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SOME OBSERVATIONS ON BEEF CATTLE AFFECTED WITH GENERALIZED EDEMA OR ANASARCA DUE TO VITAMIN A DEFICIENCY

LOUIS L. MADSEN[1] AND I. P. EARLE
WITH THE TECHNICAL ASSISTANCE OF RUSSELL E. DAVIS, HARRY BASTRON, C. W. THIES, CHARLES A. CABELL AND H. DEAN RAY
Animal Husbandry Division, Bureau of Animal Industry, United States Department of Agriculture, Beltsville, Maryland

ONE FIGURE

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The fact that prolonged vitamin A deficiency in cattle may result in edema as characterized by swelling of the legs, shoulders and brisket, on the abdomen, in the hindquarters and elsewhere has been recognized for some time. This condition is often referred to as anasarca. Hastings ('41, '44), Cady ('42) and others have more recently called attention, in the popular press, to anasarca in fattening cattle apparently due to vitamin A deficiency in rations of which the grain consisted principally of yellow corn. Similar cases have been brought to the attention of the Bureau of Animal Industry when distillery slop and low-quality roughages have been fed to fattening cattle.

In 1941 our attention (Creech and Madsen, '42; Mohler, '42; and Madsen, '42) was called to a condition characterized by extensive edema of the subcutaneous adipose tissue and musculature of beef carcasses which was encountered by

[1] Now Head, Department of Animal Husbandry, Utah State Agricultural College.

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Federal meat inspectors at several meat packing establishments. In view of the unusually large number of cases of unrecognized origin (see table 1) the senior author was called to the Corn Belt area where a number of these cattle had been fed, to determine if a nutritional basis could be established for the disease since previous bacteriological studies had been entirely negative.

TABLE 1

Beef carcasses condemned for anasarca by months from July, 1941 to December, 1946, inclusive.

	YEAR						
	1941	1942	1943	1944	1945	1946	Monthly average
January	..	2	3	9	5	5	5
February	..	3	11	5	2	12	7
March	..	9	4	4	3	9	6
April	..	6	5	5	5	10	6
May	..	9	10	7	9	6	8
June	..	5	15	9	11	5	9
July	3	12	18	18	13	10	12
August	18	5	20	3	9	8	10
September	40	6	30	3	11	3	15
October	43	9	28	5	5	7	16
November	20	4	13	7	11	10	11
December	15	5	12	6	6	12	9
Yearly total	139	75	169	81	90	97	

The present paper reports statistics on the number of beef carcasses condemned for anasarca by the Federal meat inspectors during the years 1941–1946, and presents data on the occurrence, clinical appearance and history of field cases of anasarca as they were observed during the field trip to the Corn Belt in 1941. It also describes the experimental production of edema in vitamin A-deficient cattle and presents results of observations made on the blood chemistry in both field cases of anasarca and experimentally produced cases of this condition.

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CARCASSES CONDEMNED

The data given here on condemned carcasses were furnished through the courtesy of the Meat Inspection Division of the Bureau. The records show that 651 carcasses were condemned for anasarca by Federal meat inspectors from July, 1941 through December, 1946. The distribution of these cases on a monthly and yearly basis is given in table 1. Heavy losses were encountered in the last half of 1941. Fewer cases were condemned in 1942 but losses were heavy in 1943. About the same number were condemned in 1945 as in 1946. It appears likely that the years of relatively few numbers of anasarca cases are related to the prevailing light or short-time grain feeding of steers. From these limited data it seems that more cases appear in the latter half of the year. These statistics do not represent the total loss among slaughter animals due to anasarca since in cases in which the edema is localized the carcass may be retained, trimmed as found necessary under inspection and released.

FIELD CASES OF ANASARCA

Statistics are not available on the number of cases of anasarca that develop under practical conditions. It is obvious, however, that more cases develop than are condemned at slaughter since some cases recover before slaughter because of timely changes in the ration, whereas others may die on the farm or become severely injured during convulsions while in transit. Seven farms having approximately 1200 cattle on feed were visited in the Corn Belt area by the senior author in company with Dr. John S. Koen and Dr. H. J. Classick, both veterinarians of the Bureau of Animal Industry on field assignments. Case reports from 2 of these farms are presented to illustrate (1) the development of vitamin A deficiency associated with anasarca under ordinary feed lot conditions and (2) a means of preventing serious losses by simple corrective feeding.

