of a fracture than normally and calcification is less active. There is a delay in fracture healing. Richards (1935) claimed that growth does not cease in vitamin A deficiency when the animal ceases to increase in weight. This was proved not only by direct measurements of body length of the live animal, but by direct measurements of the limb bones after death.

Wolbach and Howe (1925) observed a shrinkage of the gland cells of the pituitary in rats but no change in the architecture of the gland. Sutton and Brief (1939) pointed out that numerous investigations have shown a relationship, reciprocal in nature, between the gonad and pituitary gland. They observed a marked increase in the beta cells of the pituitary from vitamin A deficient rats. They pointed out that this work profided evidence that vitamin A deficiency exerted a direct effect on the gonads. Madsen et al (1942) studied some cystic pituitary glands of cattle and observed that a considerable portion of the functional anterior lobe was replaced by a cyst while some of the remaining portions showed increased amounts of connective tissue. Some of the glandular tissue contained pyknotic nuclei.

Wolbach and Howe (1925) stated that the specific tissue changes which follow the deprivation of fat soluble vitamin A in albino white rats and in human beings concerns epithelial tissues. The effect is the substitution of stratified keratinizing epithelium for normal

epithelium. These changes are observed in various parts of the respiratory tract, alimentary tract, eyes, paraocular glands, and the genitourinary tract. The replacement of epithelium arose from focal proliferation of cells arising from the original epithelium and not by differentiation or change of pre-existing cells. Wolbach and Howe (1933) made a study of repair of this keratinized epithelium in rats when sufficient vitamin A was added to the diet. The repair appears to take place from the lower-most cells of the epithelium. These cells, functionally correspond to the cells of the stratum germinativum of the epidermis. Wolbach and Bessey (1942) stated "the epitheliums which atrophy and which become replaced by stratified keratinizing epithelium are those having a secreting (chemical) function in addition to the role of a covering layer and whose functioning cells are without power to divide. Repair, therefore, takes place from focally distributed basal cells which multiply, spread beneath the original epithelium, and finally, through coalescence of areas thus produced, form a continuous epidermis-like layer." The histological sequences observed in the removal of cells above the stratum germinativum indicate the autolysis as shown by vacuolar degeneration, and heterolysis as shown by leucocytic infiltration. Follis (1946) reviewed the effects of vitamin A upon the trachea. The first changes appear to be an atrophy of the columnar cells. This decrease in size involves the cytoplasm and not the nucleus. Small syncytial masses of cells begin to arise from these atrophic columnar cells and these groups rapidly develop into a keratinized type of epithelium. Thorp et al (1942) noted some metaplasia of epithelium in the calices of the kidney and trachea of calves. They described conspicuous lesions in the kidney, testicle, liver, and intestine. All of these organs

showed some degeneration and necrosis along with some inflammation. Langham et al (1940) reported some degeneration and necrosis of the tubular epithelium and focal areas of inflammation in the kidneys of calves on a vitamin A deficient ration.

Wolfe and Salter (1931) and Wolbach and Bessey (1942) reported that the undifferentiated cells of the germinal epithelium are spared in rats on a vitamin A deficient ration. Bratton et al (1948) found in bulls degeneration of the germinal epithelium of the seminiferous tubules, with few spermatogonia, spermatocytes, spermatids, or maturing spermatozoa in the lumina of the tubules. Moore (1936) observed foci of inflammation and epithelial metaplasia in the prostatic acini and ducts.

Creech and Siebold (1943) found in field cases of vitamin A deficiency edema and atrophy of the peripheral nerves of the subcutaneous tissues. The peripheral arterioles and capillaries were more tortuous in appearance in the subcutaneous tissues and muscles. Hydropic and atrophic changes occurred in the skeletal muscles. There was thickening of the walls of the heart arterioles. Opper (1939) reported changes in the blood vessels in 21 out of 24 rats on a vitamin A deficient ration. There was medial degeneration in all vessels down to the larger arterioles. Necrosis of the smooth muscle cells of the media was accompanied by deposition of calcium granules in pericellular and perilamellar distribution. This was followed by the formation of large calcium plaques and formation of granulation tissue in the media. Proliferation of connective tissue in the subintima often led to complete closure of small visceral vessels. When this latter condition took place in the heart myocardial fibrosis was the result.

GENERAL EXPERIMENTAL PROCEDURE

Calves of the Holstein, Guernesy, Jersey, Aryshire, and Brown Swiss breeds from the experimental and dairy herds of Michigan State College were used in these experiments.

30 ml. of blood were drawn from the jugular vein weekly into test tubes containing potassium oxalate as an anticoagulant. Fresh smears on clean glass slides were made immediately, dried with an electric heater, and stained with Wright's stain. Standard procedures were employed for the blood counts and cell volumes as described by Kolmer (1944). The only variation to his methods was the use of Leake and Guy diluting fluid in place of Hayem's solution. Hemoglobin determinations were made using the method of Hoffman (1941). The mean corpuscular volume and mean corpuscular hemoglobin content were calculated by the formulas of Wintrobe (1942).

The blood plasma values for carotene and vitamin A were determined by the method of Boyer et al (1944). Standard carotene and vitamin A curves were made from the results obtained from reading various aliquots of these substances in the Cenco photelometer. The aliquots were produced by dissolving weighed amounts of crystalline carotene and crystalline vitamin A acetate into ether and chloroform respectively. A 410 P blue filter was employed for carotene and a 610 P orange filter was used for vitamin A.

The calves were examined daily, and treated if necessary for pneumonia and scours. The weights of the calves were taken every 10 days. The heights of the animals were measured periodically and taken at the highest point of the withers. The eyes were examined weekly

with an ophthalmoscope. Night blindness was determined by the inability of calves to avoid objects in dim light.

A necropsy was made upon each animal in order to determine the exact cause of death and to obtain tissues from certain cases for histological study. Parts of the following tissues were saved: skin, cerebrum, cerebellum, pituitary, optic nerve and foramen, spinal cord, the eyes, tongue, parotid gland, mandibular salivary gland, sublingual salivary gland, thyroid, epiglottis, trachea, lung, liver, gallbaldder, pancreas, rumen, abomasum, esophagus, various portions of intestine, kidney, urinary bladder, epididymis, testicle, penis, uterus, cervix, heart, spleen, adrenal gland, bronchial and mesteric lymph nodes, aorta, and skeletal muscle. The tissues were fixed in 10 per cent formalin, Zenker's fluid, and Carnoy's fluid. The formalin fixed tissues were used for fat stains. The Carnoy's fluid was used to preserve the glycogen in tissues so that it could be stained. All of the other stains used in this work was done on Zenker's fixed tissues.

The following stains were used according to the methods of Mallory (1938), sudan iv. for fat, hematoxylin and eosin for the usual histological features of all tissues, and Verhoff's stain for elastic tissues. For connective tissue Mallory's anilin blue (Heidenhain's azo-carmine modification) was used as described by McGregor (1929). Glycogen was stained by employing the method of Chipps and Duff (1942).

EXPERIMENT I

The Non-Colostrum Group

The Effect Of A Diet Deficient In Vitamin A

Non-Colostrum Group

Purpose

To study the effects of a vitamin A deficient ration upon calves that did not receive colostrum at birth but were supplemented with dam's whole blood or serum.

Experimental Procedure

A group of six animals of the Holstein, Jersey, and Brown Swiss breeds were placed upon a basal ration deficient in vitamin A. Two of these animals were selected as controls and received 250,000 I.U. and 100,000 I.U. of vitamin A in the form of shark liver oil in one oral dose, respectively. The other four animals were placed on the basal ration alone. The first few weeks of life the animals received whole milk followed by skim milk until they were eating the basal ration. Water was then substituted entirely for the milk.

Basal Ration

32 pounds of oats

35 pounds of barley

15 pounds of soybean oil meal

10 pounds of skim milk powder

1 pound of calcium carbonate

1 pound of bone meal

5 pounds of Brewer's yeast

15 grams of irradiated yeast (9000 I. U. per gram)

1 pound of salt mixture, the formula for which consisted of:

307.8 grams of sodium chloride

62.5 grams of ferrous sulphate

77.0 grams of manganese sulphate

6.0 grams of copper sulphate

0.3 grams of cobalt sulphate

Results

The non-colostrum group of calves did not do well as a result of the scouring and the pneumonia. There was little difference between the calves that received one large dose of vitamin A (controls) and those that received none at all. The average life span for the controls was 56 days and for the vitamin A deficient calves, 25 days. The following symptoms were observed among the 6 calves; anorexia, coughing, fever, rales, stretching of the neck, increased respirations and heart beats, inability to rise, weakness, diarrhea, and rough hair coat. The control calf C589 after the 54th day of life showed lacrimation, dilatation of the pupils, and fading of the tapeta lucida. One of the vitamin A deficient animals had some bulging of the eyes. All but one animal had pneumonia and scours (see Table II). There were no significant differences between the controls and the vitamin A deficient calves with reference to quantity of blood plasma carotene, numbers of erythrocytes and leukocytes, hematocrits, and differential leukocytic counts. Blood plasma vitamin A in the controls was higher than in the vitamin A deficient animals at the beginning. In control animal, C589, the vitamin A content of the blood plasma dropped to the deficiency range in 27 days (below 8.0

ug/per 100 ml.) in spite of the fact that this calf had received 250,000 I. U. of vitamin A in the form of shark liver oil the first day of life. The hemoglobins were lower than normal. In some of the calves with pneumonia the leukocyte count increased from normal (7 to 10 thousand) up to as high as 56 thousand. Simultaneously there was a shift of leukocytes from a high number of lymphocytes to a high number of neutrophiles. In addition there was an increase of non-segmented polymorphonuclears.

Necropsy revealed a bronchopneumonia in five of the animals which involved the apical, cardiac, intermediate, and part of the diaphragmatic lobes of both lungs. The pneumonia was of the suppurative type in three of the five cases. The intestines were greatly congested and the lumina contained very watery feces in the cases which scoured. Some of the lymph nodes were edematous and enlarged. The kidneys of three animals showed focal areas of inflammation. Calf C593 had a very marked upper respiratory infection manifested by purulent sinusitis. Control calf, C591, had an enlarged heart due to hypertrophy and dilatation of the left ventricle. (For complete records of these calves see non-colostrum group in the appendix).

Discussion

This group of calves was not of much value from the view-point of learning a great deal about the effects of vitamin A deficiency. The fact that the animals did not eat well and died early in life left much to be desired as far as the experiment was concerned. It did bring out the importance of colostrum from the standpoint of health

in calves as pointed out by Smith and Little (1924). It verified somewhat the work of Moore (1939) who found that when calves were placed on a diet low in carotene or vitamin A before the age of 40 days they usually died from an infection such as pneumonia. The red and white blood cell counts compared favorably with those of normal cattle blood as cited by Runnells (1946). The hemoglobins in most of these animals were somewhat below the normal level of 10 to 12 gm.per cent. The lower levels of hemoglobin are not surprising when one considers how poorly some of the calves were eating.

Summary

1. Six calves which did not receive colostrum were placed on a basal ration deficient in vitamin A. Two controls received a vitamin A supplement of 250,000 and 100,000 I. U. of vitamin A in the form of shark liver oil respectively.
2. The average length of life for the controls was 56 days and for the vitamin A deficient calves 25 days.
3. The animals did very poorly and most of them developed pneumonia and scours.

TABLE II

Non-Colostrum Group

Animal	Age at death	Am't of Vit. A	Pneumonia	Scours	Other Conditions
No.	days	I.U.			
		Control Calves			
C589	86	250,000	✓	✓	
C591	26	100,000	-	✓	Hypertrophy of Heart
		Basal Ration Calves			
C588	32	-	✓	✓	
C593	10	-	✓	-	Purulent sinusitis
A57	38	-	✓	✓	
A59	20	-	✓	✓	

EXPERIMENT II

Calves Received Colostrum and a Vitamin A Deficient Ration Without Hay

Purpose

To study the effects of feeding a vitamin A deficient ration, without hay, to calves that had received colostrum.

Experimental Procedure

Ten calves of the Holstein, Jersey, and Guernsey breeds were placed in this experiment at the time of birth. Calves C587 and C582 were used as negative controls and, in addition to the basal ration, received vitamin A weekly in the form of shark liver oil. The following calves, A58, C582, C595, C583, and C584 were placed on the basal ration only. The basal ration had the same formula as the one used in Experiment I. Three calves, A54, A55, and A56 were used as positive controls. These were new-born heifers that were going to be used as replacements in the regular experimental herd. They received the regular herd calf rations consisting of hay and grain. All of the animals were given whole milk until they were able to eat the dry feed. The general procedures were carried out as described earlier in this thesis.

Results

The three positive controls were apparently normal throughout the entire experiment. The two negative controls and the five basal ration calves displayed the following clinical conditions: anorexia, scouring, pneumonia, poor growth, and pityriasis. The pneumonia was characterized by coughing, moist rales, discharge of mucopurulent exudate from the nostrils, fever, increased respirations, inability to get up, and stretching of the neck. The pityriasis was recognized by

dryness of skin, loss of hair, and scaliness on the ears, neck, and withers. One of the controls, C587, developed a severe case of tongue lolling.

The basal ration calves showed some changes that were not observed in the controls. From the 84th to the 141st day of life these animals developed the following eye changes: failure of the irises to contract properly, fading of the tapeta lucida, edema and swelling of the optic discs, bulging of the eyeballs from the sockets, and lacrimation. Calf C584 developed a rolling of the eyes from side to side (nystagmus). C582 was the only animal in the group that had a clouding of the corneas which developed after the 253rd day of life. Two calves, C584 and C582, displayed typical vitamin A syncope, (a temporary suspension of consciousness for 30 seconds to a minute), at the 110th and 215th days of life respectively. Tympanites was observed only in the group of vitamin A deficient animals.

The vitamin A content of the blood plasma of the controls was well above the 10 ug. per 100 ml. of blood plasma needed for maintenance of normal health. All of the deficient animals showed blood plasma vitamin A levels, which were usually markedly below 6 ug. per 100 ml. There was little difference between the positive controls, negative controls, and basal ration calves as far as erythrocyte and leukocyte counts, hemoglobin content, hematocrit, and differential counts were concerned.

A necropsy was performed on the two negative controls and the five basal ration calves. All of them had lesions of pneumonia (Chart II). There were fibrinous adhesions between the pleurae. Some of the lymph nodes of the body were enlarged due to edema, congestion, and hemorrhage. The stomach and intestines were often congested and presented some

hemorrhages. The skin showed a pityriasis. The subcutaneous tissues of several animals were edematous and in one case edema appeared to involve the muscles. The kidneys often showed small white spots in the cortex which on section extended down through the depth of the cortex. Complete records of this group of animals are in the appendix.

Discussion

This group of calves was not of much value from the standpoint of getting a true picture of a single deficiency condition. The fact that the animals did not eat well, scoured a great deal, and were plagued with pneumonia, probably led to multiple deficiencies. This group did show many of the symptoms and conditions that have been attributed to vitamin A deficiency. An examination of the blood picture of the basal ration calves showed that blood plasma vitamin A was extremely low.

The animals developed extensive scouring similar to the calves described by Hart and Guilbert (1933). Keener et al (1942) and Thorp et al (1942). Edema was present in the subcutaneous tissues similar to that reported by Guilbert and Hart (1935), Schmidt (1941), and Creech and Siebold (1943). These calves showed some of the eye changes similar to those reported by Moore (1939), Schmidt (1941), and Jones et al (1943). Two of the animals had syncopes resembling the cases reported by Moore (1939).