Case 1. Mr. G. started feeding 41 head of yearling Hereford steers, obtained from the West, in November, 1940. They were

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started on feed with corn and fodder of the 1940 crop and about 1 pound per head per day of a mixture of equal parts molasses and alfalfa which was continued until June, 1941. When the hot weather came during the latter part of June and persisted through July and August the cattle "went off feed," but no edema was seen by the owner. At this time feeding alfalfa hay of the 1940 crop 3 times daily improved the appetite of the steers and they were soon brought back to full-feed. Early in September oat straw was substituted for the alfalfa hay. Straw feeding was continued until early in December when Dr. Koen visited the farm and suggested a change in roughage from oat straw to alfalfa on the basis of information he had received from the Bureau that anasarca may be due to vitamin A deficiency. At this time about 30 animals of the group had swollen legs and enlarged briskets. One steer could hardly walk and 1 animal had recently been shipped to Omaha and condemned for anasarca. We visited the farm on December 18, 1941, about 2 weeks after alfalfa feeding was started, and according to Dr. Koen improvement in the cattle was evident. Dr. Koen and Dr. Classick visited this farm again on January 21, 1942, and found so much improvement in the cattle that little or no edema could be observed. All of the cattle were sold about 1 week later, or approximately 8 weeks after alfalfa feeding was started, without evidence of anasarca.

Case 2. On this farm 135 yearling steers were started on feed in October, 1940. The animals were turned into a corn field where they had access to corn stalks and some blue-grass until January, 1941. At this time they were taken up to the feed lot and given Atlas sorghum silage at about 30 pounds per head daily and all the ground yellow corn (1940 crop) they would clean up. A small amount of soybean hay was also given until the supply was used, about June 1, 1941. At this time feeding of oat hay was started and corn silage was fed until August. After all the silage had been consumed, oat hay was continued as the only roughage. On November 6, 1941,

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20 of the steers were shipped to Omaha, and of this group 1 was condemned for anasarca. A few more animals were sent to Chicago about the middle of November and 1 of these was also condemned for anasarca. Another steer became very lame and a veterinarian advised that the animal had probably sprained its leg, and that the steer should be killed and used for food. The steer was butchered but the carcass was declared unfit for food owing to generalized edema.

When we visited the farm on December 19, 1941, several animals showed marked symptoms of vitamin A deficiency. One animal was blind and had swollen legs and brisket. This animal was down and died the following day. Two other steers were also markedly affected, 1 was blind and another had severe edema of the legs, brisket and shoulders.

Twelve days previous to our visit Dr. Koen had seen these cattle and suggested feeding alfalfa as in case 1. This had resulted in a marked improvement in appetite and general appearance of the steers. The foreman commented that the cattle could get up and go to the feed bunks with greater ease since they had been receiving alfalfa, and that the lameness which had been previously noted had now practically disappeared. The foreman also characterized as "fish-eyes" the peculiar protruding eyes of several of the cattle, a condition which is typical of vitamin A-deficiency blindness.

The findings presented in these 2 cases were typical of conditions noted on other farms where cases of anasarca were seen. In some instances other symptoms of vitamin A deficiency such as diarrhea, constipation, nightblindness, stiff gait and convulsions were prominent. In 1 instance impaired vision in the cattle was not suspected until difficulty was encountered in loading the cattle on a truck. On this farm the vision of an apparently blind steer improved remarkably after alfalfa hay had been added to the ration in place of oat hay. In some feed lots anasarca developed in cattle fed stored corn, but in these cases a low-carotene roughage was also used.

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EXPERIMENTAL CASES OF ANASARCA

Experimental cases of anasarca were produced by feeding either a carotene-deficient ration (diet 1) or one containing a large amount of yellow corn (diet 2), the roughage in both cases being supplied by oat straw. The concentrate portion of diet 1 had the following percentage composition: dried beet pulp, 45; cracked white corn, 40; linseed meal, 7; soybean meal, 7; steamed bonemeal, 0.5; and salt, 0.5. The concentrate portion of diet 2 consisted, in per cent, of cracked yellow corn, 80; dried beet pulp, 8.5; linseed meal, 10; steamed bonemeal, 1; and salt, 0.5. Vitamin A-deficiency edema was readily produced when cattle were restricted to either of these rations for several months. In a number of instances, after the appearance of anasarca, the diet was supplemented with 1 of several sources of carotene and vitamin A such as alfalfa leaf meal, carotene concentrate, crystalline carotene, cod-liver oil and distilled vitamin A concentrate in natural ester form. In all such cases, anasarca, as well as other symptoms of vitamin A deficiency except permanent blindness, was entirely relieved with the treatment.

A complete history is given for only 1 case (steer 411) in which the course of depletion and the development of deficiency symptoms were quite characteristic of all the other cases. Since a significant part of the story is believed to be told by the results of the blood analyses which were made at approximately monthly intervals on a number of these edema cases, these data are included in the history, although the general subject of blood analyses in these cattle is treated in a later section of this paper.

Steer 411 on which the case history of experimental edema is based was fed individually on diet 2 (80% yellow corn). This animal was a purebred Shorthorn approximately 7 months old weighing 613 pounds at the beginning of the experiment. Feed consumption, live-weight gain, together with the appearance of progressive symptoms of convulsions, diarrhea, blindness and edema are indicated in table 2 and illustrated in figure 1. Attention is called to the fact that

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TABLE 2

Summary of observations on steer no. 411 while receiving an 80% yellow corn grain ration.

OBSERVATION	DAYS OF EXPERIMENT											
	27	58	90	117	147	177	209	238	271	289	300	328 [1]
Condition of animal	Normal	Normal	Normal	Normal	Normal	Convulsions	[2]	Diarrhea	Blind	Edema	[3]	[3]
Body weight, lbs.	635	675	712	765	827	873	921	970	1033	1056	1062	1069
Average daily gain for period, lbs.	1.44	1.29	1.13	1.96	2.07	1.53	1.50	1.69	1.91	1.28	0.55	0.25
Daily feed consumption:												
Grain mixture, lbs.	7.7	8.9	10.8	11.9	12.1	13.9	14.6	15.1	15.7	16.0	17.9	14.4
Oat straw, lbs.	2.2	1.6	1.8	2.2	2.2	2.0	2.0	1.2	1.0	1.7	1.3	0.7
Plasma nitrogen fractions:												
Total plasma nitrogen, gm/100 ml	1.019	0.993	1.046	1.032	1.107	1.105	1.007	1.051	1.019	1.071	1.089	1.167
Nonprotein nitrogen, gm/100 ml	0.022	0.041	0.048	0.039	0.061	0.041	0.028	0.029	0.025	0.022	0.042	0.024
Fibrinogen nitrogen, gm/100 ml	0.052	0.060	0.088	0.063	0.074	0.060	0.055	0.075	0.064	0.133	0.142	0.133
Euglobulin nitrogen, gm/100 ml	0.089	0.081	0.082	0.081	0.106	0.076	0.119	0.096	0.081	0.083	0.085	0.159
Pseudoglobulin nitrogen, gm/100 ml	0.283	0.288	0.308	0.320	0.320	0.310	0.270	0.309	0.319	0.362	0.363	0.423
Total globulin nitrogen, gm/100 ml	0.424	0.429	0.478	0.464	0.500	0.446	0.444	0.480	0.464	0.578	0.590	0.706
Albumin nitrogen, gm/100 ml	0.573	0.523	0.520	0.529	0.546	0.618	0.535	0.542	0.530	0.471	0.457	0.437
A/G ratio	1.35	1.22	1.09	1.14	1.09	1.39	1.20	1.13	1.14	0.81	0.77	0.62
Plasma:												
Carotene, μg/100 ml	86	35	32	33	46	39	38	40	46	....	27	21
Vitamin A, μg/100 ml	25	16	23	11	13	13	9	10	7	....	8	6
Vitamin C, mg/100 ml	0.41	....	....	0.56	0.54	0.40	0.39	0.43	0.34	....	0.31	0.33
Serum:												
Calcium, mg/100 ml	....	....	13.1	10.6	11.0	12.0	10.9	10.2	11.1	....	11.5	9.8
Magnesium, mg/100 ml	2.8	....	2.6	2.6	2.4	2.5	2.4	2.3	2.3	....	2.7	2.3
Inorganic phosphorus, mg/100 ml	....	7.8	8.1	8.2	7.6	7.7	6.1	6.9	7.2	5.8	6.3	4.8
Phosphatase activity (Bodansky units)	....	4.5	3.1	4.9	4.6	4.2	3.8	4.6	4.1	4.4	3.3	3.1