Observations of the positive group of control animals brought out two important factors. First, that hay in the diet appeared to play a very important role from the standpoint of health in ruminants. Secondly, that hemoglobin values in calves appear slightly lower than those of mature animals.

Summary

1. Ten animals were used in this experiment, three positive controls (heifer calves receiving regular herd rations), two controls (receiving basal ration plus vitamin A), and five animals receiving only the basal ration low in Vitamin A.

2. All of the animals except the positive controls developed the following clinical conditions: anorexia, scours, pneumonia, and pityriasis.

3. The calves fed the basal ration low in Vitamin A in addition to having the above clinical conditions, showed eye changes characterized by failure of the irises to contract properly, fading of the tapeta lucida, edema and swelling of the optic discs, bulging of the balls, and lacrimation.

4. Necropsy of the controls and the calves which received the basal ration low in Vitamin A revealed pneumonia in all of them.

TABLE III

Colostrum Group I

Animal	Age at death	Am't of Vitamin A	Pneumonia	Scours
No.	days	I.U.		
		Positive Control Calves		
A54	living	*	-	-
A55	"	*	-	-
A56	"	*	-	-
		Control Calves		
C586	30	50,000**	/	/
C587	170	50,000**	/	/
		Basal Ration Calves (low in Vitamin A)		
A58	42	-	/	/
C582	372	-	/	/
C585	100	-	/	/
C583	140	-	/	/
C584	180	-	/	/

* Received vitamin A in form of Carotene from alfalfa hay.

** Given weekly in the form of shark liver oil.

EXPERIMENT III

The Study of Vitamin A Deficiency in Calves that Received Colostrum at Birth. These Animals Received a Basal Ration Containing a Hay Low in Carotene.

Purpose

To study the effects of a vitamin A deficient ration which contained oat hay low in carotene upon calves that received colostrum.

Procedure

Six calves of the Ayrshire, Brown Swiss, and Holstein breeds were used in this experiment. Calves C616 and C617 were employed as controls. These latter animals, in addition to the basal ration, were given 250,000 I. U. of vitamin A weekly in the form of shark liver oil. Calves C611, C613, C614, and C615 were given only the basal ration.

The basal ration consisted of 1 pound of oat hay per day, 1 pound of skim milk per 10 pounds of body weight until 12 pounds was fed, and 10,000 I. U. of vitamin D in the form of viosterol weekly. The oats and barley were fed according to the amounts of total digestible nutrients needed by the calves. 10 grams of calcium carbonate were given daily with a salt mixture containing 384.8 gm. sodium chloride, 65.5 gm. ferrous sulphate, and 6.0 gm. of copper sulphate. (The salt mixture made up 1 per cent of the grain mixture.)

The amount of the grain mixture fed was based on the weight of the animal and the total digestible nutrients needed for growth. The amount of digestible protein and total digestible nutrients in each feed substance was determined by using Morrison's Standards (1948). The digestible protein in each ingredient was as follows: skim milk 3.4, oats 9.4, barley 10.0, and oat hay 4.9 per cent. The total digestible nutrients in these feeds were as follows: skim milk 8.7, oats 70.1, barley 77.7, and oat hay 4.3 per cent. The amount of nutrients to be

fed was carried out according to the recommendations of the National Research Council (1945). The calves received skim milk during the entire experiment, except at birth, when they were allowed to nurse the cows in order to obtain colostrum. The oat hay contained 4 ug. of carotene per gm. throughout the experiment, except for one month when it contained only 1.2 ug. per gm. All of the animals were given 10 mg. of cobalt sulphate daily starting at about 7 months of age. In the last 3 months of this experiment oats were used almost exclusively for grain as barley was not available. The general procedures were carried out as described previously in this thesis (see page 27).

Results

The calves in Experiment III did not consume enough total digestible nutrients during the first 6 months of life to make satisfactory gains even though they appeared very healthy. (Table IV). Control calf C617 consumed about 1 pound less than the normal recommendation for digestible nutrients during the first 6 months. During this period the animal gained only 0.78 pounds per day, but after 10 mg. of cobalt sulphate was supplemented to the diet daily, the gain in weight increased to 1.55 pounds per day. The amount of total digestible nutrients needed per pound of gain in weight varied from 1.97 to 5.63 pounds. C616 followed practically the same pattern except that during the first 6 months the total digestible nutrients intakes were only one-half pound below normal, yet the gain was only 0.55 pounds per day. After cobalt was added the gain was 1.5 pounds per day. The amount of total digestible nutrients needed for these gains varied from 2.5 to 5.33 pounds. Calf C614 of the vitamin A deficient group gained 0.62 pounds per day during the first 6 months and 1.2 pounds per day after cobalt supplementation. The amount of total digestible nutrients

Calf No.
C617
C616
C614
C613
C611
C615

TABLE IV

Feeds, digestible protein, total digestible nutrients consumed

Calf No.	3 Month Periods	FEED INTAKE				Digestible Protein	
		Oat Hay	Oats	Barley	Skim Milk	Rec'd/day	Req./day
		pounds	pounds	pounds	pounds	pounds	pounds
						Control Calves	
C617	3	45.	36.7	34.2	940	.46	0.56
	6	91	118	141	1050	.72	0.70
	9	91	468	126	1092	1.08	0.74
	12	90	979	-	1080	1.46	0.81
C616	3	37	36.6	34.12	926	0.42	0.45
	6	91	78.5	101.5	1050	0.63	0.62
	9	91	423.0	302.5	1092	1.21	0.70
	12	89	717.0	-	1068	1.20	0.75
						Vitamin A Deficient Calves	
C614	3	29	31	22	810	.39	0.51
	6	91	78	107	1050	.64	0.65
	9	91	375	169	1080	1.02	0.73
	12	91	728	8	1102	1.22	0.78
C613	3	32	19	19	726	0.33	0.48
	6	82	69	69	1072	0.62	0.65
	9	92	246	172	1068	0.80	0.73
	12	90	489	-	1080	0.97	0.79
C611	3	34	18.25	18.25	975	0.44	0.66
	6	90	84	93	1116	0.66	0.72
	8	65	149	149	780	0.90	0.75
C615	3	26	32	24	818	0.38	0.49
	6	91	63	92	1050	0.65	0.59
	9	91	381	177	932	0.99	0.68
	12	93	511	-	1104	0.97	0.74

digestible nutrients consumed, and efficiency of feed utilization.

FEED INTAKE

lk	Digestible Protein		Total Digestible Nutrients			
	Rec'd/day	Req./day	Rec'd/day	Req./day	Req.per pound of gain	Daily gains in wt.
	pounds	pounds	pounds	pounds		pounds
	Control Calves					
	.46	0.56	1.5	2.30	1.97	0.76
	.72	0.70	3.16	4.14	3.90	0.81
	1.08	0.74	5.77	5.3	3.61	1.60
	1.46	0.81	8.62	7.3	5.63	1.53
	0.42	0.45	1.50	1.9	2.50	0.6
	0.63	0.62	2.56	3.1	5.33	0.48
	1.21	0.70	7.00	4.9	4.07	1.72
	1.20	0.75	6.70	6.4	4.75	1.41
	tamin A Deficient Calves					
	.39	0.51	1.22	2.5	1.97	0.62
	.64	0.65	2.58	3.4	4.09	0.63
	1.02	0.73	5.38	4.8	3.71	1.45
	1.22	0.78	6.77	6.3	6.64	1.02
	0.33	0.48	1.2	2.13	2.11	0.57
	0.62	0.65	2.34	3.50	2.69	0.87
	0.80	0.73	4.43	4.9	4.03	1.10
	0.97	0.79	4.89	6.2	4.33	1.13
	0.44	0.66	1.79	3.9	2.06	.87
	0.66	0.72	4.03	4.5	4.03	1.0
	0.90	0.75	4.50	5.3	4.29	1.05
	0.38	0.49	1.24	2.3	2.38	0.52
	0.65	0.59	2.33	3.0	3.88	0.60
	0.99	0.68	5.38	4.0	5.03	1.07
	0.97	0.74	4.93	4.9	-	-

needed per pound of gain varied from 1.97 to 6.64 pounds. C613 of the vitemin A deficient group was interesting in that its total digestible nutrients intake never quite reached the normal requirement yet it gained 0.72 pounds per day during the first 6 months and after cobalt was added to the diet it gained 1.11 pounds per day. The total digestible nutrients needed per pound of gain varied from 2.11 to 4.33 pounds. Calf C611 of the vitamin A deficient group only lived 8 months as a result of breaking its back during a fainting spell (syncope). This calf made the best gains in early life when compared with the other calves. During the first 6 months it gained 0.93 pounds per day. During the next 2 months it gained 1.05 pounds per day. The total digestible nutrients needed for the gain per pound of weight varied from 2.06 to 4.29 pounds. C615 of the vitamin A deficient group followed the same pattern as the other animals until the 9th month when pneumonia developed and it gradually lost weight. It showed, however, a gain in weight as a result of adding cobalt. The first 6 months it gained 0.55 pounds and from the 6th to the 9th months the gain was 1.07 pounds per day. The total digestible nutrients needed per pound of gain varied from 2.38 to 5.03.

The body weights and heights at withers were compared with normal values (see Table V). Normal growth values are not available for Brown Swiss cattle, which accounts for the lack of data in case of calf C615. The animals in both groups made fair gains in body weight, yet were below the Ragsdale standards. The heights at withers for the animals in both groups were slightly below the standards.

The blood picture showed very little difference between the

TABLE V

Growth and Carotene I

Animal	Age	Weight	Standard Weight	Per Cent of Normal Weight	Height
No.	Month	Pounds	Pounds		Inches
				Control Calves	
Holstein C617 Male	3	168	214	79	33.4
	6	252	399	64	37.2
	9	394	563	70	41.2
	12	532	741	71	45.9
Aryshire C616 Male	3	134	173	78	31.2
	6	177	321	56	34.7
	9	344	488	70	38.2
	12	467	675	70	42.0
				Vitamin A Deficient	
Holstein C614 Female	3	146	193	76	32.7
	6	203	355	57	34.4
	9	334	509	66	38.6
	12	427	632	66	42.3
Aryshire C613 Male	3	141	173	82	30.7
	6	219	321	68	34.1
	9	319	488	66	36.8
	12	422	675	63	41.9
Holstein C611 Male	3	182	214	85	33.3
	6	274	399	69	37.6
	8	342	514	67	40.1
Brown Swiss C615 Male	3	132			77 [illegible]
	6	188			84.8
	9	287			93.0
	12	280			94.8

* Calves compared with Ragsdale Standards for weight and height at wi th
** The minimum normal requirement is 16 ug of carotene per pound of body

TABLE V

ı and Carotene Intake*

; of ɔight	Height	Standard Height	Per Cent of Normal Height	Carotene**	Vitamin A
	Inches	Inches		ug/per pound	I.U.per pound
Control Calves					
	33.4	34.8	96	10.7	1488.0
	37.2	40.5	92	7.2	992.0
	41.2	44.2	93	5.0	638.0
	45.9	47.5	97	3.4	469.0
	31.2	32.7	95.5	13.5	1894.0
	34.7	37.9	92	10.3	1413.0
	38.2	41.8	91	5.5	727.0
	42.0	45.5	92	3.9	535.0
ɔin A Deficient Calves					
	32.7	34.3	95	12.3	-
	34.4	39.7	87	8.8	-
	38.6	43.5	89	5.4	-
	42.3	46.0	92	4.2	-
	30.7	32.7	94	12.3	-
	34.1	37.9	90	8.7	-
	36.8	41.8	88	5.6	-
	41.9	45.5	92	4.3	-
	33.3	34.8	96	10.0	-
	37.6	40.5	93	6.6	-
	40.1	43.1	93	5.3	-
	77 cm.			13.6	-
	84.5			9.6	-
	93.0			6.6	-
	94.5			6.6	-

height at withers.
r pound of body weight.

controls and the deficient calves (see Table VI). The vitamin A levels of the 2 controls, C617 and C616, varied between 8.6 and 13.69 ug. per 100 ml. of blood plasma. The deficient calves showed levels of vitamin A varying between 3.88 and 10.3 ug. per 100 ml. of blood plasma. The hemoglobin levels in the calves of Experiment III varied from 8.74 to 13.4 grams per 100 ml. of blood while the hemoglobins in the calves of Experiments I and II varied from 4.4 to 11.6 grams per 100 ml. of blood. The high leukocyte count in calves C617 and C614, which occurred in the last 2 months of life, could be accounted for on the basis of an infection, but in C613 no pathological condition was observed that might explain the high count. The lack of vitamin A apparently had no definite effect on the blood picture other than the low levels of vitamin A in the plasma. A summary of the blood data is given in Table VI.

The most important manifestations of vitamin A deficiency exhibited by the calves in the low vitamin A group involved the eyes. A summary of these changes with the time that each change first occurred are presented in Table VII.

TABLE VI

Summary of Blo

Animal	Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.
No.	Months	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%
							Controls
C617	3	6.32	8.65	8.03	9.2	8.74	27.59
	6	8.9	12.76	8.32	12.35	9.95	32.14
	9	8.4	9.70	9.54	14.79	11.67	35.44
	12	9.5	9.84	9.53	21.28	12.02	36.55
C616	3	12.03	13.69	6.67	8.49	9.67	31.2
	6	7.19	13.49	6.30	8.76	8.89	28.37
	9	8.37	11.96	6.06	11.70	9.66	28.5
	12	8.38	10.67	6.57	13.48	10.77	31.7
							Experimental A
C614	3	7.0	10.23	9.43	9.48	9.37	31.42
	6	8.99	6.7	9.43	11.65	9.77	32.86
	9	6.5	5.17	8.81	16.92	9.79	30.63
	12	6.36	4.12	9.23	24.84	10.57	33.9
C613	3	12.62	9.77	8.28	11.00	10.37	34.1
	6	8.57	8.78	7.00	10.00	9.8	31.75
	9	7.49	6.99	7.88	15.46	9.63	29.88
	12	9.77	5.49	7.56	24.03	11.08	33.59
C615	3	5.48	7.60	8.25	7.55	9.7	30.66
	6	5.63	6.1	8.16	10.37	9.1	29.26
	9	6.2	4.98	8.06	12.38	9.77	27.44
	12	5.0	3.88	8.02	12.49	9.77	28.5
C611	3	11.4	9.04	12.34	12.54	13.4	42.78
	6	6.79	7.44	9.59	11.67	9.7	30.81
	9	6.0	6.5	8.90	15.35	10.16	29.69

* 3 month summaries of weekly determinations.