[1] Steer died during a convulsion on the 343rd day.
[2] Beginning stages of rough hair coat, slobbering and slight edema.
[3] Edema increasing rapidly; convulsions more numerous. Ascorbic acid therapy started 303rd day.

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there was an increase in grain consumed up to the three hundredth day, but that feed consumption and rate of gain decreased after this time. No blood analyses were made after the three hundred and twenty-eighth day. Typical changes in the plasma protein fractions were observed at about the same time that edema appeared. Plasma carotene, vitamin A and

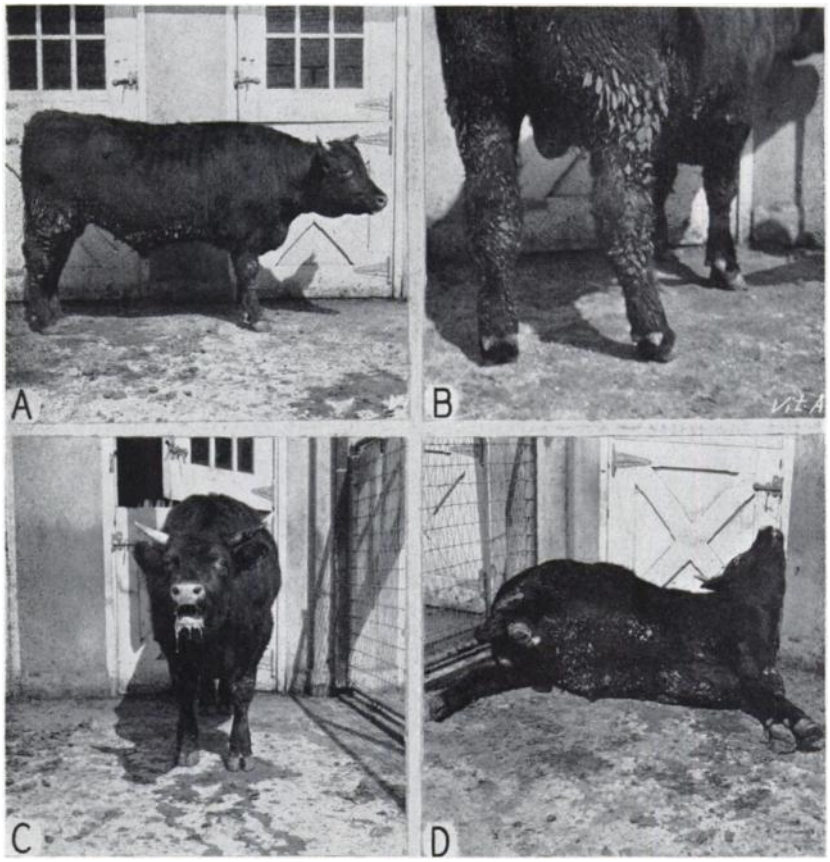

Fig. 1 These photographs illustrate symptoms of vitamin A deficiency in steer 411 after 309 days on diet 2. A. The steer is blind and has a rough, dry hair coat, but has made fairly good gains in weight. B. The hind legs are swollen due to edema. C. Rapid respiration was common and even panting occurred on slight exertion in hot weather. Note slobbering. D. Convulsions occurred frequently. The head is drawn back typically. Note left horn fracture (C) received during previous convulsions.