TABLE VI

Summary of Blood Data*

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
	Controls								
8.74	27.59	10.89	33.78	65.3	32.1	0.6	0.0	0.0	2.2
9.95	32.14	12.17	40.75	58.57	38.43	0.1	0.1	0.0	1.3
11.67	35.44	12.54	37.96	60.00	37.43	0.2	0.0	0.2	2.0
12.02	36.55	12.3	38.18	52.63	44.0	0.2	0.1	0.2	2.0
9.67	31.2	14.06	45.05	78.8	17.88	0.8	0.1	0.0	1.6
8.89	28.37	14.05	45.3	78.0	19.25	0.4	0.5	0.2	1.4
9.66	28.5	16.6	49.4	74.7	22.86	1.5	0.0	0.2	1.0
10.77	31.7	16.49	49.8	75.0	21.80	0.5	0.0	0.5	1.5
	Experimental Animals								
9.37	31.42	10.25	35.48	67.6	28.4	0.7	0.9	0.1	1.3
9.77	32.86	10.33	35.16	80.63	17.0	0.2	0.0	0.0	1.5
9.79	30.63	11.31	35.45	66.75	30.5	0.2	0.2	0.0	2.0
10.57	33.9	11.54	37.05	66.18	28.88	0.5	0.3	0.7	2.4
10.37	34.1	12.75	44.05	82.11	15.55	0.3	0.1	0.0	0.7
9.8	31.75	13.95	45.8	80.14	16.43	0.1	0.1	0.4	2.0
9.63	29.88	12.13	38.7	74.5	22.75	1.7	0.3	0.3	1.7
11.08	33.59	14.75	44.79	65.64	28.18	1.0	0.1	0.9	2.5
9.7	30.66	11.3	36.68	77.20	20.4	0.3	0.1	0.0	1.1
9.1	29.26	11.08	36.03	67.25	29.2	0.2	1.3	0.3	1.2
9.77	27.44	12.57	38.87	70.0	26.6	0.7	0.1	0.0	1.7
9.77	28.5	12.24	36.21	70.55	24.7	0.1	0.6	0.4	2.3
13.4	42.78	10.76	35.31	72.50	24.0	1.2	0.0	0.1	1.7
9.7	30.81	10.29	33.29	69.67	26.8	0.7	0.4	0.5	1.8
10.16	29.69	11.59	34.73	71.25	25.25	0.5	0.2	0.0	1.7

TABLE VII

Summary of Eye Changes

Calf No.	C611	C613	C614	C615
	Day	Day	Day	Day
Iris not contracting properly.	245	136	71	58
Night blindness.	247	65	-	-
Fading of the tapetum lucidum.	-	200	193	87
Blurring of the optic disc.	245	287	89	69
Total blindness.	-	288	246	260
Bulging of the balls.	-	289	295	311
Nystagmus.	-	-	293	262
Level of Vitamin A at time of first symptom. ug/per 100 ml.	4.8	7.2	4.8	1.9
Level of Vitamin A at death. ug/per 100 ml.	6.5	5.49	4.12	3.88

In addition to the above changes, after the animals were totally blind the diameter of the blood vessels around the optic disc became smaller and appeared more tortuous. Calf C615 also developed hemorrhages around the blood vessels.

The next important system that showed clinical manifestation of the deficiency was the nervous system. Calves C611, C613, C614, and C615 which received the vitamin A deficient ration, developed syncope or incoordination for the first time at 236th, 260th, 191st, and 254th day respectively. This syncope was characterized by fainting spells which lasted from 30 to 60 seconds.

The male calves, C613 and C615, in the deficient group apparently

had no libido since they were not interested when placed with a cow in heat. This latter condition was not true among the controls.

The only lesions found at necropsy in the controls were numerous areas of necrosis in the liver, and congestion, edema, and hemorrhage of the lymph nodes in C617. There was no pinching of the optic nerve where it passed through the optic foramen in the controls (fig. 1). The most important lesion in the deficient group was the pinching of the optic nerve where it passed through the optic foramen (fig. 2) and edema of the optic discs. Animals C614 and C615 had some chronic pneumonia. The pneumonia in C614 was confined to one small area in the right apical lobe, while C615 had it in several lobes. In addition, the latter 2 animals had small greyish-white spots in the cortex of their kidneys which on section extended down through the depth of the cortex. C613 was the only animal with a cyst in the pituitary gland. This cyst measured about 6 mm. in diameter.

The histopathological changes were confined to a few structures of the body. The part of the optic nerve that passed through the optic foramen had undergone marked atrophy and necrosis, and had been replaced by fibrous connective tissue (figs. 3 and 4). The testes of the control animals showed active spermatogenesis (fig. 5). The testes of the deficient group were characterized by complete absence of spermatogenesis (fig. 6). There were a few germinal cells left in the seminiferous tubules next to the basement membrane. The interstitial cells appeared to be intact.

Some metaplasia was observed in the epithelial structures of a few organs in the deficient animals. This condition was observed in the cervix (fig. 7), the ducts of the mandibular salivary glands (fig. 8),

and in the bronchi of the lungs where inflammatory processes were present (fig. 9). Two of the calves had microscopic changes in their pituitary glands. The changes were produced by pressure atrophy due to cystic fluid. Atrophy and necrosis of cells adjacent to the cyst both in the anterior and posterior lobes were observed. There were numerous desquamated cells in the cyst cavities. The pars nervosa of C614 had many more hyaline bodies than are normally seen. The greyish-white areas observed microscopically in the kidneys of C614 and C615 were characterized by atrophy and necrosis of the tubules. Surrounding these areas were increased amounts of connective tissue accompanied by increased numbers of lymphocytes and macrophages.

Discussion

The ration of skim milk, oat hay, barley, oats, viosterol, calcium carbonate, and salt mixture probably would have been adequate if cobalt had been in the diet in sufficient amounts at the beginning of the experiment. All of the animals in the experiment except C615 appeared to be in good health and outwardly were eating well, but were not consuming enough feed to keep up the total required digestible nutrient intakes. Table IV shows very adequately the story of these calves. In the first 6 months the total digestible nutrients and the digestible protein consumed were below the standards and the gains made by the controls and the vitamin A deficient calves were between 0.6 and 1.0 pounds per day. After the cobalt was added the gains ranged from 1.07 to 1.72 pounds per day. Cobalt is very important for the health of cattle. This has been brought out by numerous workers such as A. C. Baltzer et al (1941) and H. A. Keener et al (1944). Bowstead and Sackville (1939) (1942) showed

that the addition of cobalt to the diets of ewes increased appetite and body weight. Marginal deficiency of cobalt is very difficult to detect in cattle. It was not until these calves actually quit eating between the 6th and 7th months that cobalt deficiency was realized. There was a marked increase in appetite which occurred about the 3rd day after cobalt supplementation.

The calves of Experiment III were very efficient in their ability to produce gains when the total digestible nutrients were below normal. Both C617 and C614 required only 1.97 pounds of total digestible nutrients per pound of gain for the first 3 months. C617, however required 5.63 pounds of digestible nutrients for 1 pound of gain in the 9 to 12 month period. This increased requirement might be explained on the basis of the liver damage that developed in the last month. C615 required 5.03 pounds of total digestible nutrients for each pound of gain during the 6 to 9 month period. This inefficiency may have been due to the chronic pneumonia which occurred in the late stage of life. In this period (9th to 12th month) the calf lost weight.

It was of interest to compare these animals with the Ragsdale Standards. (Table V). C616, a control calf, was the first animal to develop anorexia and about 18 days elapsed before cobalt was given. It was interesting to observe the response to cobalt. At 6 months the weight of this calf was only 56 per cent of normal and by 1 year of age the body weight had increased to 70 per cent of normal. Control C617 was only 64 per cent of normal and after cobalt was added, body weight gradually increased to 71 per cent of normal. Calf C614 of the vitamin A deficient group showed a weight of 56 per cent of normal at 6 months which gradually increased to 66 per cent at 9 months and stay-

ed at that level. The body weight of C613 only dropped to 68 per cent of normal at 6 months of age. Cobalt supplementation did not appear to accelerate growth since at 1 year of age the weight had been reduced to 63 per cent of normal. If Table IV is examined one will see that C613 was making its best gain during the 9th to the 12th month period, (1.13 pounds per day), yet lost 3 percentage points in weight when compared to the Ragsdale Standards. C611, another calf in the vitamin A deficient group, was 69 per cent of normal in weight. This animal broke its back in the 8th month of life and had to be destroyed before the full effects of cobalt could be observed. C615 in the deficient group did not make good gains in weight which was probably due in part to cobalt deficiency and chronic pneumonia.

The long bones of the body of vitamin A deficient calves apparently continued to grow since the height at the withers of these animals was 92 per cent of the Ragsdale Standards. This compares with the work of Richards (1935), who reported that growth does not cease in vitamin A deficiency when the animal ceases to increase in weight.

There was no significant difference between the controls and vitamin A deficient animals in growth at height at withers.

The vitamin A deficient calves showed the eye changes that have been described by Moore et al (1934), Moore (1939), and Jones et al (1943). (Table VII). The earliest manifestation of vitamin A deficiency appeared to be the failure of the irises to contract properly. This condition varied with each eye of the animal. Sometimes one iris closed properly while the other did not. Night blindness, blurring of the optic discs, and fading of the tapeta lucida were the next conditions observed. The lacrimation, and the opacity and ulceration of

the corneas which have been reported by Hart and Guilbert (1933), Jones et al (1943), and Alvarez (1947) were not observed in these calves. This work confirmed the report of De Schweinitz and De Long (1934) that the caliber of the blood vessels was reduced. The optic nerves showed the pinching where they passed through the optic foramen as reported by Moore et al (1934), McNutt and Wall (1938), Moore (1939), and Wetzel and Moore (1940). This pinching of the optic nerve produced atrophy and necrosis of the nerve fibers and lead to replacement fibrosis. The destorying of the nerve at this point accounts for the total blindness.

Night blindness was only observed in two of the calves, C611 and C613, which occurred at the 247th and 65th day, respectively. In 2 of the deficient animals, C614 and C615, this condition could not be demonstrated. Night blindness was determined by attempting to run calves into objects in semi-darkness; apparently C611 must have obtained more vitamin A in colostrum than C613, as it took 192 days longer to produce night blindness in C611 than C613. Of course, there was a very good chance that night blindness was overlooked at an earlier time in C611 although this animal showed the eye changes much later than the other vitamin A deficient calves.

The deficient calves showed the characteristic syncope or incoordination that was reported by Moore and Sykes (1940), Boyer et al (1942), and Schmidt (1941), and Hodgson et al (1946). The reproductive ability of the male calves C611, C613, and C615 was greatly impaired and the testes microscopically showed that spermatogenesis had not taken place. This latter condition corresponded to the work reported by such investigators as Guilbert and Hart (1935), and Bratton et al (1948).

Chevral and Cormier (1948), and Sutton and Brief (1939) reported similar changes in the testes of rabbit and rats respectively. Two animals, C613 and C615 showed some atrophy and necrosis of the pituitary gland adjacent to the cyst. This was similar to the changes reported by Madsen, et al (1942) except no increase in connective tissue was observed.

The greyish-white lesions that have been reported in the kidneys of vitamin A deficient animals is probably due to the action of toxins from a chronic inflammatory process rather than the lack of vitamin A. Calves C614 and C615 had these lesions in the kidneys and also had small chronic areas of pneumonia in their lungs. The author has observed this latter condition on numerous occasions when calves with chronic pneumonia were necropsied.

This investigation showed that if calves are eating and growing well, even though the vitamin A and carotene are extremely low, many of the changes which have been attributed to the lack of vitamin A are eliminated. The oat hay in the diet probably kept the rumen functioning and increased the general appetite of the calves. The hay apparently produced a hay type of flora in the rumen of young calves rather than a grain type of flora when grain is fed according to Pounder and Hibbs (1949). It is this microflora that increases the digestibility of food substances and also synthesizes dietary factors such as the vitamins of the so called vitamin B-complex and vitamin K. Burroughs and associates (1949) showed that the digestibility of the dry matter of corn cobs was depressed by addition of starch, but when the same amount of starch was added to alfalfa hay the depression in the digestibility of the dry matter did not occur.

They explained these results on the basis that alfalfa hay contained more essential nutrients than corn cobs in promoting growth of rumen microrganisms. Quin (1943) and Elsden (1945) both pointed out that good quality hay increased the fermentation of glucose in the rumen of sheep. They pointed out that the difference was due to the microflora of the rumen. Louw et al (1948), using a concentrate composed of starch, casein, brewer's yeast, and a mineral mixture as a supplement to poor hay, observed that the depression of cellulose digestion by this supplement was overcome by adding fresh alfalfa. When the basal ration of Experiment III was compared with the basal ration of Experiment II, about the only difference was the presence of oat hay in the former. When this hay was present some of the clinical changes which were observed in Experiment II and many of those reported in the literature were eliminated. The rough hair and dermatitis, that were reported by Hart and Guilbert (1933), Schmidt (1941), and Keener et al (1942), had been eliminated. Scours was not a problem in this experiment as observed by such workers as Hart and Guilbert (1933), Stewart and McCallum (1938), and Keener et al (1942). Subcutaneous edema was not present in the vitamin A deficient animals although this condition has been reported by many research workers such as Bechdel et al (1928), Guilbert and Hart (1935), Cunningham and Addington (1938), Schmidt (1941), Hastings (1942), Creech and Siebold (1943) and Madsen and Earle (1947). No urinary calculi were found in the vitamin A deficient animals used in this investigation. Urinary calculi have been reported by Newsom (1938) and Schmidt (1941) in cattle. Osborn et al (1917), and Bassett et al (1946) reported a high incidence of calculi in rats and fox pups respectively. Other investigators such as Moore (1939) and Jones et al (1943) did not find calculi in their vitamin A deficient calves.

Summary

1. Six calves of the Holstein, Ayrshire, and Brown Swiss breeds were placed on a basal ration deficient in vitamin A. Two of these animals (controls) received 250,000 I. U. of vitamin A weekly.
2. The calves during the first 6 months of life did not consume enough nutrients to bring the total digestible nutrients up to the recommendations of the National Research Council. During the last 6 months, the total digestible nutrients were approximately normal or above.
3. The addition of cobalt increased the appetites of these animals and their weight gain per day increased two to three fold. The increase in appetite occurred about the 3rd day.
4. The most important manifestations of vitamin A deficiency involved the eyes. These changes were characterized by dilatation of the pupils, blurring of the optic discs, fading of the tapeta lucida, night blindness, bulging of the balls, nystagmus, total blindness, and decreased diameter of the blood vessels.
5. The vitamin A deficient animals showed periods of unconsciousness (syncope) which lasted from 30 to 60 seconds.
6. Spermatogenesis was not present in the vitamin A deficient male animals.
7. Metaplasia of the epithelium was observed only in a few organs such as the cervix, ducts of mandibular salivary glands, and the bronchi of vitamin A deficient calves.
8. The following manifestations of vitamin A deficiency which have been reported in the literature were absent in these calves: rough hair coats and skin lesions, lacrimation, scours, anorexia, edema, weak-

ness, urinary calculi, and opacity and ulceration of the cornea. The elimination of these manifestations of vitamin A deficiency was probably due to the presence of oat hay in the diet.

9. The role of hay in maintaining a normal rumen fermentation was pointed out.

Fig. 1. Normal eyes and optic nerves of control calf C616. Note that there is no pinching of the optic nerve. Life size.

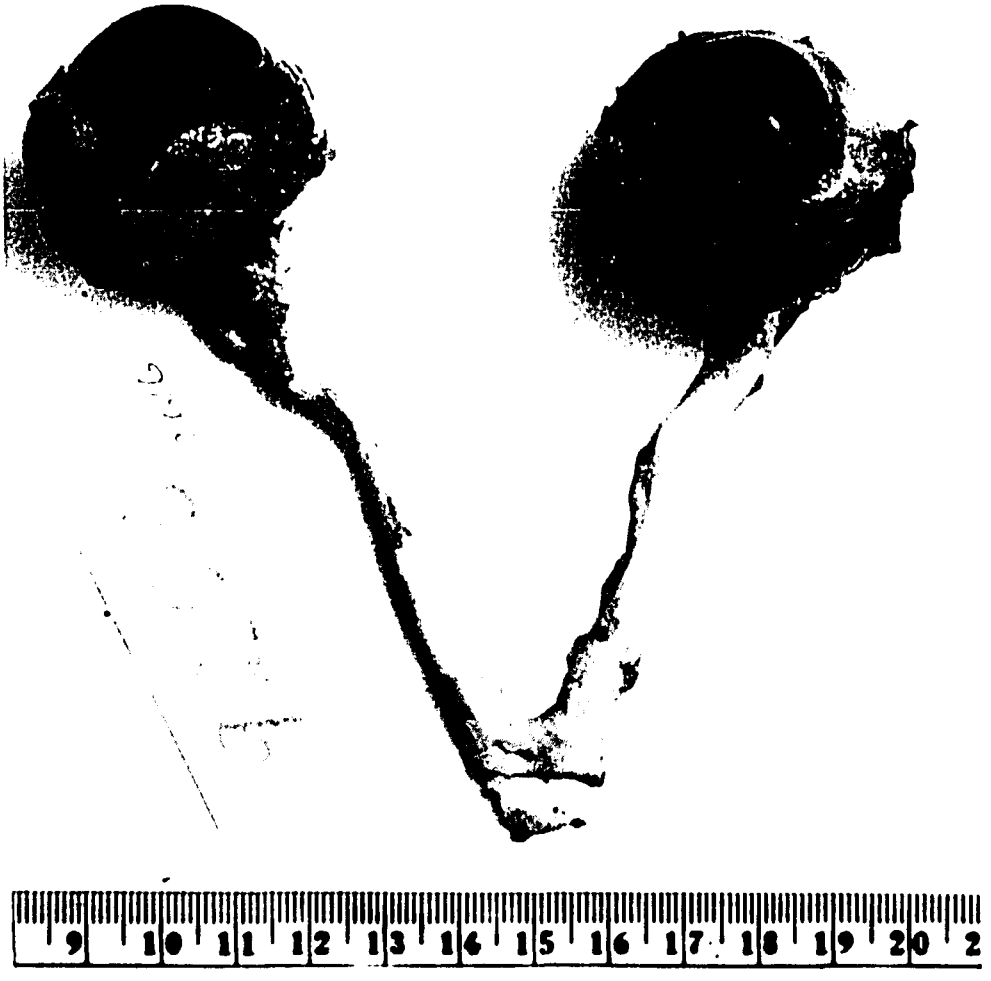

Fig. 1

Fig. 2. Eyes and optic nerves of calf 3614 that was on vitamin A deficient diet. Note the severe pinching of the optic nerve anterior to optic chiasma. Life size.

Fig. 2

Fig. 3. Longitudinal section of one of the optic nerves shown in figure 2. Note the replacement of the nerve by collagenous fibers at the point where the nerve passed through the optic foramen. Azocarmine and aniline blue stain. X6

Fig. 3

Fig. 4. Section of the same optic nerve through the point of constriction to show the increase in collagenous fibers and the disappearance of nerve structure. Hematoxylin and Eosin stain. X176.

FIG. 4

Fig. 5. Cross section of a seminiferous tubule of control calf C617 to show normal active spermatogenesis. Hematoxylin and Eosin. X700.

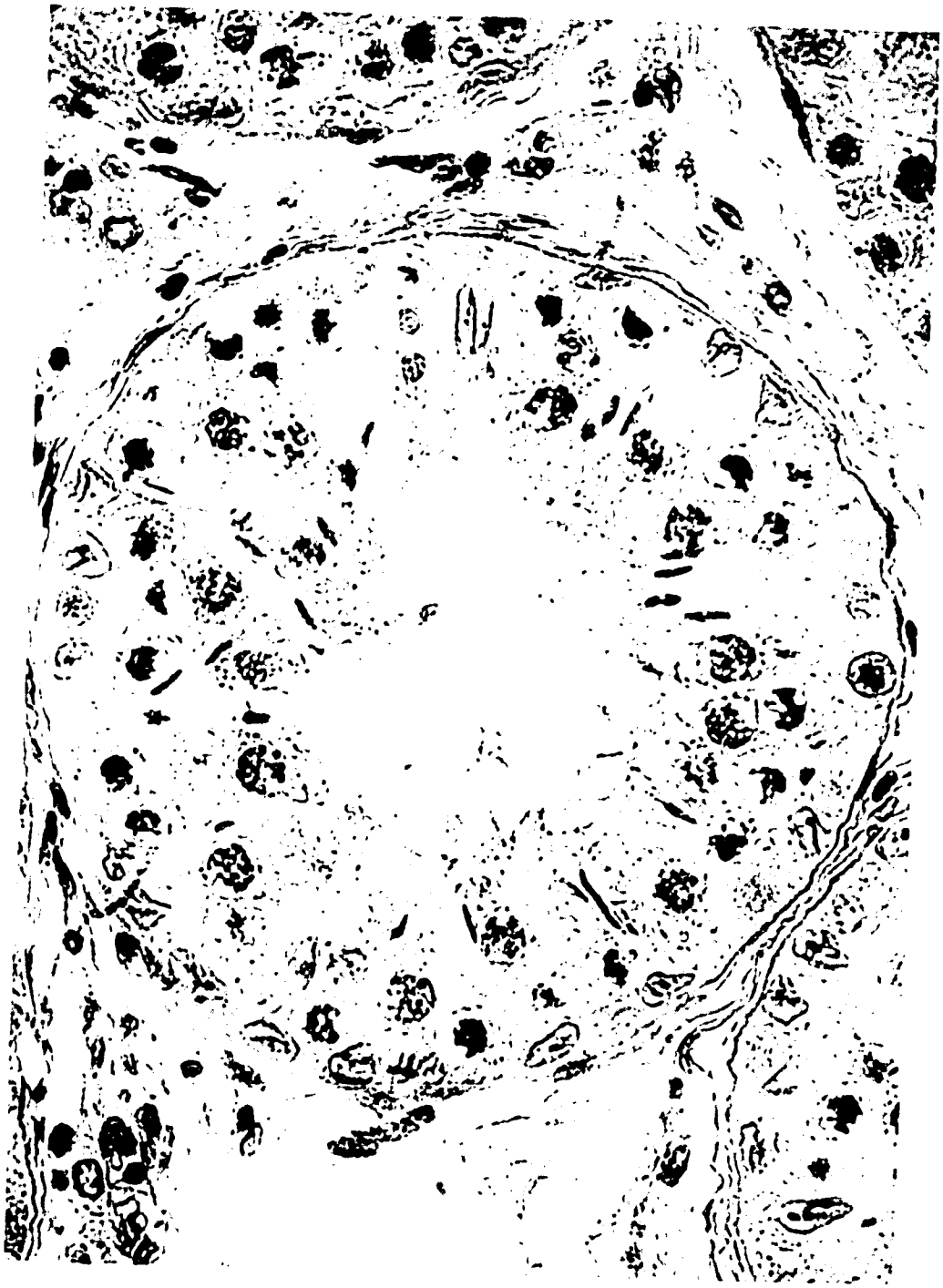

Fig. 5

Fig. 6. Cross section of a seminiferous tubule of vitamin A deficient calf C613 to demonstrate the lack of spermatogenesis. Hematoxylin and Eosin. X700.

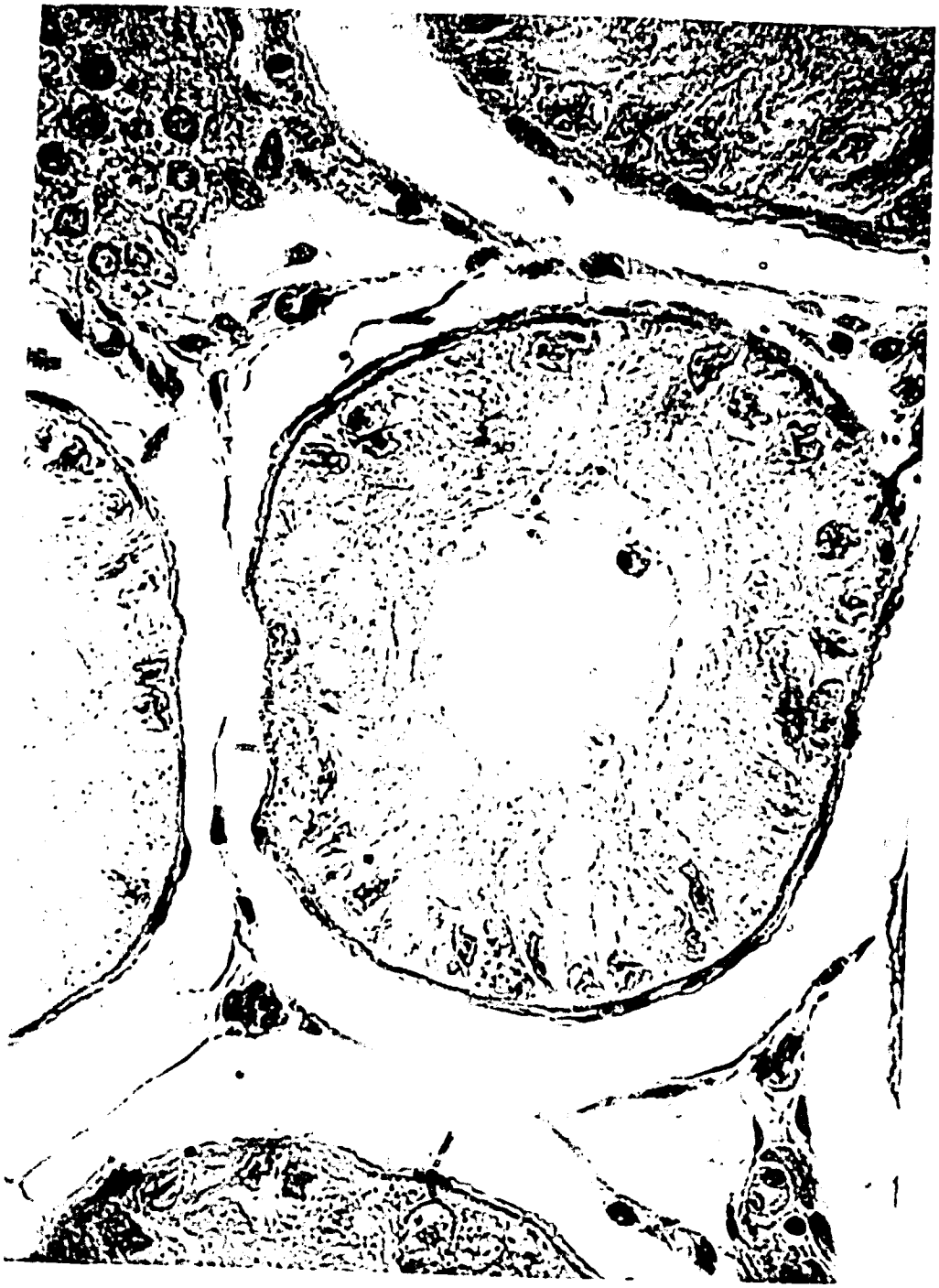

Fig. 8

Fig. 7. A portion of the cervix of vitamin A deficient calf C614. Note the replacement of the columnar epithelium by stratified squamous epithelium. Also observe how the stratified squamous epitholium starts beneath the columnar epithelium and pushes it toward the lumen. Hematoxylin and Eosin. X1442.

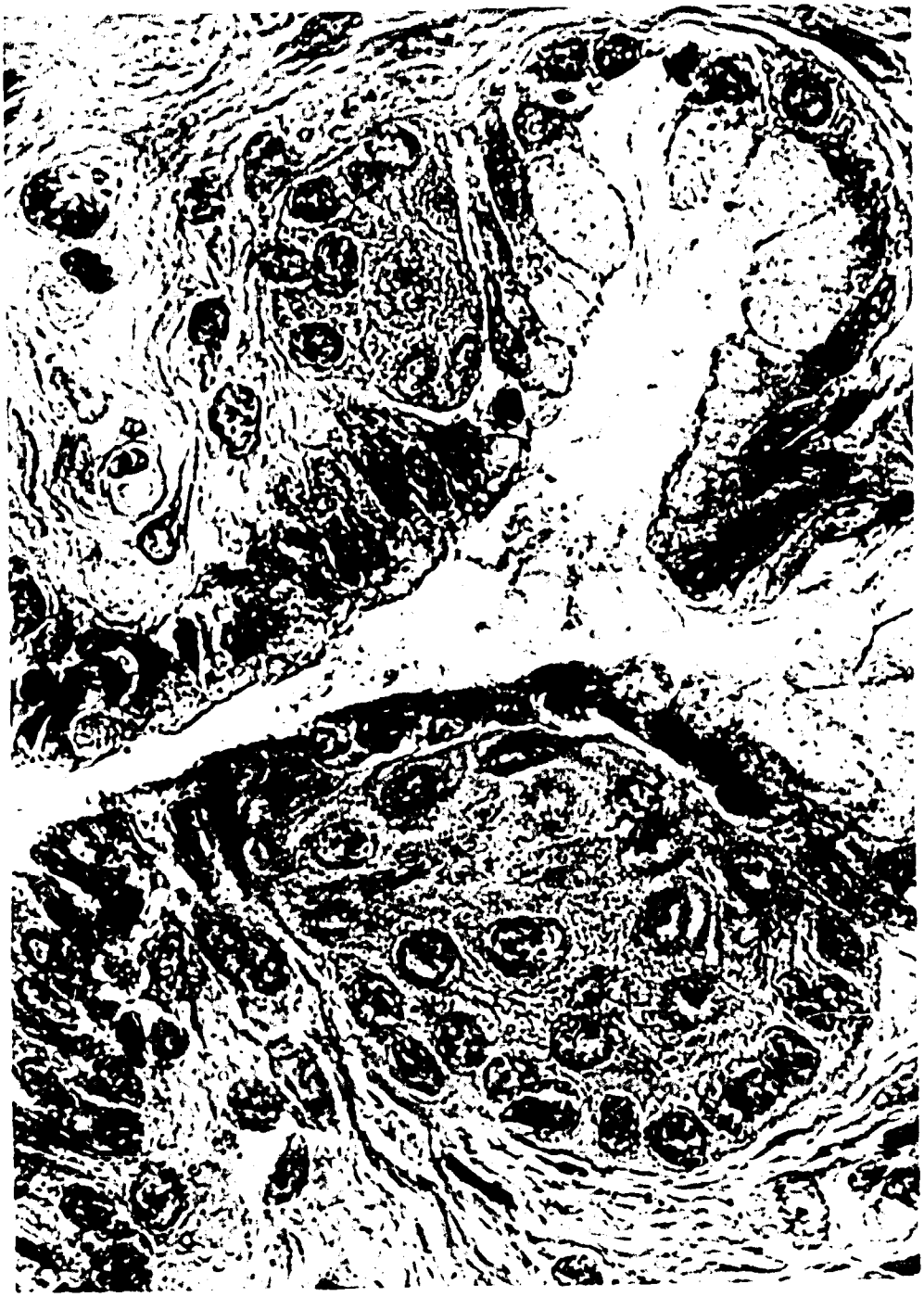

Fig. 7

Fig. 8. A cross section of a duct of the mandibular salivary gland of vitamin A deficient calf 3615. Note the replacement of the columnar type epithelium by stratified squamous epithelium. Hematoxylin and Eosin. X213.