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vitamin C decreased in the usual manner, but there were minor fluctuations in these during the experiment. Serum calcium, inorganic phosphorus and phosphatase activity tended to decrease but the changes were not marked until after about the three hundredth day at which time the food intake also decreased.

The highest average grain consumption for steer 411 was recorded for the period between the two hundred and eighty-ninth and three hundredth days. An average of 17.9 pounds of grain mixture or 14.3 pounds of yellow corn was eaten daily at this time. On analysis the corn was found to contain 2.1 μg of crude carotene per gram and on biological assay with rats a value of 1.6 International Units (I.U.) of vitamin A per gram was obtained. At the above level of yellow corn consumption, and using the assay figures just mentioned, the steer received 13.6 mg of crude carotene daily, or 10,378 I.U. of vitamin A. This amount of carotene from yellow corn was insufficient to alter the course of vitamin A depletion. Feed consumption declined, edema increased and the animal died in convulsion on the three hundred and forty-third day of the experiment. Another steer on the same ration died in convulsion on the two hundred and forty-ninth day of the experiment. In this case edema appeared about the two hundred and thirteenth day, and some impairment of day vision was evident before death.

These findings add further evidence to the field observations that vitamin A-deficiency symptoms together with edema develop in fattening cattle when their ration is made up of yellow corn, a protein supplement and a low-carotene roughage.

BLOOD ANALYSES

Before attention was directed to the field cases of anasarca occurring in the Corn Belt, some data were already available on the composition of blood from a number of experimental animals both under conditions of apparently normal health and also at various stages of vitamin A deficiency which developed during prolonged use of diet 1. These data seemed

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to indicate that there were certain characteristic changes in the composition of the blood, particularly with respect to the protein fractions, approximately coincident with the development of edema in these vitamin A-deficient animals. Analyses of blood obtained from some field cases of anasarca were undertaken primarily for the purpose of further characterization of the general physiological condition of the animals and also for the further identification of this condition with the edema occurring in the experimental animals depleted of vitamin A. Results of analyses of blood from the field cases seemed to indicate that changes had occurred here similar to those in the experimental edematous animals. In view of the feeding practices under which the anasarca cases observed had developed in the Corn Belt, blood studies were also made on animals which were fed diet 2 containing 80% yellow corn.

Blood samples for analysis were drawn from a jugular vein. For the preparation of plasma samples, exactly 30 mg of neutral potassium oxalate were used as anticoagulant for each 10 ml of blood. Plasma proteins were fractioned by Howe's ('21) micro-method with the variation later recommended by him ('23) that 0.75 molar sodium sulfate be used for the precipitation of fibrinogen in plasma instead of calcium chloride. All nitrogen determinations were made on aliquots of the filtrates by a micro-Kjeldahl method. The spectrophotometric method previously described by Madsen and Davis ('38) and Davis and Madsen ('41) was used for determination of plasma carotene and vitamin A. Plasma vitamin C was determined by the method of Mindlin and Butler ('38). Serum calcium was determined by the Clark-Collip modification of the Kramer-Tisdall method as described by Hawk and Bergeim ('37); serum magnesium was precipitated according to the method used by Godden ('37) and determined as phosphorus by the method of Bodansky ('32). Bodansky's ('33) methods were also used for the determination of serum inorganic phosphorus and serum phosphatase activity.

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RESULTS

The results of analyses on cases of experimental edema produced in 7 beef animals have been averaged and paired with the average of observations made on these same animals when they were normal. The averages are presented in table 3 together with the average results of blood analyses made on 5 field cases of anasarca.

It is obvious from the figures presented in table 3 that the plasma protein fractions in the experimental animals with edema differ significantly from those in the same animals

TABLE 3

Summary of results of blood analyses from field and experimental cases of anasarca and normal beef animals.