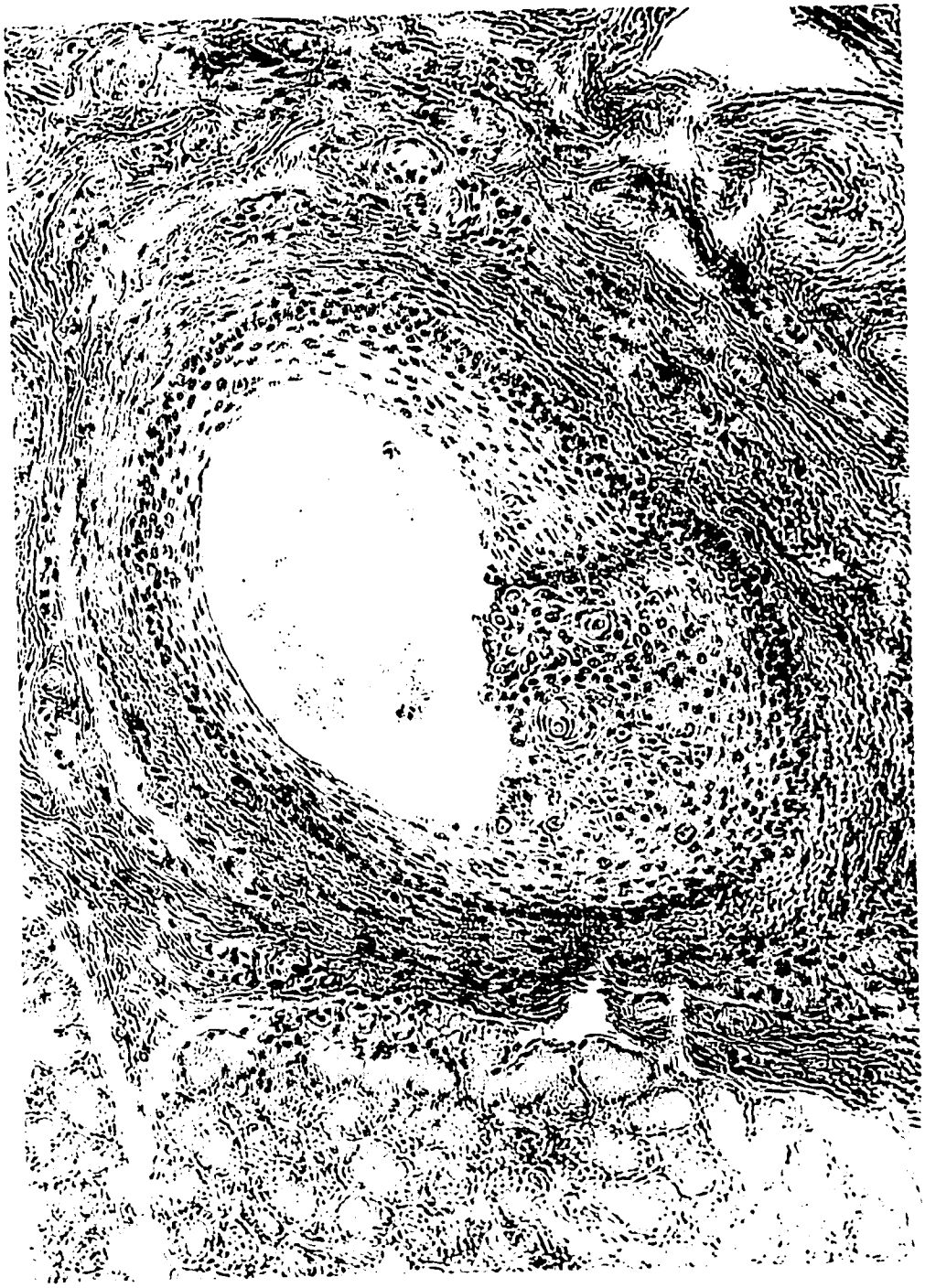

FIG. 8

Fig. 9. A section through a partially collapsed bronchus of vitamin A deficient calf 3614. In the lower portion of the photomicrograph the normal appearing respiratory epithelium is lined by cilia. In the upper portion of the photomicrograph the stratified squamous epithelium has replaced the normal epithelial structures. Note the intercellular bridges that are present in this epithelium. Hematoxylin and Eosin. X1027.

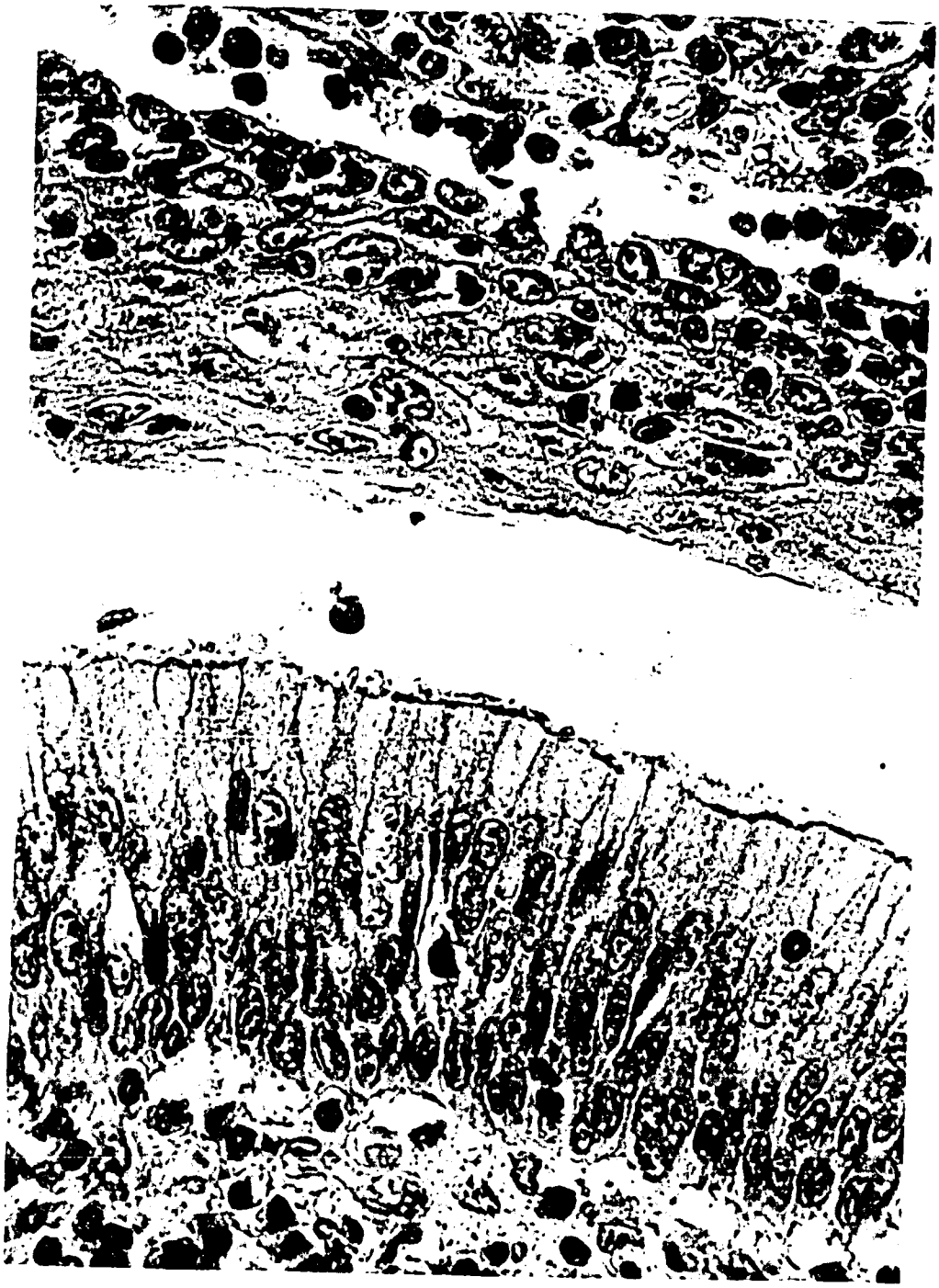

Fig. 9

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APPENDIX

Non-Colostrum Group

Holstein - Aut. 8222 - C589 (Control)

Clinical Record

Days

1. The animal did not receive colostrum, but was given 400 ml. of the dam's whole blood and 250,000 I. U. of Vitamin A in the form of shark liver oil.
6. Scouring appeared.
9. Scouring had stopped.
38. The eyes showed some lacrimation.
52. The animal had lost vigor, and had developed a cough.
54. Scouring recurred and some lacrimation continued.
56. Coughing became much harder.
65. Respirations and heart beats were very rapid. The tapeta lucida were faded and the pupils were dilated.
76. Calf developed abscess on neck.
77. The temperature was 103.1°. The animal had difficulty in rising and appeared to be stiff in all joints. The abscess on neck was becoming encapsulated and absorbed and was much smaller.
81. The temperature was 104.2°. Papillae of eyes were still normal but tapeta lucida were still faded. The right iris did not contract properly. The calf was still coughing and scouring.
86. The animal was destroyed when treatment had failed to cure the pneumonia.

Necropsy

Lung: The animal had a chronic pneumonia involving both apicals, cardiacs, the intermediate, and small portions of anterior diaphragmatic lobes. The right apical lobe contained a very large encapsulated abscess which gave off a foul odor. Small abscesses were present in all of the lobes of the right lung. The apical lobe of left lung was larger than normal and showed an encapsulated abscess.

Femur: The bone was fractured in the middle of the shaft.

Kidneys: The sinus appeared very edematous in both organs.

Gallbladder: Three brownish nodules were present in the mucosa.

Spinal cord: There was much edema around the cord, especially in the sacram.

Jersey - Aut. 8152 - C591 (Control)

Clinical Record

Days

1. The calf received no colostrum but was given 100,000 I. U. of Vitamin A in the form of shark liver oil.
2. Scouring and anorexia appeared.
3. Feces were firmer in consistency. An increase was noted in his appetite.
14. Scouring continued.
16. The feces were more firm.
17. Scouring continued.
18. The calf showed first symptoms of pneumonia such as shivering, a rattling sound when breathing, neck outstretched, and a temperature of 103.9°. Eight hours later the temperature was 105.3° and the heart was beating very rapidly.

24. The animal appeared slightly better after treatment.

26. The calf was destroyed for examination.

Necropsy

Liver: The organ was light grey in color and very friable.

Lymphnodes: Were very edematous.

Heart: Left ventricle was hypertrophied.

Lung: This organ did not have lesions of pneumonia but did have a large amount of mucous exudate in the trachea and bronchi.

Kidneys: Two small circumscribed light grey areas were observed in the cortex of the right kidney which resembled the lesions of interstitial nephritis.

Brain: A large pigmented area was present in the region of the olfactory lobe and involved the meninges.

Holstein - Aut. 8141 - C588

Clinical Record

Days

1. The calf did not receive colostrum but was given 200 ml. of the dam's whole blood.

2. 250 ml. of the dam's whole blood was given.

5. Scouring appeared.

10. Scouring continued.

23. The animal had not stopped scouring from the beginning and on this date showed the first symptoms of pneumonia. Treatment was started.

32. The calf died from pneumonia and scours.

Necropsy

Thoracic lymph nodes: These showed congestion.

Pleura: Abundant fibrinous adhesive exudate was present between the visceral and parietal pleurae in the region of apical and cardiac lobes.

Lung: On the right side pneumonia was present in the apical, cardiac, intermediate, and part of diaphragmatic lobe. Several large abscesses were present as well as some areas of necrosis on the left side. Pneumonia was present in the apical and cardiac lobes.

Stomach and intestines: These organs were greatly congested.

Kidneys: Revealed some congestion.

Brown Swiss - Aut. 8132 - C593

Clinical Record

Days

1. The calf did not receive colostrum but was given 400 ml. of the dam's whole blood intravenously.
5. The animal was not as active as usual.
8. An upper respiratory infection developed and his breathing could be heard many feet from the pen. The body temperature was 104.0°. Treatment was started.
11. Death took place from upper respiratory infection.

Necropsy

Sinuses: A purulent sinusitis was present. The nasal mucosa was greatly swollen.

Lung: This organ was congested and showed congenital atelectasis in several lobes. In the right diaphragmatic lobe there were several lobules that appeared to show some early pneumonia. The trachea and bronchi contained a frothy exudate.

Heart: The foramen ovale and the ductus arteriosus were patent.

Liver: This organ was very dark red in color and revealed some cloudy swelling on section.

Bladder: Some small yellow calculi were present.

Kidneys: The tubules and the pelvis like portion of both organs were stuffed with a yellow crystalline debris and calculi (probably sulphothiazole crystals).

Jersey - Aut. 8080 - A 57

Clinical Record

Days

1. Calf did not receive colostrum but was given 350 ml. of dam's serum.
2. 200 ml. of the dam's whole blood was given. The calf started to scour.
3. Scouring continued and calf had mild entorrhagia.
13. Still scouring.
22. The animal was very nervous and the eyes seemed to bulge. The calf had some early symptoms of pneumonia.

Necropsy

Lips: On the commissure was a small hemorrhagic area about 3 mm. in diameter.

Gums: There were several small hemorrhages and some congestion present.

Stomach: All four compartments of the stomach contained large amounts of wood shavings. The abomasum showed some inflammation characterized by congestion and increase of mucus.

Small intestine: Some petechial hemorrhages and congestion.

Large intestine: The organ showed numerous hemorrhages, and feces were very brown in color and watery.

Lung: A bronchopneumonia in the state of red hepatization was present in the apical and cardiac lobes on the right, and apical lobe of the left. Some pneumonia was present in the anterior portions of both diaphragmatic lobes.

Heart: A few small hemorrhages were present in the mitral valve.

Kidneys: Capsules stripped with difficulty. Both were congested and showed numerous pin point grayish-white foci. In addition some petechial hemorrhages were present.

Spleen: This organ was congested.

Brain: The blood vessels were markedly congested and the ventricles appeared to be dilated with servus fluid. The whole brain seemed softer in consistency.

Pituitary gland: This was found to be edematous.

Holstein - Aut. 8272 - A59

Clinical Record

Days

1. The animal did not receive colostrum.

28. Pneumonia and scouring developed and treatment was carried out.

38. Calf died of pneumonia.

Necropsy

Lung: The right lung showed consolidation of the apical, cardiac, and anterior portions of the diaphragmatic lobes. The left lung revealed a small linear streak of suppurative pneumonia in the cardiac portion. The right apical lobe was adherent to the thoracic wall by fibrin.

Stomach: Some ecchymoses were present in the fundic portion measuring from 1 to 3 mm. in diameter.

Intestines: The mucosa displayed some congestion.

Kidneys: The capsules stripped with difficulty. The surface of the cortex revealed numerous white greyish foci measuring from 1 to 5 mm. in diameter. On section the lesions extended through the depth of the cortex.

Liver: The structure showed some mottling suggestive of some degenerative processes.

Blood Picture of Calf. No.

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
1	2.5	5.27	7.88	10.0	11.6	42	14.7
10	1.25	5.78	8.70	14.5	11.6	45	13.3

R.B.C. -- Red blood cells.

W.B.C. -- White blood cells.

Hb. -- Hemoglobin in grams per 100 ml. of blood.

R.B.C.V. -- Volume of packed red blood cells expressed in percentage.

M.C.H. -- Mean corpuscular hemoglobin expressed in micromicrograms.

M.C.V. -- Mean corpuscular volume expressed in cubic microns.

Lymph. -- Lymphocytes.

Polys.

Eosin.

Juv. --

Stabs.

Mon. --

ug -- l

M/mm^3 .

T/mm^3 .

Blood Picture of Calf. No. C593

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
11.6	42	14.7	53.3	-	-	-	-	-	-
11.6	45	13.3	51.7	-	-	-	-	-	-

Polys. -- Polymorphonuclears.

Eosin. -- Eosinophiles.

Juv. -- Juveniles

ed in percentage. Stabs. -- Immature polymorphonuclears.

icromicrograms. Mon. -- Monocytes.

microns. ug -- Micrograms.

M/mm^3 -- Millions per cubic millimeter of blood.

T/mm^3 -- Thousands per cubic millimeter of blood.

Blood Picture of Calf. No. 589

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
6	2.5	14.73	12.23	12.7	10.4	46	8.5
13	3.75	13.73	8.25	9.8	9.8	40	11.7
20	8.37	9.68	9.2	8.2	9.5	36	10.3
27	1.25	7.73	8.34	14.85	8.6	35	10.31
34	0.0	6.28	9.22	12.7	8.6	34	9.32
41	5.0	5.78	6.87	13.5	7.0	29.5	10.18
55	3.75	6.76	8.27	9.9	7.5	30.5	9.06
62	0.0	4.8	7.91	12.9	7.5	28.5	9.47
69	5.0	2.24	7.9	13.9	7.6	30.0	9.68
76	5.0	3.99	7.53	35.85	6.8	27.5	9.03
86	0.0	4.8	7.93	56.95	6.95	28.0	8.94

Picture of Calf. No. 589 (Control)

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
10.4	46	8.5	37.6	72	23	1	0	0	4
9.8	40	11.7	48.4	66	32	0	1	0	1
9.5	36	10.3	39.1	59	37	0	0	0	4
8.6	35	10.31	41.9	-	-	-	-	-	-
8.6	34	9.32	36.8	66	32	0	1	0	1
7.0	29.5	10.18	42.9	61	33	1	0	0	5
7.5	30.5	9.06	36.8	63	34	0	0	0	2
7.5	28.5	9.47	36.07	-	-	-	-	-	-
7.6	30.0	9.68	37.9	43	48	0	4	2	3
6.8	27.5	9.03	36.5	34	55	0	8	3	1
6.95	28.0	8.94	35.1	19	65	0	8	7	1

Blood Picture of Calf. No. 591 (C

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
1	7.5	5.78	6.61	9.9	8.6	31	12.8
5	5.0	9.68	6.87	4.5	8.1	31	11.7
12	5.0	-	6.42	8.8	7.95	39.5	12.3
26	0.0	9.68	5.76	12.8	6.3	28	10.93

Picture of Calf. No. 591 (Control)

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
8.6	31	12.8	46.4	-	-	-	-	-	-
8.1	31	11.7	45.1	71	21	0	2	4	2
7.95	39.5	12.3	61.5	82	15	2	0	0	1
6.3	28	10.93	48.6	30	69	0	0	0	1

Blood Picture of Calf. No. C588

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.	M
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$	c
1	5.0	6.76	10.23	12.9	11.1	47	10.85	
4	7.5	7.73	11.11	18.65	10.4	43	9.36	
11	3.75	5.27	8.7	14.55	9.95	44	11.4	
18	2.5	6.28	8.33	10.2	8.9	36.5	10.6	
25	1.25	7.23	7.3	7.4	8.1	32.5	11.09	
32	0.0	4.28	7.56	5.0	8.1	32.0	10.71	

Picture of Calf. No. C588

b.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
.1	47	10.85	45.9	-	-	-	-	-	-
.4	43	9.36	38.7	77	19	1	0	0	3
.95	44	11.4	50.5	73	22	0	0	0	5
.9	36.5	10.6	43.8	69	24	1	0	3	3
.1	32.5	11.09	44.5	85	13	2	0	0	1
.1	32.0	10.71	42.3	-	-	-	-	-	-

Blood Picture of Calf. No.

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
1	0.0	6.28	8.94	12.75	8.0	29	8.94
3	10.0	6.28	8.15	9.65	7.15	27.5	8.77
10	3.75	7.73	6.88	7.8	5.5	23.5	7.99
17	6.25	7.73	7.18	4.62	5.7	24.0	7.93
24	2.5	7.73	7.08	11.75	5.45	24.0	7.69
31	0.0	3.35	7.6	21.65	5.3	23.5	6.97
38	0.0	3.35	6.57	15.8	5.0	22.5	7.61

Blood Picture of Calf. No. A59

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
8.0	29	8.94	32.4	21	74	0	2	1	2
7.15	27.5	8.77	33.5	35	59	2	0	0	4
5.5	23.5	7.99	34.1	87	12	0	0	0	1
5.7	24.0	7.93	33.42	89	7	2	0	2	1
5.45	24.0	7.69	33.8	48	42	0	5	4	1
5.3	23.5	6.97	30.9	51	34	0	4	9	2
5.0	22.5	7.61	34.2	18	59	0	5	17	1

APPENDIX

Clinical Records of Calves That Received Colostrum and a Vitamin A Deficient Ration Without Hay

Guernsey - Aut. 8078 - C586 (control)

Clinical Record

Days

1-20. Calf showed anorexia periodically.

21. Scouring started and calf appeared weak.

22. Discharged a mucopurulent exudate from nose.

24. Feces soft and yellowish white.

27. Still scouring in spite of treatment.

28. Feces were somewhat firmer in consistency but animal had not been eating.

30. Developed subnormal temperature and died.

Necropsy

The body was very thin as a result of not eating and the presence of a diarrhea.

Lungs: Pneumonia in the state of grey hepatization was observed in the right apical and cardiac lobes. On section there was some suggestion of an increase of connective tissue. Fibrinous adhesions were present between the lobes and the thoracic wall.

Pericardium: The sac was enlarged and filled with a serofibrinous exudate. A marked fibrinous epicarditis was present.

Adrenal glands: These structures were markedly congested.

Rumen: The papillae appeared a little longer than normal.

Jersey - C587 (control)

Clinical Record

Days.

2. Scouring appeared.

Guernsey - Aut. 8078 - C586 (control)

Clinical Record

Days

1-20. Calf showed anorexia periodically.

21. Scouring started and calf appeared weak.

22. Discharged a mucopurulent exudate from nose.

24. Feces soft and yellowish white.

27. Still scouring in spite of treatment.

28. Feces were somewhat firmer in consistency but animal had not been eating.

30. Developed subnormal temperature and died.

Necropsy

The body was very thin as a result of not eating and the presence of a diarrhea.

Lungs: Pneumonia in the state of grey hepatization was observed in the right apical and cardiac lobes. On section there was some suggestion of an increase of connective tissue. Fibrinous adhesions were present between the lobes and the thoracic wall.

Pericardium: The sac was enlarged and filled with a serofibrinous exudate. A marked fibrinous epicarditis was present.

Adrenal glands: These structures were markedly congested.

Rumen: The papillae appeared a little longer than normal.

Jersey - C587 (control)

Clinical Record

Days.

2. Scouring appeared.

11. Scouring was checked by treatment.

26. Feces were soft and watery.

33. Calf showed some dryness of skin and loss of hair over withers.

54. Scouring continued.

78. Coughing began.

81. Chewing and licking of wood.

90. Calf started tongue lolling.

170. Animal destroyed for examination. The only symptoms noticed from the 90th day to the present time was tongue lolling and a chronic cough.

Necropsy

Skin: There was some pityriasis around the eyes and over the withers.

Lungs: A pneumonia of the chronic type was present in the apical lobes.

Holstein - Aut. 8123 - A58

Clinical Record

Days

1. 500 ml. of the dam's blood given intravenously.

4. Scouring appeared.

12. Scouring continued.

13. Scouring continued and appetite poor.

14. Calf was eating better but diarrhea had not subsided.

23. Animal showed first symptoms of pneumonia.

33. Calf very sick with pneumonia and still scouring.

42. Died.

Necropsy

The animal died during the night. There appeared to be considerable postmortem changes.

Lymph nodes: These structures were greatly enlarged.

Pleura: Fibrinous adhesions were present between the lobes of the lung and the thoracic wall.

Lungs: Pneumonia was present in the apical and cardiac lobes of both lungs. There was extensive necrosis.

Abomasum: The mucosa was congested.

Intestines: A very marked congestion was present.

Holstein - Aut. 8335 - C582

Clinical Record

Days

115. Feces were very soft in consistency.

141. Tapeta lucida were fading.

159. Some blood showed in the feces.

165. Calf was losing weight and appeared tucked up in the abdomen. Some lacrimation was observed.

167. Scouring appeared.

168. 100,000 I. U. of shark liver oil was given daily for the next four days.

178. Marked improvement in calf had taken place as shown by decreased lacrimation, increased appetite and vigor.

181. Calf did not shed long hair coat. Some pityriasis over skin of neck.

186. Hair coat was shed some.

215. Lacrimation and incoordination were observed.

218. Scouring, syncopes, and pityriasis around the ears.

221. Calf showed loss of consciousness.

230. Animal lost consciousness when head was elevated.

232. The hair was disappearing around the eyes.

235. The animal displayed the following conditions on this date: coughing, lacrimation, dilation of the pupils, fading of the tapeta ludica, blurring of the optic discs, and scouring.

253. The corneas of the eyes were beginning to cloud over.

254. The cornea of both eyes showed white opacities and the left eye, an indentation, as though an ulcer was beginning to form. Calf had a fainting spell (syncope).

258. Scouring appeared.

270. Symptoms of pneumonia were observed.

274. 250,000 units of shark liver oil given to calf.

300. Since symptoms of chronic pneumonia persisted, 250,000 units of vitamin A were given each day for the next four days.

372. Calf was destroyed for examination.

Necropsy.

The skin over the withers was very scaly and the hair coat was rough and long. The carcass was emaciated and showed a generalized edema (anasarca).

Eyes: Edema of the optic disc was present. The tapeta lucida of both eyes had faded. The corneas were still somewhat opaque.

Lungs: A pneumonia was present in all lobes.

Liver: In one area of the organ were numerous small dull grey areas.

There appeared to be some increase in connective tissue.

Spleen: A hard lesion was present which resembled a walled-in abscess.

Holstein - Aut. 8162 - C585

Clinical Record

Days

1. Calf allowed to nurse for 15 hours.
42. There was some loss of hair and scaliness of skin along margin of ears.
48. Scouring appeared.
49. Symptoms of pneumonia were observed.
51. Pneumonic symptoms subsiding due to treatment.
58. Calf still coughing but vigorous.
71. Symptoms of pneumonia re-appeared.
73. The calf appeared to respond to treatment for pneumonia.
76. The animal was much better.
81. The pupils of the eyes were not contracting normally and the tapeta lucida of both eyes had faded.
83. Coughing continued.
86. Some lacrimation and scaliness showed around the eyes.
93. The eyes were bulging from the sockets and looked starry. They were rolling back and forth (nystagmus). The calf had difficulty in getting up.
94. Pneumonia flared up again.
100. Since the animal was paralyzed in hind limbs, it was destroyed for examination.

Necropsy

Lymph nodes: The renal, hepatic and mesenteric nodes revealed some edema, congestion, and hemorrhage.

Lungs: A chronic pneumonia was present with a large abscess in the right apical lobe.

Liver: A few red areas were scattered throughout the lobes.

Kidneys: The capsules stripped with difficulty and on section the cut surface bulged. Numerous small white spots were present which on section extended through the depth of the cortex.

Bladder: Petechial hemorrhages were quite numerous.

Intestines: These structures were congested and showed some hemorrhages.

Muscle: Somewhat edematous.

Pituitary: Contained a large cyst.

Holstein - Aut. 8080 - C583

Clinical Record

Days

72. Optic papillae was not as clear as normal.

90. Ruminal bloat was present.

98. Tapeta ludida faded somewhat.

105. Tapeta lucida were definitely faded and the optic papillae were still swollen.

118. The skin of the ears and neck was becoming scaly (pityriasis). The changes of the eyes were more marked.

132. There was some lacrimation. The calf showed some coughing and a discharge from the nostrils.

136. The papilla of the left eye was very blurred, while that of the right eye was not quite so markedly affected. Symptoms of pneumonia were noted.

137. Anorexia appeared.

139. Calf showed bloating.

140. Died of pneumonia and tympanites.

Necropsy.

Lungs: Pneumonia in the state of grey hepatization was located in the left cardiac and anterior portion of the diaphragmatic lobes. In the other lobes the pneumonia was in the state of red hepatization.

Abomasum: A few small ulcers were present in the mucosa.

Nesenteric lymph nodes: These structures were enlarged and edematous.

Kidneys: Some small white lesions were revealed in the cortex.

Heart: A few small hemorrhages were located on the tricuspid and bicuspid valves.

Eyes: The optic discs showed some edema and the tapet lucida were faded.

Optic nerve: There appeared to be some pinching of the nerve where it passed through the optic foramen.

Brain: Some congestion.

Holstein - Aut. 8131 - C584

Clinical Record

Days

84. Slight fading of tapeta lucida.

99. Calf showed bloating.

102. Eyes showed some blurring of optic papillae. Tapeta lucida had faded markedly.

108. Animal had difficulty in rising.

110. Calf developed loss of consciousness for first time and showed some lacrimation. Pityriasis was developing around the eyes and upon the ears.

115. Animal continued to show incoordination and bloat.

118. Calf showed tympanites.

120. Scouring syncope and lacrimation were observed.

121. Calf showed very severe bloat.

126. Coughing began.

128. Calf showed bloating.

134. Still continued to bloat. The tapeta lucida were faded and the optic papillae were blurred.

141. Symptoms of acute pneumonia and anorexia were seen.

Necropsy.

Subcutaneous tissues: Some edema was present.

Lungs: The organs showed a very extensive bronchopneumonia involving all the lobes except small portions of the diaphragmatic lobes.

Liver: The organ was mottled and showed some petechial hemorrhages.

Kidneys: Capsules stripped with some difficulty. There were a few petechial hemorrhages and some mottling of surface due to small greyish-white foci which on section could be seen extending down through the cortex.

Gallbladder: The bile was unusually concentrated and viscous.

Small intestine: There was a section of the ileum approximately 1

foot in length about 6 inches from the ileo-cecal valve that showed a marked inflammation characterized by congestion, increased mucus and a swollen condition of mucosa.

Spinal cord: There seemed to be an increase in fluid as well as some gelatinous material around the cord.

Brain: Congestion of vessels.

Eyes: Some edema of disc.