CONSTITUENTS	AVERAGE OF 5 FIELD CASES OF ANASARCA	EXPERIMENTAL CASES		
		Averages of 7 with edema	Averages of 7 when normal	Calculated odds — edematous vs. normal [1]
Nitrogen fractions in plasma:				
Total plasma nitrogen, gm/100 ml	1.263	1.150	1.076	104
Non-protein nitrogen, gm/100 ml	0.038	0.036	0.037	
Fibrinogen nitrogen, gm/100 ml	0.151	0.150	0.061	191
Euglobulin nitrogen, gm/100 ml	0.166	0.180	0.136	51
Pseudoglobulin nitrogen, gm/100 ml	0.494	0.384	0.318	1999
Total globulin nitrogen, gm/100 ml (including fibrinogen)	0.811	0.714	0.515	2499
Albumin nitrogen, gm/100 ml	0.414	0.400	0.524	587
A/G ratio	0.51	0.56	1.02	
Vitamins in plasma:				
Carotene, μg/100 ml	31	21	67	26
Vitamin A, μg/100 ml	8	7	23	9999
Vitamin C, mg/100 ml	..	0.25 [2]	0.37 [2]	13
Constituents in serum:				
Calcium, mg/100 ml	..	10.01	11.00	160
Magnesium, mg/100 ml	..	2.63	2.72	
Inorganic phosphorus, mg/100 ml	..	5.60	6.74	40
Phosphatase activity (Bodansky units)	..	2.28	4.24	232

[1] Student's method used for estimating significance between means of paired observations in experimental cases of anasarca and corresponding normal control values.

[2] Average for only 4 animals.

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when normal. Attention is called to the increases in fibrinogen and total globulin and to the decrease in albumin in the animals with edema.

Low values for plasma carotene and vitamin A were observed in all cases of anasarca. Calculated odds for estimating the significance of differences in the pairs of average values for plasma carotene and for vitamin A were greater for vitamin A than for carotene. This may be explained by the uniformly low values for vitamin A and carotene in all cases of anasarca as contrasted with uniformly high values for vitamin A and high, but variable, values for carotene in normal animals. The carotene intake of the normal cattle was not standardized, but in all cases was above a deficiency level. In the field cases of anasarca the average blood carotene and vitamin A were higher than in the experimental cases, a condition which is probably due to the fact that alfalfa hay feeding had already been started in some of these animals when blood samples were taken.

Average content of vitamin C in plasma was found to be lower while edema was present than when it was absent in the same animals. The significance of the difference in the 2 averages is shown by the calculated odds not to be great, however, probably because data for this comparison were available from only 4 pairs of observations and because the degree of vitamin A deficiency was not the same in all the animals when the observations were made. In the 4 cases observed there was always a decrease in plasma vitamin C as the deficiency of vitamin A progressed but the drop was not uniform in all cases. This decrease in plasma vitamin C in cases of vitamin A deficiency was first demonstrated by Phillips and coworkers ('38).

While serum calcium, magnesium and inorganic phosphorus tended to decrease slightly from the levels observed when the same animals were normal, the values still remained well within the normal range. There was, however, a decrease from normal in phosphatase activity of the serum in all cases of experimental anasarca studied.

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DISCUSSION

Losses due to anasarca associated with vitamin A deficiency are economically important. Nevertheless they can be prevented by proper feeding. Supplying fattening cattle with a small amount of well-cured alfalfa hay successfully avoided serious losses in 2 instances cited. Any other well-cured hay having a good green color, well-made silage, green pasture or a marine oil such as cod-liver oil would no doubt have accomplished the same purpose. The prevention of anasarca or of other symptoms of vitamin A deficiency by the use of proper feeds does not necessarily discourage the highly economical practice of feeding low-carotene roughages such as ordinary cereal hay, straw and corn stover, etc. When these roughages are used, however, it is necessary to supply a supplementary ration of well-cured green hay to avoid possible vitamin A depletion in the latter part of the feeding period.

Opinions have been expressed in the literature and have been prevalent among some cattle feeders that anasarca is caused by feeding stored corn. In both field and experimental cases of anasarca presented in this paper, however, yellow corn of the current season's crop failed to prevent the development of either anasarca or other symptoms of vitamin A deficiency. This observation confirms the report by Hastings ('44). The real error in practical feeding appears to be not in the use of old corn but in the exclusive use of low-carotene roughages such as oat hay and straw and the failure to supplement these low-carotene roughages with green hay less than 1 year of age. Average yellow corn usually contains no more and often less carotene than a low grade hay. Corn should not be depended upon to supply enough carotene to satisfy the vitamin A needs of cattle.