Blood Picture of Calf. No. A

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
1	0	15.6					
18	20	17.1	7.99	10.25	9.95	43	12.45
37	12.5	9.51	5.24	4.55	8.8	33	16.8
44	25.	9.69	9.97	8.75	8.1	39.5	8.1
51	17.5	9.68	10.59	7.55	8.6	40.0	8.3
58	55	15.6	10.28	8.66	9.2	41.0	8.9
65	34	14.24	9.58	8.75	10.1	37.0	10.5
72	35.5	11.72	8.74	9.50	10.4	45.0	11.9
79	45	17.4	9.47	6.70	11.1	41.5	11.7
86	55	16.89	9.36	11.40	9.8	40	10.4
93	55	20.4	9.39	11.40	10.1	41	10.8
107	83.75	17.58	8.38	12.05	9.5	32.5	11.3
114	87.5	12.3	8.13	13.00	9.8	34.5	12.0
127	72.5	17.57	7.18	9.55	9.5	32.6	13.2
135	88.75	7.12	6.7	6.40	7.8	29.0	11.6
145	87.5	16.2	7.61	10.80	9.2	32.5	12.0
152	112.5	20.6	7.79	9.60	9.2	32.6	11.8
222	90	26.45	5.99	11.80	7.3	25.4	12.15
243	72.5	15.27	7.11	7.65	8.3	31.5	11.65

Blood Picture of Calf. No. A54 (✓ control)

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
9.95	43	12.45	53.8						
8.8	33	16.8	62.7						
8.1	39.5	8.1	39.6						
8.6	40.0	8.3	37.7						
9.2	41.0	8.9	39.8	76	20	0	0	1	3
10.1	37.0	10.5	38.6	74	23	0	0	0	3
10.4	45.0	11.9	47.4	67	30	0	1	1	1
11.1	41.5	11.7	43.8	70	28	0	0	0	2
9.8	40	10.4	41.1	59	37	0	0	0	4
10.1	41	10.8	43.7	70	28	0	1	0	1
9.5	32.5	11.3	38.8	68	29	1	0	0	2
9.8	34.5	12.0	42.4	71	25	0	0	0	3
9.5	32.6	13.2	45.4						
7.8	29.0	11.6	43.2	62	32	3	1	0	2
9.2	32.5	12.0	42.7	61	37	1	0	0	1
9.2	32.6	11.8	41.8	67	28	0	2	0	3
7.3	25.4	12.15	42.4	65	32	0	0	0	3
8.3	31.5	11.65	44.3	80	15	1	0	0	4

Blood Picture of Calf. No. A55

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
14	17.5	5.9	5.33	5.45	9.8	-	18.4
21	20.0	7.73	7.79	5.30	9.8	-	12.5
28	25.0	10.58	9.12	7.25	10.1	44.	11.07
35	37.5	10.15	9.74	5.05	10.1	50.	10.3
42	30.0	12.7	8.56	5.00	9.8	38.	11.4
49	37.5	14.24	9.16	7.00	10.4	41.	11.3
56	37.5	14.24	10.03	6.95	10.1	41.	10.0
63	37.5	13.42	10.36	9.25	9.8	34.	9.4
70	61.25	19.94	8.25	8.25	10.3	36.	12.5
84	77.5	16.01	11.18	14.25	11.4	38.	11.9
91	55.0	19.86	7.26	10.20	10.8	41.0	14.8
104	77.5	14.52	7.84	9.00	8.8	30.7	11.2
111	58.75		6.71	7.65	8.3	31.0	12.3
121	30.0	10.72	10.16	13.70	9.2	38.0	9.0
128	58.75	19.45	8.52	6.05	9.2	36.5	10.79
149	85.0	24.21	6.74	10.15	7.95	32.0	11.8
175	127.5	17.42	7.85	12.65	8.1	31.0	13.3
208	77.5	28.07	7.32	10.50	7.5	26.5	10.2
215	67.5	15.27	6.49	12.15	6.5	22.5	10.0

lood Picture of Calf. No. A55 (✓ control)

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
9.8	-	18.4							
9.8	-	12.5							
10.1	44.	11.07	48.2						
10.1	50.	10.3	51.3	74	23	0	0	2	1
9.8	38.	11.4	44.3	67	30	0	0	1	2
10.4	41.	11.3	44.8	84	12	0	0	1	2
10.1	41.	10.0	43.8	84	16	0	0	0	5
9.8	34.	9.4	32.8	65	35	0	0	0	4
10.3	36.	12.5	43.6	61	36	0	0	0	5
11.4	38.	11.9	33.9	77	19	0	0	0	4
10.8	41.0	14.8	56.4	77	17	1	0	0	5
8.8	30.7	11.2	39.1						
8.3	31.0	12.3	46.2	87	7	1	0	0	5
9.2	38.0	9.0	37.4	65	32	0	0	0	3
9.2	36.5	10.79	42.8	66	28	2	2	0	2
7.95	32.0	11.8	47.4	64	35	0	0	0	1
8.1	31.0	13.3	39.4	64	24	5	0	0	5
7.5	26.5	10.2	36.2	78	10	11	0	0	3
6.5	22.5	10.0	34.5	77	15	6	0	0	2

Blood Picture of Calf. No. A56

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.	
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$	
1		3.9	10.62	5.87				
3	8.75	14.63						
8	16.2	15.76	11.23	7.70	8.6	44.5	7.6	
15	10.0	13.42	10.64	10.05	8.9	41.	8.3	
22	10.0	7.73	9.75	7.70	10.1	37.	10.3	
29	25.0	11.7	7.19	8.45	9.2	35.5	12.8	
43	45.0	11.64	8.27	7.50	8.1	29.5	9.9	
50	21.5		8.05	7.20	8.6	32.5	10.6	
64	35.75	8.68	7.38	10.45	8.1	33.5	10.9	
71	39.8	4.8	8.55	8.05	8.9	35.2	10.4	
81	50.0	13.86	8.51	7.50	8.4	33.0	9.8	
88	87.5	17.58	8.55	9.40	8.6	32.5	10.05	
102	45.0	19.94	7.19	7.95	9.5	36.0	13.02	
130	85.0	23.67	7.28	12.20	8.45	33.0	11.6	
158	68.75	32.99	7.67	5.70	7.8	32.0	10.15	
179	58.75		7.36	8.00	7.8	28.5	10.5	
193	36.25	18.72	7.55	11.75	8.3	28.	10.99	
200	27.5	22.04	6.70	8.70	7.8	27.75	11.6	

lood Picture of Calf. No. A56 (/ control)

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
8.6	44.5	7.6	39.6						
8.9	41.	8.3	38.5						
0.1	37.	10.3	37.9						
9.2	35.5	12.8	49.3	69	28	1	0	0	2
8.1	29.5	9.9	35.6	61	38	0	0	0	1
8.6	32.5	10.6	40.3	89	9	0	0	0	2
8.1	33.5	10.9	45.3						
8.9	35.2	10.4	41.1	65	35	0	0	0	1
8.4	33.0	9.8	38.7	84	15	0	0	0	1
8.6	32.5	10.05	37.88	75	23	0	1	0	1
9.5	36.0	13.02	50.06	53	44	0	0	0	3
8.45	33.0	11.6	45.19						
7.8	32.0	10.15	41.7	74	25	0	0	0	1
7.8	28.5	10.5	38.7	83	15	0	0	0	2
8.3	28.	10.99	37.02	52	46	1	0	0	1
7.8	27.75	11.6	41.4	82	16	1	0	0	1

Blood Picture of Calf. No. 587

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
2	10	19.45	8.85	10.15	8.6	36.5	9.71
9	1.25	14.73	7.55	8.80	7.8	33	10.33
16	5.0	25.21	6.85	7.00	7.0	28.2	10.2
23	10.	22.07	6.91	6.45	7.8	32	11.28
30	2.5	23.07	7.05	7.60	7.8	23.5	11.06
37	1.25	22.07	8.1	7.35	8.9	43	10.98
44	5.0	23.28	8.21	9.50	8.5	39	10.35
58	3.75	30.72	8.47	7.70	9.2	37	10.7
65	15.0	28.57	9.12	8.05	9.6	35.5	10.5
72	11.5	24.25	9.55	7.40	10.8	41.5	11.3
79	12.5	16.58	9.56	11.80	10.4	37.5	10.8
86	8.75	28.57	10.09	7.80	10.1	39.	10.0
93	11.25	S	10.65	7.50	10.95	40.5	10.2
100	5.0	30.72	10.09	10.10	11.1	40	11.
107	5.0	28.57	8.43	9.10	10.4	37	12.3
114	15.0	S	8.45	9.25	10.1	38	11.9
121	5.0	19.94	9.01	11.95	10.1	34.25	11.2
128	6.25	12.71	8.91	8.65	10.1	37	11.3
149	7.5	S	8.96	10.65	11.1	33	12.38
165	5.0	24.25	9.17	12.25	11.1		12.1
170	2.5	24.25	7.68	9.05	11.1	38.3	14.4

Blood Picture of Calf. No. 587 (- control)

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
8.6	36.5	9.71	37.8						
7.8	33	10.33	43.7	61	37	0	0	0	2
7.0	28.2	10.2	41.1	78	20	0	1	0	1
7.8	32	11.28	46.3						
7.8	23.5	11.06	47.5						
8.9	43	10.98	53.08	82	14	0	0	0	4
8.5	39	10.35	47.5	65	25	0	0	0	8
9.2	37	10.7	43.1	62	37	0	0	0	0
9.6	35.5	10.5	38.9						
10.8	41.5	11.3	43.4	85	9	1	1	0	4
10.4	37.5	10.8	39.2	71	23	4	0	0	1
10.1	39.	10.0	38.6	89	7	1	0	0	2
10.95	40.5	10.2	38.02	74	23	2	0	0	1
11.1	40	11.	39.6	86	5	8	0	0	1
10.4	37	12.3	43.8	89	9	0	0	0	2
10.1	38	11.9	44.9	87	10	2	0	0	1
10.1	34.25	11.2	38.01	80	18	1	0	0	1
10.1	37	11.3	41.5	72	26	0	0	1	1
11.1	33	12.38	36.8	70	29	1	0	0	1
11.1		12.1		66	32	0	1	0	0
11.1	38.3	14.4	49.8	78	5	7	0	0	0

Blood Picture of Calf. No

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
Born	0	7.13	10.3	7.40	12.9	47	12.5
9	5	7.73	11.03	11.20	12.1	46	10.97
23	0	6.67	12.15	25.40	12.1	46	9.9
30	1.25	7.73	8.28	4.25	9.8	40	11.8
37	5	7.23	9.25	6.80	8.1	33.5	8.75

Blood Picture of Calf. No. A58

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
12.9	47	12.5	45.6						
12.1	46	10.97	41.7	78	19	0	0	0	3
12.1	46	9.9	37.7	32	67	0	0	0	0
9.8	40	11.8	48.3	71	28	-	-	-	1
8.1	33.5	8.75	36.2	55	37	1	3	-	3

Blood Picture of Calf. No

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
14	17.5	9.5					
28	14.0	10.6	4.47	5.00	6.8	24	15.21
49	15.0	17.1	7.13	3.65	7.5	29	10.52
68	12.5	9.5	5.21	6.60	7.5	28	14.4
75	20	8.71	6.58	5.50	8.1	36	12.3
82	10	8.7	7.86	5.90	7.5	33.5	9.5
89	19	11.72	8.16	7.25	8.9	40	10.9
96	10	9.68	7.86	6.25	7.3	33.5	9.2
103	17.5	11.15	8.18	8.10	6.0	29.5	7.3
110	17.5	10.15	9.03	6.45	9.5	36.0	10.5
117	5.0	7.73	11.33	5.95	9.8	39.0	8.6
124	5.0	6.76	7.65	3.60	9.2	33.0	12.03
138	7.5	7.3	7.05	9.75	9.8	35.0	13.9
145	7.5	7.74	6.46	7.55	8.9	31.0	13.7
158	10.0	3.99	6.36	4.75	8.1	28.0	12.7
166	8.75	2.24	6.92	8.25	8.6	34.0	12.4
176	6.25	3.99	6.06	9.90	8.3	32.5	13.6
183	10.0	5.78	5.11	7.40	7.8	27.8	13.2
197	5.0	19.94	4.63	6.55	6.3	25.5	13.6
204	7.5	10.72	6.12	9.50	7.5	29	14.6
211	7.5	9.68	6.22	11.90	7.5	28	12.6
218	5.0	8.7	6.35	6.85	8.2	31.5	12.9

Blood Picture of Calf. No. C582

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
6.8	24	15.21	53.7						
7.5	29	10.52	40.69						
7.5	28	14.4	53.7						
8.1	36	12.3	54.7						
7.5	33.5	9.5	42.6						
8.9	40	10.9	49.0						
7.3	33.5	9.2	42.6	91	6	0	0	0	3
6.0	29.5	7.3	36.06	80	18	1	0	0	2
9.5	36.0	10.5	39.8	76	21	1	0	0	2
9.8	39.0	8.6	34.0	84	11	0	1	0	4
9.2	33.0	12.03	43.1						
9.8	35.0	13.9	49.6	67	31	0	0	0	2
8.9	31.0	13.7	47.9						
8.1	28.0	12.7	43.7	71	24	0	5	0	1
8.6	34.0	12.4	49.1	80	17	0	0	0	3
8.3	32.5	13.6	53.6	88	5	0	1	0	6
7.8	27.8	13.2	47	95	1	0	0	0	3
6.3	25.5	13.6	55.8	87	7	0	0	0	6
7.5	29	14.6	56.6	85	11	1	0	0	3
7.5	28	12.6	45.0	73	25	1	0	0	1
8.2	31.5	12.9	49.6	83	10	1	0	0	6

Data on Calf No. C582 (conti

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
225	2.5	6.28	5.49	9.60	8.1	31.0	14.75
232	2.5	5.78	5.64	8.20	7.4	30.5	13.13
239	6.41	5.0	5.71	12.85	7.0		12.25
253	5.0	6.76	6.57	8.10	6.9	25.5	10.5
260	2.5	4.33	6.07	7.45	6.9	31.5	11.3
267	5.0	3.35	5.64	6.40	8.1	31.0	14.3
274	5.0	4.8	4.48	10.20	6.3	23.	14.06
281	2.5	5.78	4.54	11.15	5.85	21.5	12.8
288	5.0	4.8	4.39	9.05	4.8	20.0	10.9
295	10.0	5.78	3.83	6.77	4.4	17.5	11.48
302	1.25	8.7	4.70	8.55	6.3	23.75	13.4
309	5.0	11.72	5.11	6.10	6.15	22.	12.03
316	5.0	3.35	4.57	8.75	5.7	22.0	12.4
323	5.0	4.33	5.31	8.35	6.5	23.5	12.24
344	5.0	7.73	6.44	5.85	8.1	31.5	12.57
360	0.	8.7	5.25	5.20	7.8		14.8
365	0.	9.68	5.68	11.80	8.8	32.0	15.4
372	5.0	3.35	3.90	12.80	8.2	29.5	21.0

Data on Calf No. C582 (continued)

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
8.1	31.0	14.75	56.49						
7.4	30.5	13.13	54.07	83	12	0	0	0	5
7.0		12.25	53.2						
6.9	25.5	10.5	38.8	69	30	0	0	0	1
6.9	31.5	11.3	51.3						
8.1	31.0	14.3	54.9	81	15	0	0	0	4
6.3	23.	14.06	51.3	56	35	0	6	1	2
5.85	21.5	12.8	47.4	47	45	0	6	1	2
4.8	20.0	10.9	45.5	55	39	1	2	1	2
4.4	17.5	11.48	45.6	66	31	1	1	0	1
6.3	23.75	13.4	50.5	84	14	0	0	0	2
6.15	22.	12.03	43.05	88	11	0	0	0	1
5.7	22.0	12.4	48.1	76	21	1	0	0	2
6.5	23.5	12.24	44.25	64	31	1	0	4	0
8.1	31.5	12.57	48.9	80	17	1	1	0	1
7.8		14.8		55	38	1	5	0	1
8.8	32.0	15.4	56.3	40	58	0	0	2	0
8.2	29.5	21.0	75.6	46	47	0	1	5	1

Blood Picture of Calf. No. C

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
0	5.0	7.3	9.9	12.30	11.1	44	11.2
14	12.5	5.78	8.35	8.80	10.4	40	12.4
22	1.2	6.28	9.47	5.75	9.2	38	9.7
32	8.75	5.87	10.48	18.15	11.4	45	10.87
39	5.0	5.78	9.22	7.05	8.3	33	9.03
53	5.0	13.23	8.69	9.80	8.6	36.5	9.89
60	3.75	4.8	7.45	19.25	8.45	35.5	11.34
67	5.0	9.11	6.66	8.40	7.45	31.4	11.15
74	5.0	5.78	7.04	14.85	7.95	32.5	11.29
81	3.75	5.78	7.36	11.53	7.3	31.5	9.9
88	0	6.76	7.35	8.85	6.7	30.8	9.11
95	0	5.33	6.25	4.35	6.4	26.0	10.24
100			8.41	6.55	7.95		9.45

Blood Picture of Calf. No. C585

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
11.1	44	11.2	44.4						
10.4	40	12.4	47.9	88	9	-	-	-	4
9.2	38	9.7	40.1	79	19	-	-	-	2
11.4	45	10.87	42.9	82	17	-	-	-	1
8.3	33	9.03	35.7	67	29	1	1	1	1
8.6	36.5	9.89	42	60	39	-	-	-	1
8.45	35.5	11.34	47.6	64	26	-	3	-	7
7.45	31.4	11.15	47.1	60	33	1	4	-	2
7.95	32.5	11.29	46.1	59	37	-	-	-	4
7.3	31.5	9.9	45.5						
6.7	30.8	9.11	41.9	33	64	0	1	1	1
6.4	26.0	10.24	41.6	74	24	1	-	-	1
7.95		9.45							

Blood Picture of Calf. No.

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
5	12.5	10.74	8.21	8.00	9.5	43.0	11.56
24	10.0	5.78	5.42	7.35	8.6	34.	15.9
31	16.25	7.24	8.9	5.60	8.6	45.	9.6
38	10.0	7.73	8.14	8.30	7.3	38.	8.9
45	19.0	8.70	8.08	5.60	8.0	38.	9.9
52	10	10.68	8.97	9.65	8.6	39.	9.5
59	16.2	9.68	8.6	8.95	8.9	40	10.3
66	12.5	5.26	10.89	7.65	8.6	36.5	7.8
73	10.	4.8	9.0	10.00	8.6	35.4	9.5
80	8.75	5.78	8.56	6.05	8.9	35.0	10.4
94	6.25	4.8	9.11	6.70	8.3	31.5	11.9
101	7.5	3.35	10.44	7.95	8.3	32.0	7.9
114	8.75	1.9	7.99	5.65	7.5	28.3	9.38
123	1.2	1.9	7.54	8.55	7.5	31.	9.9
132	5.0	3.99	7.97	6.40	7.4	30.5	9.3
139	3.75	3.99	6.7	5.85	7.0	30.1	10.5

Blood Picture of Calf. No. C583

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
9.5	43.0	11.56	52.4						
8.6	34.	15.9	62.7						
8.6	45.	9.6	50.5						
7.3	38.	8.9	46.6						
8.0	38.	9.9	47.4	85	10	-	-	-	5
8.6	39.	9.5	39.5	76	21	1	-	-	2
8.9	40	10.3	54.07	75	24	-	-	-	1
8.6	36.5	7.8	33.5	83	16	1	-	-	-
8.6	35.4	9.5	37.6	68	30	1	-	-	1
8.9	35.0	10.4	40.9	86	8	1	-	-	5
8.3	31.5	11.9	34.2	72	24	1	1	1	1
8.3	32.0	7.9	30.6						
7.5	28.3	9.38	35.4	62	31	0	4	2	1
7.5	31.	9.9	41.1	71	23	1	-	-	-
7.4	30.5	9.3	38.3	73	18	1	1	3	4
7.0	30.1	10.5	45	58	38	2	-	-	2

Blood Picture of Calf. No.

Age	Carotene	Vitamin A.	R.B.C.	W.B.C.	Hb.	R.B.C.V.	M.C.H.
days	ug/100ml.	ug/100ml.	M/mm^3	T/mm^3	gm.%	%	$gm.x10^{-12}$
1	5.0	1.0	10.59	8.60	11.8	59	11.1
4	5.9	11.46	11.09	10.70	11.1	59.5	10.0
11	19.0	9.15	8.64	12.90	10.8	54	12.5
18	5.0	6.28	10.18	12.70	12.1	45	11.8
25	10.0	11.9	9.54	11.05	10.4	43.5	10.9
32	6.25	7.73	10.52	9.85	11.1	42	10.5
38	10.0	3.99	10.75	11.50	9.5	37.9	8.8
45	10.0	8.7	9.55	10.64	10.4	40.5	10.9
59	6.25	9.11	9.01	9.95	10.1	39.5	11.2
66	-	-	9.32	11.15	11.1	36.0	11.9
79	4.0	3.99	8.07	8.05	8.5	35.0	10.5
87	8.75	1.9	7.95	7.65	8.6	35.0	10.8
97	6.25	5.27	8.87	8.35	8.7	34.0	9.5
104	5.0	7.73	10.06	8.00	8.1	31.0	8.05
118	3.75	5.78	6.67	7.65	7.4	29.0	11.09
125	0	4.8	5.73	8.05	6.15	25.0	10.7
132	2.5	4.8	5.26	12.65	6.0	24.4	11.4
139	5.0	7.73	4.84	8.70	5.3	20.5	10.9

Blood Picture of Calf. No. C584

Hb.	R.B.C.V.	M.C.H.	M.C.V.	Lymph.	Polys.	Eosin.	Juv.	Stabs.	Mon.
gm.%	%	gm.x10^{-12}	cu/ug	%	%	%	%	%	%
11.8	59	11.1	47.9						
11.1	59.5	10.0	53.6						
10.8	54	12.5	62.5	75	23	1	0	0	1
12.1	45	11.8	44.2	65	33	-	-	-	2
10.4	43.5	10.9	45.5	84	14	-	-	-	2
11.1	42	10.5	39.9	74	21	1	-	1	3
9.5	37.9	8.8	34.4	80	18	-	-	-	2
10.4	40.5	10.9	42.5	78	20	-	-	-	1
10.1	39.5	11.2	43.8	82	17	-	-	-	1
11.1	36.0	11.9	38.6						
8.5	35.0	10.5	43.3						
8.6	35.0	10.8	44.0	75	23	-	-	-	2
8.7	34.0	9.5	38.3	50	48	-	-	-	2
8.1	31.0	8.05	30.8	82	14	2	1	-	2
7.4	29.0	11.09	43.4	87	12	-	-	-	1
6.15	25.0	10.7	43.6	67	30	-	-	-	3
6.0	24.4	11.4	46.3	64	29	1	2	1	3
5.3	20.5	10.9	42.3	51	45	-	1	-	3

APPENDIX

Clinical Records of Calves that Received Colostrum at Birth and a Basal Ration Containing a Hay Low in Carotene

Aryshire - Aut. 9255 - C616 (control)

Clinical Record

Days

157. Animal did not eat grain well.

161. Calf was consuming grain only every other day.

175. 10 mg. of cobalt sulfate was given orally.

178. Appetite was greatly improved.

367. Calf was destroyed for examination.

Necropsy

This was an unusual case in that no gross lesions of any kind were observed.

Holstein - Aut. 9288 - C617 (Control)

Clinical Record

Days

13. Symptoms of pneumonia.

20. Calf appeared to make good recovery from pneumonia.

196. Anorexia developed.

198. Anorexia continued.

200. The animal was given 10 mg. cobalt sulphate per day.

203. Appetite returned to normal.

256. Blood vessels of the eyes were about twice the diameter of those of the deficient animals.

318. Developed abscess on abdomen due to injury on feed box.

376. Destroyed for examination.

Necropsy

Liver: The organ appeared mottled and somewhat swollen. The surface showed numerous dull grey areas surrounded by a red zone of inflammation. On section the lesions were scattered through the depth of organ.

Lymph nodes: The mandibulars, parotids, and some of the mesenteric nodes were enlarged, hemorrhagic, and congested.

Eyes and Optic nerves: The optic nerves did not looked pinched where they passed through the optic foramen. The nerves at this point were slightly narrower than other points of the nerve. The optic foramen measured about 4 mm. in diameter.

Pituitary gland: The structure measured 11 by 13 by 21 mm. and weighed 3.6 gm.

Holstein - Aut. 9008 - C611

Clinical Record

Days

44. Tapeta lucida were very yellow (normal).

105. Lacrimation began.

235. Lacrimation continued and partial syncope.

236. Animal lost consciousness (syncope).

238. Anorexia developed.

239. Since animal was not eating well, started administering 10 mg. of cobalt sulphate per day.

242. Calf was eating normally again.

244. Showed syncope.

245. The irises did not contract properly. The optic discs were somewhat blurred. The tapeta lucida were still yellow in color.

247. Calf showed symptoms of nightblindness.

250. Animal had syncope and injured back so that he had to be destroyed.

Necropsy

There were some bruised areas scattered over the legs at various points characterized by hemorrhage and edema.

Spinal Column: There was a complete fracture at the first lumbar vertebra.

Abomasum: The mucosa of the organ was congested.

Aryshire - Aut. 9210 - C613

Clinical Record

Days

35. Tapeta lucida were only about one-half as yellow as the controls and C611.

59. A slight pityriasis was present on the margin of the ears.

65. Night blind.

136. Pupils were dilated more than normal in bright light.

200. The tapeta lucida were faded some.

215. The eyes were about the same.

258. The eye reflexes were not as sharp as the controls.

260. Animal had first attack of syncope.

261. The reflex of the left eye was not as strong as the right eye.

264. Animal lost consciousness when bled.

266. The eyes were starry and bulged from their sockets. The tapeta lucida were faded still more.

287. The optic papillae for the first time appeared blurred.

288. The animal was totally blind.

289. The right eye bulged more than the left.

301. The optic papillae were foggy and the large veins coming in at the top of the disc appeared more tortuous when compared with the normals.

320. The blood vessels of the eyes were more tortuous than normal and only about one-half the normal diameter. The remainder of the eye changes were about the same as mentioned previously.

325. Loss of consciousness (syncope).

327. There appeared to be an increase in the number of blood vessels in the eye grounds.

332. Calf fainted when handled for bleeding.

358. Destroyed for examination.

Necropsy

The only lesions found in this animal were confined to the eye and the pituitary.

Pituitary: This structure contained a cyst measuring 6 mm. in diameter.

Optic nerve: There was a narrowing of these structures at the place where the nerve passed through the optic foramen.

Holstein - Aut. 9224 - C614

Clinical Record

Days

52. Some pityriasis was present on the margin of the ears.

71. The irises were not contracting properly because the pupils remained open in bright light.

89. The optic discs were slightly blurred.

129. The pupils were dilated.

193. The tapeta lucida were beginning to fade when compared with the normals.

218. Some discharge of a mucopurulent exudate from the nostrils.

229. The eyes were about the same.

244. The optic discs were very foggy.

246. Calf was completely blind.

248. Optic disc was blurred and could be cleared at 1.5 diaopters. The pupils were dilated.

254. Calf had typical vitamin A deficient syncope. Its left horn was broken off.

259. Animal showed marked loss of consciousness. It bumped into stall when walking. No reflexes of eyes were present. The optic discs were enlarged, very irregular in outline, foamy, and showed congestion of vessels. The discs were made clear at 1½ diaopters. The tapeta lucida still had some yellow color.

266. Calf was very nervous.

280. The blood vessels of the eye were smaller in diameter than the controls. The remainder of the eye conditions were about the same.

290. Calf lost consciousness.

293. Animal had rolling of the eye balls (nystagmus).

295. The eyes were bulging and appeared almost pointed at the front. The optic discs were blurred, irregular in outline, and many of the vessels were indistinct. The other eye changes were about the same.

297. Calf did not eat grain.

298. Animal was not eating grain so 10 mg. of cobalt sulphate was given.

300. Started eating its grain.

307. The appetite of the animal was back to normal.

314. Eyes were about the same.

322. The blood vessels of the eyes were much narrower than previously.

329. The feces were soft in consistency.

331-361. The calf did not show any new changes during this period.

363. The eyes were examined just before the animal was destroyed and they were about the same. The tapeta lucida did not completely fade in this calf. The blood vessels were about one-third the normal diameter.

Necropsy

Skin and subcutaneous tissues: There were no skin lesions and no edema present in the subcutaneous tissues.

Thyroid glands: These organs appeared smaller than normal.

Lungs: The right apical lobe revealed a chronic pneumonia with some bronchiectasis. The bronchi and bronchioles contained a very thick tenacious purulent exudate. Some of this exudate was present in the trachea.

Kidneys: The capsules stripped fairly easy. The cortex showed scattered petechial hemorrhages. The surface was mottled due to numerous small white areas which on section extended in streaks through the depths of the cortex.

Optic nerves: These structures were pinched where they passed through the optic foramen. The nerves were very dark at this point suggestive of degenerative changes.

Brown Swiss - Aut. 9227 - C615

Clinical Record

Days

16. The optic discs of this animal were not as clear as the other animals right from the start.
50. Scouring began.
52. Scouring continued.
58. The irises were not contracting normally. Calf was still scouring.
64. The feces were firmer in consistency.
69. Pupils were dilated and the optic discs were blurred so that it took about 0.5 diaopters to clear.
87. The eyes were lacrimating and the tapeta lucida were beginning to fade.
127. Scouring reappeared.
129. The feces were firmer again.
137. Left eye reflex was not as strong as the right.
159. Animal was not eating its grain properly.
166. Still not eating well.
176. The calf was given 10 mg. of cobalt sulphate orally and was to receive this much each day in the diet from now on.
179. Appetite was greatly improved.
191. Tapeta lucida were slightly faded and the optic papillae were

blurred when compared with the controls. Calf showed syncope for the first time.

206. Eyes were about the same.

235. It didn't consume its grain.

242. The tapeta lucida have lost most of their yellow color.

249. Eye reflexes were not as sharp as controls.

254. Syncope.

257. Two hemorrhagic areas were in the left eye near the optic disc.

260. The calf was completely blind.

262. The eyes were rolling (nystagmus) and the irises did not contract at all. Both optic discs were edematous. The right eye showed an ecchymotic hemorrhage.

263. The hemorrhage of the right eye was much more diffuse.

272. Animal did not eat grain as well as normal.

276. The optic discs were much larger in diameter and the outer margins were very irregular and blurred. The blood vessels of the eyes were smaller in diameter than the controls. The hemorrhages of the eyes were practically cleared.

283. Scouring began.

291. The eyes were about the same.

297. Some mucopurulent discharge was coming from the nostrils.

303. Syncope.

311. The eyes were bulging from the sockets and were starry. The tapeta lucida still had some yellow color.

326. Animal did not consume grain well.

342. The blood vessels were smaller in diameter than seen previously in the eyes.

365. Destroyed for examination.

Necropsy

Lungs: There were scattered areas of pneumonia in the apical, cardiac, diaphragmatic lobe of each lung and the intermediate lobe.

Kidneys: The organs appeared much smaller than normal. The hilus was occupied by a very watery fat. A few small petechial hemorrhages and some small white foci were present in the cortex.

Jejunum: The mucosa was reddened and contained an excess of mucus.

Testicles: The structures appeared smaller than normal.

Eyes and optic nerves: The optic nerves were pinched where they passed through the foramen.