The clinical symptoms and blood findings presented suggest that the field cases and experimental cases of anasarca probably arose from the same condition. All cases of anasarca observed evidenced low levels of blood carotene and vitamin A and, in addition, showed the same changes in blood protein

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fractions. These changes consisted essentially of an increased level of total globulins, due principally to increased fibrinogen and, to a lesser degree, to increases in the euglobulin and pseudoglobulin fractions. At the same time there was a definite, although not in every case large, decrease in plasma albumin. The total protein was usually slightly elevated when edema was present.

The relation of the altered protein picture to vitamin A deficiency is not apparent. Increases in fibrinogen in conditions in which destruction and inflammation of tissues occur and increases in other globulin fractions, either with or without increased fibrinogen, in cases of chronic infections, are usually accompanied by some reduction in albumin concentration, an effect presumably secondary to the increase in globulin. Since a reduction in blood albumin is a primary factor in some types of edema, the question arises as to the relation between the lowered albumin and the development of edema in these cattle. The concept of an increased tendency toward transudation and accumulation of fluid in the interstitial tissues with a lowering of the plasma colloid osmotic pressure is a familiar one. In this relation, the concentration of albumin is of greater significance than that of globulins, since the albumin exerts about 2.4 times as great an osmotic pressure as the globulins. It is estimated that at the so-called critical levels for albumin nitrogen and for total plasma nitrogen in the human (i.e., levels below which edema usually appears), the plasma colloid osmotic pressure is reduced to roughly two-thirds of the normal level (Moore and Van Slyke, '30).

If it is assumed that approximately similar relative levels for albumin and for total protein are critical for cattle, it becomes apparent that the average reduction in albumin observed in the anasarca animals is not great enough to account for the appearance of edema, especially in view of the increase in total protein. Values for colloid osmotic pressure calculated by application of the formula of Wells, Youmans and Miller ('33) to data obtained on concentration of proteins in

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serum of these animals (unpublished data), also suggest that increases in globulins are great enough to compensate in large measure for the reduction in albumin. It is believed, therefore, that the occurrence of edema in these animals is not entirely related to a lowering of the colloid osmotic pressure of the plasma brought about by a lowering of the albumin concentration.

SUMMARY

Statistics are presented showing that 651 beef carcasses were condemned for generalized edema or anasarca by Federal meat inspectors during the period of July, 1941 to December, 1946, inclusive. Other symptoms of vitamin A deficiency were found to be prevalent among cattle with anasarca in the Corn Belt area. This edematous condition was observed to occur 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 or straw. Alfalfa hay was highly effective in curing the condition. It is concluded, therefore, that the major dietary error in the production of this deficiency disease is the exclusive use of low-carotene roughages throughout the feeding period rather than the use of old yellow corn.

Cases of anasarca were readily produced experimentally by feeding a carotene-deficient ration or a grain ration containing 80% of new-crop yellow corn together with oat straw as roughage.

Results of blood studies made on field and experimental cases of anasarca produced by vitamin A deficiency indicate a marked similarity in the 2 conditions. Affected animals showed: (1) deficiency levels of blood plasma vitamin A and carotene and a decrease in plasma vitamin C as the vitamin A deficiency progressed, (2) an increase in total plasma globulin due usually to a marked increase in plasma fibrinogen with smaller increases in the other globulin fractions, (3) a decrease in plasma albumin, and (4) an increase in total plasma nitrogen.

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In the serum of affected cattle, calcium and inorganic phosphorus decreased slightly; phosphatase activity was reduced while magnesium was relatively unchanged.

It is pointed out that the edema of vitamin A deficiency in cattle appears not to be entirely related to a lowered colloid osmotic pressure of the plasma through reduction in the albumin, since the increases in other protein fractions are estimated to compensate largely in this respect for the decrease in albumin.

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